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CHAPTER 25 – FOOT AND ANKLE

SECTION A Biomechanics

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This section discusses the biomechanical linkage of the joints of the foot and ankle and their effects on the lower extremity. The ankle joint, subtalar joint, transverse tarsal joint, and metatarsophalangeal joints are uniquely interrelated so that function in one reliably alters the mechanics of the others. The foot and ankle complex initially helps absorb the impact of ground contact and later in stance provides the body with a stable platform from which to function. During walking, the toes are lifted from the floor, but in athletics, forceful push-off facilitates rapid acceleration and deceleration, direction changes, and jumping.

Dysfunction of the foot and ankle complex may result in an altered gait pattern, degradation of athletic performance, and compensatory changes in the knee and hip joints. The ankle and subtalar joint complex function as a universal joint, linking pelvic, thigh, and leg rotation to hindfoot motion and longitudinal arch stability. This allows the ankle joints to compensate for some degree of dysfunction in the hindfoot, and vice versa. Athletics, however, requires maximal performance from these systems, and dysfunction of ankle and foot mechanics often leads to pain, injury, and loss of performance.

Athletics is distinguished from normal walking by the stresses applied to the joints. The stresses can be repetitive, as in a long distance runner, or impulsive, as occurs in the push-off foot of a shot-putter. The vertical force involved in running is 2 to 2.5 times body weight compared with 1.2 times body weight in walking, [1] and can be higher for many sports with extreme push-off, such as a football lineman engaged in blocking or a basketball player engaged in rapid accelerating and jumping activities (Fig. 25A-1). The nature of these forces depends on the activity and includes vertical force, fore-and-aft shear, side-to-side shear, and torque forces. These forces are measured in a variety of ways, including force plate analysis or thin film pressure transducers placed in a shoe. [2] The foot and ankle complex must be supple enough to absorb impact and rigid enough to transmit muscle forces, or injuries such as sprains, strains, stress fractures, and fascial tears may result. Athletic training can help to attenuate these forces and minimize the risk for injury. [3]

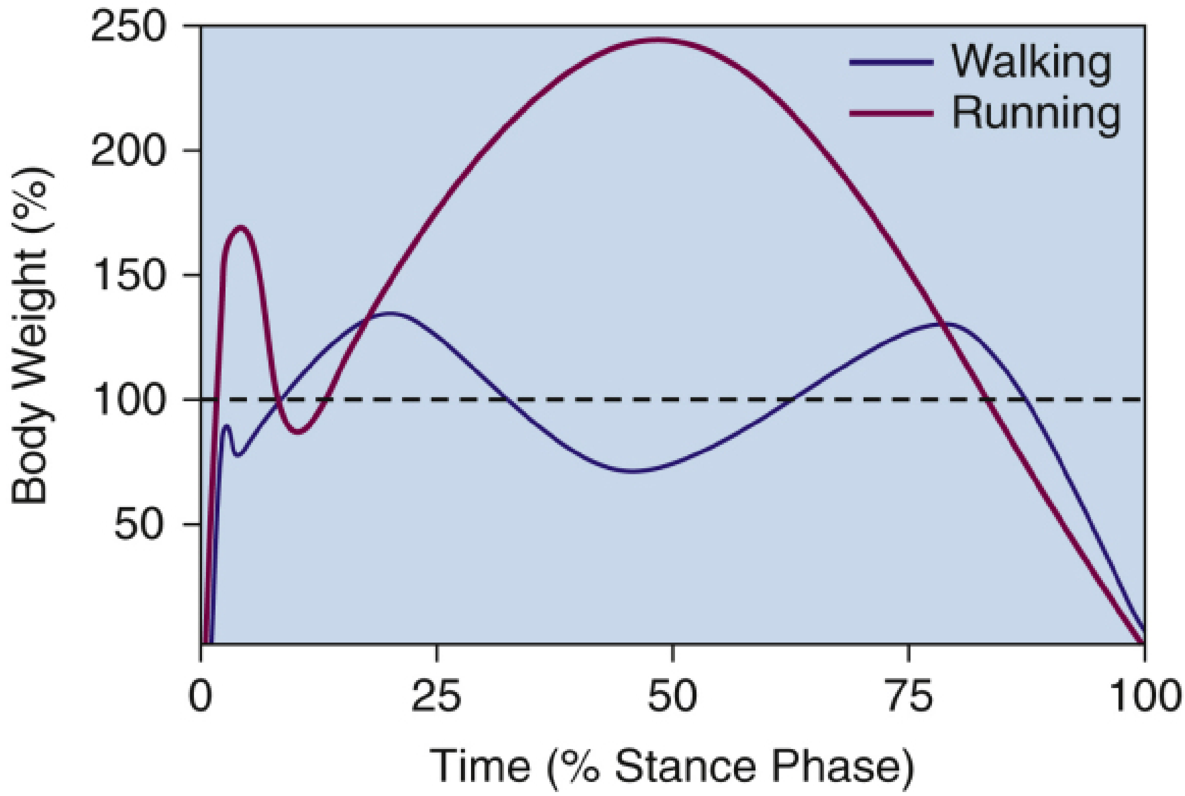

Figure 25A-1 Comparison of vertical ground reaction force for walking (*blue line*) compared with jogging (*red line*). The horizontal axis is scaled as a percentage of total time in stance phase for walking (0.6 sec) and running (0.24 sec). The vertical axis is shown as a percentage of body weight. *(From Mann RA, Haskell A: Biomechanics of the foot and ankle. In Coughlin MJ, Mann RA, Saltzman CL [eds]: Surgery of the Foot and Ankle, 8th ed. Philadelphia, Mosby, 2007.)*

ANKLE JOINT

The ankle joint allows sagittal plane motion of 20 degrees of dorsiflexion and 50 degrees of plantar flexion; however, there is a great deal of variability among individuals. The ankle joint is not a simple hinge joint, but rather the trochlear surface of the talus is a section from a cone whose apex is based medially (Fig. 25A-2). [4] The talus is stabilized within the ankle mortise by bony and soft tissue restraints. The congruity of the ankle mortise leads to considerable inherent bony stability. [5] [6] Ligament support includes the deltoid ligament medially [7] and three separate ligamentous bands laterally: the anterior and posterior talofibular ligaments and the calcaneofibular ligament. [8]

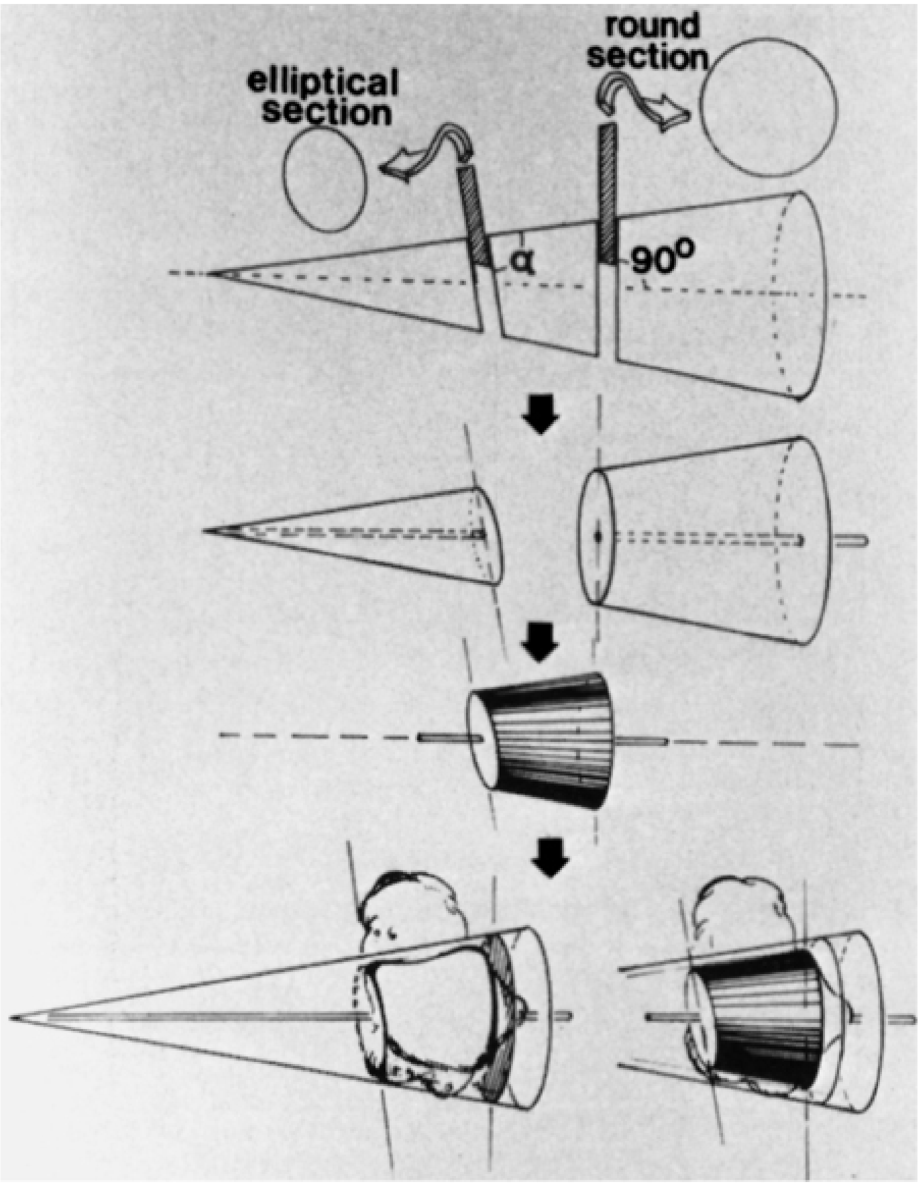

Figure 25A-2 The trochlear surface of the talus is a section from a cone. The apex of the cone is directed medially, and the open end is directed laterally. *(From Stiehl JB [ed]: Inman's Joints of the Ankle, 2nd ed. Baltimore, Williams & Wilkins, 1991.)*

The anterior talofibular ligament is taut with the ankle joint in plantar flexion, when it is in line with the fibula; and the calcaneofibular ligament is taut with the ankle joint in dorsiflexion, when the ligament is in line with the fibula (Fig. 25A-3). [4] The anterior talofibular ligament is injured most frequently during ankle sprains, in part because the ankle has less intrinsic bony stability in plantar flexion when this ligament is under tension. [9] Isolated injuries to the calcaneofibular ligament are less frequent, although they often occur in conjunction with anterior talofibular sprains.

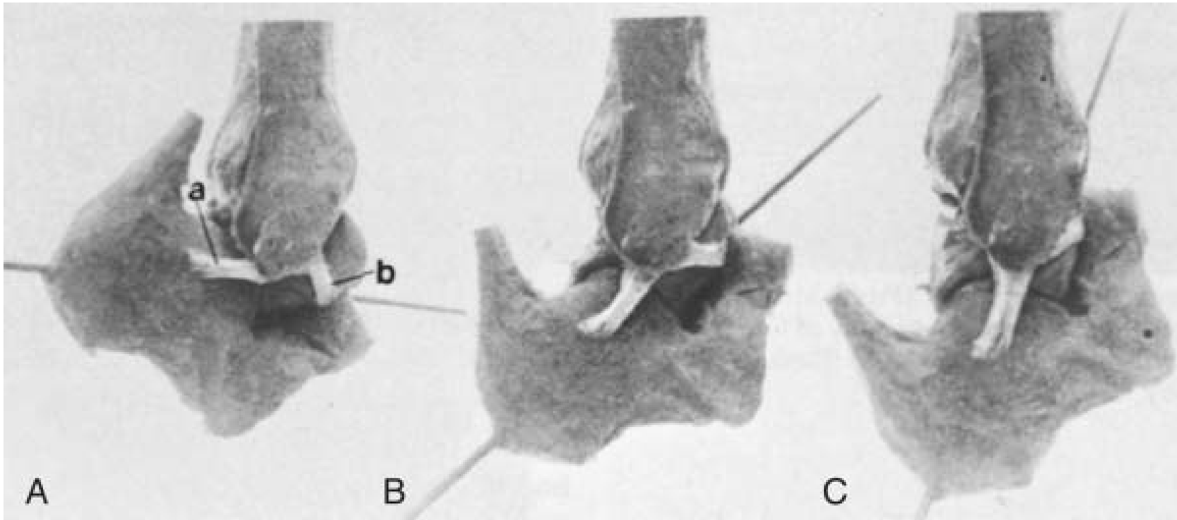

Figure 25A-3 Calcaneofibular (a) and anterior talofibular (b) ligaments. **A,** In plantar flexion, the anterior talofibular ligament is in line with the fibula, thereby providing most of the support to the lateral aspect of the ankle joint. **B,** When the ankle is in neutral position, both the anterior talofibular and the calcaneofibular ligaments support the joint. The obliquely placed structure depicts the axis of the subtalar joint. It should be noted that the calcaneofibular ligament parallels the axis. **C,** When the ankle joint is in dorsiflexion, the calcaneofibular ligament is in line with the fibula and supports the lateral aspect of the joint. *(**A** to **C,** From Stiehl JB [ed]: Inman's Joints of the Ankle, 2nd ed. Baltimore, Williams & Wilkins, 1991.)*

Dorsiflexion and plantar flexion occur at the ankle joint during gait. At heel strike, the dorsiflexed ankle rapidly plantar flexes. This ends at foot flat, after which progressive dorsiflexion occurs. Dorsiflexion reaches a maximum at 40% of the walking cycle, when plantar flexion begins as the heel rises, and continues until toe-off, when dorsiflexion occurs again during the swing phase (Fig. 25A-4).

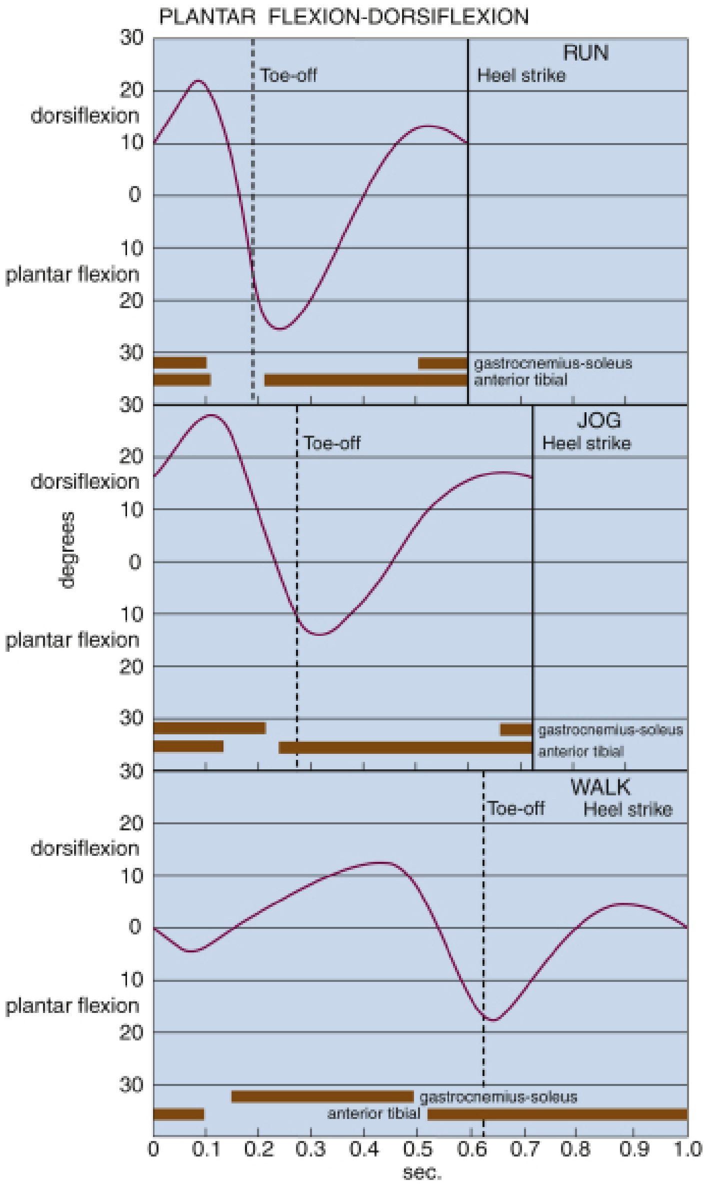

Figure 25A-4 Ankle joint range of motion for walking, jogging, and running. The muscle function of the anterior and posterior compartment is noted on the bottom of the graph. *(Redrawn from Mann RA: Biomechanics of running. In American Academy of Orthopaedic Surgeons: Symposium on the Foot and Leg in Running Sports. St. Louis, CV Mosby, 1982.)*

The force applied across the ankle joint during walking has been measured at about 4.5 times body weight. [10] This maximal stress occurs just before and just after the onset of plantar flexion of the ankle joint. If this force were extrapolated to running, in which the ground reaction force is over double that of walking, we would see stress across the ankle joint that approaches 10 times body weight.

Muscle control of the ankle joint can be divided into the anterior and posterior compartments. The anterior compartment consists of the tibialis anterior, extensor hallucis longus, and extensor digitorum longus. The posterior compartment consists of the gastrocnemius-soleus group, tibialis posterior, flexor digitorum longus, and flexor hallucis longus. The lateral compartment, consisting of the peroneus longus and brevis, functions with the posterior compartment.

During normal walking, the anterior compartment muscles become active late in the stance phase and in the swing phase to bring about dorsiflexion of the ankle joint by a concentric (shortening) contraction (see Fig. 25A-4). [11] This muscle group remains active after heel strike to control the rapid plantar flexion of the ankle joint that occurs by an eccentric (lengthening) contraction. This eccentric contraction during plantar flexion helps to dissipate the forces on the limb at initial ground contact. The anterior compartment becomes electrically silent by foot flat. The posterior compartment muscles are active after foot flat (15% to 20% of gait cycle), during which time the ankle joint is undergoing dorsiflexion, and they remain active until about halfway through the cycle, at which time about 50% to 60% of ankle joint plantar flexion has occurred. [12] This muscle group initially undergoes an eccentric contraction controlling forward movement of the tibia over the foot, then a concentric contraction when plantar flexion begins. The electrical activity of the posterior calf group ceases before full plantar flexion has occurred, indicating that the last portion of plantar flexion is a passive phenomenon.

As the speed of gait increases to steady running and sprinting, several changes occur. The ankle joint starts in slight dorsiflexion and, at heel strike, rather than the ankle plantar flexing to a foot flat position, the ankle remains dorsiflexed (see Fig. 25A-4). The tibia moves forward, and the foot flat position is achieved. As running speed increases, the magnitude of the motion increases, the stance phase is reduced significantly, and the period of double limb support gives way to a period lacking limb support. The electrical activity of the anterior compartment still begins late in stance and continues through swing, but it now lasts through about the first third of the stance phase. The posterior calf muscles show a significant change in their activity in that they become active late in swing phase and remain active until about halfway through ankle joint plantar flexion. [13] This increased activity in the posterior calf musculature probably results in increased stability of the ankle joint at the time of initial ground contact.

SUBTALAR JOINT

The subtalar joint is a complex joint that permits inversion and eversion. The axis of the subtalar joint is variable but is about 42 degrees to the horizontal plane and passes from medial to lateral at about 16 degrees (Fig. 25A-5). [10] [14] Measurement of subtalar joint movement during walking and running is difficult and is based on many assumptions. Although the overall pattern of motion appears to be consistent, the magnitude of motion is variable. [15] The range of motion of the subtalar joint in its pure form, inversion and eversion, is 30 degrees of inversion and 15 degrees of eversion, with considerable variability among people. To measure this motion accurately, it is important to start with the calcaneus in line with the tibia.

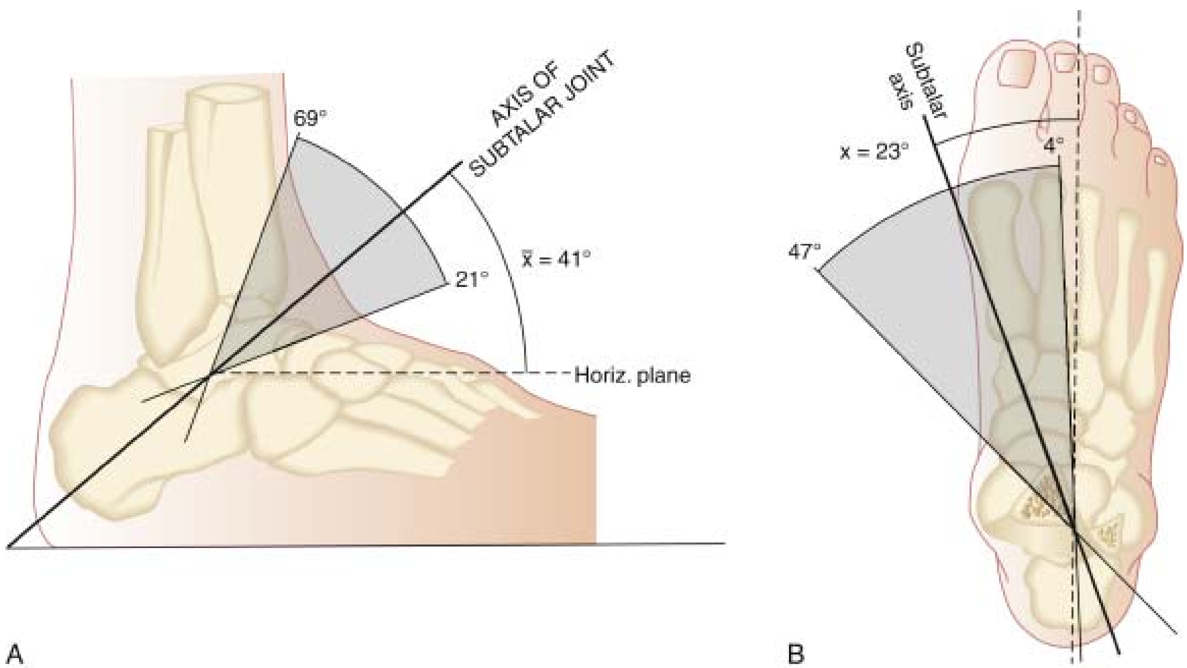

Figure 25A-5 Variations in the subtalar joint axes. In the horizontal plane (**A**), the axis approximates 45 degrees and (**B**) passes about 23 degrees medial to the midline. *(**A** and **B**, Adapted from Isman RE, Inman VT: Anthropometric studies of the human foot and ankle. Bull Prosthet Res 10:97, 1969.)*

The subtalar joint is less constrained than the ankle, being stabilized primarily by the joint configuration and the interosseous ligament. [16] [17] This joint is stable when the long axis of the tibia passes medial to the obliquely placed subtalar joint axis. In the normal foot, subtalar joint eversion ceases with weight-bearing owing to the configuration of the joint surfaces and the interosseous ligament. When the weight-bearing line is lateral to the subtalar joint axis, inversion stability depends on lateral ligament support and active muscle function.

The subtalar joint has been likened to an oblique hinge that functions to translate motion between the transverse tarsal joint distally and the ankle joint and leg proximally (Fig. 25A-6). [18] [19] This linkage is important for energy dissipation at heel strike. At the time of initial ground contact during walking, the slightly inverted subtalar joint undergoes rapid eversion, the tibia undergoes internal rotation, the transverse tarsal joints become supple, and the medial longitudinal arch flattens (Fig. 25A-7). These are passive energy-absorbing mechanisms. In a person with flatfoot and increased eversion of the subtalar joint (Fig. 25A-8), an increased amount of tibial internal rotation may occur, which can affect the knee, patellofemoral, or hip joint in selected cases. An orthotic device that supports the longitudinal arch with medial heel posting may restrict subtalar joint rotation and decrease the internal rotation of the lower extremity, possibly resolving knee or hip pain.

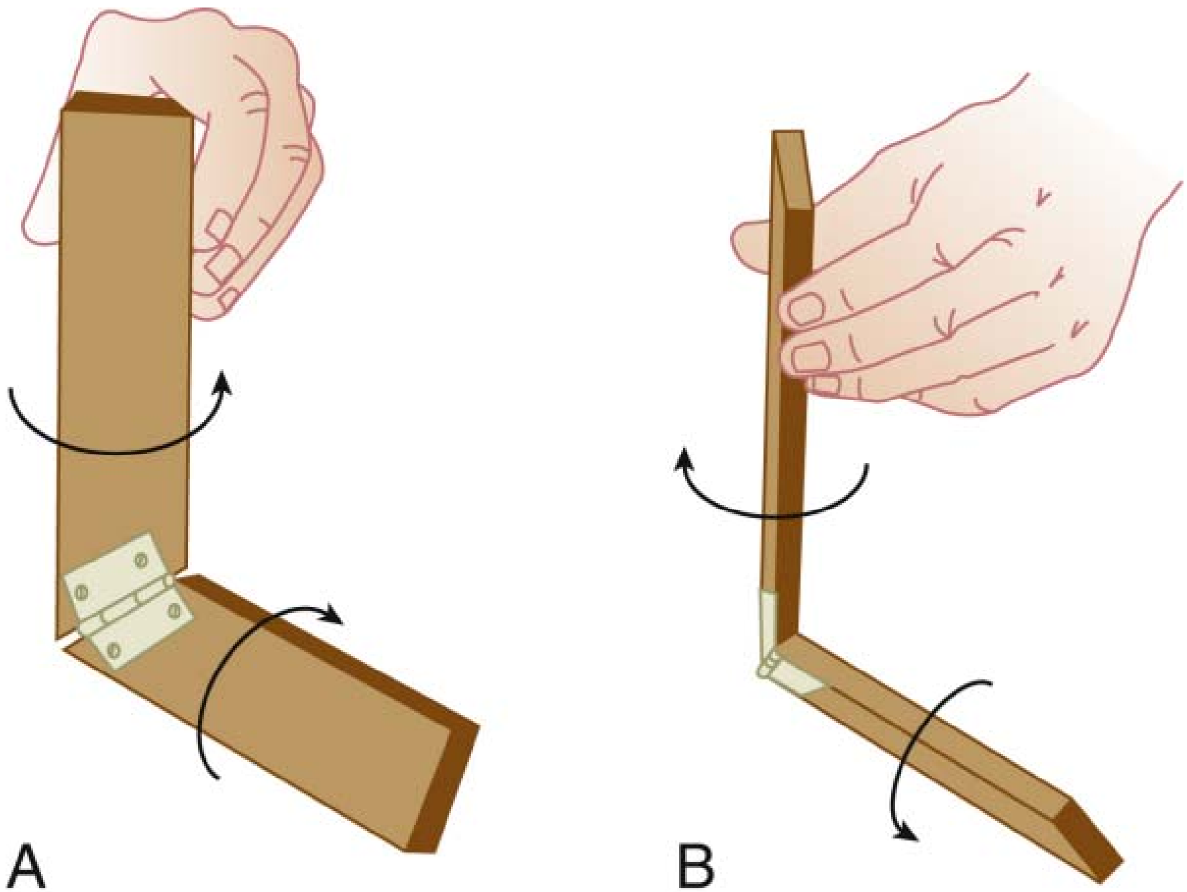

Figure 25A-6 Mitered hinge effect of subtalar joint. The joint acts as a mitered hinge, converting motion in the calcaneus below into the tibia above and, conversely, from the tibia above into the calcaneus below. *(**A** and **B,** Redrawn from Mann RA, Haskell A: Biomechanics of the foot and ankle. In Coughlin MJ, Mann RA, Saltzman CL [eds]: Surgery of the Foot and Ankle, 8th ed. Philadelphia, Mosby, 2007.)*

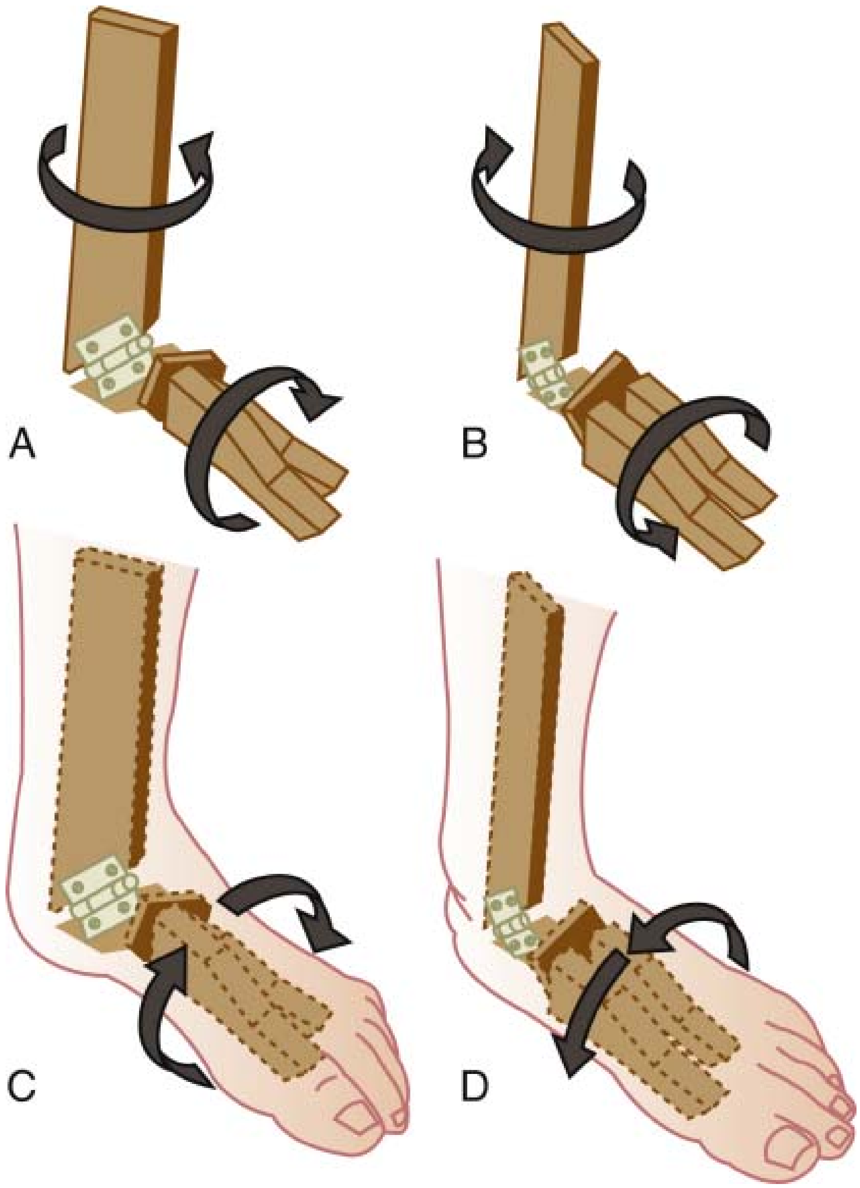

Figure 25A-7 Model demonstrating flattening and elevation of the longitudinal arch. **A** and **B,** Flattening of the longitudinal arch occurs at the time of heel strike with eversion of the calcaneus and internal rotation of the tibia. **C** and **D,** Elevation and stabilization of the longitudinal arch are associated with the outward rotation of the tibia, causing inversion of the calcaneus and locking of the transverse tarsal joint. *(Redrawn from Mann RA, Haskell A: Biomechanics of the foot and ankle. In Coughlin MJ, Mann RA, Saltzman CL [eds]: Surgery of the Foot and Ankle, 8th ed. Philadelphia, Mosby, 2007.)*

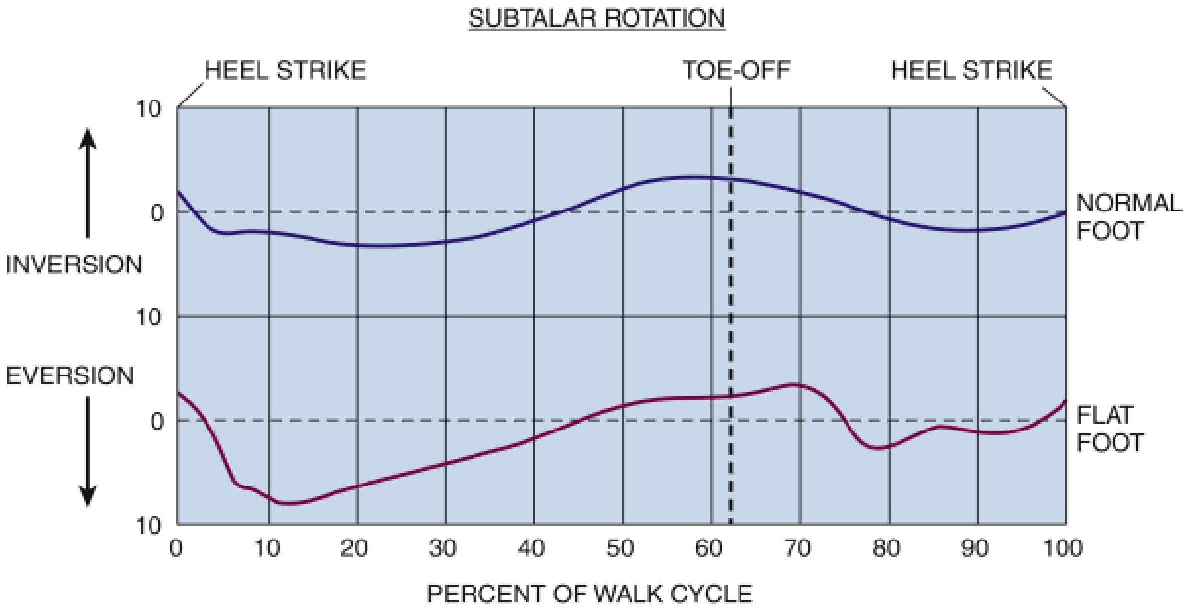

Figure 25A-8 Graph of subtalar joint motion in the normal individual and in a flatfooted individual. *(Redrawn with permission from data in Wright DG, Desai ME, Henderson BS: Action of the subtalar and ankle joint complex during the stance phase of walking. J Bone Joint Surg Am 46:361, 1964.)*

This linkage is also important for efficient energy transfer during heel rise and toe-off. After the initial eversion, the subtalar joint undergoes progressive inversion, which reaches a maximum at toe-off, when eversion begins again. This movement increases the stability of the transverse tarsal joints and medial longitudinal arch, stiffening the foot and allowing it to act as a rigid extension of the leg. Although the initial eversion is passive, the inversion that follows appears to be both passive and active. The inversion results from an external rotation torque from the lower extremity above, which is transmitted across the ankle joint and is translated by the subtalar joint into inversion. The plantar aponeurosis mechanism and the oblique metatarsal break enhance the inversion, as described later.

The muscle function around the subtalar joint can be appreciated best by looking at the muscles in relation to the subtalar joint axis (Fig. 25A-9). Muscles medial to the axis are invertors, muscles lateral to it are evertors, and the function of the muscles on the axis is determined by the position of the subtalar joint. The main invertors are the tibialis posterior and the gastrocnemius-soleus complex, and the main evertor is the peroneus brevis and, to a much lesser extent, the peroneus longus, which is mainly a plantar flexor of the first metatarsal. The tibialis anterior lies on the subtalar joint axis and has little influence on the subtalar joint, although it is the only functioning muscle at heel strike and, as such, besides controlling plantar flexion of the ankle joint, may resist eversion at the subtalar joint. The inversion that occurs during the last half of stance is due to the passive mechanisms noted previously, along with the input from the gastrocnemius-soleus complex and the posterior tibialis. The patient who lacks posterior tibial tendon function cannot initiate standing on tiptoe but can maintain the position when it is achieved. It can be concluded that posterior tibial tendon function is necessary to initiate inversion, and the gastrocnemius-soleus complex is necessary to maintain it.

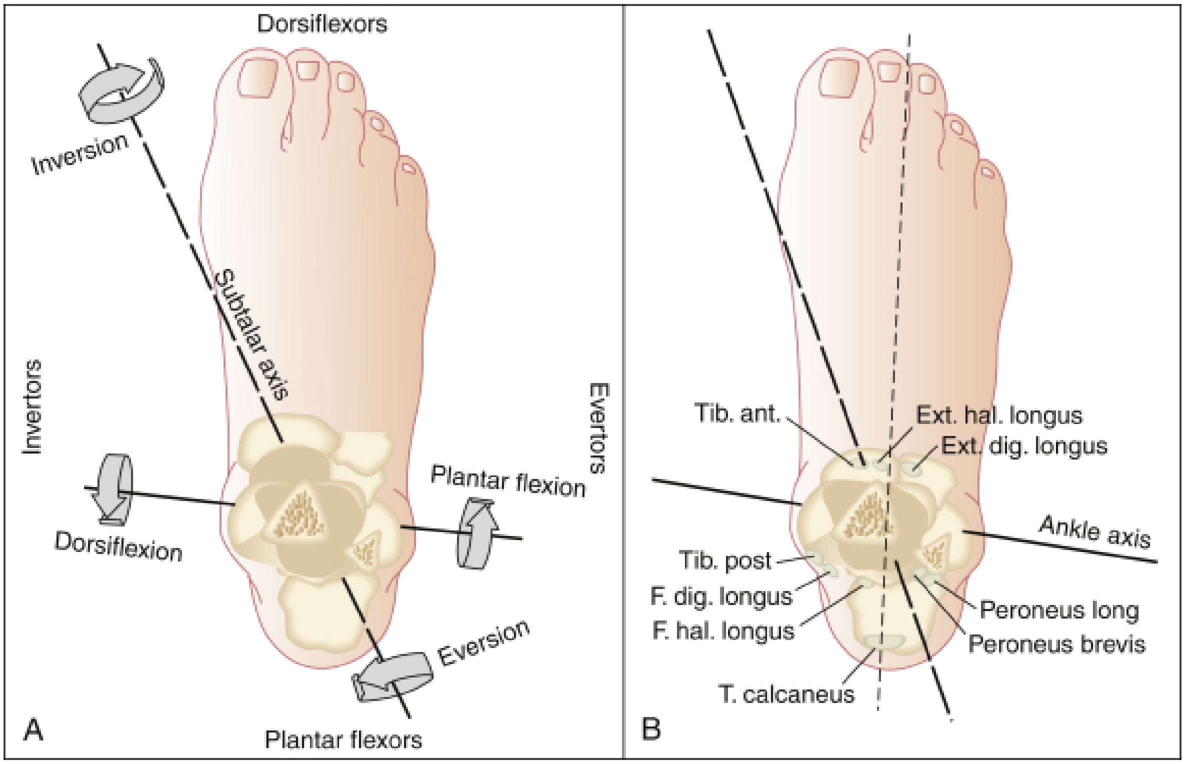

Figure 25A-9 **A,** The location and the types of rotation that occur about the ankle and the subtalar axes. **B,** The relationship of the various extrinsic muscles about the subtalar and ankle joint axes. Ext. dig. longus, extensor digitorum longus; Ext. hal. longus, extensor hallucis longus; F. dig. longus, flexor digitorum longus; F. hal. longus, flexor hallucis longus; T. calcaneus, tibialis calcaneus. *(Redrawn from Haskell A, Mann RA: Biomechanics of the foot. In American Academy of Orthopaedic Surgeons: Atlas of Orthoses and Assistive Devices. Philadelphia, Elsevier, 2008.)*

As noted in the discussion of ankle function, with running, the posterior calf muscles become active late in swing phase and remain active through most of stance (see Fig. 25A-4). Besides providing stability to the ankle joint, these muscles probably bring about some inversion of the subtalar joint before initial ground contact.

TRANSVERSE TARSAL JOINT

The transverse tarsal joint, consisting of the talonavicular and calcaneocuboid joints, lies distal to the subtalar joint and is influenced strongly by it.[20] The motion of the transverse tarsal joint is that of adduction and abduction and is measured with the calcaneus in neutral position and the forefoot parallel to the floor. Normal motion is about 20 degrees of adduction and 10 degrees of abduction.

The main support of the joint is ligamentous, but its stability is derived from subtalar joint inversion without much direct muscle control. The axes of the transverse tarsal joint are aligned such that when the calcaneus is in an everted position, the axes are parallel, permitting more motion to occur around this joint system. During normal walking, this occurs at heel strike, creating a flexible foot to absorb the energy of impact. When the calcaneus is inverted, the axes of the transverse tarsal joint are nonparallel, creating a stable joint system (Fig. 25A-10).[20] This occurs at heel rise and toe-off, creating a rigid foot to effectively lengthen the limb and assist in propulsion during running.

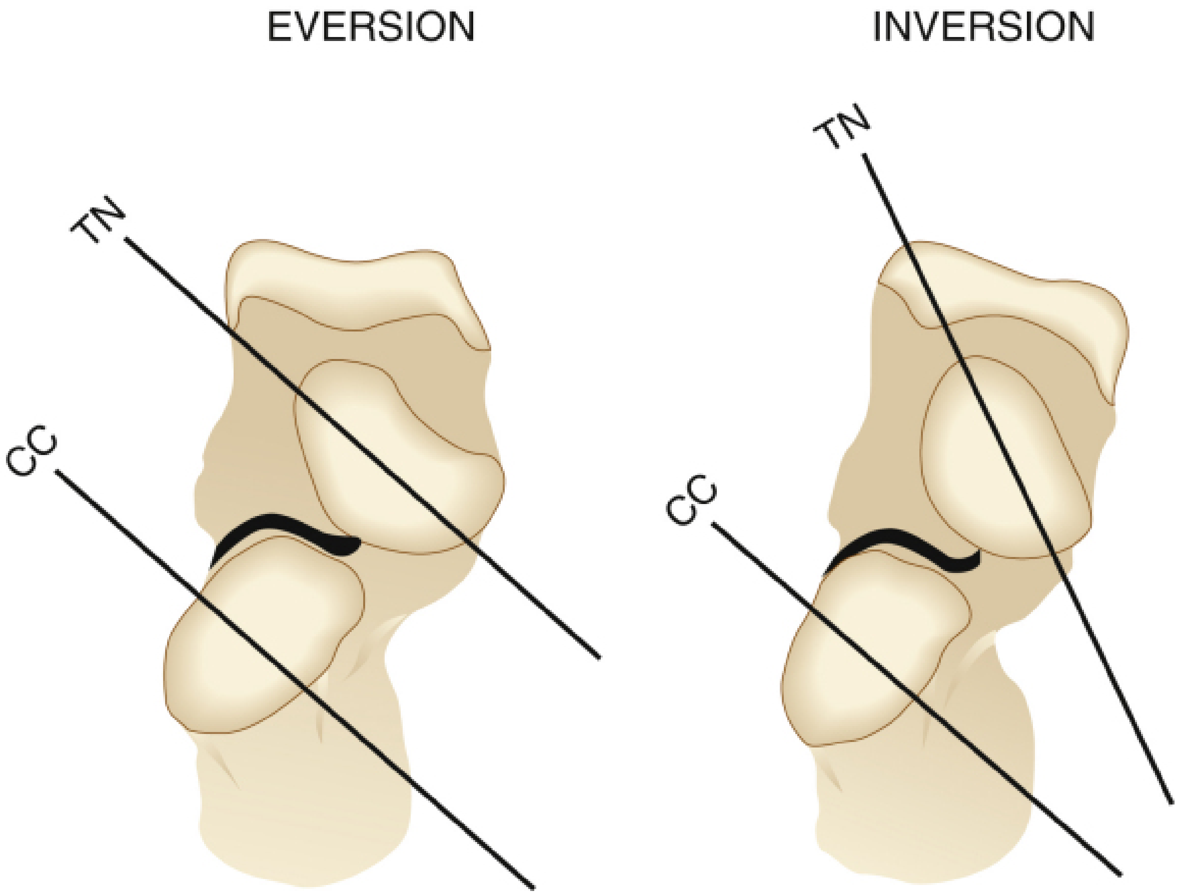

Figure 25A-10 The function of the transverse tarsal joint as described by Elftman. When the calcaneus is in eversion, the resultant axes of the talonavicular (TN) and calcaneocuboid (CC) joints are parallel or congruent. When the subtalar joint is in an inverted position, the axes are incongruent, giving increased stability to the midfoot. *(Redrawn from Mann RA, Haskell A: Biomechanics of the foot and ankle. In Coughlin MJ, Mann RA, Saltzman CL [eds]: Surgery of the Foot and Ankle, 8th ed. Philadelphia, Mosby, 2007.)*

WINDLASS MECHANISM AND METATARSAL BREAK

The windlass mechanism describes the function of the plantar aponeurosis during gait. The plantar aponeurosis arises from the tubercle of the calcaneus and inserts into the base of the proximal phalanges (Fig. 25A-11). After heel rise, the metatarsophalangeal joints dorsiflex, tightening the plantar aponeurosis. This depresses the metatarsal heads, elevates and stabilizes the longitudinal arch, and helps to bring the calcaneus into an inverted position (Fig. 25A-12). [21] The inverted calcaneus causes the transverse tarsal joint axes to diverge, helping to stabilize the midfoot at toe-off.

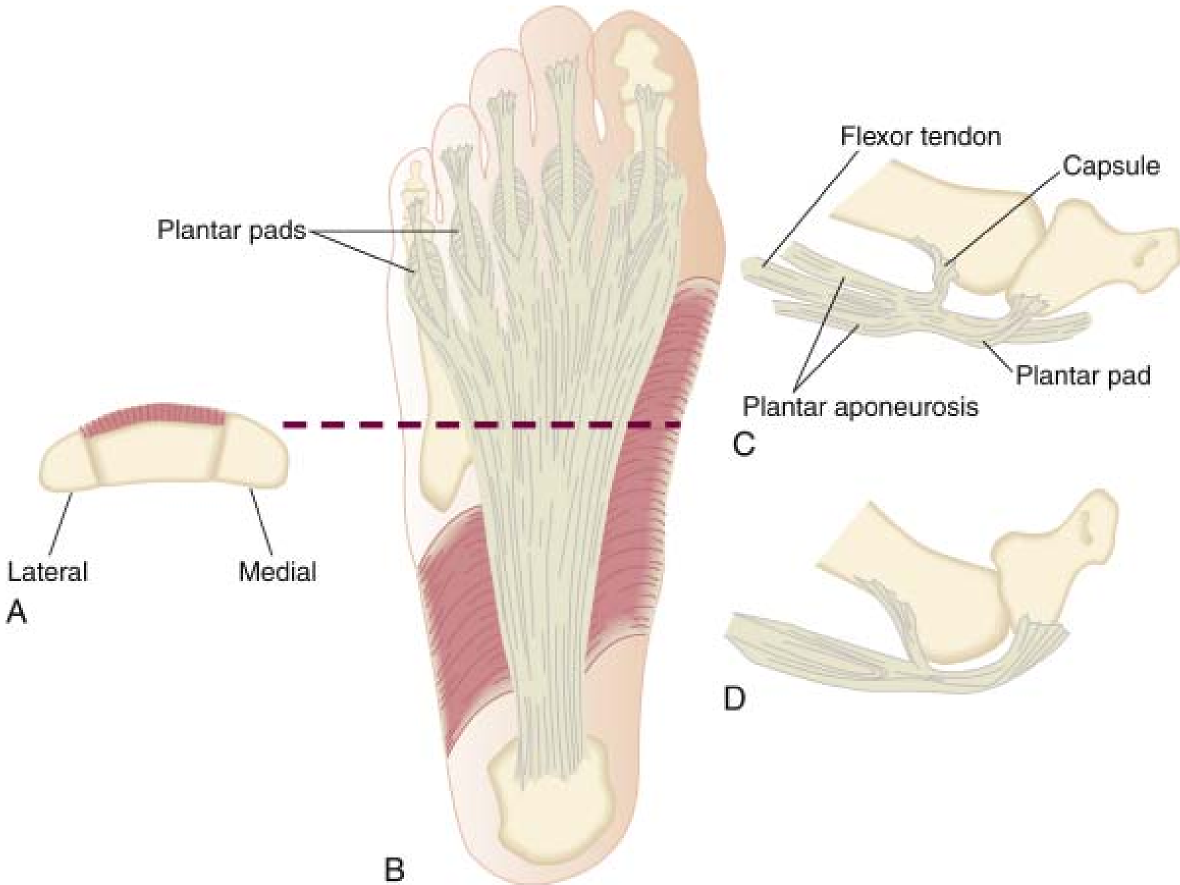

Figure 25A-11 Plantar aponeurosis. **A,** Cross section. **B,** The plantar aponeurosis originates from the tubercle of the calcaneus and passes forward to insert into the base of the proximal phalanges. The aponeurosis divides, permitting the long flexor tendon to pass distally. **C,** Components of the plantar pad and its insertion into the base of the proximal phalanx. **D,** Extension of the toes draws the plantar pad over the metatarsal head, pushing it into plantar flexion. *(From Mann RA, Haskell A: Biomechanics of the foot and ankle. In Coughlin MJ, Mann RA, Saltzman CL [eds]: Surgery of the Foot and Ankle, 8th ed. Philadelphia, Mosby, 2007, p 24.)*

Figure 25A-12 The function of the plantar aponeurosis. The *brown outline* shows the medial column with the foot at rest. The *red figure* shows the medial column with the first ray dorsiflexed. Note that dorsiflexion of the metatarsophalangeal joints tightens the plantar aponeurosis, which results in depression of the metatarsal heads, elevation and shortening of the longitudinal arch, inversion of the calcaneus, and elevation of the calcaneal pitch.

The oblique metatarsal break is created by the lateral slope formed by the metatarsophalangeal joints two through five (Fig. 25A-13).[18] This oblique line creates a cam-like action as the body weight is brought over the metatarsal heads, further enhancing external rotation of the lower extremity and inversion of the calcaneus.

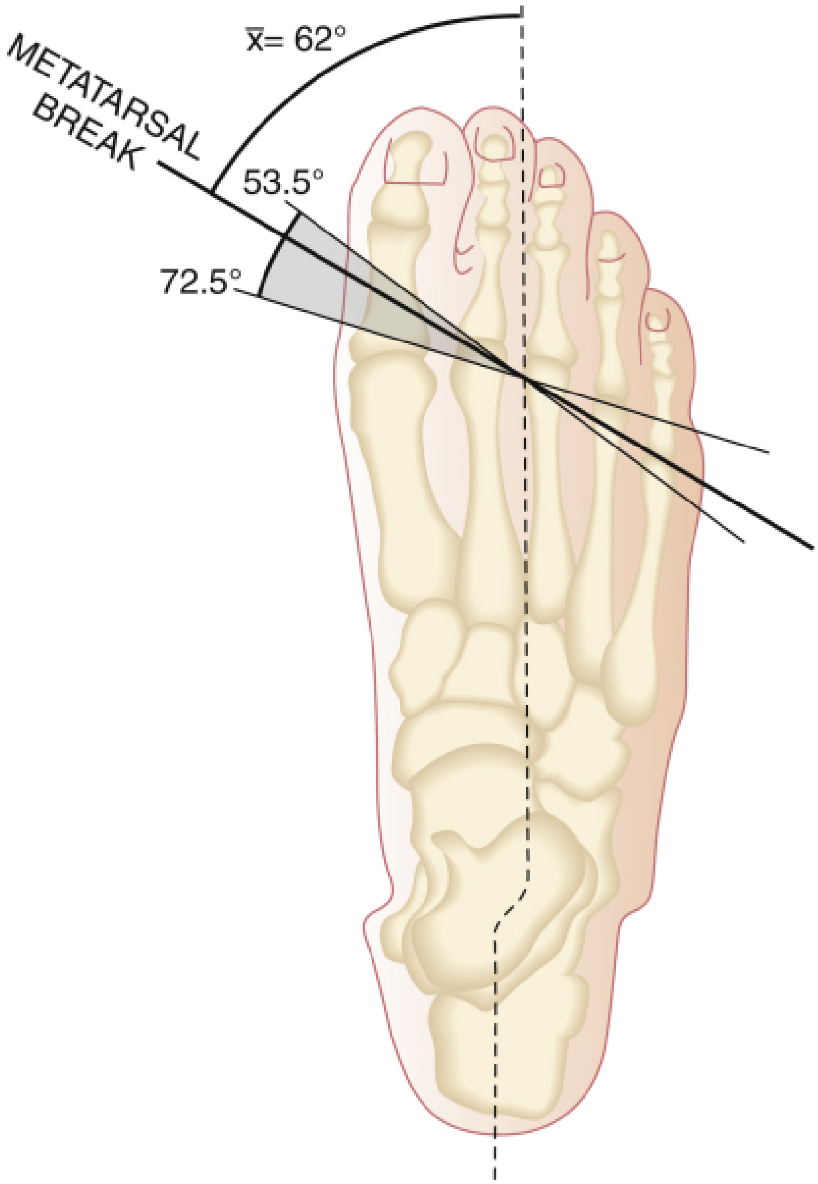

Figure 25A-13 The metatarsal break passes obliquely at an angle of about 62 degrees to the long axis of the foot. *(Adapted from Isman RE, Inman VT: Anthropometric studies of the human foot and ankle. Bull Prosthet Res 10:97, 1969.)*

LINKAGE OF THE FOOT AND ANKLE

The functions of the ankle joint, subtalar joint, transverse tarsal joint, and plantar aponeurosis have been examined and their interdependence described. The linkage between these joints should be emphasized further. The terms *pronation* and *supination* describe a coordinated series of movements of the foot and ankle that facilitate its two main functions during gait, namely, energy absorption at impact and energy transfer during stance (Table 25A-1). These joint motions are enhanced further by the function of the plantar aponeurosis, the transverse metatarsal break, and the muscles of the leg and foot. It is this linked series of movements that enables the athlete to absorb the forces of impact, yet create a rigid platform from which to push off.

TABLE 25A-1 -- Comparison of Foot Characteristics Based on Foot Position

Characteristic	Foot Position	
	Pronation	Supination

Characteristic	Foot Position	
	Pronation	Supination
Joint position	Ankle dorsiflexion Subtalar eversion Transverse tarsal abduction	Ankle plantar flexion Subtalar inversion Transverse tarsal adduction
Arch stiffness	Supple	Rigid
Gait cycle	Heel strike	Foot flat to toe-off
Function	Energy absorption	Energy transfer to ground

These joint linkages are essential for high-performance function of the lower extremity. If one of these linkages within the system fails to function properly, stress is placed on the joints proximal and distal to it. Although we speak of ankle joint dorsiflexion and plantar flexion, only about half of this motion comes from the ankle joint; the remainder comes from the movement occurring within the subtalar and transverse tarsal joints. [22] If there is diminished motion of the ankle joint, perhaps from an anterior impingement, degenerative changes within the joint, or fusion, the subtalar and transverse tarsal joints compensate for the lost motion. If there are degenerative changes within the subtalar or transverse tarsal joints, any loss of ankle joint motion is magnified. This compensatory increase in motion of the neighboring joints often leads to pain, loss of function, and degenerative changes over time. [23]

Moving distally, if the motion in the subtalar joint is restricted, its ability to translate rotation proximally and distally is impaired, placing increased stress on the ankle and transverse tarsal joints. Talocalcaneal coalition can lead to a spastic peroneal flatfoot or ball-in-socket ankle because of the effect of lack of subtalar motion. The degree of ankle joint dorsiflexion and plantar flexion also is affected, and the ankle can become arthritic from the abnormal stresses. [24]

Impairment of the transverse tarsal joint impairs subtalar joint motion because for subtalar motion to occur, rotation must occur around the talonavicular joint as well as the calcaneocuboid joint. If an isolated arthrodesis of the talonavicular or calcaneocuboid joint is carried out, most subtalar joint motion is lost. [25] When performing an arthrodesis around the hindfoot, sparing the talonavicular joint when appropriate usually leaves the patients with a more functional foot.

The metatarsophalangeal joints also are affected by loss of motion. First metatarsophalangeal joint dorsiflexion is lost in hallux rigidus, a degenerative arthritis of the first metatarsophalangeal joint. This leads to a compensatory external rotation of the foot during gait to relieve the stress across the involved area. This compensation, in turn, can affect the alignment of the lower extremity.

The theory behind orthotic use for many conditions involving the foot, ankle, knee, hip, and back is the effect it has on this linkage system within the lower extremity. Soft orthoses and compliant shoe material help absorb the impact of initial ground contact. For individuals engaged in repetitive sports, such as long-distance running, a material that helps absorb some of this impact could be beneficial if the athlete is having problems related to impact, such as heel pain, metatarsalgia, or shin splints. However, softer material paradoxically can lead to greater vertical impact when landing from jumps in an attempt to improve balance and stability. [26]

On a more sophisticated level, the use of a medial heel wedge, whether in the shoe or within an orthotic device, may have some influence on the rotation of the subtalar joint. [27] Because at the time of initial ground contact rapid eversion of the subtalar joint and flattening of the longitudinal arch occur, a buildup of material along the medial arch that prevents some of this rotation from occurring, in theory, would decrease the amount of internal rotation being transmitted to the lower extremity, affecting the ankle, knee, and hip. From a clinical standpoint, some patients appear to benefit from an orthotic device, although the benefit may be in part psychological. [28] A runner with chronic knee pain may be helped by an orthotic device that limits eversion of the calcaneus, which, in turn, diminishes internal rotation of the tibia and affects the patellofemoral joint. Reliable data to support this theory are lacking.

CRITICAL POINTS

- At heel strike, the foot and ankle help absorb the force of contact with the ground.
- During the heel rise and toe-off phases of gate, the foot becomes more rigid, providing a stable platform for the body.

- The subtalar joint links motion of the leg and foot such that eversion of the hindfoot causes internal rotation of the tibia at heel strike, and external rotation of the tibia causes inversion of the hindfoot at heel rise.
- Eversion of the hindfoot unlocks the transverse tarsal joints, making the foot supple; inversion of the hindfoot locks the transverse tarsal joints, making the foot more rigid.
- The muscles of the leg and foot contract both concentrically and eccentrically during the gait cycle to control the rate of ankle plantar flexion and dorsiflexion during walking and to provide stability during running.
- Athletics increases the normal stresses on the foot and ankle and can lead to acute or overuse injuries.

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SECTION B Sports Shoes and Orthoses

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There should be little surprise that an entire section of one chapter would be devoted to a discussion of sports shoes. The athletic shoewear industry has grown to such an extent that it has reached one of the pinnacles of achievement in our society—the front cover of *Sports Illustrated* (Fig. 25B-1). [1] With this achievement, both increasing recognition from Wall Street investors and harsh criticism alleging consumer exploitation surfaced. [30] [31] [32] Sports shoes became “high tech” and rode a wave of advertising to become a status symbol. [32] [33] Among today’s youth, the “right” shoe may vary from week to week. Athletic shoe sales rose to approach $8 billion in 1998, with fully half of that total brought in by the top two athletic shoe manufacturers. Not surprisingly, the top retailer of athletic shoes spent more on promotions and advertisements (some $163.2 million) than the next nine producers combined. [6] Major shoewear manufacturers have paid six-figure salaries to high-profile athletes and coaches to endorse their products. [32] [35] Financial benefits from shoe contracts have become a major consideration for college athletic programs and their coaches. [7] In this distorted environment, it is often difficult to wade through the hype to discover the contributions of merit in shoewear technology. This section attempts to do just that. A foundation of relatively stable information is provided to guide the reader through this subject despite the constant changes fueled by fashion trends and advertising gimmickry as well as scientific research. To understand where we are and where we are headed, some historical perspective is necessary.

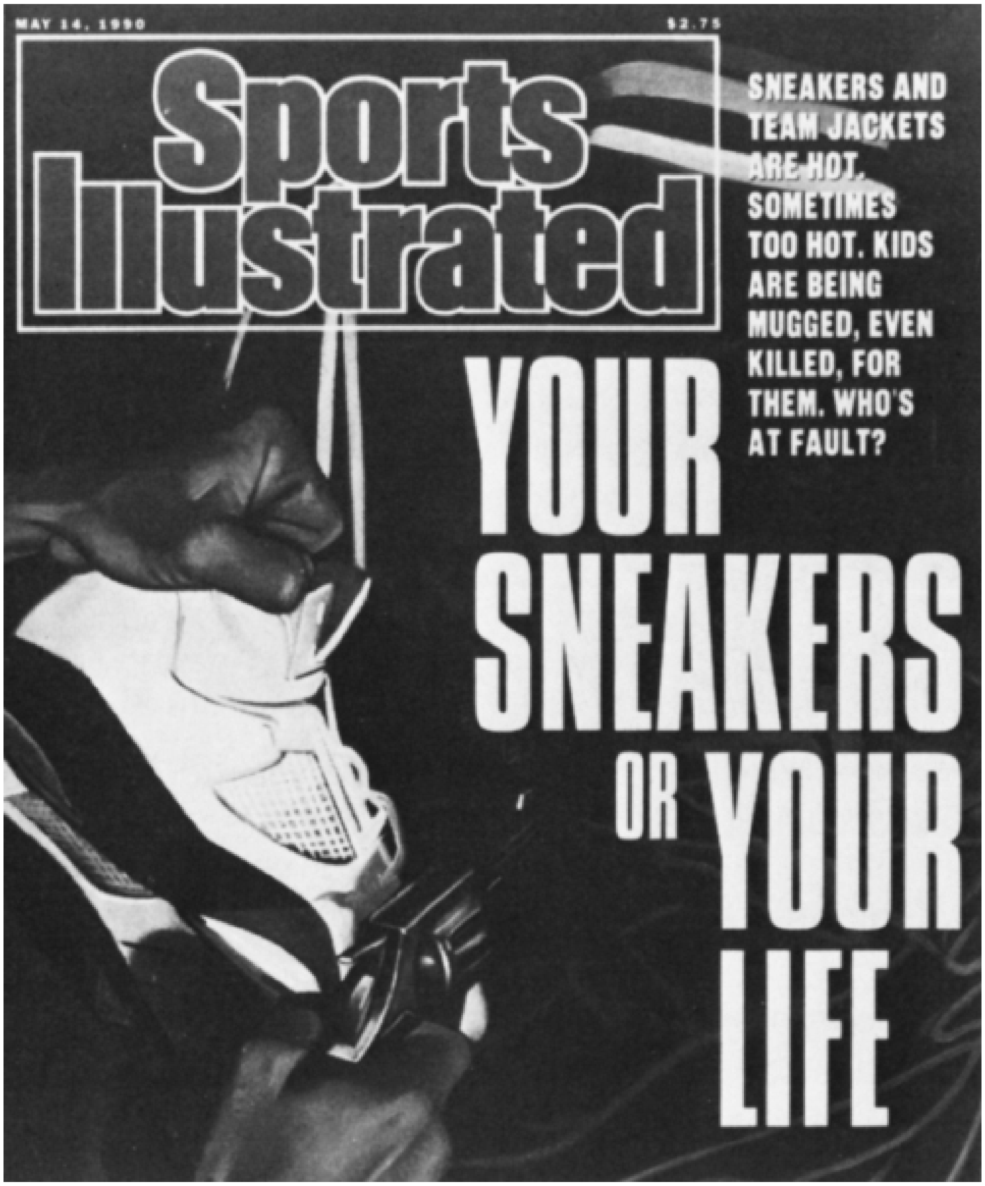

Figure 25B-1 *Sports Illustrated* cover indicating the notoriety of sports shoes. *(Illustration by Julian Allen. From Sports Illustrated, vol 72, May 14, 1990.)*

HISTORY

The history of sports shoes parallels the history of shoewear itself. According to legend, shoes were originally designed after an Arab chief dismounted from his camel onto a thorn and declared that all the earth would be covered with leather. Seeing the error in this logic, the chief's main advisor decided to make something that would cover just the feet. Although this makes a good story, it has not been supported by the discovery of shoes in the Fertile Crescent.[8] Indeed, the earliest footwear was discovered in south central Oregon in 1932 by anthropologist Luther Cressman—a sandal made from sagebrush bark (Fig. 25B-2).[36][37] This find dates back 10,000 years to pre-Columbian times, but design features indicate a much earlier origin. It supports the notion that the shoe's primary function is to protect the sole from the hazards of the environment.

Figure 25B-2 Earliest existing footwear, dating back some 10,000 years. It is made from sagebrush bark and was found in south central Oregon by anthropologist Luther Cressman in 1932. *(Photograph by Steve Bonini. Courtesy of University of Oregon Museum of Natural History and State Museum of Anthropology.)*

A more ancient type of shoe was a hide shoe made by folding the skin or hide of a beast around the foot. This is the forerunner of what we call a *moccasin*, a term derived from the Algonquin Indians and introduced into English literature in 1612 by John Smith's "Map of Virginia." [10] Examples of this form of shoe come from excavations in Denmark of early Bronze Age oak-log coffins dating to about 1000 BC. [10] Although earlier examples do not appear to have been preserved, one can assume that the early hunters of the Stone and Ice Ages must have been capable of seeing the advantages of covering the foot for protection. What could be more logical than using the hide of their prey to provide a suitable foot covering?

Rock carvings have provided evidence that these hide shoes were secured to the foot by lashing them around the instep and arch. [10] Cave paintings found in Spain dating to 15,000 BC depict boots made of animal skin and fur. [11] More recent descriptions of shoemaking from animal hides are provided in the works of Xenophon, Niebuhr, and Pinkerton. [40] [41] [42] Examples of the bear paw used as a shoe are seen in Figure 25B-3 from the Musée de l'Homme in Paris. [10] This bear paw with claws attached could be considered the first shoe with cleats.

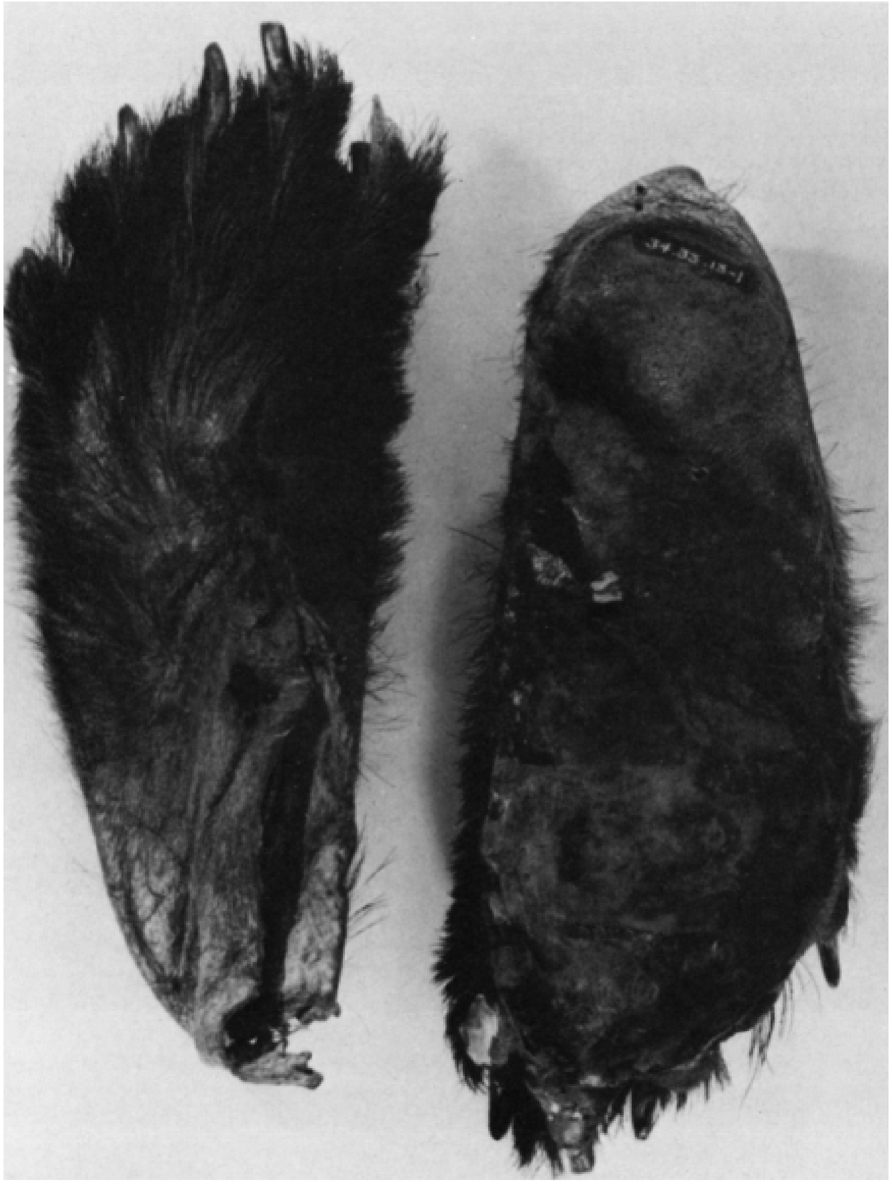

Figure 25B-3 Example of the bear paw shoe. *(Courtesy of the Musee de l'Homme in Paris.)*

A later development in shoewear and the second broad type of shoe is made of two components, an upper and a sole. [10] These are joined together at the lower edge of the foot. The appearance of this shoe occurs in Roman times, when a hide shoe reinforced by an extra piece of sole material was used. [11] Furthermore, during this time, the insole appears as a layer added for comfort and protection against chafing. These design features were a product of necessity owing to the abuse to which soldiers' feet were subjected. Similarly, modern design features were generated to protect the athletes' feet, particularly those involved in distance running.

Because early humans were largely dependent on hunting, one can postulate that the earliest footwear was used in running. With civilization's advancement and socialization, shoes took on symbolic functions. [11] Papyrus sandals for religious ceremonies and jeweled sandals for high-fashion gatherings have been discovered in the burial holdings of Egyptian pharaohs. [15] Although these have little to do with sports shoes, they do foreshadow the current specialization, trendy colors, and designs incorporated into athletic shoewear construction. Competitors in the early Greek games competed barefoot according to early drawings found on vases of that period (Fig. 25B-4). [9] Inasmuch as shoemaking was a well-developed trade by this time, it appears that early athletes eschewed comfort for the presumed benefits of barefoot performance, that is, less weight, better feel for the surface, and improved traction.

Figure 25B-4 Competitors in the early Greek games competed barefoot, according to early drawings found on vases from that period. *(Courtesy of the Metropolitan Museum of Art, Rogers Fund, 1914.)*

Robbins and Waked revived interest in barefoot running with their hypothesis that the excessive cushioning found in modern shoewear prevents appropriate sensory feedback and results in a "pseudoneurotrophic" effect. [16] The sensibility of the plantar foot is a key reason that gymnasts and dancers perform with bare or minimally shod feet. While an individual is running, plantar tactile reflexes and the intrinsic shock absorption system of the body complement one another and result in behavior modification to control load magnitude. Specifically, humans dramatically reduce impact force by altering knee and hip flexion at ground contact. [16] A series of studies by Robbins and coworkers have proposed that cushioned shoes lead to negligible decreases in load because subjects decrease flexion to accommodate the instability produced by softer surfaces. [43] [44] A recent study demonstrated that subjects presented with a "deceptive" advertisement of the ability of a surface to cushion impact led individuals to increase the ground reaction force of a barefoot footfall when compared with a "warning" and "neutral" message. This was despite the fact that the surface was covered with an identical thickness of ethyl-vinyl acetate surfacing material. [17]

Although the notion that "deceptive" advertising can lead to potentially harmful behavior associated with shoewear is intriguing in light of the enormous sums spent on advertisements by shoe companies, noted authorities on running and running shoes have not been impressed with this theory of the importance of sensory feedback. [18] They point out that biomechanical abnormalities such as excessive pronation, excessive Q angle at the knee, forefoot varus, and so on are the primary causes of running injuries, not a lack of sensory feedback. Furthermore, it is only with shoewear adaptations that these abnormalities can be corrected, according to these experts. [18] Ironically, one of the editors of *Runner's World* magazine recorded his shoewear experience over a 20-year period in a shoe diary and noted that a 5-year period of barefoot running was his healthiest period. [9] There is even a Web site now that promotes the benefits of shoeless running: www.runningbarefoot.com. [19] Although it is clear that Western-style shoes have contributed to many of the foot ills of modern society such as bunions, corns, calluses, and neuromas, [48] [49] there is circumstantial evidence to suggest that improvements in running shoe construction have reduced the prevalence of Achilles tendinitis and allowed greater numbers of average citizens to participate in the sport of distance running. [9]

Tracing the history of the running shoe is an enlightening look at the shoe industry itself, at the role of sports in society, and at the international trade competition surrounding sport and its premier athletes. The most thorough sources of information in this area are *The Running Shoe Book*, written in 1980 by Peter Cavanagh, and *The Complete Book of Athletic Footwear*, written by Melvyn Cheskin in 1987. [37] [50] Both trace the evolution of running shoes through the footraces of 16th-century fairs and the pedestrian races of the later 1800s to modern-day track and field competition. Important landmarks in this history can be picked out along the way. The turnshoe construction technique was firmly established by the 12th century. It allowed a shoe to be made with the seams on the outside and the smooth material inside next to the foot. The shoe was turned inside out to produce the finished product. [37] [50] By the 14th century, shoe construction had incorporated small strips of leather called welts to allow a replaceable outsole to be added to the upper. [23] Since the 14th century, shoemaking has been fairly standardized, with shoes consisting of the following:

1. Two parts: upper and lower
2. Four processes: cutting, fitting, lasting, and bottoming
3. Eight tools: knife, awl, needle, pinchers, last, hammer, lapstone, and stirrup [24]

With the Industrial Revolution, the craft of shoemaking went from an in-home trade to a model of manufacturing method with the use of machines and mass production. Entire books have discussed the significance of this method to the development of industry in the United States. [53] [54] Leather was the mainstay of shoemaking throughout this period and continued as such into the 1900s. A change in shoemaking and the origin of the sneaker were presaged by the first patent, granted in 1832, for attaching rubber to the sole of the shoe. Unfortunately, the material was too unstable and lacked durability. [11] In 1839, Charles Goodyear's vulcanization process turned rubber into a usable material, but it was more than 100 years before rubber replaced leather as the most desirable outsole material for running shoes. [9]

Cavanagh cites the development of the Spencer shoe, a spiked shoe found in England's Northampton Museum, as the 1865 precursor of modern track shoes (Fig. 25B-5). [9] This shoe shows the separation of running shoes into a line separate from street shoes, although a spiked shoe used for cricket was patented in England in 1861. Spiked shoes were used in the short races popular in that day. Longer distances became popular in the latter part of the 19th century. Races around circular tracks for 144 straight hours became a spectator event imbued with international flavor. These pedestrians, as the participants were called, wore high-top leather boots and thick wool socks reminiscent of combat boots used in the military. Although pedestrian races faded in popularity, long-distance racing gained an audience, and the Olympics were reborn in Athens, Greece, in 1896. A marathon was included as a race of 40 km to commemorate the legend of Pheidippides. Cavanagh marks this Olympic race as the impetus for development of the distance shoe, or training shoe, we use today. [9]

Figure 25B-5 The Spencer shoe, a spiked shoe found in England's Northampton Museum, was the 1865 precursor to modern track shoes. *(Courtesy of the Northampton Museum, Northampton, England.)*

The popularity of the marathon prompted the Spalding Company to introduce a long-distance shoe for the general public in 1909. [9] Three shoes were advertised, two being high-tops (Fig. 25B-6). They had leather uppers and rubber soles and were priced at $5 to $8. A retired shoemaker named Richings began custom making a shoe for distance runners near the 1930s that predated the custom shoewear used by elite athletes of our day. [9] By the early 1900s, production of running shoes was in full swing, and the 1915 Spalding catalog advertised shoes for sprinting, middle-distance running, jumping, and pole-vaulting (Fig. 25B-7). [22] Competition entered the scene at about the same time when Sears Roebuck entered the catalog shoe sales market and began the continuing controversy over who makes the best running shoe. [22] Endorsements by famous athletes were seen much earlier, but notable shoes were the Kiki Cuyler, Jr., basketball shoe and the Chuck Taylor All-Star shoe. We continue to see society's ongoing enchantment with famous athletes and their shoewear, as evidenced by the incredible popularity of the "Air Jordan" and "Bo Knows" campaigns of the Nike shoe company in the late 1980s and early 1990s. Despite recent cuts in advertising budgets for most of the major shoe manufacturers, [27] the fact that basketball shoe sales alone are responsible for as much as 25% of total athletic shoe revenue [28] ensures that certain National Basketball Association stars will expand their talent into marketing.

Figure 25B-6 Long-distance shoes introduced by the Spalding Company for the general public in 1909. *(From Cheskin MP: The Complete Handbook of Athletic Footwear. New York, Fairchild Publications, 1987.)*

Figure 25B-7 Page from the 1915 Spalding catalog advertising shoes for sprinting, middle-distance running, jumping, and pole-vaulting. *(From Cheskin MP: The Complete Handbook of Athletic Footwear. New York, Fairchild Publications, 1987.)*

A shoe designed for sports alone did not come into existence until the latter half of the 19th century. Croquet was a popular recreation during the Victorian period, and a croquet sandal appeared during this time.[11] Known as the *sneaker,* it was in use by the 1860s and had a fabric upper, a rubber sole, and laces.[37] [39] [57] Further sports development in the late 1800s spawned the need for durable but lightweight shoes with variable traction requirements depending on the playing surface. Wilcox provides several illustrations of these specialized sports shoes of the late 1800s in his book, *The Mode in Footwear* (Fig. 25B-8).[29] From these developments, we can trace the roots of the multibillion-dollar sports shoe industry and can conclude that the protection of our feet and fashionable design have always been important concerns of humankind. From this foundation, an explosion occurred in sports-specific footwear that has provided us with today's shoes for basketball, rock climbing, tennis, snowboarding, soccer, gymnastics, fishing, rollerblading, skating, jumping, sprinting, and so forth (Fig. 25B-9).

Figure 25B-8 Illustrations of specialized sports shoes of the late 1800s. *(Adapted from Wilcox RT: The Mode in Footwear. New York, Charles Scribner and Sons, 1948.)*

Figure 25B-9 Photograph from typical shoe store with wall of sport-specific shoes in all varieties. *(Photograph by Andrew Borom.)*

ANATOMY OF THE SPORTS SHOE

Just as the anatomy of the human body is the basis on which the surgeon's skill rests, the anatomy of the sports shoe is critical to those who must understand athletes and their injuries. Although the process of shoe manufacturing has evolved into a multibillion-dollar industry, the basic shoe remains the same. This section first looks at the anatomy of the basic sports shoe and then discusses the shoe features unique to particular sports. The actual manufacturing process is discussed briefly.

Most of what has been written about athletic footwear has concentrated on shoes designed for the runner. Therefore, the prototype shoe for this section is the running shoe, and shoes for other sports are described in similar terms with specific modifications. Figure 25B-10 illustrates the components of the shoe.

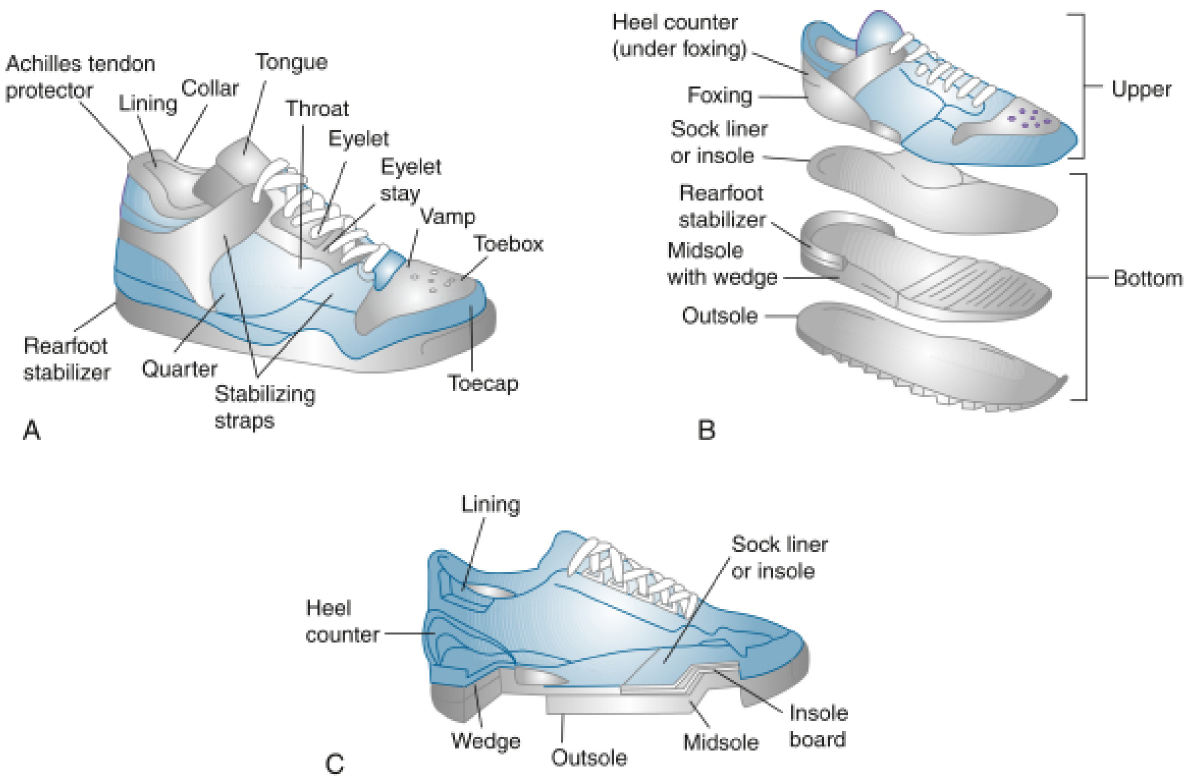

Figure 25B-10 Illustrations of athletic shoes. **A,** Overview of external appearance. **B,** Separation of shoe into component parts. **C,** Sectional view of interior of shoe.

For the sake of simplicity, the shoe can be broken down into two basic components: the upper and the bottom. The upper covers the foot, whereas the bottom cushions it and provides the interface between the foot and the surface. These two basic components are then subdivided into their various parts. The upper is composed of toe box, toe cap, vamp, quarter, saddle or arch bandage, eyelet stay, eyelets, throat, tongue, collar, Achilles tendon protector, heel counter, foxing, forefoot and rearfoot stabilizers, and lining. The bottom consists of sockliner or insole, insole board, midsole, wedge, and outer sole. Although some of these names differ from those used in traditional shoemaking, the actual construction of a sports shoe does not vary remarkably from the traditional shoe manufacturing process (see Glossary in Box 25B-1).

Box 25B-1

GLOSSARY OF FOOT AND SHOE TERMINOLOGY [*]

Abduction: To move away from the midline of the body.

Achilles notch: A depression cut into the back of the heel collar to provide a secure fit and prevent irritation of the Achilles tendon.

Adduct: To move toward the midline of the body.

Adduction: Moving a part toward the midline of the body.

Adhesive (cement): Substance capable of holding materials together by surface attachment.

Air: First introduced in 1979, Nike's cushioning concept of encapsulated air units in the midsole isn't actually air, it's Freon. Depending on the model, the air units may be in the heel or forefoot, or both.

Air ball: An air-pressurized ball imbedded in the heel of Hi-Tec Badwater models for additional shock absorption.

Anatomical: Pertaining to the structure of the body.

Anatomical last: A stabilizing footbed contoured in such a way that the heel sits down in the midsole, rather than resting atop a flat platform. Developed and used extensively by Turntec.

Anatomy: The study of the structure of the body and the relationships between its parts.

Anterior: Front portion.

Anterior heel: Type of metatarsal bar, also known as *Denver bar* or *Denver heel;* the apex coincides with the posterior edge under the posterior half of the metatarsal shafts.

ARC: Avia's stabilizing system, which is made of plastic in one of two configurations. Placed in the rearfoot, the "fingers" of the ARC spread out on impact to absorb shock and stabilize the foot.

Arch bandage: Reinforcing strips of fabric stitched inside the shoe on the medial and lateral quarters.

Arch cushion (cookie): Support pad for the medial arch of the foot.

Asymmetric: In shoemaking this applies to lasts and patterns that have uneven shapes, the right side different from the left.

ATP (heel horn): Extended padding at the back heel collar to protect the Achilles tendon (Achilles Tendon Protector).

Autoclave: Vessel or oven in which chemical reaction or cooking takes place under pressure such as in the vulcanizing construction method.

Axis: A reference line for making measurements. Ground reaction forces are usually evaluated relative to a set of three orthogonal axes: vertical, longitudinal (direction of motion), and transverse (right angle to direction of motion).

Backpart (rear foot): Portion of the last extending rearward from the break of the joint to the back of the last.

Backpart width: The width of the heel end measured parallel to the heel featherline plane at a specified distance from the heel point.

Bal (Balmoral): Front-laced shoe in which the meeting of the quarters and the vamp is stitched or continuous at the distal end of the throat. Bal is the abbreviation of Balmoral, the Scottish castle where this style was first introduced.

Ball: Widest part of the sole, at the metatarsal head.

Ball girth: Circumference measure around the last encompassing the first to the fifth metatarsal area.

Bar, comma: Comma-shaped bar wedged laterally and posteriorly, also known as *Hauser.*

Bar, Denver: See Anterior heel.

Bar, Jones: Metatarsal bar placed between the inner sole and outer sole.

Bar, Mayo: Metatarsal bar with the anterior edge curved to approximate the position of the metatarsal heads.

Bar, metatarsal: Rubber, leather, or synthetic bar applied transversely across the bottom of the sole, with the apex immediately posterior to the metatarsal heads.

Bar, rocker: Sole bar having its apex beneath the metatarsal shafts causing rocking instead of flexing action.

Bar, Thomas: Narrow metatarsal bar with abrupt anterior and posterior drop-offs.

Bar, transverse: See Bar, metatarsal.

Base plane: The plane to which the last in its proper attitude is referenced for the purpose of defining certain terms.

Bias cut: Cut away upswept heel.

Bilateral: Affecting both right and left sides.

Biomechanics: The study of the internal and external forces acting on the human body and the effects produced by these forces.

Blind eyelet: A metal or plastic eyelet concealed beneath the top surface of the shoe leaving only a small, rimless hole.

Blown rubber: The lightest kind of rubber outsole material. As the outsoles are manufactured, air is injected into the rubber to lighten and soften the outsole. Few outsoles today are made with full blown rubber because it lacks durability, but many outsoles have blown rubber in the forefoot and midfoot for lightness and a harder carbon rubber in the high-wear area of the heel.

Blucher: Front-laced shoe in which the quarters are not attached distally to the vamp, giving more allowance at the throat and instep in fitting. Opposite of bal style. Front quarters or tabs are stitched over the vamp for a short distance at the throat.

Board last: One of three ways shoes are constructed. A fully board-lasted shoe is constructed by gluing the upper to fiberboard before it is attached to the midsole. Board lasting promotes stability and provides a good platform for orthotics but lacks flexibility. Few new models are fully board lasted. (See combination last and slip last for information about the more common types of last.)

Boot, high top: High quarter shoe in which the quarters cover the malleoli.

Bottom: The sole up to the breast of the heel. On a wedge sole, the term covers the complete sole.

Bottom filler: Material that fills the cavity between the outer and inner soles.

Bottoming: The operation of attaching the completed sole to the upper.

Bottoming out: When the midsole material has worn out and is too soft relative to a runner's size, it compresses too quickly, which results in compromised shock absorption and support.

Box toe: Hardener used to maintain shape of front toe area.

Break: Flex point or path; creasing formed at the vamp when the shoe is dorsiflexed.

Breastline: An arbitrary line defining the forward boundary of the heel seat.

Breathability: The ability of a material to absorb and ventilate foot moisture; not to be confused with porous.

Bumper: Rubber toe strip attached over front toe area.

Calfskin leather: Leather made from the skin of calves.

Cantilever: A concave outsole design in which the outer edges flare out on impact to dissipate shock. Used extensively by Avia.

Carbon rubber: The most durable kind of rubber outsole material. It's a solid rubber with a carbon additive that makes the material stronger. If an outsole is not full carbon rubber, it probably has a carbon-rubber heel pad.

Celluloid: A thermoplastic material.

Cellulose: Natural polymeric.

Cement process: Construction stuck-on bottoming method.

Center of pressure: An outsole design in which the middle area is bordered by an elevated tread pattern along the outer rim to promote stable footing. This also makes for a lighter shoe by exposing the midsole and eliminating unnecessary outsole material in the center. Used in some Nike, Diadora, New Balance, Hi-Tec, Avia, and Adidas models.

Certified pedorthist: One who is certified by the Board for Certification in Pedorthics (BCP).

Chainstitch: Sewing method used for stitching uppers to soles.

Chukka: Three-quarter Blucher boot with two or three eyelets or a strap with a buckle.

Circular vamp: Design vamp extended from toe to heel breast.

Coefficient of friction: A number between 0 and 1 indicating the slip resistance of a material such as a shoe sole on a particular surface. The greater the value, the less likely any slipping.

Collar: Top line of the shoe quarters. Many are padded. Narrow strip of material stitched around the proximal edge of the quarter.

Combination last: Last with wider forepart and narrow heel fitting. Indicates the shoe is board lasted in the rearfoot for stability, but slip lasted in the forefoot to promote flexibility. Remove the sock liner of your shoe; if it is combination lasted, you will find a fiberboard in the rearfoot and stitching in the front. The last deviates from standard proportions, usually to accommodate feet with narrow heels.

Compaction: Permanent flattening and deformation of sole material (bottoming out).

Composition: Scraps that are pulverized, compressed, and held with a binder to form a sheet material for insoles, midsoles, heel bases, and other components.

Compression deflection: The amount of deformation observed in a material after it has been subjected to a compressive or impact load.

Compression mold: Shaping materials by heat and pressure.

Compression set: The amount of permanent deformation observed in an unloaded material following a single or multiple load application.

Conformability: Ability of a material to mold itself to the shape of the foot.

Contact face: The surface brought into contact with another surface or object.

Contoured midsoles: Similar to anatomic lasts, contoured midsoles are shaped to the foot. This promotes stability.

Contour insoles: Foam insoles capable of retaining pressure pattern of the foot.

Cookie: Arch pad in shoe; wafer-shaped longitudinal arch support.

Copolymer: A natural or synthetic compound.

Cordovan: Leather not made from hide or skin but from a ligament-like shell found under the buttocks of animals such as the horse, mule, and zebra. Often referred to as a “shell” and tanned in a burgundy (cordovan) color.

Cork: Made from the bark of the cork tree, which may be combined with other materials. Available in various forms such as sheet cork, natural cork, cushion cork. Different names are given to the cork according to the binders used.

Corrective shoe: Shoe with special features designed to help correct some type of foot disorder. The term has been displaced by other terms such as a *prescription shoe* or a *modified shoe*.

Corset, ankle: Reinforcement to preserve the shape of the quarter’s counter. Laced fabric within the shoe intended to retain the hindfoot on the inner sole.

Counter, long lateral: Counter extended anteriorly beyond the breastline on the lateral side.

Counter, long medial: Counter extended anteriorly beyond the breastline on the medial side.

Counter (pocket): See Heel counter.

Crepe rubber: Natural rubber soling material; latex rubber compounded for use as soles and heels.

Crest, toe: Convex cushion under the plantar phalangeal-sulcus.

Curved last: Refers to the shape of a shoe. Curve-lasted shoes are shaped somewhat like a banana and offer less medial (inner) support but greater foot mobility. Generally, curved-lasted shoes are for biomechanically efficient, faster runners who want a responsive shoe.

Cushioning: The ability to absorb shock. Because a runner generates a force of about 3 times the body weight on impact, this is a crucial shoe characteristic. Cushioning is primarily a function of the midsole.

Custom shoes: Shoes made to the customer’s specifications.

Dellinger web: Embedded into the midsole of some Adidas models, the Dellinger web is a fabric that maintains the durability of the midsole. Designed by University of Oregon coach Bill Dellinger.

Denier: Weight of synthetic fibers (measure of fineness).

Density: Weight per unit volume of a substance. The measure of the firmness of the midsole material. Many shoes have midsoles of varying densities. For example, a two-density EVA midsole will usually have the firmest material (designated by a darker color) on the medial side to control pronation.

Derby: Design quarters overlapping vamp and tongue.

Design: Pattern or cut of upper.

Die cutting: Cutting of upper or sole materials with metal dies.

Differential loading: The application of forces of varying magnitudes.

Dip construction: Direct injection process.

Distal: Part farthest from the central portion of the body.

Dorsal: Top of foot, the upper surface of the foot.

Dorsiflexion: Moving the toes up toward the distal end of the foot toward the leg.

Doubler: Interlining placed between the vamp and vamp lining for additional reinforcement; interfacing between upper material and lining.

Drop-off: Anterior vertical edge of a metatarsal.

Duo process: Method of upper assembly construction by cementing instead of stitching edge.

Durometer scale: A method of determining material hardness on a scale of 0 to 100, with lower readings indicating softness.

Dutchman: Lateral sole wedge.

DVP: Direct vulcanizing process.

Elasticized material: Resilient fabric used for goring in panels and inserts for shoe uppers.

Elastomer: Term used for synthetic rubber.

Electrodynograph: An instrumentation system consisting of individual sensors to measure pressure at selected locations on the bottom of the foot.

Electromyography: The measurement of the electrical activity associated with muscular contractions.

Elevation: Material added to the entire sole or heel.

Elvalite: A new foam developed by DuPont that's being used as a midsole material in some Reebok models. Elvalite has the cushioned feel of EVA but is more durable.

Elvaloy: Resin modifier added to PVC.

Embossing: Depressing a specific pattern in leather or fabrics.

EVA: Ethylene vinyl acetate (EVA) is the most common midsole foam used in running shoes. Compression-molded EVA is heated and compressed into the shape of the midsole. It is light, resilient, and has good cushioning properties. Nearly every running-shoe company uses EVA in at least some of its midsoles.

Eversion: Turning out the plantar aspect of the foot from the midline of the body.

Evert: To turn out the plantar aspect of the foot so that it faces away from the midline of the body.

Exercise physiology: The study of the effects of exercise on the biochemical function of the body and its parts.

Expanded vinyl: Soft, nonbreathable PVC (stretchy base) material (as opposed to nonexpanded vinyl: harder, nonbreathable [rigid base] material).

Extended eyestay: A design wherein the eyestay is extended to form the toe cap.

External: Outer part; lateral.

External heel counter: A rigid, plastic collar that wraps around the heel of the shoe for support and to control pronation.

Eyelets: Holes for lacing (blind) with metal reinforcements or eyelet hooks.

Eyestay: Reinforcement around lacing holes.

Fabric: Woven or nonwoven flexible material.

Feather edge: Last bottom profile.

Finish: Coating on leather or synthetic material.

Finishing: End of manufacturing process.

Flanging: The edge where the upper is turned outside for attachment to outsole or midsole.

Flare: Widened heel or sole base.

Flared heel: Wider flanged heel for landing.

Flex: To bend.

Flex grooves: Strategically placed ridges in the midsole of the forefoot that make the shoe more flexible at toe-off.

Flexibility: A shoe's ability to bend; the rigidity of the shoe bottom composite usually evaluated in the forefoot region of the shoe.

Flex path (break): Girth area at the main metatarsal of foot, which must flex as foot pushes off from ground.

Flexion energy: The energy required to bend a shoe or object through a flexion cycle.

Flow molding: The construction method of molding PVC-coated materials as an exact replica of original uppers.

Footbridge: A stability device. As used by Nike in the Air Structure and Air Span II, the footbridge is molded into the midsole across the rearfoot. Reebok uses a footbridge in its Ventilator, but it's placed under the arch on the medial side. Asics uses a variation of the footbridge in the Gel-MC.

Footframe: An extension on top of the midsole or an additional piece that cradles the foot for added support and prevents the foot from rolling over.

Force: A pushing or pulling effect that produces motion or deformation of an object or material.

Forefoot stability strap: A leather or plastic overlay on both sides of the ball area of the shoe that reinforces the upper and offers stability and support.

Forepart: Area of foot from the ball to the toe; portion of the last extending from the ball to the toe.

Forepart centerline: The best line of symmetry of the forepart bottom pattern.

Frontal plane: The vertical plane that passes through the body dividing into a front and a back half.

Functional anatomy: The study of the effects of body structure on performance.

Functional shoe: Shoe designed to serve a specific purpose.

Gait laboratory: A testing lab equipped with specialized equipment for the study of walking and running.

Gel: The primary cushioning system used by Asics in all of its performance shoes. It is a pad of silicone in the midsole, which, depending on the model, is found in the heel or the forefoot, or both.

Geometric last: Last using a geometric rather than the traditional arithmetic grading system. Intended for better shoe fit and to facilitate automated manufacturing.

Girth: Circumferential dimension measured around the last; widest part of the last.

Goniometer: An instrument used to measure angles. It can be employed to evaluate motions of the joints, particularly the knee and ankle joints.

Goodyear welt: Construction method of stitching uppers to sole; shoemaking process in which the joining of the upper, inner sole, and outer sole is accomplished with a welt.

Goring: Elastic fabric inserted in the front or sides of an upper, the expansion of which allows a larger opening to insert the foot.

Grade increment: The change per size and shoe width of any last dimension.

Grade rate: The ratio of the change in girth per size to the change in length per size.

Grading: Method used by designers to size original patterns.

Heel breast: Anterior margin of heel; front face of the heel or anterior portion at the shank.

Heel counter: A firm, usually plastic cup that is encased in the upper and surrounds the heel. It controls excessive rearfoot motion. It may be notched to accommodate the Achilles tendon.

Heel counter pocket: Rearpart upper material pocket containing heel stiffening material.

Heel curve: A side-view profile of the back end of the last from the top of the last to the heel seat or featherline.

Heel curve angle: Angle between the heel featherline plane and heel point 2½ inches (63 mm) up from the heel point intersecting the heel curve.

Heel elevation: Modification measured in a vertical line at the center of the heel; the vertical distance between the base plane and the heel point is the heel elevation.

Heel featherline: A line that defines the heel seat shape.

Heel height: Vertical measurement from the plantar surface to the heel seat at the anterior surface of the heel, usually in increments of eighths of an inch.

1. Spring heel, 3⁄8 to 6⁄7 inch; heel base lies under the outer sole eliminating a definite heel breast.
2. Flat heel, 6⁄8 to 10⁄8 inches.
3. Military heel, 10⁄8 to 13⁄8 inches.
4. Cuban heel, 13⁄8 to 14⁄8 inches.
5. Wedge heel, 4⁄8 to 14⁄8 inches; slopes upward from ball to posterior heel.

Heel pad: Resilient material to cushion or raise the heel.

Heel pitch: Slant at the posterior aspect of the heel; amount of rise at back of last when last is held level.

Heel plug: Found in multidensity outsoles, where the most durable rubber is placed in the high-wear area of the heel.

Heel point: The rearmost point of the heel featherline.

Heel seat: Area of the shoe upon which the foot rests; the bottom surface of the heel end of the last from the breast line back.

Heel seat width: The greatest width of the heel seat measured from featherline to featherline perpendicular to the heel centerline.

Heel, Thomas: Heel with anteriorly curved medial border.

High cut: Over the ankle shoe design.

Horizontal plane: See Transverse plane.

Hytrel: A resilient and durable polymer plastic developed by DuPont and used for a variety of shoe components by several companies. It's most commonly used as forefoot support straps.

IMP (injection): Injection molding process form of shoe construction (see also Lasting insole).

Inflare: Asymmetric inward swing of last shape; last or shoe whose forepart provides more medial than lateral surface area.

Injection molded: Shoe construction whereby a heat-softened plastic is injected into a mold, then compressed against the mating surface of a concentric mold and allowed to cool and harden.

Inlay: Prefabricated removable material upon which the foot directly rests inside the shoe. In some shoes, the inlay is an integral design component.

Inner sole: Material conforming to the size and shape of the last bottom upon which the foot rests (see also Insole).

Insert: A type of orthosis, although the term has been used interchangeably in some circles with inlays and insoles to designate an off-the-shelf device placed inside the shoe. The Health Care Financing Agency defines it as a total contact, multiple-density, removable inlay that is directly molded to the patient's foot or a model of the patient's foot and that is made of a suitable material with regard to the patient's condition.

Inserts: Metal threaded retainers for spikes or studs.

Insole: Integral design component (layer) of the shoe that is the shoe's structural anchor to which is attached the upper, toe box, heel counter, linings, and/or welting.

Instep: Medial inside arch area of the shoe.

Instep: Portion of the upper over the midfoot.

Instep girth: The dimension around a last passing through the instep point.

Interface: The surface forming the common boundary between two bodies or spaces.

Internal: Inner part; medial.

Inversion: Turning the plantar aspect of the foot in toward the midline of the body.

Invert: To turn in the plantar aspect of the foot so that it faces the body's midline.

Ionic cushioning: Pillars of polyurethane in the midsole used by Saucony to add durability.

Ionomer resins: A family of thermoplastic resins.

Iron: Dimension used for measuring sole thickness, 1⁄48-inch; thus a 6-iron sole is 1⁄8-inch thick.

Isoprene: Fundamental rubber molecule.

Joint girth: The greatest dimension around the last passing through the break joint.

Kinematics: The science of pure or abstract motion.

Kinetic energy: Energy associated with motion.

Lace locks: Plastic devices on the upper that maintain tension on the laces.

Lace stay: Portion of the upper containing eyelets for lacing.

Lace-to-toe: Low- or high-quarter shoe laced to the toe; a design in which the eyestay is extended down to the toe box area.

Last: Three-dimensional facsimile of the foot; model approximating the shape and size of the weight-bearing foot, made of wood or plastic, over which a shoe is formed. The shape of the last determines the shape of the shoe. The straighter the last, the greater the medial support. Generally, faster, lighter runners who need less support prefer curved-lasted (or semicurved) shoes. Runners who need maximal medial support and those who overpronate opt for straight or slightly curved shoes.

Last bottom centerline: A line defined by the toe and heel point.

Last bottom featherline: A line that defines the bottom shape of the last (last bottom pattern).

Last bottom width: The width across the ball area of the last bottom at its widest point.

Lasting: Fitting and shaping of the upper to the last.

Lasting allowance: Extra material on shoe patterns to fit around and under the bottom edge of the last.

Lasting insole: An insole used to attach an upper to an insole before bottoming; the bottom surface of the upper.

Lasting margin: See Lasting allowance.

Last joint break: Point located at the intersection of the shank and the forepart, tangent to heel point and perpendicular to last centerline.

Last systems: Methods of sizing last dimensions: Arithmetic, Geometric, Dynametic, Europoint.

Lateral: Outer side of the foot or limb; the side away from the midline of the body.

Leather: Material created by tanning a hide or skin.

Length: Dimension on the center of the last bottom from toe point to heel point.

Levy mold: Full-length inlay that conforms to contour of the plantar foot.

Lightweight trainer: A training shoe that weighs less than 10 ounces. It can be used for training or racing, but it's not as durable or supportive as most training shoes.

Lining: The inside backing material for uppers.

Lockstitch: A method of sewing the upper to the bottom.

Long heel girth: The dimension around a last passing through the instep and heel featherline point.

Long heel plate: A sheet metal bottom surface extending from the heel to midway of the shank area.

Longitudinal arch: Curvature of hind and midfoot.

Longitudinal force: The force generated in the direction of motion by a walker or runner during foot contact and related to the slip characteristics of a shoe. Also referred to as the anteroposterior force.

Low cut: Below the ankle shoe design.

McKay: A shoe construction method that uses tacks and a stitched sole; the upper is tacked, stapled, or cemented, and the sole is attached with chainstitches.

MCR: Microcellular rubber.

Medial: The side closest to the midline of the body; inside area of the foot.

Memory: The speed and extent to which a material recovers to its original shape after load compression.

Mesh: Woven or knitted nylon material for uppers.

Metatarsal pad: A soft wedge of material placed under the ball of the foot to add shock absorption and comfort for forefoot strikers.

Metatarsals: The long bones of the foot between the toes and ankle.

Midsole: The layer of material between the upper and outsole. It's the most important component of the shoe because it provides most of the cushioning. The midsole is usually made of EVA or polyurethane or some combination of the two.

Moccasin: A method of construction whereby the upper is placed under the last and extended up and around to form the quarter and vamp.

Modification: Alteration, change, or addition.

Mold, mould: That which is shaped, molded, or formed; a cavity used to shape plastic or rubber by pressure and heat.

Molded shoe: Shoe made from a model of the foot.

Monk strap: Shoe with a wide buckled strap across the instep.

Motion analysis: The analysis of total or partial body movements for the purpose of better understanding how the body functions. The analysis is usually done in conjunction with high-speed filming and computers.

Motion control devices: Materials and designs that control the inward rolling (overpronation) of the foot.

Nap: The surface pile or layer of textile fabric.

Negative heel: Heel with plantar surface lower than the ball of the shoe.

Neoprene: Synthetic, rubber-like material, very durable, used for outsoles, heels, and other components; oil-resistant; an elastomer, polychloroprene.

Neutral position: The most efficient functional position for the foot producing the least amount of stress on the joints, ligaments and tendons.

Open toe: Shoe design with no front center seam.

Orthopaedic shoe: Shoe designed with features to accommodate or reposition foot abnormalities.

Orthosis: Corrective device that is used to protect, support, or improve function of parts of the body that move.

Outflare: Last or shoe whose forepart provides more lateral than medial surface area.

Outsole: Bottom, ground-contacting portion of the shoe; the black material on the bottom of the shoe that strikes the ground. Carbon rubber is the most common outsole material because it is firm and resilient. Blown rubber has a more cushioned feel but is less durable.

Oxford: A shoe design with a laced, low-cut shoe; low quarter-laced shoe.

Pad: A device placed inside a shoe to provide support or relieve pressure from a specific location such as a longitudinal arch pad or a metatarsal pad. They are made of various materials and come in a variety of shapes and sizes.

Pattern: The cut-out pieces making up the design of the upper.

Pedorthics: Allied health profession concerned with the design, manufacture, fit, and modification of footwear and related appliances.

Pedorthist: Practitioner of pedorthics.

Phylon: A foam similar to EVA and the name of the midsole material that Nike uses in several models. Hi-Tec also uses Phylon in one model.

Pivot point: The rotation area on sole under ball of foot.

Plantar: The bottom or sole of the foot.

Plantar flexion: The downward movement of the toes or distal end of the foot away from the leg.

Plastic: Synthetic material or human-made polymeric substance, excluding rubber.

Platform: Elevated sole.

Polyethylene: A thermoplastic material or ethylene.

Polymer: A molecular compound, natural or synthetic.

Polypropylene: A tough lightweight plastic.

Polystyrene: A transparent thermoplastic.

Polyurethane: A synthetic rubber that's a common midsole material. It is firmer, heavier, and more durable than EVA, but it's not as cushioned. Polyurethane is often used with EVA in many popular models, such as the Nike Air Pegasus, Avia 2200, and New Balance 997, with the polyurethane in the rearfoot for firmness and durability and the EVA in the forefoot for flexibility.

Polyurethane resins: A family of resins from which polyurethane is produced.

Polyvinyl: A semirigid plastic used for some heel counters.

Polyvinyl acetate: A thermoplastic material.

Polyvinyl chloride: Thermoplastic material with various applications such as soles and heels and as a coating for uppers and linings.

Porous: Material having pores.

Posterior: Behind, back.

Posting: The use of a firmer material (usually midsole material) to slow foot motion in the rear or middle of the midsole. Posting is usually used to limit overpronation.

Potential energy: Energy associated with position.

Prefabricated: A sole unit built from more than one layer.

Prescription: Legal order, requesting specific treatment, signed by a medical doctor, podiatrist, or osteopath.

Prescription shoe: Footwear prescribed by a medical practitioner, either stock or custom-made.

Prewelt construction: Shoe construction in which the upper and welt are joined by chainstitches, the insole and upper are cemented, and the outer sole is lockstitched to the welt.

Pronation: A complex multijoint action of the foot that is usually estimated from the inward rotation of the heel relative to the leg producing the inward rolling motion that takes place in the foot and ankle joint following footstrike during running; a triplane motion of the foot or part of the foot that consists of simultaneous movements: abduction, dorsiflexion, and eversion; basically, a movement away from the midline of the body, up and out; lowering the medial foot arch; the opposite of supination. Nearly all runners pronate to some degree, or should. If the foot rolls too far inward, however, injuries can result. Overpronation, or the extreme inward roll of the foot, places a strain on tendons and ligaments.

Proportional last: Last with geometric, rather than arithmetic, grading that conforms better to proportional size increments.

Prosthetic foot: An imitation foot closely resembling the shape, texture, flexibility, and weight of a human foot. Used in testing procedures.

Proximal: Closest to a reference point such as the center of the body.

PU: Polyurethane (cellular plastic).

Pump: Low-cut shoe not built above the vamp line and usually held onto the foot without fastenings.

Push rod: A rod that functions in conjunction with a cam to open or close valves.

PVC: Polyvinyl chloride (plastic material).

Quarter: Posterior aspect of the upper; the major pattern piece making up the sides of the upper.

Rearfoot stability: The ability of the shoe to control foot pronation during the initial 40% to 50% of the support phase.

Resiliency: The ability to regain quickly the original shape (return energy rebound).

Resin: Solid organic products of natural or synthetic origin.

Ridge: A well-defined intersection of the wall and the conical section of the forepart.

Rigid shank: Firm, stiff, inflexible area of the shoe between the heel breast and ball.

Rocker bar: See Bar, rocker.

Rocker bottom: See Bar, rocker.

Rubber: An elastomer or natural rubber compound; resilient natural or synthetic material.

Running machine: A piece of equipment, used to test shoe characteristics, that simulates the actions of running on a shoe.

Sagittal plane: The vertical plane that passes through the body from back to front dividing it into a left and right half.

Seam: Sewing that joins together pieces of the upper.

Semi cut: A design cut just on or over the ankle. (Also called three-quarter cut.)

Shank: The reinforcement under the arch between the heel and the sole; the bottom area of the last between the breastline and the joint break.

Shank piece: Rigid reinforcement of the shank.

Shank plug: A metal piece inserted in the shank in order to clinch metal shank fasting staple.

Shearing force: A force that causes or tends to cause two parts of a body to slide relative to each other.

Shoe size:

Prewalkers: 3000-4

Big boys: 5½-11

Infants: 1-8

Growing girls: 3½-10

Children: 8½-12

Men: 6½-16

Misses: 12½-4

Ladies: 4-13

Youths: 12½-4

Boys: 3½-6

Silicone: A slippery polymeric material used in treating shoes for water repellency.

Skive: The thinning down of edges of leather or poromeric material; to cut in thin layers or to a fine edge.

Slip last: The most flexible type of shoe construction. With a slip-lasted shoe, the upper is stitched together like a moccasin and glued to the midsole. Slip lasting allows for a better fit. Lasting method whereby a closed upper is formed before being stretched over the last.

Sneaker: The American name for vulcanized, canvas rubber shoe.

Sockliner: The material (regularly called an insole) inserted between the foot and lasting insole next to the foot; material covering the dorsal surface of the inner sole.

Sole: Bottom or ground contact area of footwear.

Sole leather: Heavy leather, usually cattle hide that is dry-finished and used for outer soles.

Speed lacing: A lacing method that uses D-rings.

Splint, Denis-Browne: Rigid bar between both shoes used to abduct the feet.

Splint, Friedman-counter: Flexible strip attached to both counters; used to limit internal rotation.

Split: The flesh or the underside of the leather hide after the grain side has been removed.

Split leather: See Sole leather.

Stability: The ability of the shoe to keep the foot moving in a forward direction, rather than allowing for excessive side-to-side movement.

Stabilizer: An ingredient used in formulating elastomers and synthetics.

Standard deviation: A standard measure of dispersion of a frequency distribution around the average value. A distribution is typically made up of 3 standard deviations on either side of the average value.

Stitchdown: A method of sewing the uppers to the bottom.

Straight last: A last that is relatively straight on the medial side to add stability. The straighter the last, the greater the medial support. 1. Form for constructing a shoe that can be worn on either foot. 2. Form for constructing a shoe in which the medial border approximates a straight line.

Studs: Large knobs protruding from the sole.

Suction cups: Indentations on the outsole that provide traction on smooth surfaces.

Supination: A triplane motion of the foot or part of the foot that consists of simultaneous movements: adduction, plantar flexion, and inversion; basically, a movement toward the midline of the body, down and in; elevation of the medial foot arch; the opposite of pronation. Oversupination occurs when the foot remains on its outside edge after heel strike instead of pronating. A true oversupinating foot underpronates or does not pronate at all, so it does not absorb shock well. It is a rare condition, occurring in less than 1% of the running population.

Symmetrical: In shoemaking, this applies to lasts or patterns that have even sides, the right side the same as the left side.

Synthetic: Something resulting from synthesis rather than occurring naturally; a product (as drug or plastic) of chemical synthesis.

Tanning: Process of converting raw hides and skins into leather by a combination of chemical and mechanical means.

Tensile strength: The pulling force expressed in measuring leathers or fabrics; the resistance of a material to being pulled apart.

Terminal wear condition: A condition in which the outsole of a shoe is worn completely through to the midsole or underlying material.

Thermoplastic: Material capable of being repeatedly softened by heat and hardened by cooling; a type of rigid, durable plastic used for most heel counters.

Thomas bar: See Bar, Thomas.

Throat: Entrance of the shoe where normally the vamp and quarters meet; the topline of the vamp in front of the instep.

Throat opening: The distance in a straight line from the vamp point to the back seam tuck.

Toe box: Reinforcement used to retain the original contour of the toe and guard the foot against trauma or abrasion.

Toe cap: An additional protective device on the frontal toe area.

Toe recede: The slope of the top surface of the last extending from the toe point to the point of full toe thickness.

Toe spring: The vertical distance between the base plane and the toe point of a last having the desired heel elevation; the vertical distance between the ground and the toe point giving the shoe frontal pitch.

Tongue: A layer of upper material that protects the top part of the foot from pressure from the laces. Some tongues on Asics shoes are now split to allow the foot to expand.

Tongue guide: The tag or slit in the tongue through which the laces are slotted to hold the tongue in place (lace keeper).

Top line: The open area of the shoe around the ankle.

Torque: A force that causes or tends to cause rotation of an object about an axis. The torque (also called moment) is the result of the magnitude of the force, its direction, and distance from the axis of rotation.

Torsion: The stress caused by twisting a material.

Torsional rigidity: The amount of stiffness in the shank and waist of a shoe.

Torsion system: The flagship technology of Adidas. It's a system designed to allow the forefoot and rearfoot to move independently of each other to encourage freedom of movement. Shoes designed with the Torsion system have a groove cut into the midsole where the foot bends naturally during the running gait. The Torsion Bar is a Kevlar strip embedded lengthwise into the midsole to control excessive twisting of the foot.

TPR: Thermoplastic rubber.

Traction: The amount of friction or resistance to slip between a shoe outsole and the contact surface.

Transverse arch: Curvature of metatarsal heads.

Transverse force: The force generated at a right angle to the direction of motion by a walker or runner during foot contact and most closely related to rearfoot stability. Also referred to as the mediolateral force.

Transverse plane: The horizontal plane that passes through the body from side to side and back to front dividing it into an upper and lower half.

Tread: The soling configuration of the outsole.

Treadmill: A rotary machine-driven belt that allows subjects to run in a confined space.

Treadpoint: Point of the bottom forepart of last or shoe in contact with the treading surface.

Tricot: Knitted fabric commonly used for linings in women's shoes.

Unit sole: The bottom unit with sole and heel portions molded together as a single piece.

Universal last: A standard last used by sports shoemakers for all width fittings.

Upper: The material making up the "top" part of the shoe.

Urethane: Plastic used for uppers, soles, top lifts, and other components; commonly known as *polyurethane.*

Urethane: A resin combination with polymers.

U-throat: A lacing eyestay pattern at the front of the shoe.

Vamp: Forepart of the upper. The top or front part of the upper over the toe and lacing area.

Vamp length or depth: The distance measured along the toe profile from the vamp tack to the toe point.

Vamp tack: An arbitrary point on top of a last forepart marked by a tack, measured from the toe.

Vegetable tanning: Tanning that uses plant or vegetable materials; uses materials derived from plant life such as oak, chestnut, quebracho, myrobalans, or divi-divi.

Velcro: Nylon hook and loop tape fastener that clings on contact.

Vertical force: The force perpendicular to a level surface. The dominant force generated by a walker or runner during foot contact and most closely related to the shock absorption characteristics of a shoe.

Vinyl: A PVC material that's available in expanding and nonexpanding types.

Vulcanize: A method of shoemaking in which the rubber sole and/or foxing is cured by heat after attaching to the upper; bonding of the outer sole to the upper from a sole mold in which the soft rubber molds to the shoe, then is allowed to cool and harden. Common in footwear such as sneakers.

Waist: Section of the last or shoe between the ball and instep.

Waist girth: The smallest dimension around a last between the joint girth and the instep girth.

Wall: Straight sides around the periphery of the forepart of certain style lasts.

Water repellent: Shoes treated so that they will shed water.

Waterproof: Shoes treated so that water cannot penetrate.

Wear tester: A piece of equipment used to evaluate the resistance of the outsole of a shoe to abrasion.

Wedge: Tapered leather, rubber, or other material used to elevate one side of the sole or heel; replaces the heel.

Wedge angle: The angle between the heel featherline plane and the base plane, with the last positioned on the base plane.

Wedging: Insertion of wedges inside the shoe or on the sole or heel.

Weighted shaft: A shaft with a weighted end that is used to impact shoes or material.

Welt: A narrow strip around the outside of the sole, stitched between the upper and the sole.

Width: Measurement of circumference around the ball of the foot; a coded method for shoe girth sizing.

Width sizing: Most running shoes are available in just one width; a few selected models are offered in two. New Balance is the only company that offers its shoes in several widths. For men: from AA to EEEE. For women: AA to EE.

Wing tip: The design of the toe cap.

ZO2: A Turntec feature; it is a silicone pad that is used for cushioning in the insole.

∗ This glossary has been compiled from: An A to Z guide to shoe terminology. Runner's World, 25:48-49, 1990; Cheskin MP: The Complete Handbook of Athletic Footwear. New York, Fairchild Publications, 1987, pp 163-245; Prescription Footwear Association/Board for Certification in Pedorthics: 1992/93 Desk Reference and Directory. Columbia, Md, Prescription Footwear Association, 1992, pp 65-78; and Janisse D (ed): Introduction to Pedorthics. Columbia, Md, Pedorthic Footwear Association, 1998.

To understand the shoe itself, it is necessary to review the steps by which the shoe is made. Integral to this process is the last (from the Old English *laesk*, meaning sole or footprint), which acts as an artificial foot form. [50] [58] This allows the shoe upper to be created in the proper shape, size, and dimensions. Important measurements with respect to the last are the toe pitch or toe spring, the girth, and the heel height or pitch (Fig. 25B-11). [50] [59] The variation in toe spring and heel height can affect the movement of the foot by improving or impairing the rocker action of the foot during gait. [31]

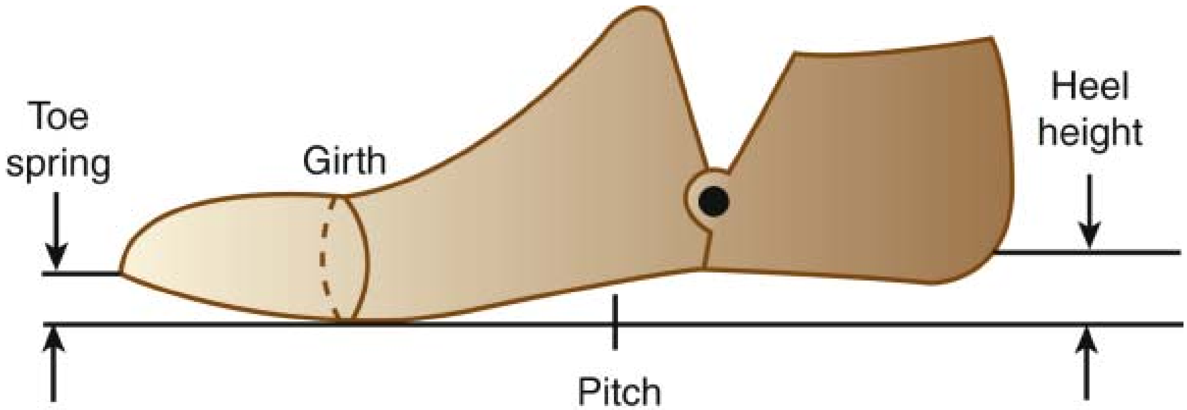

Figure 25B-11 Important measurements with respect to the last are toe pitch or toe spring, girth, and heel height or pitch. *(Adapted from Cheskin MP: The Complete Handbook of Athletic Footwear. New York, Fairchild Publications, 1987; and Stacoff A, Luethi SM: Special aspects of shoe construction and foot anatomy. In Nigg BM: Biomechanics of Running Shoes. Champaign, Ill, Human Kinetics, 1986; Copyright © 1986 by Benno M. Nigg.)*

Shape is also a critical consideration in analyzing the form for the last of a shoe. This shape can be either straight-lasted or curve-lasted depending on the amount of inward curve built into the last. Because most feet have a slight inward curve, the curved type of last provides better comfort and fit for most feet. The curved last allows the most shoe flexibility and is particularly well suited to the athlete with a highly arched or cavus foot. When the last is straighter, it translates into better medial support for the foot and is best suited to the flatter foot or to the person with an overpronated foot. Figure 25B-12 depicts the difference between straight and curved lasts. Sports shoe manufacturers use a curve of about 7 degrees in a curved last.[22] Variations exist now as slightly curved and semicurved lasts.

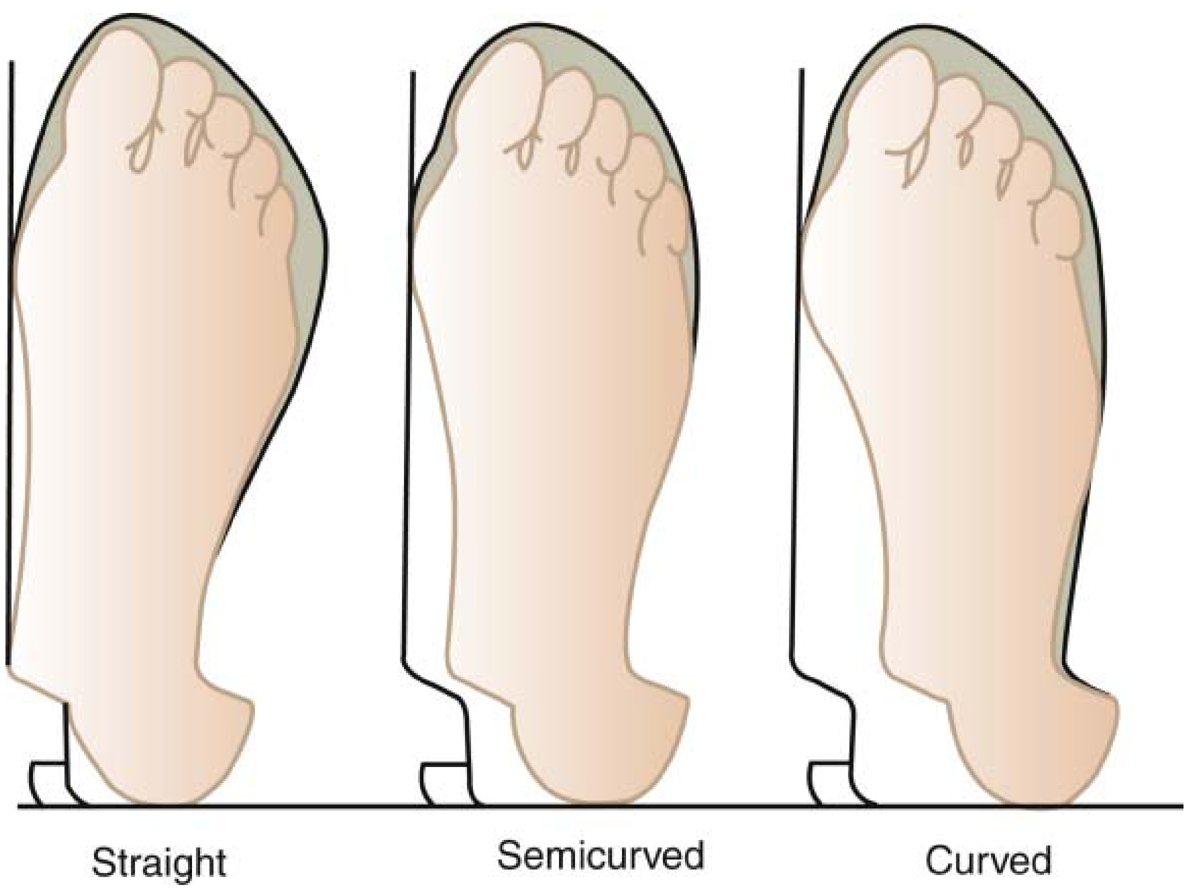

Figure 25B-12 Illustration of the difference between straight, semicurved, and curved last shoe.

As we examine the last, the next consideration is the shape of the toe box. The various alternatives are depicted in Figure 25B-13 . [22] It is clear that this feature has an important bearing on fit and comfort. The best example of this is the need for the athlete with clawing in the toes or even a single hammer toe to have a toe box of sufficient height to prevent chafing.

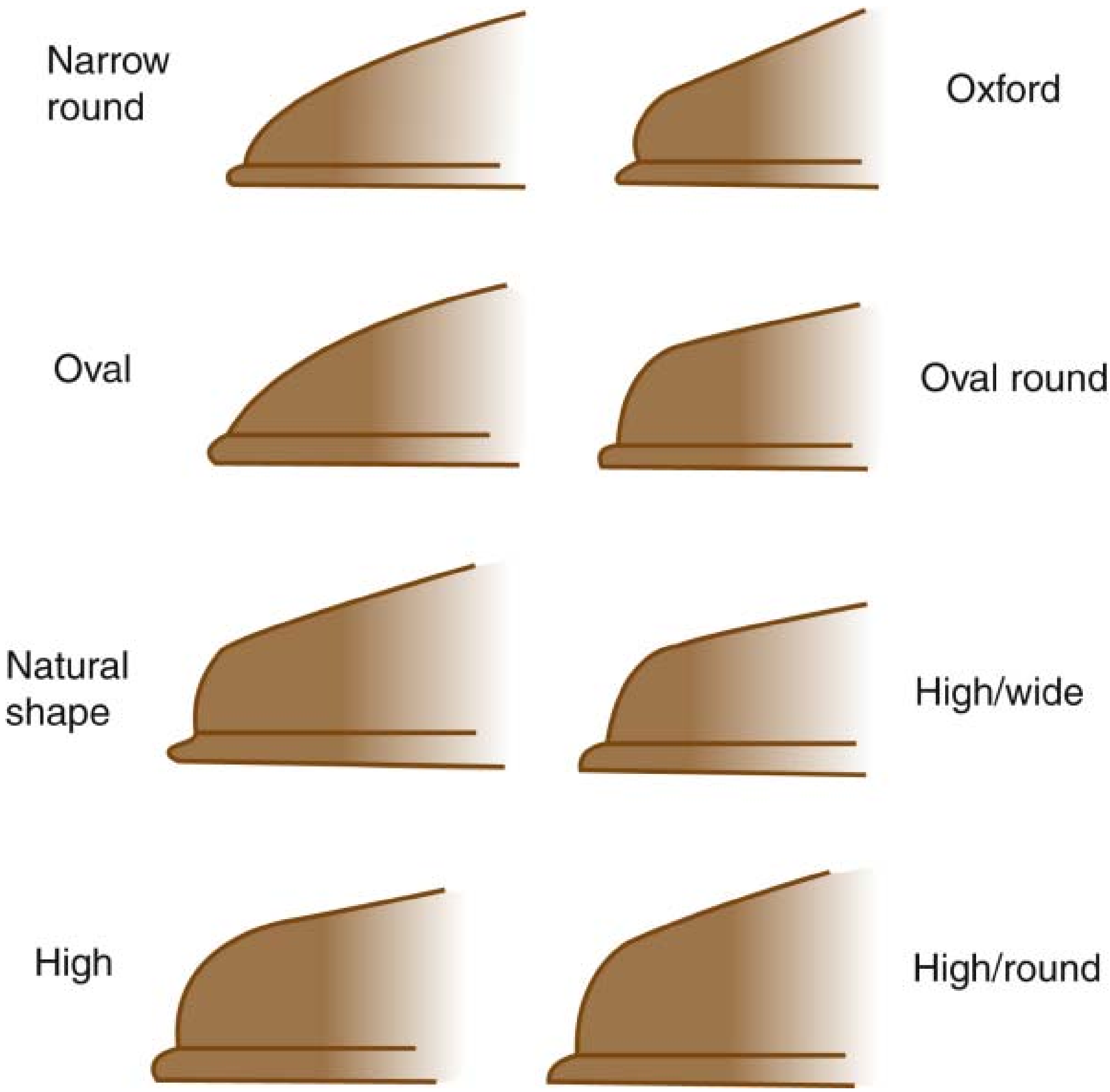

Figure 25B-13 Various alternatives for the shape of the toe box. *(From Cheskin MP: The Complete Handbook of Athletic Footwear. New York, Fairchild Publications, 1987.)*

In the evolution of shoewear, the last was originally chiseled out of stone. [31] Later models were whittled from wood. A machine used in shaping gunstocks was converted to make lathes and led to the first lastmaking plant in Lynn, Massachusetts, in 1820. [31] Today most lasts are made from plastic, a process developed by the Sterling Last Corporation in 1969. [31] Metal lasts are used when direct- or injection-molded soles are attached to the upper because the heat used in this process is poorly tolerated by wood or plastic. [22]

The dimensions of lasts are based on the average measurements of the segment of the population to whom the shoe will be marketed (e.g., men or women). [37] [50] In the past, women's shoes were based on scaled-down versions of lasts derived from the male foot anatomy. Recent investigations have noted several structural differences between the male and the female foot. Specifically, the female foot typically has a narrower Achilles tendon, a narrower heel in relation to the forefoot, and a foot that is narrower in general than its male counterpart. [32] In addition to these dimensional discrepancies, women have proportionately shorter leg length to total body height than do men, necessitating more foot strikes per distance covered. Because of their smaller feet, the heel-to-toe gait cycle is completed more quickly. Consequently, the cumulative ground reaction force is increased in the female runner, particularly in elite women runners, who tend to be midfoot strikers. [33] The repetitive nature of running causes these factors to be magnified tremendously over the life of a typical athletic or running shoe. Until recently, women's shoe manufacturers typically scaled down all key internal dimensions of a male athletic shoe in fixed proportion. This practice, termed *scaling* or *grading*, persists today. Fortunately, most major athletic shoe companies now have divisions devoted to female athletic footwear, and many have developed lasts based on the anatomy of the female foot. [32] Now, the last is divided and measured in ways that more closely duplicate the average shape of the foot, and a method exists for custom-making a last and producing a shoe to individual specifications. This is frequently done for elite athletes and particularly for athletes in certain sports such as figure skating. It is considered cost ineffective and unnecessary for the general public. [22]

The divisions and measurements used for the last are shown in Figure 25B-14 . [22] Individualized lasts are made from an outline of the weight-bearing foot, a weight-bearing impression (to determine pressure distribution), a profile showing the height of the big toe and the instep contour, measurements of overall width and length, and specific girth measurements. Girth measurements are made from (1) the joint—around the metatarsophalangeal joints, (2) the waist—the smallest circumference behind the metatarsophalangeal joints, (3) the instep—the smallest circumference around the arch, (4) the long heel girth—the circumference from the lower edge of the heel around the instep, (5) the short heel girth—the circumference from the lower edge of the heel around the ankle at the lowest crease line, and (6) the ankle—the circumference around the malleoli that is used in high-top shoes and boots. [37] [50]

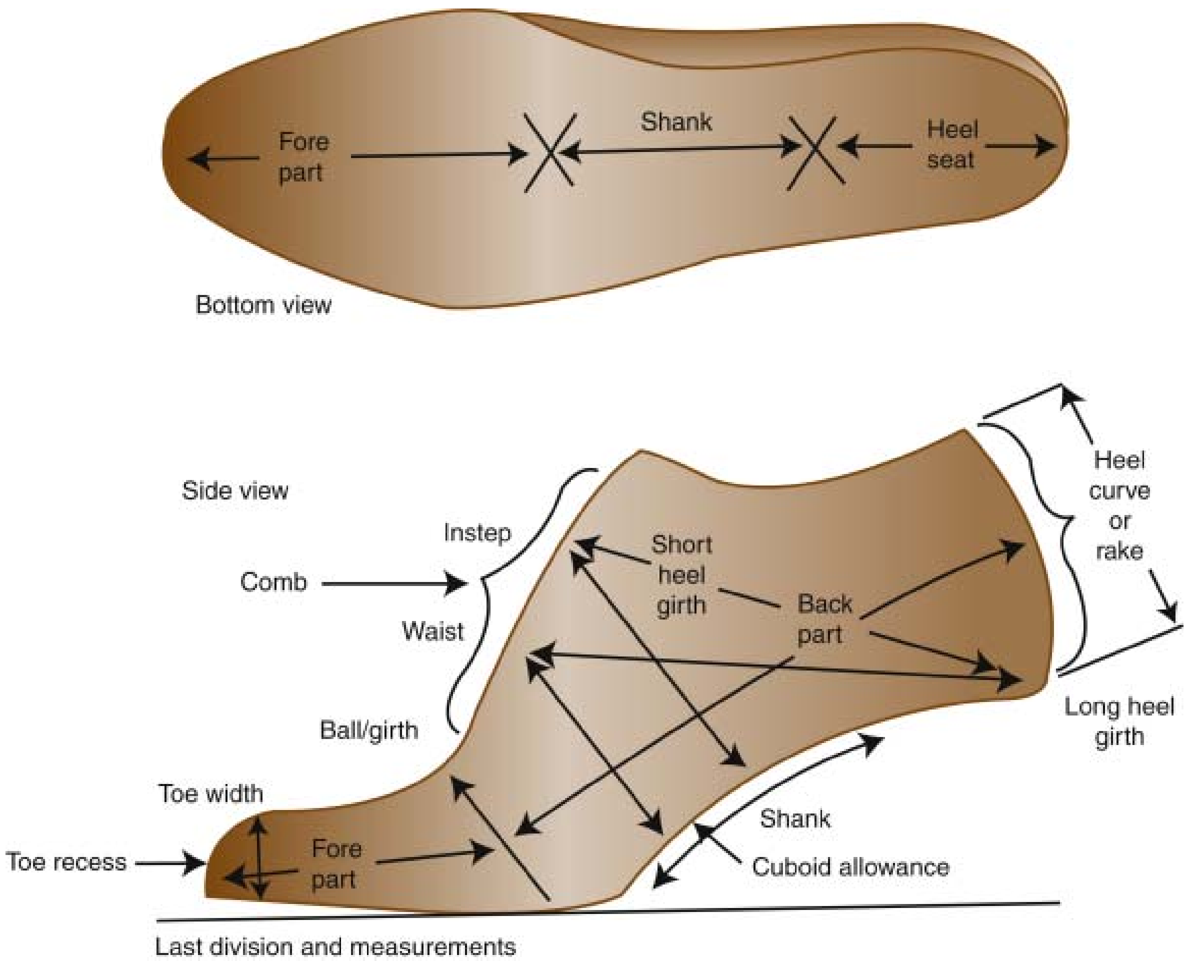

Figure 25B-14 The divisions and measurements used with the last. *(From Cheskin MP: The Complete Handbook of Athletic Footwear. New York, Fairchild Publications, 1987.)*

The process of lastmaking has achieved some level of automation with respect to the upper, for which computer technology has been used more effectively. Computer-aided design and manufacturing processes produce three-dimensional designs for the upper and allow direct transference of this information into automated methods of pattern grading and cutting. [22] This reduces the work of a formerly labor-intensive operation and foreshadows further innovations in computer-assisted production of sports shoes.

Figure 25B-10 illustrates shoe parts.

Specific Shoe Parts: Upper

The upper of the shoe is the material that covers the foot. As such, the most important consideration is the specific material used and its relationship to the purposes for which the shoe is purchased. A shoe with a nylon mesh upper is far from ideal for a cold-weather hiking boot, and thick leather is, by the same token, less than ideal for a running shoe in warm climates. These material considerations are discussed in a later section.

Toe Box

The front part of the shoe upper is crucial to the health of the toes. Adequate depth is necessary to prevent chafing of the skin over the bony prominences at the interphalangeal joints. Reinforcements of the toe box in the form of stiffeners can vary from being nonexistent in running shoes to being quite stiff in hockey skates or hiking boots. An inserted stiffener protects the toes and prevents the collapse of the upper material onto the toes. Its disadvantages are added weight and stiffness.

Toe Cap

The addition of material called foxing to the front of the shoe protects the toes and increases the durability of the toe box. The toe cap is usually an isolated component in running shoes and is made of suede or rubber stripping.

Vamp

One of the 16 to 20 pieces of material forming the upper of a shoe is called the vamp. It is the piece (or two pieces) of material forming the front part of the upper and sewn to the eyestays and quarters. Most shoes use a single piece of material for the vamp to eliminate a seam in the toe box area. The vamp is sewn to the quarters at the midfoot level, and these seams are usually hidden by the various trademark stripings of different companies. Split-vamp construction has been popularized as a method allowing better shoe fit for some individuals. It splits the vamp into two separate pieces with separate lacing systems (Fig. 25B-15).

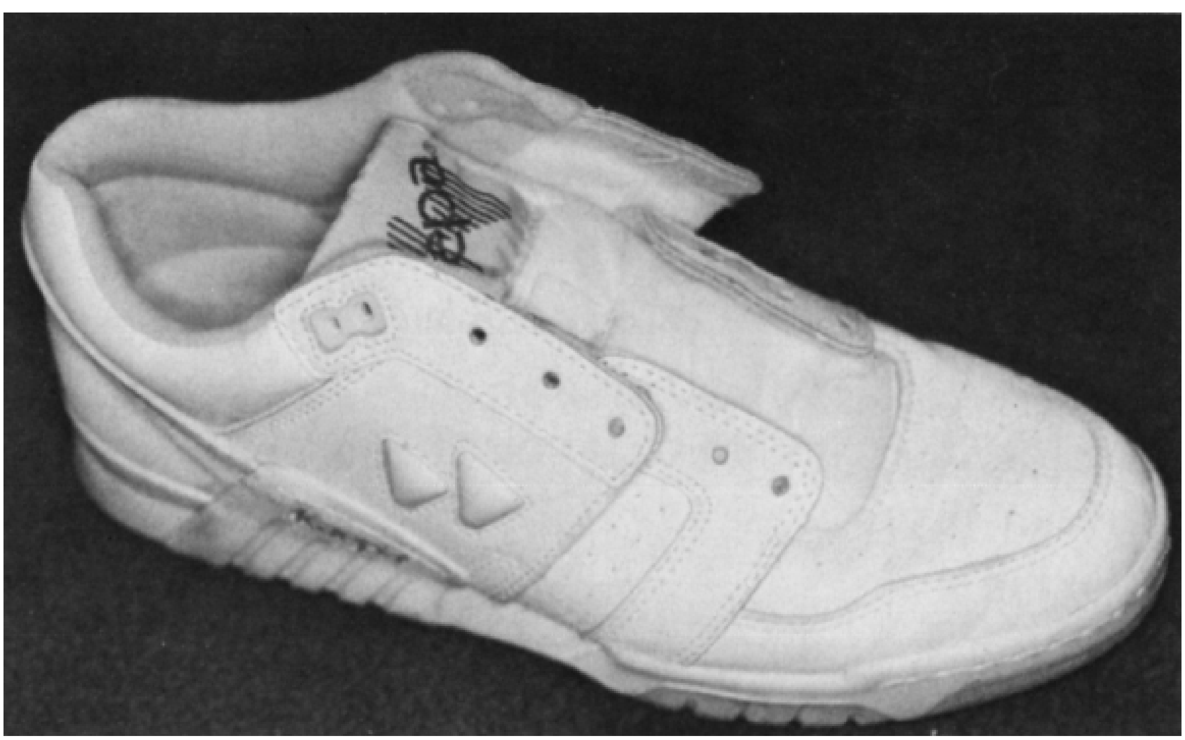

Figure 25B-15 Split vamp shoe with two separate pieces on the upper with separate lacing systems. *(Photograph by Thomas O. Clanton.)*

Quarter

This is the other major piece of material composing the upper. Two pieces form the sides of the shoe and conform to the midfoot and arch area of the foot. In shoes designed for side-to-side movement, the vamp and quarter are usually reinforced by extra material (usually leather) called the saddle or arch bandage.

Eyelet Stay and Eyelets

The eyelet stay reinforces the holes or eyelets used for lacing. It can be incorporated into the reinforcing material of the saddle and provides additional support for the forefoot and midfoot. Holes are replaced with plastic or metal rings or hooks in some athletic footwear to allow quicker or more forceful lacing. Many athletic shoes have extra eyelets for individualization of a snug but comfortable fit. Widening the reinforcing layer and the eyelets has allowed variable-width lacing to accommodate some of the width sizing difficulties seen in sports shoes. Variable-width or dual-lacing systems can be used with conventional or nontraditional lacing patterns to accommodate variations in foot size, bone spurs, nerve irritation, or other problems. Widely placed eyelets allow the laces to pull the quarter tighter for narrow feet, and narrowly placed eyelets are better suited to a wider foot (Fig. 25B-16). [32]

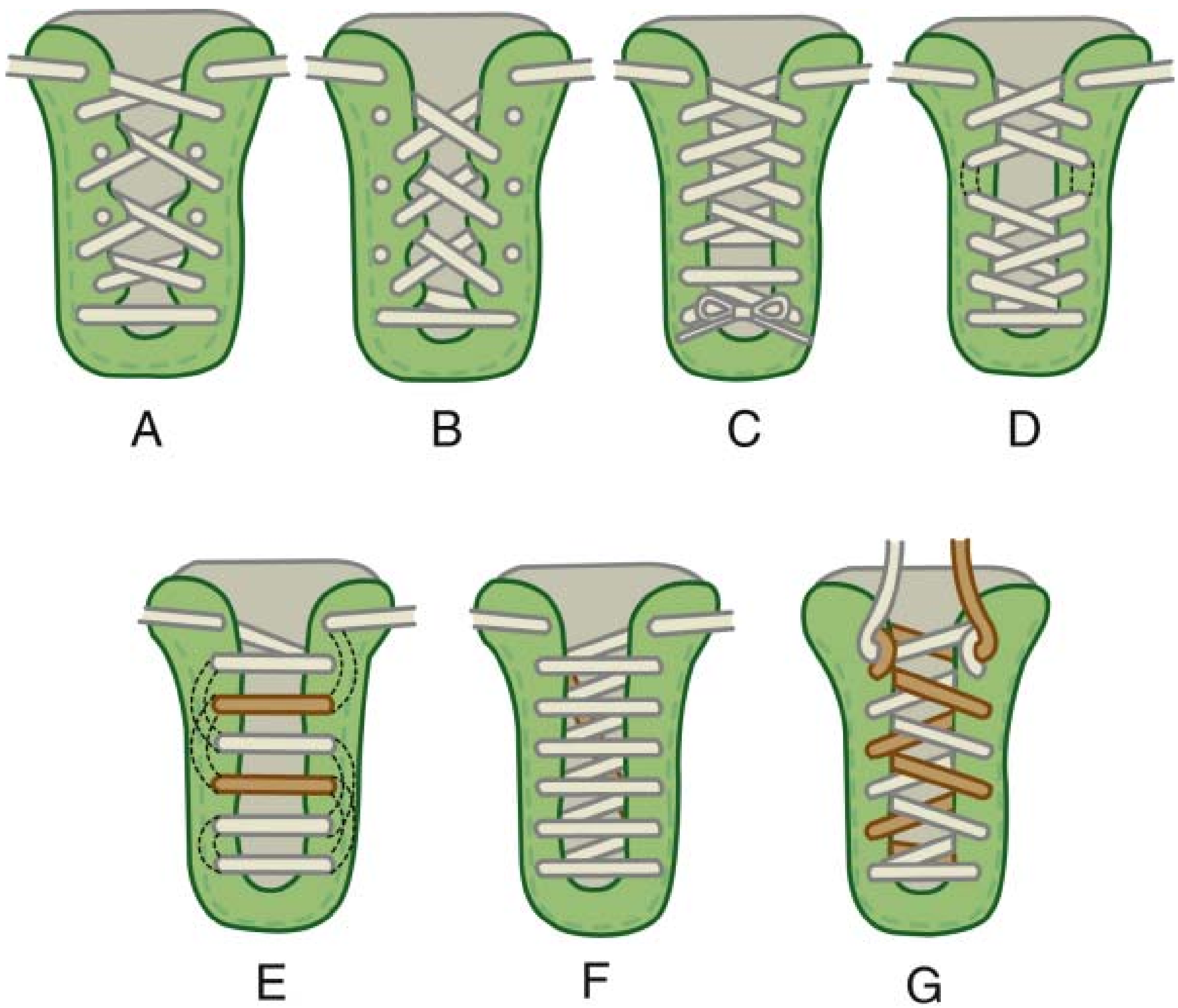

Figure 25B-16 Examples of lacing patterns for use in certain forefoot conditions. **A,** Variable-width lacing used for wide foot. **B,** Variable-width lacing used for narrow foot. **C,** Independent lacing system using two separate laces. **D,** Crisscross lacing pattern designed to avoid area of dorsal prominence or pain. **E,** Lacing pattern useful in highly arched foot so that laces never cross over the top of the foot. **F,** Lacing pattern designed to pull the toe box up to relieve pressure on the toes. **G,** Crisscross and loop lacing system used to hold the foot snugly in the heel of the shoe to treat or prevent heel blisters or chafing. *(Redrawn from Frey C: Foot health and shoewear for women. Clin Orthop 372:32-44, 2000.)*

Tongue

The portion of the upper that extends under the laces is called the tongue. It may be padded to reduce irritation to the dorsum of the foot. The tongue is often slit in a way that allows the laces to anchor the tongue and prevent it from sliding laterally.

Collar

The collar forms the uppermost part of the quarters and is the part through which the foot enters the shoe. When excessively stiff or high, the collar can irritate the hindfoot or ankle malleoli. It often has extra padding.

Achilles Tendon Protector

The extended area on the back of the shoe acts as a pull tab and protection for the Achilles tendon. It should be both molded and padded well to prevent irritation of this area. The high tab design that caused irritation of the Achilles tendon has been replaced by a "bunny-ear" design with a dip in the center. [34] The cutaway should be wide enough to prevent friction on the sides of the Achilles tendon.

Heel Counter

This reinforcement to the upper of the shoe is located in the heel area. It is a stiffened material of fiberboard or plastic that is molded to the heel and provides greater rearfoot control. The size of the heel counter and the quality of the material vary between shoes.

Forefoot and Rearfoot External Stabilizers (Footframe)

Material is used as a reinforcing component to cup the rearfoot or forefoot of the shoe for greater stability. This is a molded material that may or may not be an integral part of the midsole.

Lining

This is the material that acts as the inside backing for the material of the upper. It must be smooth and nonirritating because it is in direct contact with the foot.

Specific Shoe Parts: Bottom

The bottom, or sole, of the shoe is important in protecting the foot from the environment. Therefore, it requires materials that are both comfortable and durable. In modern athletic footwear, these two features are accomplished by using multiple components.

Sockliner or Insole

This material cushions the foot and is the layer between the foot and the bottom of the shoe. Various materials have been used for this layer and are discussed in the next section. Most sports shoes come with removable sockliners to allow them to be replaced when they are worn out or when a corrective orthosis is necessary. The insole reduces friction and provides some degree of shock absorption. It also absorbs perspiration and can provide some canting or control of overpronation. The capability of this material to mold to the shape of the foot can be a source of additional comfort and control.

Insole Board

The cellulose fiberboard to which the upper is attached in the conventional lasting process is called the insole board; hence the term *board-lasted*. This process provides the greatest stability, as opposed to slip lasting, which has no insole board under the sockliner, and leads to improved comfort and flexibility. The intermediate option is combination lasting, in which there is an insole board in the hindfoot with stitching in the forefoot where it has been slip lasted (Fig. 25B-17).

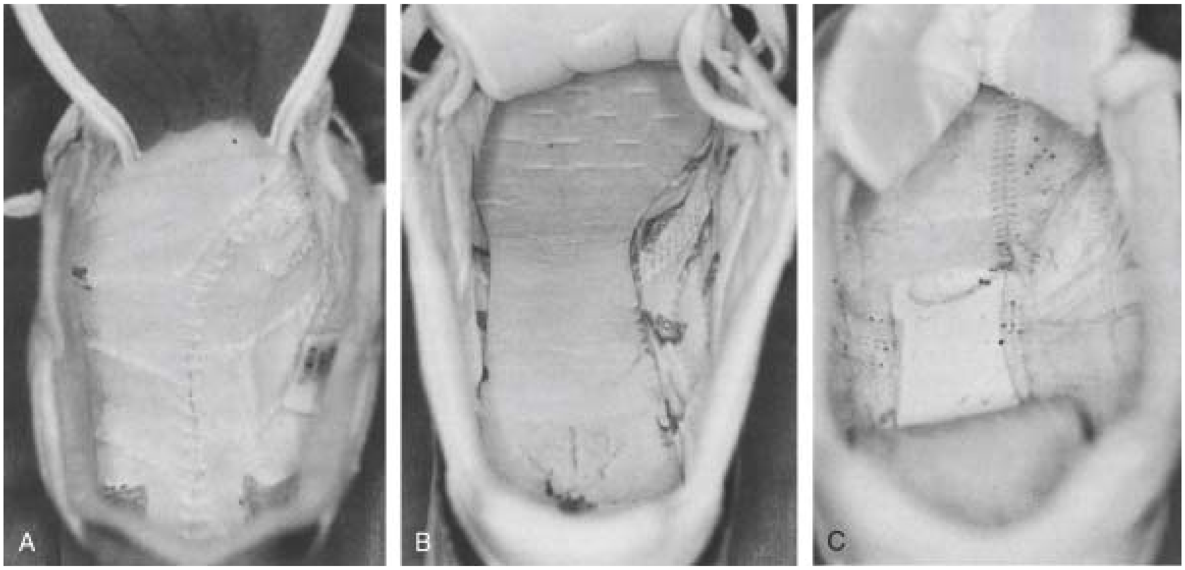

Figure 25B-17 Examples of different lasting methods for running shoes. **A,** Slip-lasted shoe. **B,** Board-lasted shoe. **C,** Combination-lasted shoe. *(Photographs by Thomas O. Clanton.)*

Midsole

Known as the *heart* of the running shoe, the midsole is sandwiched between the upper and the outsole of the shoe and provides the bulk of shock absorption. With the wedge, this component also produces the desired heel lift, rocker action, and toe spring. Through the use of canting and variable hardness, the midsole can control foot motion. With the use of anatomic contouring, even greater stability and comfort can be achieved. Variations in the materials used add another dimension to what the midsole can do for the foot.

Many of the more significant and recent design advances have occurred through alteration of the midsole.[35] These modifications are seen as significant enough by the manufacturers that they are frequently incorporated in the name or advertising campaigns of the various shoe products (Table 25B-1). One particular midsole modification has even contributed to the most recognizable nickname of one of the greatest basketball players of all time—Michael “Air” Jordan.

TABLE 25B-1 -- Materials Encapsulated in Midsole Cushioning Designs

Shoe Manufacturer	Trade Name	Material and Design
Asics	Gel	Silicone resin in a pad
Avia	Arc	DuPont Hytrel in polyurethane
Brooks	Hydroflow	Silicone fluid in a two-chambered plastic bladder
Converse	He:01	Helium gas in polyurethane and nylon
Etonic	Soft Cell	Combined gel and ambient air
New Balance	ENCAP	Ethylene vinyl acetate core in polyurethane shell
Nike	Air	Freon gas in polyurethane
Puma		Honeycomb pads
Reebok	Hexalite	Honeycomb pads
Reebok	DMX	Pads connected by tubing, which allows air flow during stride

Compiled from Heil B: Running shoe design and selection related to lower limb biomechanics. Physiotherapy 78:406-417, 1992; Frey C: Footwear and stress factures. Clin Sports Med 16:249-257, 1997; and shoe product brochures and the Web sites of the named shoe manufacturers.

Outsole

This is the bottom layer of the shoe that makes contact with the ground. The outsole can be constructed with different materials, patterns, colors, and densities. These factors, excluding color variations, can be used to modify the shoe's stability, flexibility, comfort, and shock absorption. These features are discussed in greater detail later in this chapter.

MATERIALS USED IN SHOES AND SHOE INSERTS

The petroleum industry has affected many areas of society, including shoe construction. Traditional materials such as leather, rubber, and cotton (canvas) still have their place, but the search for lighter, more durable, more shock-absorbing materials has led to the development of complex foams with high-tech names such as Elvalite, Hexalite, Hydroflow, Hytrel, Kevlar, Millithane, Phylon, and ZO2. [50] [64] [65] [66] [67] [68] Technologic developments in the field of materials science have progressed at such a rate that it is virtually impossible for the athletic shoe salesperson, the equipment manager, or the sports medicine specialist (much less the average runner) to maintain a current working knowledge in this area. A comprehensive discussion of materials science and the various materials of shoe construction is beyond the scope of this textbook and beyond the attention span of either the author or the reader. Therefore, this section is designed to educate the reader on the fundamentals while providing resources for those interested in further research.

Polymer Science, in Brief

The history of polymer science has been summarized succinctly by Cheskin, although he differs with the *Encyclopaedia Britannica* about some dates. [22] Various landmarks are listed in Table 25B-2 .

TABLE 25B-2 -- History of Polymer Science

1493-1496	Christopher Columbus notes Indians playing with balls made from gum of a tree during his second voyage —rubber discovered by Western man
1615	Spaniard describes the use of tree milk in Indian footwear and cloaks
1735	French geographical team to South America describes “caoutchou” as the condensed juice of the *Hevea* tree
1835	Preparation of vinyl chloride
1839	Discovery of vulcanization process for rubber by Charles Goodyear
1860	Basic component of rubber discovered and named isoprene
1862	First plastic produced by English chemist Alexander Parkes, called *Parkesine* and later *Xylonite*. It was a nitrocellulose softened by vegetable oil and camphor.
1869	Plasticizing effect of camphor recognized by John Wesley Hyatt; cellulose nitrate patent for celluloid introduced
1879	French chemist Bonchardat introduces process of heat-cracking rubber.
1909	Heat and pressure patent for phenolic resins introduced by Backebrend (Bakelite)
1920s	Discovery of foam rubber made by confining gaseous bubbles within the rubber
1921	First injection molding machine produced and automated by polymerization
1922	German chemist Studinger writes that rubber is a chain of isoprene units. This provided theoretical background for polymerization.
1928	DuPont begins laboratory study of polymers leading to “superpolyamide” or nylon.
1931	Polychloroprene (Neoprene) first marketed commercially
1935	Germany produces synthetic rubber: Buna S from styrene butadiene, Buna N from nitrate butadiene
1937	Polyurethanes produced
1939	British invention of polyethylene
1940s	Redox process for low-temperature polymerization introduced in Germany to provide more uniform product.

Rubber and Plastic Technology for the Outsole and Midsole

Rubber is an organic substance that is a primary component of most athletic footwear. It can be obtained in nature as the milky latex produced by tropical and subtropical trees, or it can be manufactured synthetically. [69] [70] Natural rubber has the empirical formula C_5H_8, as assigned by Michael Faraday in 1826. [43] The synthetic rubber most commonly used in outsoles is styrene-butadiene rubber. [50] [71] Styrene's empirical formula is C_8H_8, whereas butadiene's is C_4H_6. [71] [72] Different properties of styrene-butadiene rubber can be created by altering the ratio of these two substances or by varying other elements within the manufacturing process. The addition of carbon black as a filler in the final curing process improves the elasticity and tensile strength of rubber, resulting in improved durability. [43]

According to the *Encyclopaedia Britannica*, plastics are "synthetic materials that are capable of being formed into usable products by heating, milling, molding and similar processes. The term is derived from the Greek *plastikos*, 'to form.'" [45] They have come into increasing use in the shoewear industry in large part as a result of their ability to soften but not melt when heated, which allows for a change in shape without a loss of cohesiveness or mechanical properties and allows for processing into a stable new form on cooling.

These synthetics include a vast array of materials that have properties of diverse usefulness. They can be grouped into thermoplastic or thermosetting varieties, [46] the basics of which are outlined in Table 25B-3 . Regardless of their type, plastics are dependent on the process of polymerization for their existence. This is the process whereby two or more molecules are joined into chains or networks of repeating units (the monomer). [45]

TABLE 25B-3 -- Synthetic Material Properties

Group	Properties	Examples
Thermoplastic	Soft when heated	Ethylene vinyl acetate, polyvinyl chloride, polyethylene
	Hard when cooled	Polypropylene, some polyurethanes
	Heating and reheating repeatable (heat labile)	
Thermosetting	Shaped by heating	Melamine, phenolic and furan resins, aminoplastics, alkyds, epoxy resins
	Retains shape when cooled	Polyesters, silicones, most polyurethanes
	Once set, process not repeatable (heat stable)	

In an effort to increase the shock absorbency of hard rubber soles, gaseous bubbles were introduced into liquid rubber in a process that produced foam rubber in the 1920s. [43] When the structure of the material has openings to environmental air (like a sponge), the material is defined as open-cell foam. Closed-cell foams differ in that their structure is not open to environmental air. [47] This cellular construction provides cushioning as a result of the compressibility of the cellular structure as well as the encapsulated gas. Repetitive impact stress can cause a breakdown in the foam material owing to compaction of the foam cell structure. This can occur in as little as 1 hour in some open-cell, lightweight foams. This effect has been studied in running shoes and indicates a loss of 25% of the initial shock absorption after just 50 miles and a loss of more than 40% after 250 to 500 miles, according to machine testing. [48] Most experienced runners do not change shoes during this mileage range because it may be reached within 1 month of purchasing the shoe and there may be little external appearance of wear. This type of breakdown has stimulated a search for new and improved polymers for the midsole as well as alternative cushioning systems.

Polyurethanes are one of the most versatile groups of synthetic rubber polymers. [50] [69] They are produced by the polymerization process, in which diisocyanates react with polyols (multiple OH groups) into polyesters or polyethers. [45] A liquid catalyst or resin hardener is used to initiate the chemical process. In liquid or semiliquid form, the polyurethane rubber can then be cast, mixed, milled, or vulcanized. Variations in the process can result in materials with a wide range of properties, ranging from hard plastics to soft foams with consequent differences in rigidity and flexibility. Polyurethanes can be either thermoplastic or thermoset resins. Because of their versatility, these compounds have become an integral part of the athletic footwear industry with uses as both midsoles and outsoles. Relatively lightweight and quite durable, polyurethane can be injection-molded into specific shapes or used as a flat sheet. This allows it to be processed into multistudded athletic shoes and intricately patterned running shoe outsoles. [49] Companies continue to support research and development departments to find the ultimate polyurethane for cushioning and durability without sacrificing additional weight. [66] [67]

Ethylene vinyl acetate (EVA) is a copolymer made from ethylene and vinyl acetate in a high-pressure addition polymerization process. [22] It is used most commonly as a foam produced by dispersal of gaseous bubbles within the liquid plastic. With a density less than that of polyurethane, it is lighter and less expensive while providing good resiliency and cushioning properties. [41] [78] It can be adjusted to provide varying hardness, and this has been incorporated into "dual-density" and "tridensity" midsoles. [58] [66] [67] The harder foam usually has a darker color to denote this difference for the educated consumer. When placed in certain key areas of the midsole, these firmer midsole components can theoretically add control features to the shoe. Because EVA has a tendency to deform with repetitive stress, a process of compression molding is often added, using a combination of heat and pressure to improve the memory and the durability of this foam. [30]

The competitiveness between shoewear manufacturers and the expanding field of polymer science are irrepressible stimuli to advances in the materials used in athletic shoewear. New forms of lightweight and durable polyurethane have already been introduced and are gradually replacing EVA midsoles in the more expensive running shoe lines. [66] [67] [68] [78] In nonrunning shoes, in which wear and weight are less important, polyurethane midsoles and outsoles are already quite common. [79] [80] [81] Foams made from new polymers are being introduced with such rapidity that it is hard to keep them all straight. Shoewear companies traditionally attach proprietary names to their technologically based cushioning systems and materials, and it can be difficult to obtain relevant background information.

The introduction of air encapsulation in 1979 by Nike signaled a new era in shock absorption technology. Despite early problems with instability, other companies rapidly adopted this encapsulation using other materials (see Table 25B-1). Encapsulation of a cushioning material such as air or gel avoids or delays the compaction seen with traditional midsole foams, thus improving a shoe's durability. [63] [82] [83] [84] [85] Whether such innovations prevent injuries remains open to speculation, but one can be certain that the athletic shoewear industry will continue to merge science and technology with marketing to give us more sophisticated names and polished promotions.

Heel Counter Materials

The next shoe region in which materials play an integral role is the heel counter. The increasing attention placed on rearfoot stability by research resulted in the incorporation of more rigid materials into the heel cup of the shoe. Originally, the heel counter was found only in the running shoes made by the Dassler brothers at Adidas and Puma and consisted of a fiberboard construction. [9] The fiberboard lost its stiffness with repetitive wear and constant moisture, leading to the use of more durable plastics. Today, molded plastics take the place of sheet plastic to provide better fit. Heel counters are made from a variety of synthetics, including polyethylene, polyvinyl chloride, and other thermoplastic materials. [50] [58] [67] The stability of the heel counter is enhanced by foxing, external stabilizers, or footframes made of leather or synthetic materials.

Upper Materials

Leather remains the most commonly used material in general shoewear construction, particularly for the upper. [75] [86] It has gradually been replaced in many athletic shoes, in which synthetics provide certain specialty features such as better breathability or reduced weight. [22] Leather comes from the skin of an animal and goes through a tanning process to fix the proteins in the skin and eliminate components that would promote degradation. This process can be varied to affect the properties and texture of the finished product. Two layers of the skin are available for use in shoemaking once the skin has been split (Fig. 25B-18). [50] [75] This process reduces the thickness of the material and changes its properties, the inner split being softer but tending to fray more readily.

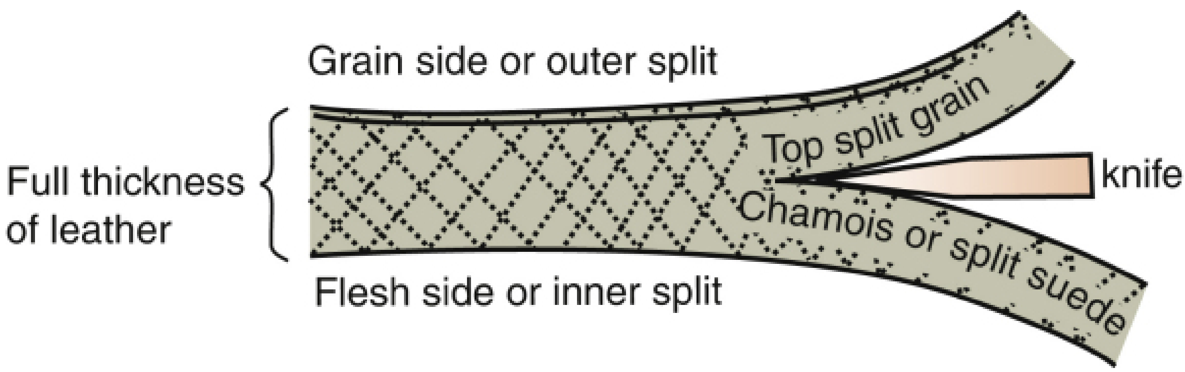

Figure 25B-18 Splitting of leather into two layers of skin for use in shoemaking. *(Redrawn from Cheskin MP: The Complete Handbook of Athletic Footwear. New York, Fairchild Publications, 1987; and Philps JW: The Functional Foot Orthosis. New York, Churchill Livingstone, 1990.)*

Leather's usefulness is apparent from its universal applicability to footwear construction since ancient times. It has the ability to adapt to the shape of the foot and maintain the altered configuration. Leather transmits perspiration ("breathes") and can be treated to resist or repel water. [50] [86] Tensile strength is outstanding (up to 4 tons per square inch), flexibility is excellent, and puncture and abrasion resistance is superior. [22] The tanning process, along with the finishing and dyeing of the leather, can accentuate one or all of these properties. The disadvantages of leather are its deformability under stress, its tendency to crack when successively moistened and dried, its weight, and cost variations.

Nylon-weave uppers are made from polyamide resin fibers woven together into a taffeta and doubled or interfaced with a thin foam or tricot lining. [22] In the assembly process, these layers are "flamed," or heat-bonded. This bonds the laminates together, resulting in a material more flexible and absorbent than if the layers were glued. [34] The variables in this composition process are the exact material used in the thread, the size of the thread, the number of threads in the bundles, and the number and orientation of the bundles going lengthwise and widthwise (Fig. 25B-19). [9] The closeness of the weave affects the mechanical properties of the fabric. The nylon weave of a taffeta upper has good durability, softness, and flexibility and is lightweight, making it a superior replacement for leather in many athletic shoes. [37] [50]

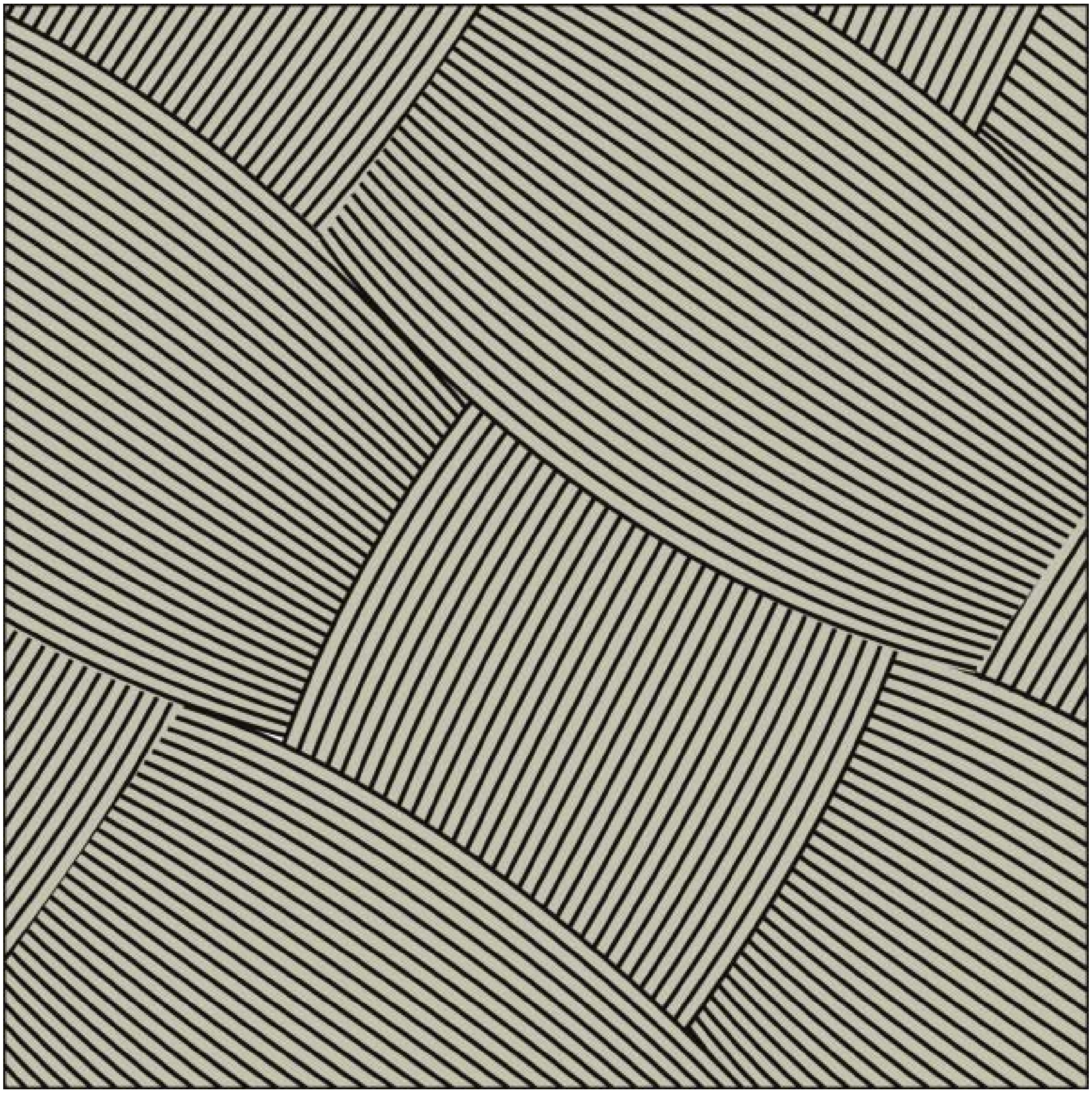

Figure 25B-19 Nylon-weave uppers made from fibers woven together into a taffeta. *(Redrawn from Cavanagh PR: The Running Shoe Book. Mountain View, Calif, Anderson World, 1980.)*

Nylon mesh is made from the same nylon threads, but in a knitted rather than a woven process (Fig. 25B-20). [9] This knitted process adds space within the strands without compromising strength. Thus, the breathability of the upper is improved. It can be used as a single-, double- or triple-mesh knit. Increasing the denier of the thread adds body and strength to the fabric. Whether used in mesh or woven form, the nylon upper is generally combined with a thin foam and a tricot lining for improved fit and comfort. [22]

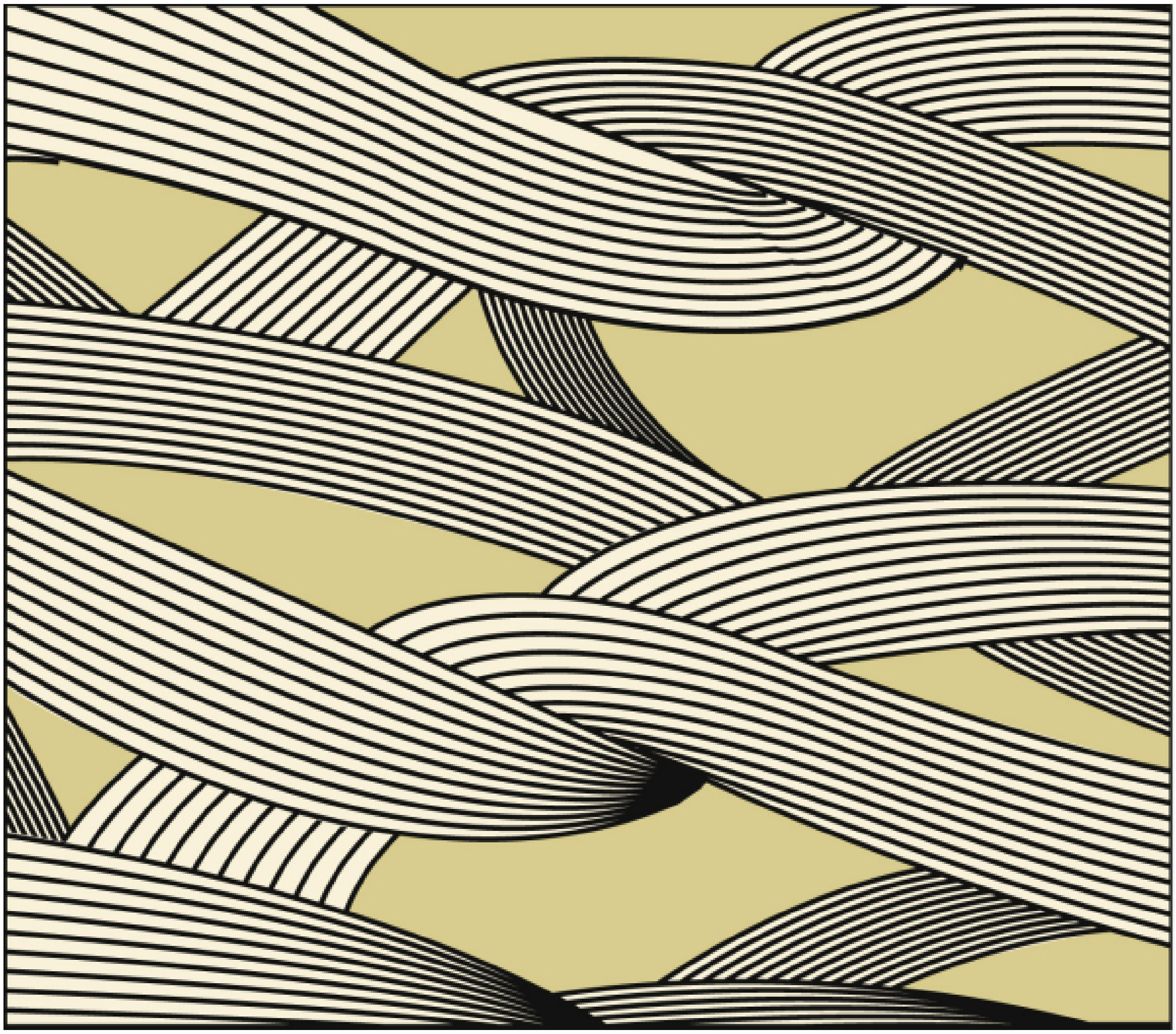

Figure 25B-20 Nylon mesh made from threads with a knitted rather than a woven process. *(Redrawn from Cavanagh PR: The Running Shoe Book. Mountain View, Calif, Anderson World, 1980.)*

Other synthetic materials are finding their way into athletic shoe uppers, including Gore-Tex (W. L. Gore and Associates, Elkton, Md). [30] Thermoplastic vinyl has uses in golf shoes in coated or laminated applications to leather or fabric. Slush- and dip-molded uppers of thermoplastic materials are used in recreational ice skates and certain waterproof footwear, whereas injection-molded thermoset plastic is the norm for ski boots. [22] Depending on the shoe's intended use, the shoewear manufacturer can provide a full range of breathability, extending from complete breathability up to complete insulation and waterproofing. The upper can be easily or barely deformable. Weight can be varied over a wide range, and the foot can be protected minimally or maximally.

Inlays, Inserts, Insoles, and Orthoses

Since ancient times, it has been known that the addition of leaves, moss, or animal skin to the inside of the shoe could provide cushioning for the foot and protection from environmental stresses. [10] For soldiers on long marches, this extra protection might mean the difference between life and death. For modern-day runners and athletes, this cushioning is intended to protect the most readily identifiable weak link in the kinetic chain. Many athletes would provide testimonial support for the merits of their orthotic devices or inserts in preventing injury or enhancing performance. Although it has been more difficult to document these beneficial effects objectively, [59] [87] [88] scientific evidence of the shock absorption properties of the various materials used in these devices does exist. [82] [85] [89] [90] This section provides some acceptable definitions to help make sense of this confusing area and then describes some of the materials used. The following section discusses their biomechanical properties.

Because of the confusion that surrounds this area, we have chosen to use the definitions of terms accepted by the Pedorthic Footwear Association.[63] The *insole* is the integral design component (layer) of the shoe that is the shoe's structural anchor to which is attached the upper, toe box, heel counter, linings, and welting. The *inlay* is a prefabricated removable material upon which the foot directly rests inside the shoe. In some shoes, the inlay is an integral design component. The *insert* is a type of orthosis, although the term has been used interchangeably with inlays and insoles in some circles to designate an off-the-shelf device placed inside the shoe. For the purpose of this chapter, we use the definition for an *insert* supplied by the Health Care Financing Agency: a total contact, multiple density, removable inlay that is directly molded to the patient's foot or a model of the patient's foot and that is made of a suitable material with regard to the patient's condition. An *orthosis* (or orthotic device) is a device that is used to protect, support, or improve function of parts of the body that move. A common error is to use the adjective *orthotic* as a noun. A *pad* is a device placed inside a shoe to provide support or relieve pressure from a specific location such as a longitudinal arch pad or a metatarsal pad. Pads are made of various materials and come in a variety of shapes and sizes.

Inlays and inserts can be made from a single material or a composite of several materials. The most commonly used materials are leather, cork, foam, felt, and plastic.[41] Based on the previous definitions, inlays are "off-the-shelf," whereas inserts are "custom made." The latter can be subdivided into those made from a casting of the foot and those made from an impression of the foot (Fig. 25B-21). [69] [75] [92] These can be formulated in a weight-bearing, partial weight-bearing, or non–weight-bearing fashion. A recent trend in orthosis manufacture uses digital foot scanners, obviating the need for a negative mold of the foot. A topographic reading of the plantar foot is obtained using laser scanning, digitized force-plate gait and pressure analysis, or digital analysis of multiple air pegs. These manufacturing devices employ computer-assisted design in the final conversion of data to orthosis. [93] [94] [95] [96] The devices, which are at present quite expensive, largely rely on the subtalar neutral position as a starting point for data collection. The subtalar neutral position has been variously defined as the position from which there is equal inversion and eversion range of motion, the position from which there is twice as much inversion as eversion, or the position from which the talar head is most fully covered by the tarsal navicular when palpating the foot. It is unclear how much a small variation in this point affects readings and ultimately function. Adequate data in the form of prospective, randomized, controlled studies comparing the use of orthoses manufactured with these systems and inserts made with traditional methods are lacking. There are no data confirming their superiority or justifying their cost.[69] Given the uncertainty surrounding the multitude of theories guiding orthotic prescriptions, it would be helpful to have some scientific support for the ability of foot orthoses to accomplish their stated objective.

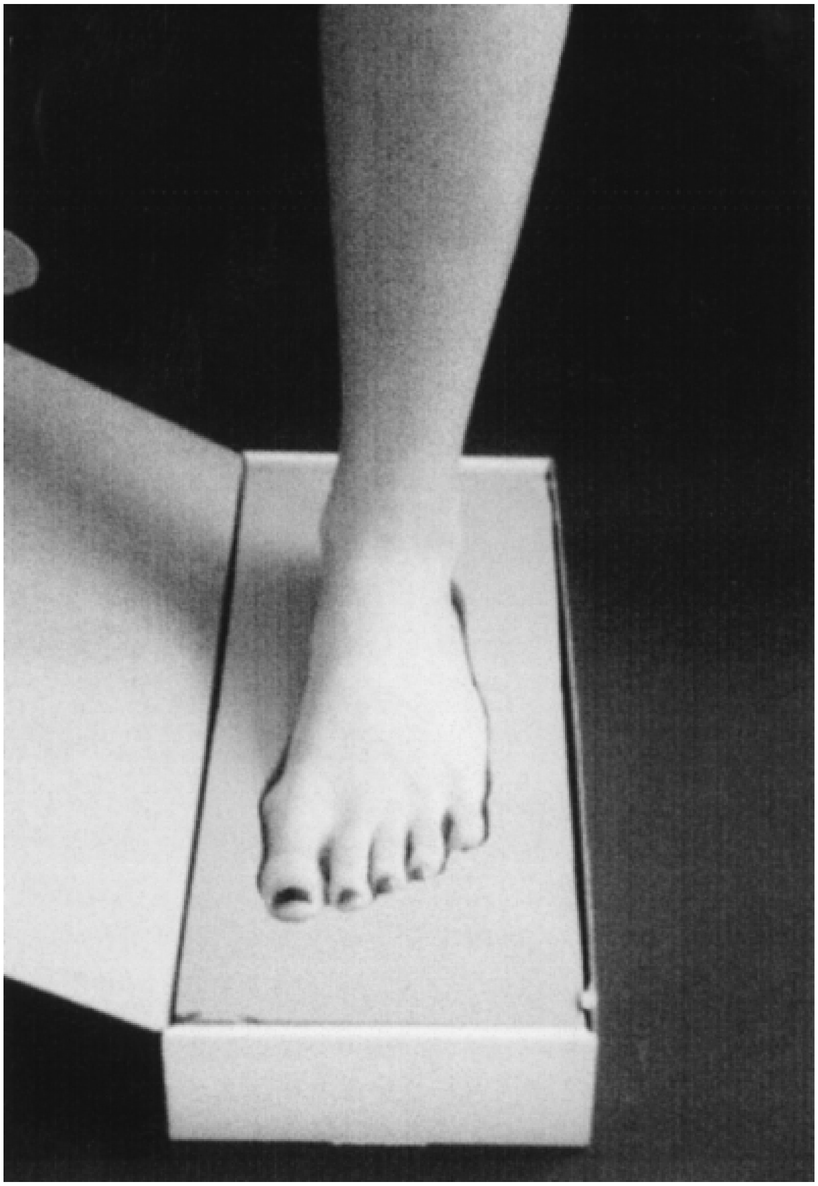

Figure 25B-21 Foam box used for taking an impression of a foot to make a custom-made orthosis. *(Photograph by Andrew Borom.)*

For general classification purposes, inserts can be further divided into accommodative or functional varieties. *Accommodative* devices are those that are designed with a primary goal of conforming to the individual's anatomy, whereas *functional* devices are designed with the primary goal of controlling an individual's anatomic function, such as providing support or stability, or assisting ambulation. [75] [91] [98] Different materials can be used for these purposes, leading to an additional subdivision into rigid, semirigid, and soft, in which the material becomes the critical factor.

As discussed previously, the materials used in shoes, and now in orthotic devices, can be either natural or synthetic. [41] The most commonly used natural materials are leather, rubber, cork, metal, and felt. Synthetics include plastics and foams (both closed and open cell), which can be manufactured with varying qualities of hardness, density, durability, and moldability. The following paragraphs consider the advantages and disadvantages of these materials.

Natural Materials

Leather is extremely durable and conforms well to the contours of the foot. It is readily available, is tolerated well by the skin, and combines well with other materials as a composite. [69] [86] On the negative side, leather is relatively expensive and provides very little shock absorption. [47]

Rubber has been discussed in the preceding section. It provides good shock absorption with durability but is heavy, and its qualities vary considerably according to the exact process used in its production. [69] [70] Rubber foams are made by the addition of chemical additives or air. Closed-cell foam rubbers are more stable and durable. [47]

Cork is a lightweight cushioning material made from the outer bark of a tree. [71] Although it does not work well when in direct contact with the skin, it is usually used in combination with leather and has a good history of use as an insert. Synthetic forms of cork are now used. Although cork was the material used in the first formal shoe insert in the 18th century, [72] it has fallen into disfavor because of its mediocre shock and shear absorption capacities in comparison with some of the newer products.

Synthetic Materials

Among synthetic materials, there are a wide variety of plastics and foams that have been used in the fashioning of orthoses, inlays, inserts, pads, and insoles. Included in this group are various synthetic rubbers, polyolefins, thermoplastics, thermosetting materials, viscoelastic materials, polyurethane foams, and certain copolymers and composites. [69] [75] [82] [92] Because many of these have been described previously, the present discussion is confined to materials in common use that have not been previously described.

Styrene-butadiene rubber is a synthetic rubber that has found considerable applicability in orthotic devices both as a basic shell and as a posting material. [41] Neoprene is a special rubber made of polychloroprene and is primarily known for its use as an inlay or insole material in the form of a closed-cell foam commonly known as *Spenco* or as an open-cell foam called *Lynco* (Table 25B-4). [69] [82] [101] It functions well to reduce friction and attenuate shock but is somewhat hot (particularly in the closed-cell form).

TABLE 25B-4 -- Neoprene Foams

Name	Qualities	Company
Spenco	Closed cell, nylon cover	Spenco Medical Corp Waco, Tex
Lynco	Open cell	Apex Foot Products, Englewood, NJ

The polyolefins consist of polyethylenes, polypropylenes, and their copolymers. [69] [102] When fabricated as foam, these materials fill a multitude of uses. Because of the modifications available in the manufacturing process, it is possible to vary the mechanical properties of the material over a wide range. Therefore, these foams can be constructed as flexible, semirigid, or rigid orthoses. Table 25B-5 lists some of the trade names for polyethylene foams that are in common use along with their manufacturers. [41]

TABLE 25B-5 -- Polyethylene Foams

Name	Qualities	Company
Plastazote	Three densities	Apex Foot Products, Englewood, NJ
Pelite	Three densities	Durr-Fillauer Medical Inc., Chattanooga, Tenn
Aliplast	Four densities	AliMed Corp., Dedham, Mass
Berkezote	Medium density	Foot & Ankle Orthopaedic, Bedford Hills, NY
UCOlite	Medium density	UCO International, Prospect Heights, Ill
UCOplast 10	Very firm	UCO International, Prospect Heights, Ill

Thermoplastic materials are materials that become malleable when heated and hold the set configuration when cooled. They allow reheating for further remodeling. [73] [74] Polyvinyl chloride (PVC) is one example of such a material. As a plastic, PVC is frequently found in the heel counter of athletic shoes. Polyethylene thermoplastics are one of the most commonly used orthotic materials because they can be fashioned to conform to the patient's foot and maintain this position under weight-bearing stress. [41] The material is relatively stable yet possesses adequate flexibility and strength. Depending on the manufacturing process, the polyethylene thermoplastic can have a low, medium, high, or ultrahigh density. For orthoses, ultrahigh-density polyethylene is most commonly used. Trade names for these products include Ortholen, Subortholen, and Vitrathene. [69] [75]

Thermosetting materials differ from thermoplastics in that they can be molded after heating, but once set, they do not allow repetition of this process. Therefore, they are heat stable.[46] The material can be processed into either a plastic or foam and is used in both forms. The most common thermosetting resins are phenolics, furan resins, aminoplastics, alkyds, allyls, epoxy resins, polyurethanes, some polyesters, and silicones.[45] The original thermosetting material was a combination of phenol and formaldehyde (Bakelite) formed by high pressure and high temperature.[73] [74] The physical properties of these plastics can be varied by altering the filler material.[46] Polyurethanes can be either a thermosetting resin or a thermoplastic.[45] Their principal application is in the form of an open-cell or closed-cell foam. There are a variety of trade names for these products (Table 25B-6). The materials come in various densities and thicknesses, and their mechanical properties are described later in this section.

TABLE 25B-6 -- Polyurethane Foams

Name	Qualities	Company
PPT	Open cell, single density	Langer Biomechanics Group, Deer Park, NY
Poron	Open cell, single density	Rogers Corp, East Woodstock, Conn
Axidyne		Force 10-Polymer Dynamics, Allentown, Pa
OVA-FLEX	Low to medium density	UCO International, Prospect Heights, Ill
OVA-FIT	Medium density	UCO International, Prospect Heights, Ill

Viscoelastic materials are materials that combine the mechanical properties of a viscous fluid and an elastic solid. When subjected to a constant deformation or load, their response varies in relationship to the time of application.[75] This is an important mechanical property that is also exhibited by tissues of the human body. The viscoelastic nature of a material allows both the storage and dissipation of mechanical energy.[59] This would seem to be a desirable quality in an inlay or insert, leading to the claims of manufacturers that it is the perfect material for shock attenuation in running or jumping. Because the time required for stress relaxation to take place is a critical factor in functional shock absorption, however, one cannot assume that these claims are entirely accurate. The trade names for the most commonly used viscoelastic orthoses are given in Table 25B-7 .

TABLE 25B-7 -- Viscoelastic Materials

Name	Company
Sorbothane	Sorbothane Inc., Kent, Ohio
Viscolas	Chattanooga Corp., Chattanooga, Tenn

Copolymers are substances formed by a process of copolymerization wherein two unlike molecules are united in either a randomly or regularly alternating sequence within a chain.[74] An example of this is the nylon acrylic resin Rohadur, which is made from methylmethacrylate and acrylonitrile.[41] It is heat moldable and rigid and is primarily used as a functional orthosis. According to Levitz and coauthors, this material was developed in Germany in the 1950s and sold in the form of resin pellets. The different companies that marketed the product for orthopaedic use turned the pellets into sheets of resin.[41] When the original material was discovered to be possibly carcinogenic, it was reformulated and is now a clear mahogany-colored plastic. It is widely used in making orthoses and comes in sheets varying from 2 to 5 mm in thickness. It is one of the most rigid materials available.[47]

Although each of these materials can be found among the various orthotic devices on the market today, each has specific advantages and disadvantages. One foam may have excellent cushioning properties yet be inordinately heavy or hot, and one plastic may be very strong and stable while being difficult to work (Table 25B-8). For this reason, the use of composite orthoses has emerged to combine the properties of two different materials to their best advantage. The ideal orthosis is moldable to the foot, durable, lightweight, effective in maintaining the proper foot position, and capable of eliminating detrimental stress. Because it appears that no one material can combine all these properties, we can expect an increase in the types and complexities of these composites.

TABLE 25B-8 -- Copolymer Foams

Trade Name	Qualities	Manufacturer
Polyolefin Foams		

Trade Name	Qualities	Manufacturer
Evazote	Closed cell	Apex Foot Products, Englewood, NJ
	EVA material	
Polyvinyl Chloride Foam		
S.T.S.	Open cell	Apex Foot Products, Englewood, NJ

BIOMECHANICAL ASPECTS OF SHOES AND ORTHOSES

An understanding of biomechanics as it applies to athletic shoes and the use of orthotic devices requires some understanding of gait and the history of its study, which dates back to the time of Aristotle. Cavanagh has reviewed this history in his book *Biomechanics of Distance Running*, wherein he points out the influence of such men as Leonardo da Vinci, Isaac Newton, Giovanni Borelli, the Weber brothers, Etienne Jules Marey, Vierordt, Braune and Fischer, Eadweard Muybridge, A. V. Hill, Wallace Fenn, Nicholas Bernstein, Herbert Elftman, O. Boje, and Rodolfo Margaria.[76] Contemporary workers in this field include Barry Bates, David Brody, Peter Cavanagh, Tom Clarke, Ed Frederick, John Hagy, Harry Hlavac, Verne Inmann, Stan James, Roger Mann, Benno Nigg, Merton Root, Don Slocum, Thomas Sgarlato, John Weed, and others. It would take an entire chapter to review the contributions of these men—hence the interested reader is referred to their original works, the references from Mann's section on biomechanics, and the preceding section on the causes of injury to the foot and ankle. Without the contributions of these individuals, there would be no foundation on which we could base further research.

Whether one is talking about shoes or orthoses or a combination of these, from a biomechanical standpoint, one is primarily concentrating on (1) the effect of shoes or orthoses on reducing the forces present at foot strike, (2) their ability to improve functional motion within the foot, and (3) their efficacy in preventing or treating pathologic conditions in the lower extremity. Although it is known that certain other biomechanical parameters such as running economy and speed can be altered by shoes and orthoses, these factors are not discussed in any depth in this section.

Nigg reviewed the available literature and combined this with nearly 2 decades of his own investigations in the field of shoewear research to formulate a new concept for inserts and orthoses. Although the interested reader is referred to this excellent review for details, the Human Performance Laboratory in Calgary proposes that the traditional view of the ability of orthoses or inserts to align the skeleton is not supported in the available literature. Rather, the concept that an orthosis or insert functions most effectively if it minimizes muscle work is advanced by these investigators.[77] The basics of this proposed concept are found in Table 25B-9 . The situation-dependent variables can be influenced by the shoe, insert, orthosis, or selection of the movement task, whereas the subject-dependent variables, by definition, vary from individual to individual. If this proposal is accepted, the authors suggest that it is possible to conclude the following regarding orthoses:

- The skeleton has a preferred path for a given movement task (e.g., running).
- If an intervention supports the preferred movement path, muscle activity is decreased. Interference with the preferred path increases muscle activity.
- An optimal insert or orthosis decreases muscle activity.
- An optimal insert feels comfortable owing to decreased muscle activity with resultant decreased fatigue.
- With an optimal insert, performance should increase in association with decreased muscle activity and fatigue.

TABLE 25B-9 -- Proposed Concept for Inserts and Orthoses

Situation-Dependent Variables	Subject-Dependent Variables
A force signal acts as an input variable on the shoe, based on the chosen movement.	The soft tissue and mechanoreceptors on the plantar surface of the foot act as a third filter.
The shoe acts as a first filter for the force input signal.	The filtered information is detected by the central nervous system, which provides a subject-specific dynamic response.
The insert or orthosis acts as a second filter for the force input signal.	The subject performs the movement task at hand.

From Nigg BM, Nurse MA, Stefanyshyn DJ: Shoe inserts and orthotics for sport and physical activities. Med Sci Sports Exerc 31(Suppl 7):S424-S428, 1999.

This proposal is clearly innovative and controversial because it is unsupported by adequate experimental evidence. Further research matching subject and insert characteristics to identify the optimal solution for insert and orthosis fitting will, it is hoped, fill this evidence gap. [77]

As mentioned previously, the functional orthosis is fabricated based on a specific biomechanical theory. It usually uses the "subtalar neutral" position as the starting point. This theoretically aligns the hindfoot with the forefoot and allows the foot to function in its most biomechanically advantageous position. The functional orthosis changes the position of the foot with respect to the weight-bearing surface. The accommodative orthosis, in contrast, brings the surface up to meet the foot in its steady-state position in an effort to improve weight distribution and alleviate symptoms. The accommodative device must be fabricated from a material that will mold easily to the surface of the foot because one of its primary purposes is to accommodate deformities. In contrast, the functional orthosis must be rigid enough to maintain the foot in the position chosen for maximal function. This makes it apparent why plastic is usually selected for the functional device and a polyethylene or polyurethane foam is more commonly the choice if accommodation is the goal.

Shock Absorption

Loading of the athlete's body during sports activities has been implicated as a significant causal factor in pain and injury. [106] [107] [108] [109] The study of this relationship is an essential element in the field of sports biomechanics. [104] [110] [111] Numerous works have been published on the biomechanics of walking and running, [111] [112] [113] [114] [115] [116] [117] [118] and a like number have documented the forces acting on the foot during various activities. [107] [116] [117] [118] [119] [120] [121] [122] [123] [124] [125] Measurement of the pressure under the foot during gait dates back to 1882 with the work of Beely, who used crude manual methods. Since then, measurement of load has progressed to the use of force plates with the aid of piezoelectric transducers or strain gauge technology with computer analysis. [125] [126] The vertical force component produces skeletal transients beginning at heel strike or foot strike, and these have been theorized to produce injury through their resultant shock and shear waves. [127] [128] [129] [130] [131] [132] Experimental support for this analysis was provided by the finding that osteoarthritis developed in the joints of sheep housed on concrete. [133] [134] The association between chronic repetitive trauma, exercise, and arthritis has been the source of considerable controversy. [135] [136] [137] [138] Although controlled studies have failed to demonstrate a clear relationship between osteoarthritis and the loads generated in sports such as running (in otherwise healthy participants), [137] [138] [139] [140] [141] [142] this relationship has nevertheless served as an important catalyst for the athletic shoe industry to improve the shock absorption qualities of shoes.

Although increasing cushioning in shoewear seems intuitively appealing as a method to diminish shock transmission to the skeleton, Robbins and coworkers have produced a sizeable volume of work outlining the potentially detrimental effects of soft materials in shoes. [45] [143] [144] [145] [146] Robbins and Hanna proposed that shoewear creates a pseudoneurotrophic condition that eliminates the plantar tactile response from the human system designed to minimize impact loading through alteration of musculoskeletal response. [119] It has been demonstrated that although certain interface materials reduce vertical impact from inanimate objects dropped on them, human landing paradoxically increases these forces. [120] Gymnasts landing on a 10-cm thick mat demonstrated a 20% increase in vertical impact compared with a rigid surface. [121] Behavioral modifications that can either amplify or reduce vertical impact include variation in amplitude of hip and knee flexion. [122] Bending at these joints is decreased when landing on soft surfaces. This stiff-legged landing serves to heighten impact, whereas landing on hard surfaces results in increased hip and knee flexion to absorb energy. [121] Stability is part of this equation. When soft materials are placed beneath the plantar surface of individuals on a force platform, stability declines as measured by increased sway. [123] Human balance improves when placed on thin, stiff surfaces. [152] [153] To accommodate these factors, humans adopt landing strategies to deal with the landing surface. Decreased hip and knee flexion is used momentarily to increase stability by compressing the interface material. [121] Robbins and Waked examined ground reaction force in 12 men without disability using a 4.5-cm foot fall onto a force platform covered with one of four materials. [121] Vertical impact was inversely related to surface stiffness, with the softest surface producing the greatest impact. They concluded that balance and vertical impact are closely related and hypothesized that landing on a soft surface is accompanied by an attempt to render it more stable by compressing the material, decreasing its thickness and increasing its stiffness. Although recognizing the impracticality of barefoot activity, they propose that currently available athletic shoes are too soft and thick and recommend redesign. [121] In a further study by the same investigators, a "deceptive" advertising message associated with an EVA-covered platform was shown to produce higher vertical impact forces than the identical platform with a preceding "neutral" or "warning" message. [17] Although this theory has not been universally accepted, there is evidence that shoe manufacturers are incorporating thinner midsole shoes into their product lines, with the purported advantage of "more speed and stability." [55] Despite this opposing viewpoint, there are scientific data to support the incorporation of elements of cushioning for the reduction of certain overuse injuries.

Experimental Work

Available independent data on laboratory testing of various materials used in shoes and orthotic devices are somewhat limited compared with other areas of research in the fields of sports medicine, orthopaedics, and podiatry. In one of the earliest studies of this kind, Brodsky and coworkers studied the effects of repeated compression and the effects of repeated shear and compression on the behavior of five commonly used materials for shoe inserts. [54] They also determined the force-attenuation properties of the new and used materials. The materials tested were Plastazote (Apex Foot Products, South Hackensack, NJ), Pelite (Durr-Fillauer Medical Inc., Chattanooga, Tenn), PPT (Langer Biomechanics Group, Inc., Deer Park, NY), Sorbothane (Sorbothane, Inc., Kent, Ohio), and Spenco (Spenco Medical Corp., Waco, Tex). To test compression, the authors used an Instrom testing machine and subjected the materials to cyclic loads. The greatest degree of compression was seen with the soft-grade Plastazote, which went from an original thickness of 6.6 mm to 4.55 mm after 5000 cycles. Lesser amounts of compression were seen with medium-grade Pelite, Spenco, and Sorbothane, and the least change was found with PPT.

In the same study, the authors also looked at the resilience of the different materials. Resilience is a measure of a material's ability to resume its original shape after having been distorted. This is an important property in an insole or orthotic device. The resilience of the above materials was determined by remeasuring their thickness after a period of rest. Both Plastazote and Pelite showed good rebound in thickness after a 12-hour rest, which allowed a return to 6.0 mm for the Plastazote (a 70% return). Unfortunately, after rebound takes place, accelerated compression occurs when the material is again subjected to the same stress. Some of the results of this study are displayed in Table 25B-10 . [54]

TABLE 25B-10 -- Maximum Loss in Thickness Expressed as Percentage of Original Thickness after 10,000 Cycles

Material	Compression (%)	Shear Compression (%)
Plastazote (soft)	55	45
Pelite (medium)	15	16
Spenco	15	3.6
Sorbothane	3	10.2
PPT	0	0

From Brodsky JW, Kourosh S, Stills M, et al: Objective evaluation of insert material for diabetic and athletic footwear. Foot Ankle 9:111-116, 1988.© American Orthopaedic Foot and Ankle Society.

Foto and Birke recently investigated resilience for the most commonly prescribed multidensity material combinations used in manufacturing orthotic devices. Dynamic strain, or the material's percent deformation per cycle, as well as strain loss (or compression set), reflective of the material's permanent deformation, was measured for each of four combinations of materials. Cyclic loading to 10,000 and 100,000 cycles demonstrated marked differences in the temporary and permanent deformation of the various combinations. Although the materials tested are more commonly used in the treatment of diabetic plantar pressure problems, the authors point out that an ideal pressure-relieving orthosis should demonstrate a dynamic strain of 50% or better at 350 kPa of pressure and that compression set should be minimal. Excessive compression set corresponds to losses in posting, pressure relief, or accommodation of deformity. [55] Furthermore, composite material performance is affected by the overall thickness as well as the ratio of the individual materials.

The forces on the foot during walking, running, and other sports activities are rarely only compressive in nature. Therefore, a more physiologic test measures how these materials withstand both shear and compression. This process was examined in the study by Brodsky using a special jig designed for this purpose. [54] The highest degree of change in thickness was again seen in Plastazote with a lesser amount of change in the samples of Pelite and Sorbothane, minimal change in Spenco, and essentially no change in PPT. Rebound was again noted in the materials after a period of rest. One interesting finding was that although Plastazote and PPT showed similar results in both the compression and the shear-compression tests, a notable difference was evident between the Spenco and the Sorbothane. Spenco tended to resist shear better than compression, whereas Sorbothane showed a better resistance to compression than shear. [54]

From the standpoint of the patient and the clinician, the most pertinent information concerning a given insole material is how much reduction in force it affords, that is, how much of the force that is transmitted is actually experienced by the foot? Bench research confirms that all materials (Plastazote, Pelite, PPT, Spenco, UCOlite, ZDEL, and Sorbothane) reduce the force transmitted to the load cell protected by these materials by 10% to 60%. [82] [85] [154] [155] [156] Nigg and colleagues, in a review of the literature, examined the available data on shock absorptive insert materials and concluded that the typical reduction of impact loading is in the 10% to 20% range. They questioned whether these small reductions are capable of reducing injury, and suggest that material alteration of inserts may produce an effect through adjustments in the muscular response of the locomotor system. [77] The ability of different materials to accomplish shock absorption can change depending on the amount of force involved and the study methodology, with the exception of Sorbothane, which transmits the highest forces over the entire range. [82] [85] In contrast, an in vivo study of British Royal Marine recruits evaluating in-shoe pressures found Sorbothane to be significantly better in attenuating peak pressure at heel strike for both marching (23% decrease) and running (27% decrease). [62] All materials demonstrate a reduction in shock absorbency after cyclic stress (a phenomenon known as *stress relaxation*), although this reduction is considerably more apparent in the softer grades of polyethylene foams such as Plastazote, which can lose more than 50% of its shock absorbency after 25,000 cycles. [82] [154]

The actual amount of cushioning contributed by a given material varies according to several factors that have significant implications for their applicability to sports. In general, greater cushioning occurs with increasing thickness of the material, but this also increases weight and affects the stability, comfort, and flexibility of the device or the shoe. Furthermore, it has been shown that softer shoes may allow increased pronation compared with shoes with a firm midsole, for example, and this could produce rather than prevent certain types of injuries. [157] [158] [159] As stated earlier in this chapter, the correlation between laboratory testing conditions and the physiologic situation in the athlete may be rather poor. [148] [158] [159] [160] [161] [162] [163] [164] [165] Such factors as the size of the missile head used in the impact tests, the shape of the head, and the height from which the missile is dropped can all affect the results of the tests. [134] Other factors influencing load magnitude include running velocity, joint kinematics, running strategy, choice of surface, and pattern of foot contact. [51] [100] [78] [121] [158] [160] [161] [162] [163] [166] [167] [168] Therefore, one must be cautious in interpreting test results and include in the equation the data gained from experience and clinical trials.

Clinical Work

The clinical influence of improved shock absorption provided by shoes and inserts is reviewed to some extent in Chapter 25C . Other studies dealing with the effect of cushioning on injury and pain are discussed here.

Stress fractures are an obvious example of an injury for which one would expect to see a reduction in incidence with the use of improved cushioning in shoes or with shock-absorbing insoles. Unfortunately, studies of stress fractures in military recruits have shown inconclusive results. Milgrom and colleagues studied the effect of a semirigid, composite orthotic device on the incidence of stress fractures in Israeli Army recruits. [141] There was a reduction in the incidence of femoral stress fractures, but the effect on tibial and metatarsal fractures was insignificant. In fact, the average number of stress fractures per recruit was identical in both groups of recruits, those with the orthotic device and those without. In a subsequent study, Milgrom and coworkers prospectively randomized 390 recruits to train in either standard issue military boots or a modified basketball shoe. The latter group sustained significantly fewer metatarsal stress fractures and overuse injuries of the foot. Prevention was limited, however, to injuries related to vertical impact loading. In particular, tibial stress fractures were not prevented because they are the result of bending stress. The overall incidence of lower extremity overuse injuries was likewise unaffected. [142] Milgrom and associates prospectively examined stress fracture incidence in Israeli army recruits fitted with either a soft or semirigid custom-made functional orthosis, and compared them with a group who wore no biomechanical orthosis. All recruits trained in a modified infantry boot whose sole design resembled a basketball shoe. Although their data appeared to suggest a protective benefit in the soft orthosis group, more than half of the subjects failed to complete the study. The leading cause of failure to complete the study was dissatisfaction with the orthosis, although the soft orthoses were better tolerated than the semirigid variety. [62] In a separate study, Nigg prospectively followed 131 runners for 6 months. Although no difference in injury frequency was seen for subjects with low-, medium- or high-impact force peaks, those runners with a high loading rate sustained roughly 50% fewer injuries than those with a low loading rate (Fig. 25B-22). [143] Gardner and coworkers found no reduction in stress fractures with the use of a viscoelastic insole placed in the shoes of military recruits. [144]

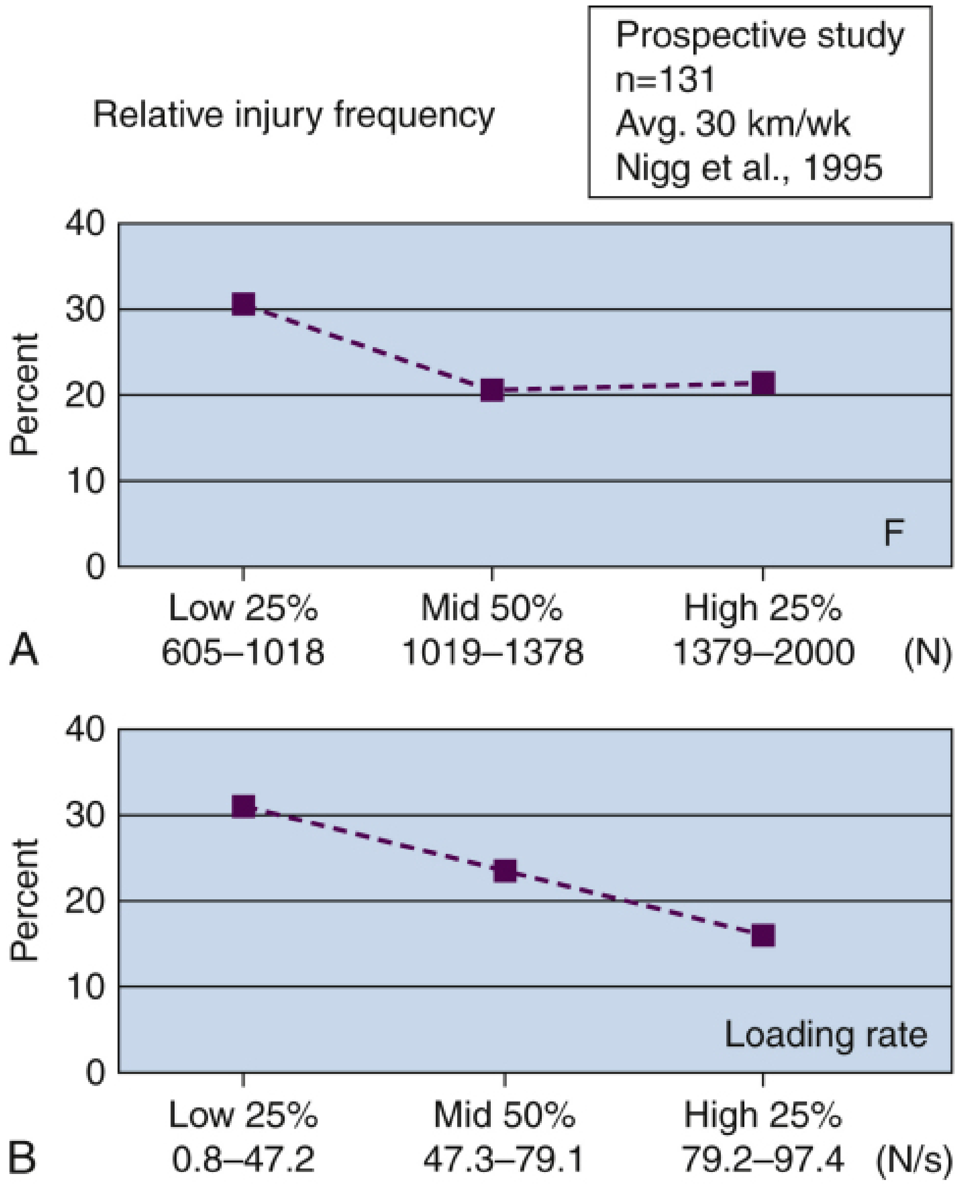

Figure 25B-22 Relative injury frequency for groups with (**A**) high-, medium-, and low-impact force peaks, and (**B**) high-, medium-, and low-maximal loading rate. *(Redrawn from Nigg BM, Kahn A, Fisher V, Stefanyshyn D: Effect of shoe insert construction on foot and leg movement. Med Sci Sports Exerc 30:550-555, 1998.)*

Another element of shock absorption relates to foot pressure and involves proper distribution of load. Locally concentrated forces can cause pathologic lesions such as intractable plantar keratoses, corns, and in the diabetic population, ulceration. Additionally, comfort may be affected by concentration of local pressure. Plantar pressure assessment has played a key role in the management of diabetic patients with neuropathy. [173] [174] [175] [176] [177] Running shoes, by virtue of their design, have been used to better distribute plantar pressures in this patient population in the hopes of avoiding ulceration. [147] Shoewear that reduces peak plantar pressure can do so either by reducing the force or by increasing the area over which the force is distributed. The ability of an insole or insert to distribute peak pressures and cushion the plantar foot, although related to the composition and thickness of the insert material, seems more dependent on the plantar tissue thickness. [150] Although the footwear industry is known to evaluate in-shoe pressure, this information (like so much else in the athletic shoe industry) is considered proprietary and is not available to the scientific community. [146] Although pressure assessment does not seem necessary or practical in the normal population, Figure 25B-23 demonstrates the ability of a moderately priced running shoe to distribute pressure more effectively over its plantar surface than an inexpensive sports shoe. [146]

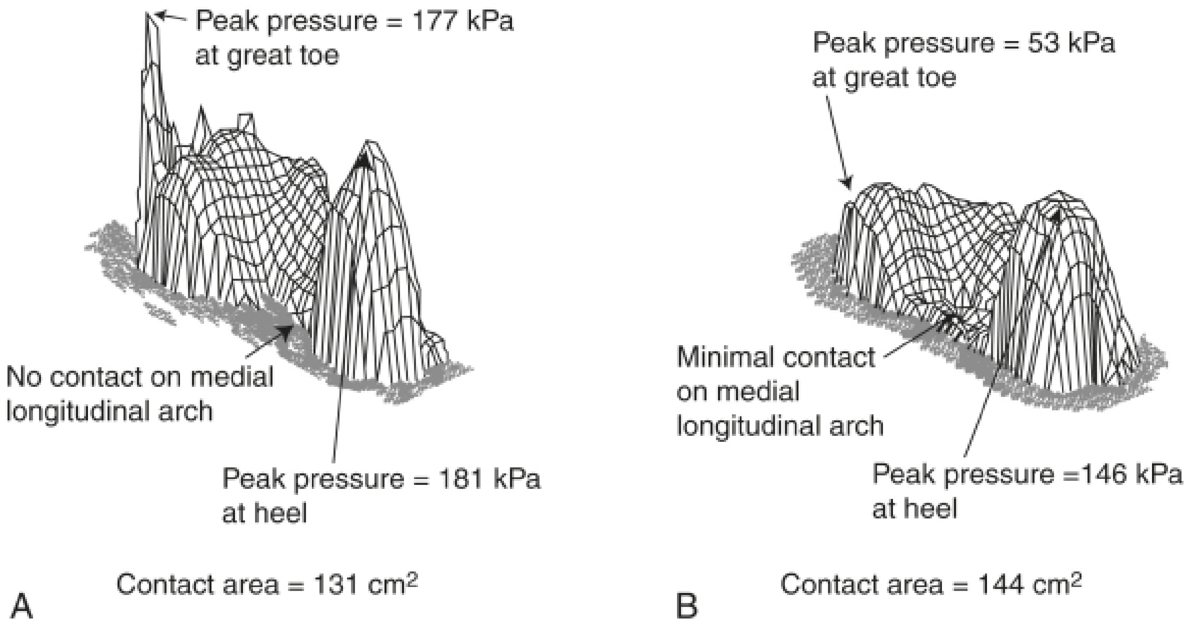

Figure 25B-23 Example of peak pressure measurements comparing different shoes. **A,** Shoe with limited cushioning properties. **B,** Shoe with improved cushioning properties. *(Redrawn from Mueller MJ: Application of plantar pressure assessment in footwear and insert design. J Orthop Sports Phys Ther 29:747-755, 1999.)*

Considering the amassed results of bench and clinical studies, there is considerably less correlation than one would expect for the hypothesis that shock attenuation reduces injury. At best it would appear that improvement in a shoe's shock-attenuating characteristics can decrease vertical impact-related lower extremity injuries and can change the pattern, but not the overall incidence, of overuse injury.

Alignment and Control

As noted in the chapter sections on the causes of injury to the knee, lower leg, and foot and ankle, there is a suspected association between positional abnormalities in the feet and injuries occurring in other areas. As more attention is focused on sports and their attendant injuries, many workers have sought an explanation for the causes of these injuries and a method of treatment that would allow continued participation in sports. The running shoe industry has capitalized on this proposed association between injuries and the feet and has developed entire shoe lines as well as specific shoe features based on the idea of providing improved "control" for the foot. Biomechanical theories have provided the rationale for this development as well as a method of treatment using prescription orthoses. The concept is that there is an ideal functional position for the various articulations of the lower extremity. Theoretically, when the foot is in a properly balanced position (achieved by having the subtalar joint in a neutral position), the foot has the greatest ability to adapt to the stresses placed on it in weight-bearing. In this subtalar neutral position, the maximal amount of inversion and eversion is available and can be used to dissipate the forces of weight-bearing and to transfer load properly to other areas of the lower extremity.

Alignment has particular implications for problems about the knee because pronation through the subtalar joint results in internal rotation of the tibia. Excessive pronation has therefore been implicated as a causal factor in anterior knee pain, [179] [180] [181] [182] [183] [184] [185] iliotibial band syndrome, [167] [179] [183] [184] [185] [186] pes anserinus bursitis, [155] and popliteal tendinitis (see Chapter 25C). [179] [183] [187]

The problems attributed to excessive pronation have not been limited to the knee. In the leg, ankle, and foot, excessive pronation has been associated with posterior tibial tendinitis, [179] [181] [184] [188] [189] [190] overuse syndromes in runners, [181] [191] medial tibial stress syndrome, [190] [192] tarsal tunnel syndrome, [193] [194] cuboid syndrome, [195] [196] plantar fasciitis, [181] [184] [197] Achilles tendinitis, [157] [179] [181] [190] metatarsalgia, [162] and stress fractures. [198] [199] From this listing of virtually every known medical condition affecting the lower leg, it should become obvious to the astute reader that excessive pronation is a seemingly disastrous condition for the athlete. As such, it is natural to expect a plethora of laboratory and clinical studies offering scientific confirmation of the relationship between excessive pronation or rearfoot instability and these pathologic conditions. Unfortunately, the few studies that have addressed this question have failed to provide a conclusive answer.

Although most studies that have been done on the use of rearfoot control features and orthotic devices have focused on the advantages of these features in the treatment of the pronated foot, shoe features and orthotic devices have also been used to treat the opposite foot condition—the cavus or highly arched foot. As discussed in Chapter 25A , the cavus foot type tends to be more rigid and has less available motion to dissipate the forces of weight-bearing. Consequently, it is the cavus foot that has been implicated in the production of plantar fasciitis, [179] [191] [197] [200] [201] stress fractures, [198] [199] [202] and medial tibial stress syndrome. [162] Because the cavus foot is generally more rigid and is commonly associated with a plantar flexed first ray and a varus hindfoot, orthotic support is designed to alleviate these problems. A less rigid orthosis is preferable to provide improved shock absorption and allow some degree of flexibility. Although reports have found that up to 75% of patients who have pronation-related problems benefit from the use of an orthotic device, there has been a considerably less favorable response to the use of orthoses in the cavus foot population. [181] [203] [204] [205] Clinical experience has been the source of most of the evidence supporting the use of orthotic devices for the treatment of a variety of conditions that plague the athlete, particularly the runner.

Although much of the scientific and anatomic basis for the use of orthoses can be traced to the work of Manter, [206] [207] Elftman, [206] [208] [209] [210] Hicks, [211] [212] Close, [213] [214] [215] and Inman, [71] [73] [216] it has primarily been orthotists and podiatrists who have experimented with various shapes and materials in an effort to develop a practical approach to the prescription of orthoses. The field of orthotic prescriptions has a pseudoscientific aura. This is created by many factors: erudite yet ambiguous terminology, seemingly contradictory theories, failure to establish what constitutes the normal foot (much less the abnormal), lack of recognition of normal anatomic variation, confusing concepts of what is compensated and what is not, and limited use of the scientific method in establishing the criteria for employment of orthoses and evaluation of their usefulness. [59] [98] [217] [218] [219] [220] [221] [222] [223] Faced with this conundrum, it is valuable to reflect on the former foundational work while viewing current shoe and orthotic research with a combination of skepticism and open-mindedness. One can then investigate the available experimental and clinical work, which either supports or refutes the scientific basis for prescribing orthoses or using particular modifications in athletic shoewear.

Experimental Work

Studies concerned with control of alignment and maintenance of rearfoot stability must begin by determining an acceptable indication of foot pronation. [196] Because pronation is a complicated triplane movement, it is difficult to quantify this movement with currently available techniques. Therefore, it has been generally agreed in the research community to use the degree of calcaneal eversion (valgus) as the indicator of pronation. [217] [225] [226] By using heel eversion alone, the associated abduction and dorsiflexion are discounted. [179] Nevertheless, this appears to be the most practical method and the one that has gained acceptance.

Measurement is done by observing the subject from a posterior viewpoint using reference markers on the lower leg to define its axis and a second set of markers on the calcaneus to denote its position (Fig. 25B-24). Gait analysis is then performed using high-speed film cinematography, video cameras, or optoelectronic systems to visualize the markers during each phase of gait. [199] The marker positions can then be plotted using anatomic landmarks and sent to a computer for analysis. This is the process of digitalization, from which are derived the specific angles exhibited at specific points in time in the gait cycle.

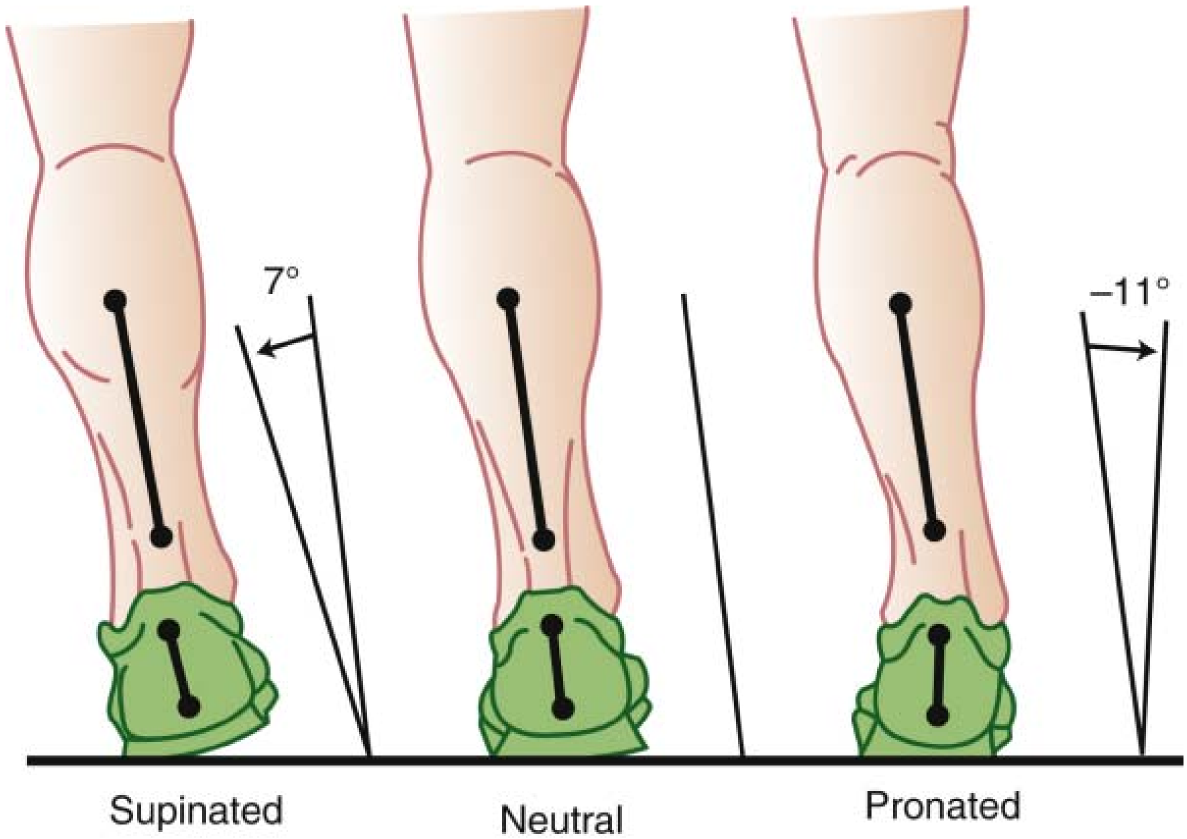

Figure 25B-24 Position of reference markers on the lower leg to define its axis and a second set of markers on the calcaneus to denote its position in kinematic analysis of gait. *(Redrawn from Sport Research Review. Beaverton, Ore, Nike Sport Research Laboratory, Nov/Dec 1989.)*

In the cinematographic system originally used, the manual plotting and calculations needed for each frame of film made this an incredibly time-consuming and laborious method subject to a certain degree of human error. Kinematic analysis provides information on a number of variables including initial Achilles tendon angle, maximal Achilles tendon angle, initial pronation, total pronation, initial pronation velocity, and so on. [95] By employing a video system, one can use a video processor to analyze the film and eliminate part of the tedious process of manual plotting. In the more sophisticated optoelectronic systems, the markers are actually infrared light-emitting diodes and are filmed by infrared-sensing cameras, thereby allowing further automation of the kinematic analysis. [197]

Regardless of the visualization method used, certain important factors must be taken into consideration. Sampling must be performed at a rate that is at least twice the frequency of the movement being analyzed, requiring a minimal rate of 200 Hz for rearfoot movement. [199] The accuracy of the data collection system must be ensured both by the equipment manufacturer and by the on-site testing facility. Calibration, marker-to-marker distance testing, optimization of the collection environment, and meticulous attention to detail are just some of the factors necessary to ensure validity in kinematic testing. [199] For example, the markers can be placed using either a relative method, wherein four markers are arbitrarily placed on the posterior foot or shoe and the posterior calf, or an absolute method, which uses standard anatomic landmarks. Furthermore, it should be remembered that testing is done in a variety of conditions including different test subjects, different speeds from walking to sprinting, different surfaces ranging from treadmill to various over-ground conditions, in shoed and shoeless conditions, in varying types of shoewear, and in shoes with or without orthoses. [189]

This background is necessary to understand some of the information provided by the studies on rearfoot control. Although it is easy to see how a determination of rearfoot position could be performed in the barefoot runner, an obvious problem exists when the heel is hidden inside a shoe. [200] How does one determine the proper position for the markers? This question has been answered by studies that have used a window in the heel of the shoe to allow visualization of the calcaneus position. [196] One such study reported by Nigg analyzed measurements using one type of shoe in three test subjects with three trials per subject. A 2- to 3-degree shift was noted between the subject's heel and the shoe itself. [95]

Because this shift was systematic, it was not believed to invalidate the test method. In a more recent study, Stacoff placed intracortical bone pins into volunteers to monitor movement coupling between shoe, calcaneus, and tibia. Apart from the difficulty of recruiting volunteers for this type of invasive monitoring, they observed considerable individual differences in coupling between these areas and suggested that we have yet to unravel the details of this complex interaction. [201]

Shoes and shoe design characteristics can have considerable effect on the kinematics of the foot in running and other sports activities including rearfoot control. Some of this effect is simply the result of displacement of the foot away from the contact surface by the shoe. It has been shown by several investigators that the Achilles tendon angle is decreased in the shoeless condition. [217] [225] [228] The total rearfoot movement and rate of pronation are also reduced in the barefoot runner. [123] [217] [225] [228] [230] This suggests that wearing shoes increases not only pronation but also other temporally related variables. [217] [225] This effect of shoewear is demonstrated in Figure 25B-25 .

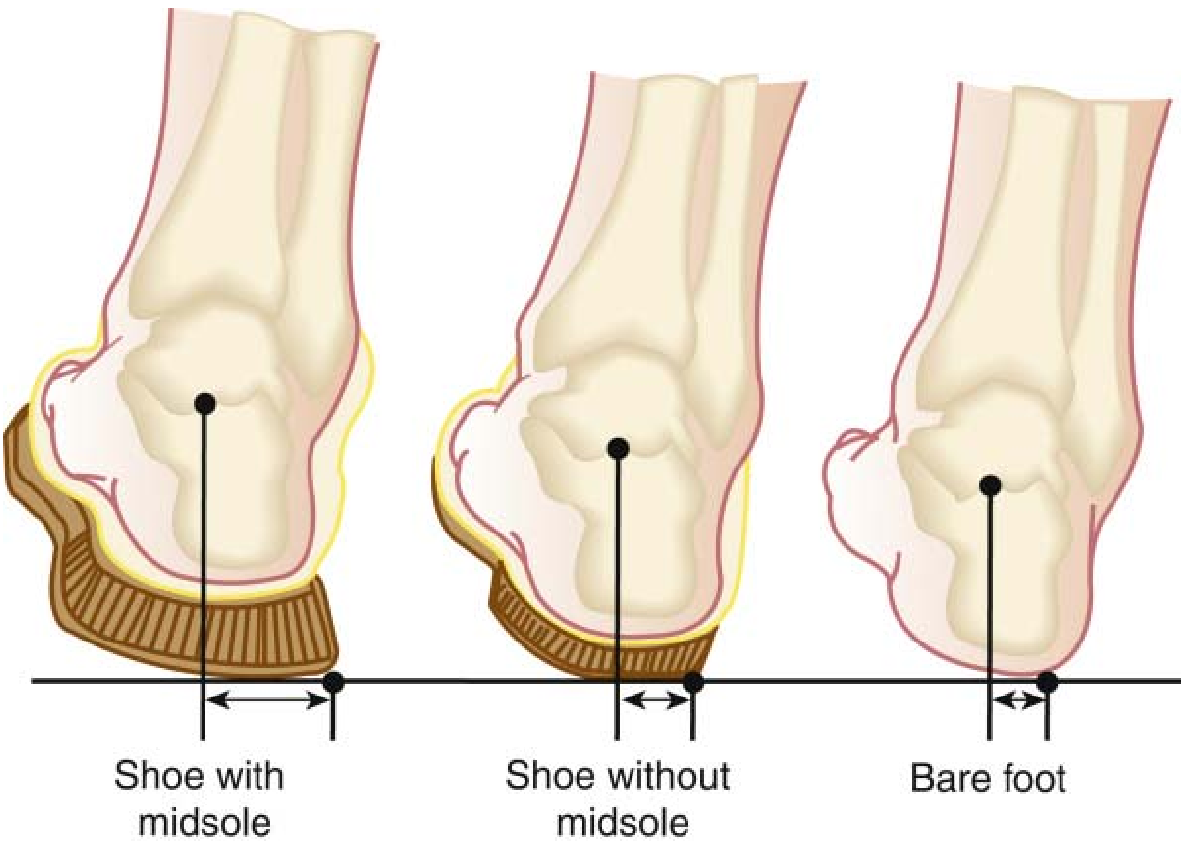

Figure 25B-25 Depiction of the effect of shoewear in increasing pronation from the barefoot condition. *(Redrawn from Sport Research Review. Beaverton, Ore, Nike Sport Research Laboratory, Nov/Dec 1989.)*

The shoe design variables that have been investigated most thoroughly with respect to kinematic effect include sole hardness, heel height, use of a heel flare, width of the midsole, and torsional flexibility. [217] [225] Sole hardness is measured in terms of durometer, a 25 shore A durometer sole being considerably softer than a 45 shore A durometer sole. Clarke and associates showed that the softer the sole, the greater the degree of maximal pronation and total rearfoot movement. [129] A similar study by Nigg found exactly the opposite result, the softer sole having less total pronation. [95] A later study by Nigg and colleagues confirmed their previous results, and further elaborated that the total foot eversion was roughly double for hard versus soft inserts. [143] The harder inserts allowed for more individual variation of movement and did not force a preset movement pattern to the foot. Despite significant interindividual variance, the subjects with a flexible foot were more likely to have diminished tibial rotation. This study concluded that individual variation must be taken into account when matching feet to inserts. Because other factors such as overall shoe stiffness, shoe construction techniques, and variations in sole geometry can have significant effects on the kinematic function of the foot, it is easy to surmise how different results can be forthcoming from different laboratories when performing similar tests. [189]

Potential confusion in test results also occurs in the relationship of heel height to rearfoot control variables. Bates and coworkers found that raising the heel height in relation to the forefoot could reduce both maximal pronation and the period of pronation. [200] Stacoff and Kaelin analyzed the effect of heel height over the range from 18 to 43 mm and found the same effect in the range from 23 to 33 mm but the opposite effect on pronation at the upper and lower ends of heel height. [203] To cap things off, Clarke and associates discovered no significant effect of varying heel heights between 10 and 30 mm. [129] It is apparent that there are inconsistencies in the studies generating dissimilar results and adding to an already confusing picture.

With the introduction of the New Balance 305 Interval shoe in 1975, a new variable appeared in the shoe-foot control equation. [9] The flared heel, seen originally in this shoe, was added primarily to improve the stability of the shoe by widening the base of support when the foot was on the ground. Following the principle that “if a little bit is good, then more will be better,” Nike introduced a shoe in the late 1970s that had a full 1-inch lateral heel flare. Unfortunately, a negative byproduct was quickly perceived in the increasing incidence of lateral knee pain that occurred with this shoe. [9] Nigg and Morlock discovered the underlying problem with the flared heel in a scientific study of 14 runners reported in 1987. [156] Using three shoes identical except in their degree of lateral heel flare, the authors noted increased initial pronation with wider flare but no difference in total pronation or impact forces at heel strike. Increasing the lateral heel flare increases the lever arm on the axis of motion across the subtalar joint, resulting in earlier initiation of a pronating movement, greater rearfoot angulatory velocity, and an increased initial Achilles tendon (touchdown) angle. A similar study by Nigg and Bahlsen confirmed this result but indicated that heel flare was less important when a softer midsole was used. [204] This study supported the earlier work of Cavanagh on a rounded heel design by demonstrating a reduction of more than 15% in the time of peak force with use of this modification. [217] [232]

Two shoe design characteristics that have been introduced recently are related to midsole width and torsional flexibility. Both have been supported by shoe companies—Nike promoting the wider midsole and Adidas promoting running shoes with greater torsional flexibility. [68] [224] [233] According to the results provided by the Nike Sports Research Laboratory, reductions in maximal pronation and maximal pronation velocity can be obtained by increasing sole width to about 90 mm. [197] Torsional flexibility is a new concept related to evaluating the foot in three dimensions rather than by the traditional two-plane analysis of rearfoot inversion and eversion. Work by Stacoff and colleagues has demonstrated that large torsional movement occurs between the forefoot and hindfoot in the barefoot state. [202] This three-dimensional linkage between the forefoot and hindfoot is mediated through the tarsometatarsal (Lisfranc’s) and transverse tarsal (Chopart’s) joints and in a shoe can be influenced by shoe sole construction factors.

Shoe soles that are stiff in the longitudinal direction restrict the torsional movement normally produced when the forefoot adapts to the ground. Adidas has taken this concept and developed an entire line of running shoes with “a unique construction that controls the natural torsion, or twisting of the foot.” [205] They also claim “the foot moves as nature intended it to. The natural twisting of the front part of the foot is controlled so that it no longer strains joints and tendons. Performance greatly improves, whereas injury and muscle strain are substantially diminished.” [205] This seems to be an exaggerated claim for a shoe design feature that affects a foot movement that itself is not entirely understood, much less how it is changed by the shoe design and how that relates to other kinematic values. More time and research will be necessary to establish the importance of such factors as shoe width and torsional flexibility because they appear to be mutually exclusive based on examination of the shoes produced with this technology (Fig. 25B-26).

Figure 25B-26 Example of wide versus narrow midsole widths used in the Nike (*right*) and Adidas (*left*) running shoes. *(Photograph by Thomas O. Clanton.)*

The fabrication of orthotic devices to control rearfoot stability, thereby treating various disorders of the lower extremity, has a rather short history. In 1962, Rose presented one of the earliest studies on the ability of a position-modifying device (the Schwartz meniscus) within the shoe to alter the rotation of the lower leg. [206] Sheehan, writing in the Preface to *The Foot Book* by Harry Hlavac, traced the origin of biomechanical therapy to the early 1970s and the development of sports podiatry. [207] He theorized that the increasing problem with anterior knee pain in runners was the key to the development of orthotic devices for the foot. The ability of a shoe insert to provide benefit when a surgical solution was not forthcoming led to "the ascendancy of orthopedic medicine." [207] Ascend it did, as orthoses for sports scaled new heights in cost, in numbers, and in varieties of conditions for which they were recommended. At present, many athletes and coaches think that the orthotic device is standard athletic equipment. Athletes come to the clinician with one sort of complaint or another and a self-made diagnosis requesting "orthotics." In this environment, it is critical that individuals involved in the field of sports medicine understand not only the biomechanical principles behind the use of orthotic devices but also the relevant literature from gait laboratory studies.

As in the evaluation of shoewear, kinematic analysis of various parameters is essential to the documentation of orthotic effects on gait. According to Stacoff and Luethi, it was Nigg who first determined the effect of shoe inserts on gait using film analysis. [31] Nigg's study concluded that a properly functioning insert should change gait characteristics toward values consistent with those of normal feet. Cavanagh and colleagues presented a paper at the 1978 meeting of the American Orthopaedic Society for Sports Medicine in Lake Placid, New York that showed a reduction in maximal pronation and maximal velocity of pronation in runners who used a properly placed felt shoe pad as a medial support.[189] Nigg and associates reported in 1986 on the use of various conditions and positions (from no support to anterior to posterior positions) for medial supports within the shoe, demonstrating that placing the pad (elastic cork in this instance) more posterior reduced initial pronation and maximal pronation to a lesser extent (Fig. 25B-27).[208] Taunton and coworkers found a similar reduction in maximal pronation with the use of an orthosis.[209] These studies support the ability of an orthosis to affect kinematic parameters.

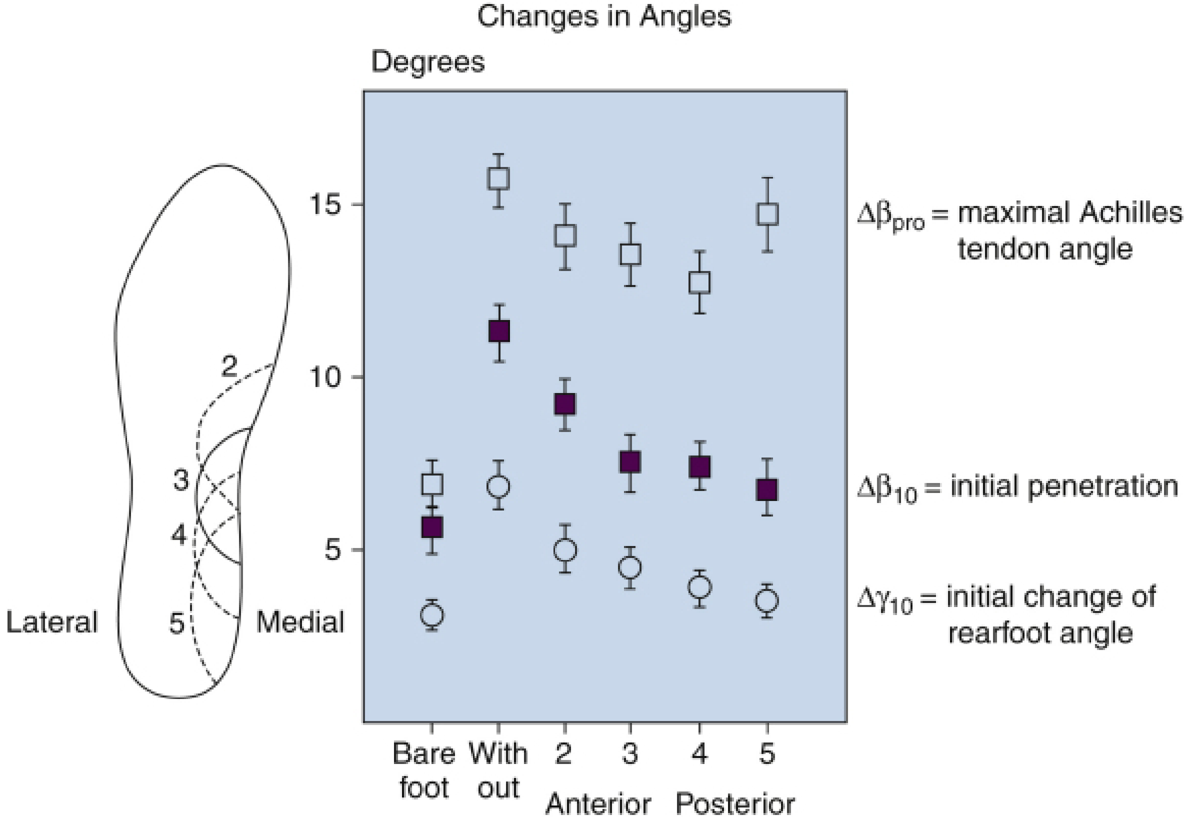

Figure 25B-27 Graphic demonstration that placing an insert more posteriorly reduces initial pronation and maximal pronation. *(From Nigg BM: Biomechanics of Running Shoes. Champaign, Ill, Human Kinetics, 1986. Copyright © 1986 by Benno M. Nigg.)*

Further work in this area was provided by Smith and colleagues, who studied 11 well-trained runners using soft or semirigid orthoses while running on a treadmill at a 6- and 7-minute mile pace. [238] [239] They found that calcaneal eversion was reduced in their subjects, who had an average rearfoot varus posting of 4.2 degrees. This reduced maximal pronation from a mean of 11.3 degrees in controls to 10.5 degrees for the soft orthosis to 10.1 degrees for the semirigid device, only a 1% change. Maximal velocity of pronation was reduced from a mean of 540 degrees per second in controls to 430 degrees per second for the soft orthosis to 464 degrees per second for the semirigid group, an 11% change. Although the reduction in maximal pronation is quite small compared with the expected result for this amount of posting, the reduction in pronation velocity may play a larger role in symptomatic relief provided by orthoses—an effect that can be achieved as adequately by the less expensive soft orthosis as by the semirigid orthosis. Although these studies further augment the role of orthoses in adjusting kinematic variables, other authors have had results that are less supportive.

One of the most widely recognized studies of foot orthoses and their effect on gait is that done by Bates and associates in 1979.[163] This study failed to confirm a significant reduction in maximal pronation with a custom-molded orthotic device compared with shoes in six symptomatic runners. The study compared its results with control values obtained in a previous study of 10 asymptomatic runners.[200] Their results also suggested that an increased velocity of pronation occurred with the orthotic device in contrast to the findings of the previously mentioned studies. Rodgers and LeVeau produced a similar study using 29 runners fitted with custom-made semirigid orthoses made from polypropylene.[212]

Subjects ran in their own shoes on an outdoor track and were filmed with 16-mm film at 120 frames per second. Runners completed three randomly sequenced runs in the following conditions: (1) barefoot, (2) shoe without orthosis, and (3) shoe with orthosis. Values obtained for maximal pronation, angulatory velocity of pronation, and time spent in pronation were not significantly different among the three conditions. There was, however, a trend toward decreased maximal pronation and time in pronation in the shoe with orthosis group. Smith and colleagues pointed out some of the flaws in the various studies related to inadequate control of confounding variables such as type of shoe, type of orthosis, and physical measurements of test subjects.[211] Given the number of variables that are being analyzed in these gait studies of running, there should be little surprise that some discrepancies exist. More studies will be necessary to determine which of the variables is important in relation to the clinical situation and the prevention or treatment of injuries. Progress in this field would proceed at a more rapid rate if there could be some universal agreement on terminology, methods of measurement, normal values, and sharing of information without commercial self-interest.

Clinical Work

Although there is a widespread belief in sports medicine circles that biomechanical abnormalities are a significant causal factor in lower extremity injuries, clinical studies have had paradoxical results. The military formerly believed that the pronated flat foot was more susceptible to injury, but recent work has disproved this belief. DeVan and Carlton suggested as early as 1954 that stress fractures of the metatarsals were equally common among pronated, normal, and cavus foot types.[213] Bensel[214] and Gilbert and Johnson[215] reached the same conclusion from their research. Further confirmation of this conclusion has come from the Israeli Army study of 295 recruits, which indicated that the low-arched foot might even protect against the development of stress fractures.[174] The overall incidence of stress fractures in their study was 39.6% in recruits with a high arch, 31.3% in those with average arch height, and 10% in those with a low arch. It is important to note that in these studies, the criteria for defining arch height were subjective, and determinations were made in a non–weight-bearing position.

A continuation of the Israeli Army study was reported in 1989 with a more quantitative determination of the longitudinal arch based on radiographic analysis.[171] This study reported a higher number of metatarsal stress fractures in recruits with low arches, whereas the incidence of femoral and tibial fractures in these recruits was less than that in those who had a higher arch. This result seemingly contradicts the findings of the previous studies. It should be noted that both of the Israeli Army studies excluded those with marked pes cavus or pes planus from the outset. A subsequent Israeli Army study examined the use of custom soft or semirigid functional orthoses and suggested a benefit in reducing stress fractures; however, fewer than half the recruits completed the study using their assigned orthoses (25% dropped out because of dissatisfaction with the orthosis). Nearly 50% of the arriving recruits were already using orthoses (30% custom-made).[62] All of this adds further confounding variables complicating interpretation of the various clinical studies and extrapolation of an association between biomechanical parameters and symptomatology.

Among runners, it is evident that a number of risk factors have been implicated in the production of injuries (Table 25B-11).[244] [245] [246] Because so much emphasis is placed on the relationship between overpronation and injury, it is natural to expect confirmation of this in the epidemiologic surveys of running injuries. One of the first studies to focus attention on the role of pronation in runners was that of James and colleagues in 1978.[153] They reported a 58% incidence of pronation in 180 patients evaluated for a variety of complaints. It is important to note that one cannot draw the conclusion that most injured runners have pronated feet because there is no way of determining the overall incidence of pronation in the running population. Despite this fact, it is our opinion that this study was instrumental in focusing the attention of sports medicine specialists on the role of alignment in sports injuries. Unfortunately (or perhaps fortunately for the overpronator), no such relationship has been clearly determined in epidemiologically valid studies.[217] One of the most comprehensive studies of running injuries is the Ontario cohort study, which included 1680 runners. Anthropometric measurements including femoral neck anteversion, pelvic obliquity, knee and patella alignment, rearfoot valgus, pes cavus and pes planus, somatotype, and running shoewear pattern were made in 1000 of these runners. The study concluded that "none of the anthropometric variables measured was significantly related to risk."[218] The most consistent risk factor for a running-related injury is weekly training mileage, and this has been proved in study after study.[244] [245] [246] [247] [248] When weekly distance reaches 64 km or 40 miles per week, the risk for injury increases by 3 times.[244] [246] Additionally, no correlation has been shown between shoe characteristics (e.g., varus wedge or waffle sole) or shoe expense and injury reduction in these studies.[172] [246] [249] Given all this information, what role does rearfoot stability provided by shoes or orthoses play in the prevention or treatment of athletic injury?

TABLE 25B-11 -- Risk Factors in Running Injuries

Characteristics of Runners	Characteristics of Running	Characteristics of Running Environment
Age	Distance	Terrain

Characteristics of Runners	Characteristics of Running	Characteristics of Running Environment
Gender	Speed	Surface
Structural abnormalities	Stability of pattern	Climate
	Form	Time of day
Body build	Stretching, weight-training, warm-up and cool-down	Shoes
Experience		
Individual susceptibility		
Past injury		

From Powell KE, Kohl HW, Caspersen CJ, et al: An epidemiological perspective on the causes of running injuries. Physician Sportmed 14:100-114, 1986.

Although the scientific approach of systematically establishing a basis for the use of orthoses has failed, there does appear to be some practical basis for prescribing orthoses to injured athletes. Seventy-eight percent of the injured runners in the oft-quoted series of James and colleagues reported some benefit from the use of either rigid or flexible orthoses.[153] D'Ambrosia subdivided patients by diagnosis and analyzed the numbers who benefited from the use of an orthosis. The subgroup with pes planovalgus had the most benefit (90% of patients), whereas the subgroup with pes cavus had the least benefit (25% of patients) (Table 25B-12).[175] Another study from the Louisiana State University Medical Center Runner's Clinic reported a 72% improvement in symptoms with the use of orthoses, although these were prescribed for only 10% of their total patient population.[222] Blake and Denton surveyed 180 patients and reported that 70% claimed that they were "definitely helped" by treatment with functional foot orthoses.[223] Donatelli and coworkers used a similar questionnaire survey of patients to document a 90% relief of pain in patients treated with semirigid orthoses alone.[224] A recent multicenter study compared custom-made and prefabricated orthoses in the treatment of plantar fasciitis. Patients were treated with stretching alone or stretching with one of four different orthoses, three of which were prefabricated and one of which was custom-fitted. Stretching alone produced improvement in 72%, whereas those who combined stretching with a silicone heel cup showed the most improvement (95%). Subjects who stretched and used a custom orthosis improved only 68% of the time. Stretching alone or combined with a prefabricated inlay of any of the types tested was significantly more likely to lead to improvement than use of a custom orthosis with stretching.[225] Santilli and Candela found that 100% of 40 athletes complaining of metatarsalgia were relieved of symptoms by a custom-molded polyurethane insert that enabled them to resume sports within 3 weeks.[226] Kelly and Winson found that a prefabricated foam inlay with the ability to individualize the placement of the metatarsal support was more effective in relieving metatarsalgia than a prefabricated silicone orthosis, which could not be "semi-customized."[145] A study by Gross and associates reported that 75% of 347 long-distance runners had complete resolution of or great improvement in their symptoms with various types of orthoses (mostly flexible).[176] Similar success rates of 70% to 90% have been reported by other clinics using orthotic prescriptions for various athletic injuries.[59] [204]

TABLE 25B-12 -- Effectiveness of Orthoses

Diagnosis	No. of Patients (%)	Percentage Improved with Orthosis
Posterior tibial syndrome	55 (27.5)	77
Pes planovalgum	23 (11.5)	90
Metatarsalgia	30 (15)	87
Plantar fasciitis	20 (10)	
Calcaneal spur	18 (91)	81
Iliotibial band tendinitis	14 (7)	66
Cavus foot	13 (6.5)	25
Leg-length inequality	10 (5)	NA
Chondromalacia patellae	6 (3)	NA
Achilles tendinitis	4 (2)	NA
Miscellaneous	7 (3.5)	NA

Diagnosis	No. of Patients (%)	Percentage Improved with Orthosis
Total	200 (100)	

From D'Ambrosia RD: Orthotic devices in running injuries Clin Sports Med 4:611-618, 1985.

NA, not available.

Based on this evidence that orthoses can improve symptoms and generate documentable alterations in defined kinematic variables, one would assume that a positive attitude exists supporting their continued use. The inconsistency is that "different schools of applying inserts seem to have equal success despite the fact that their inserts look quite different." [31] Support for the value of expensive running shoes (incorporating some of the same principles as orthoses) to treat injuries or symptoms is harder to generate. [221] Injury rates for runners have shown no decrease during the past 3 decades of improved shoewear technology, although many factors are at work in this statistic. [37] [244] [245] [246] [248] [249] [255] [256] [257] These include the entry of the less physically fit into, and an aging of, the running population. Comparative studies of injury rates in runners have been performed for two separate periods by the University of British Columbia Sports Medicine Clinic. [255] [256] The results indicate a reduction in foot and ankle injuries in the more recently studied group but an increase in knee injuries. This finding begs the question originally proposed by Cavanagh of whether the increasing technology applied to shoewear has actually contributed to this change. [9] The work of the Human Performance Group in Quebec appears to substantiate the idea that increasingly well-cushioned and controlled athletic footwear has tended to produce injuries rather than protect the athlete from them. [143] [144] [145] [146] [147] [257] Perhaps an enterprising shoe company will spend part of its advertising budget on a cooperative study with a runners' clinic to produce a study that will help answer the question of whether these technologic advancements are helping or hurting. Realistically, the number of variables involved presents an almost insurmountable barrier to epidemiologically valid studies of this nature. Although much has been accomplished in this field in a relatively short time, there are still many unanswered questions and ample opportunities for the inquisitive researcher.

Energy Return

Although several shoe companies either state or imply the ability of their products to return energy to the athlete, little support exists for this in the scientific literature. [105] [258] [259] Any shoe that would return energy of a beneficial nature to its user would need to return this energy at the correct location, time, and frequency. Unfortunately, materials used to increase shock absorption tend to be poor for energy return. [260] [261] Additionally, the location of maximal possible energy storage (the rearfoot) is not ideal for use of any returned energy. [259] [261] Nigg and Segesser have demonstrated that running with running shoes actually requires 3% to 5% more energy than running barefoot. [231] They have calculated that the maximal amount of returnable energy from shoewear is less than 1%, which is not even sufficient to overcome the energy requirement of accelerating the shoe itself, or to make up the additional vertical movement added by the shoe. [231] Consequently, claims of a shoe's ability to improve or alter performance through return of energy to its wearer should be viewed with skepticism.

It appears that energy considerations may be better met through limitation of energy loss. [233] Stefanyshyn and Nigg examined the effect of increased bending stiffness in the midsole, through insertion of a carbon-fiber plate, on jump height and loss of energy through the metatarsophalangeal joint. [233] They surmised that stiffening the metatarsophalangeal joint would decrease the amount of energy lost and increase jumping performance. The former was indeed the case for both running and jumping, and vertical jump height was increased by 1.7 cm in the stiffest shoe tested. No comment was made on the potential for increased risk for injury to the Achilles tendon complex, as has been suggested in stiff-soled designs. [34]

Friction and Torque

This aspect of shoewear is thoroughly covered in Chapter 25C and will not be repeated here. Obviously, shoe design features carry major significance in this area. Nigg and Segesser suggested that frictional loads on the human body are of greater importance in the production of sports-related pain and injuries than impact loads. [234] The interested reader is referred to the previously noted chapter for a discussion of friction and torque related to athletic shoewear.

PROPER FIT AND SHOE PURCHASE DECISIONS

Although biomechanical abnormalities have caught the attention of both the athlete and the sports medicine practitioner in recent years, these problems are much less likely to be the source of day-in and day-out problems compared with the difficulties created by poorly fitting shoes. It is the poorly fitted shoe that creates such commonplace annoyances as blisters, ingrown toenails, certain forms of calluses, metatarsalgia, nerve compression syndromes, “black toes,” corns, and a variety of other unnecessary ills. Most of these conditions are preventable with a working knowledge of shoe-fitting techniques.

The most classic case of a footwear-related problem is the bunion. Although there are certainly individuals who have an anatomic predisposition to develop this condition, it has become evident from accumulated scientific research that the improperly fitted shoe is of major significance in the causes of bunions, or hallux valgus. Hoffman’s study of barefooted peoples demonstrated “progressive characteristic deformation and inhibition of function” in people who wore shoes compared with those who remained shoeless.[235] Kato and Watanabe pointed out the relationship between the development of hallux valgus as a clinical entity in Japan and the introduction of the Western-style shoe to replace the traditional geta sandal.[21] Other studies have reached similar conclusions, but this has had little effect on the shoe manufacturing industry or the consuming public, who continue to believe that the dainty foot is the most attractive foot.

Shoe fit is governed primarily by sizing systems used in the manufacturing process. The English system, originated in 1324, was based on the length of barleycorns (one barleycorn equals 1⁄3 inch).[50] [102] Today there are numerous systems of sizing in use around the world, making it difficult to fit the foot based on size alone. The most common systems are the English, the American standard, the Continental sizing, and the centimeter systems.[22] When determining the proper size to try at the shoe store, it is traditional to use a device such as the Brannock, the Ritz, or the Scholl to size the foot. These devices measure the overall length of the foot, the position of the metatarsal break (related to arch length and toe length) and the girth or width of the foot. The latter is designated by a letter in most circumstances, with AAA being the most narrow, C and D being standard medium widths, and EEE being the widest.[22] This form of width sizing is relatively standard and corresponds to various last width standards as seen in Table 25B-13 and Figure 25B-28 .

TABLE 25B-13 -- Last Width Standards (Generally Accepted)

American Men’s			Inches (cm)		Inches (cm)		Widths Available		
8A			8½	(21.6)	8 11⁄16	(22.1)	AAAAA	A	E
8B	9A		8¾	(22.2)	8 15⁄16	(22.7)	AAAA	B	EE
8C	9B	10A	9	(22.9)	9 1⁄8	(23.2)	AAA	C	EE
8D	9C	10B	9 1⁄8	(23.2)	9 3⁄8	(23.8)	AA	D	EEE
8E	9D	10C	9¼	(22.5)	9 7⁄16	(24.0)			EEEE
8EE	9E	10D	9½	(24.1)	9 11⁄16	(24.6)			
8EEE	9EE	10E	9¾	(24.8)	10	(25.4)			

From Cheskin MP: The Complete Handbook of Athletic Footwear. New York, Fairchild Publications, 1987.

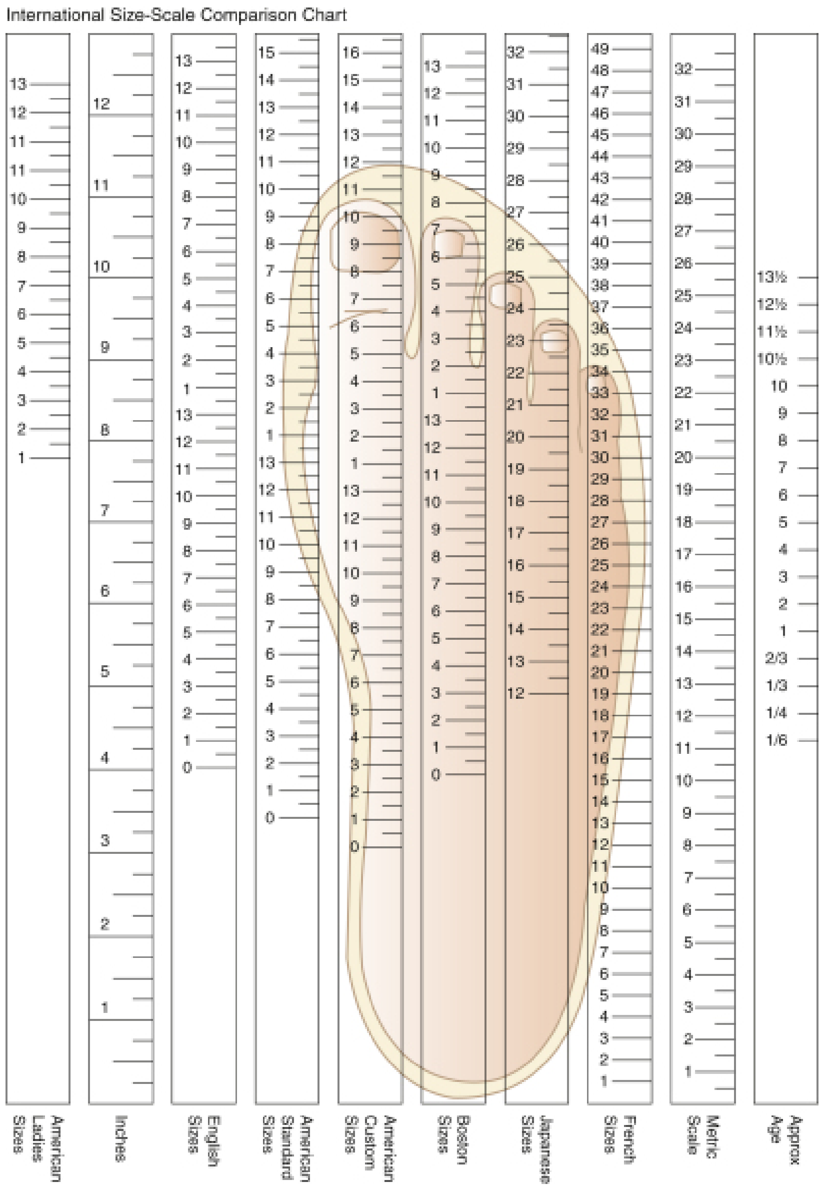

Figure 25B-28 International size scale comparison chart. *(From Cheskin MP: The Complete Handbook of Athletic Footwear. New York, Fairchild Publications, 1987.)*

In athletic shoewear, it is uncommon to find width sizing available. The New Balance Company has made width sizing a relatively successful marketing strategy in the running shoe market. At present, most athletic shoes are made on a standard D last for men and a C last for women. Adjustability for width is often incorporated into the lacing system. A variety of lacing strategies can be used to accommodate a range of temporary or permanent foot shape considerations, and these are demonstrated in Figure 25B-16 . [32] Additionally, newer, round “laceroni” shoelaces have found their way into shoes. These slide more easily than flat laces when used with the newer loop and web eyelet designs. Pressure is thus more evenly distributed on the dorsal foot than with flat laces, and problems with kinking are minimized. Nevertheless, these lace designs have the slight disadvantage of untying more easily. [56] Although women’s shoes are made on a narrower last than men’s, many athletic women have a foot shape more like a man’s and therefore are more properly fitted by the use of a man’s shoe.

The manufacturing process has a major impact on how the shoe fits, specifically with regard to the exact last that is used. Different manufacturers use different lasts, which naturally affect the size and fit of the shoe and cause the discrepancies seen in the fit of the same size shoes from different companies. It is for this reason that when an athlete finds one particular make and model of shoe that is satisfactory, he or she will not vary from it except in extraordinary circumstances. This has led to such practices as a famous athlete endorsing a particular company’s shoes while wearing the shoe of a different company, or having his shoes modified by a logo change to resemble the shoes being endorsed. [22] Clearly, the proper fitting of the shoe is indispensable to maximal performance in the eyes of many sports participants.

Because fit and comfort are so critical, it would seem that the popular shoe surveys would try to determine how to achieve proper fit for the benefit of their readership. Evidently, this is a matter of individual preference and is not quantifiable in the same sense that shock absorption and pronation control can be analyzed. There are too many variables among individuals and even major differences between right and left feet in the same individual (Fig. 25B-29). Comfort and fit are a matter of individual preference—some people like a snug fit, whereas others like a more loosely fitting shoe; some like the feel that a soft insole provides, whereas others do not like this sensation; and some like the feel of a higher heel, and others cannot tolerate this. In our own dealings with patients, it has become evident that although we can make suggestions based on reasonable empirical considerations, it is impossible for us to predict with accuracy which shoe a specific patient with a specific foot type will select as the most comfortable.

Figure 25B-29 Right and left feet with different shapes and shoe fit requirements. *(Photograph by Thomas O. Clanton.)*

It should be apparent from the earlier discussion that much of what goes into the fit and comfort of the shoe is derived from the lastmaking process (see the earlier section entitled "Anatomy of the Sports Shoe"). Six measurements are taken into consideration in this process: the ball girth, the waist girth, the instep girth, the long heel girth, the short heel girth, and the heel-to-toe length or stick length (Fig. 25B-30). [22] Other important specifications are the toe spring, the heel breast, the heel height, and the heel pitch. [50] [59] These measurements are then used by the lastmaker to turn a piece of unfinished rock maple or other wood into the finished last. The variability of design produces endless possibilities for fit. Stacoff and Luethi calculated that if 20 different elements in shoewear construction were varied by five systematic steps, more than 95 trillion test shoes would be produced. [31] Traditionally, a great deal of craftsmanship has gone into the area of lastmaking. Now, with computer technology, standardization has been introduced that will ideally lead to better fit and greater comfort.

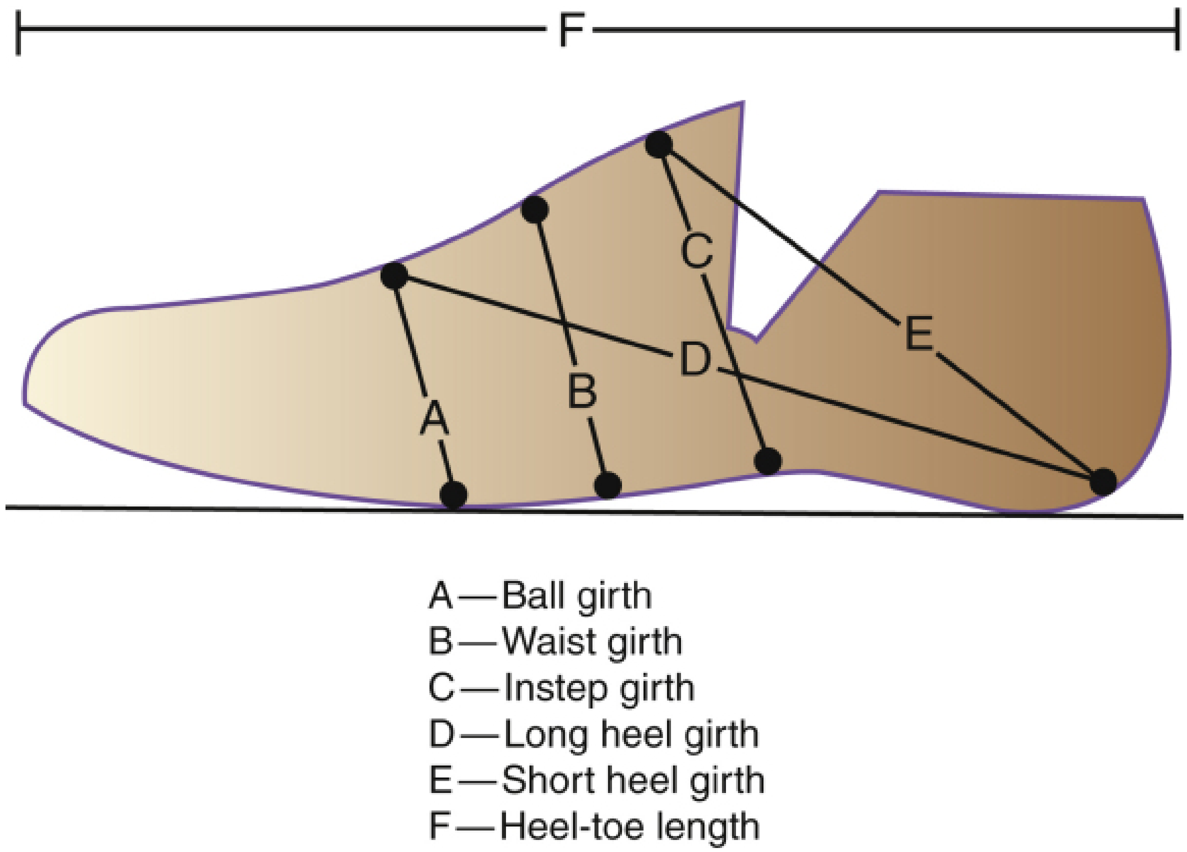

Figure 25B-30 Measurements used in proper fitting of a shoe include the ball girth, the waist girth, the instep girth, the long heel girth, the short heel girth, and the heel-to-toe length or stick length. *(Redrawn from Cavanagh PR: The Running Shoe Book. Mountain View, Calif, Anderson World, 1980.)*

In determining the proper fit of a pair of shoes, it is commonly believed that the shoe may be somewhat uncomfortable on the first fitting but can then be "broken in" over time. Conversely, it is often thought that the shoe that feels comfortable in the store the first time will then fit comfortably for the rest of its natural life. Unfortunately, both of these concepts are subject to error. For these reasons, it is important to approach thoughtfully the purchase or fitting of a pair of athletic shoes. The decision to purchase a particular shoe should be based on the quality of construction (brand name may or may not be a factor in this). One should avoid buying a shoe simply because it is made by a specific company or endorsed by a certain athlete. Many universities and professional sports teams receive shoes at considerable discounts or even have them donated for the publicity derived by the shoe company. In this situation, it is not uncommon to encounter fitting problems in certain athletes who simply do not have a foot that fits well into the selected shoe. Also, some athletes' feet require shoes with greater stiffness or other specific characteristics that are not available in the offered shoes. Rather than forcing athletes to adjust to the shoe for the sake of conformity, it is preferable to let them participate in the selection of a shoe that they know will fit well and that will allow them to perform to the best of their ability. Although this is seldom possible in intercollegiate or professional sports, the sports medicine specialist should be sensitive to the relationship between poorly fitting shoes and certain complaints of the athlete as well as the need to switch to a more supportive shoe when appropriate.

There are three basic determinations to be made in the fitting of shoes. One must first ascertain that the length is correct. This can be guided by the "rule of thumb" test performed by pressing on the end of the shoe while the wearer is applying full weight. There should be between half and a full width of the examiner's thumb between the end of the longest toe and the end of the shoe (Fig. 25B-31). It is essential to note that for many people, the second toe is longer than the great toe. Another important test for length is to have the athlete kick the plantar forefoot into the ground as he would in a sudden stop. If the toes jam uncomfortably into the end of the shoe, the shoe will not last long, the toes will suffer, or the shoe will be shelved. It is wise to do this test before the shoe is purchased. The next step in the fitting process is to determine proper width. The "pinch" test helps with this. The individual stands in the shoe while the examiner tries to pinch a small amount of material in the upper between the thumb and index finger across the forefoot of the shoe (Fig. 25B-32).

Figure 25B-31 Demonstration of proper fitting of a shoe for length. Between half width and a full width of the examiner's thumb can be placed between the end of the longest toe and the end of the shoe. *(Photograph by Thomas O. Clanton.)*

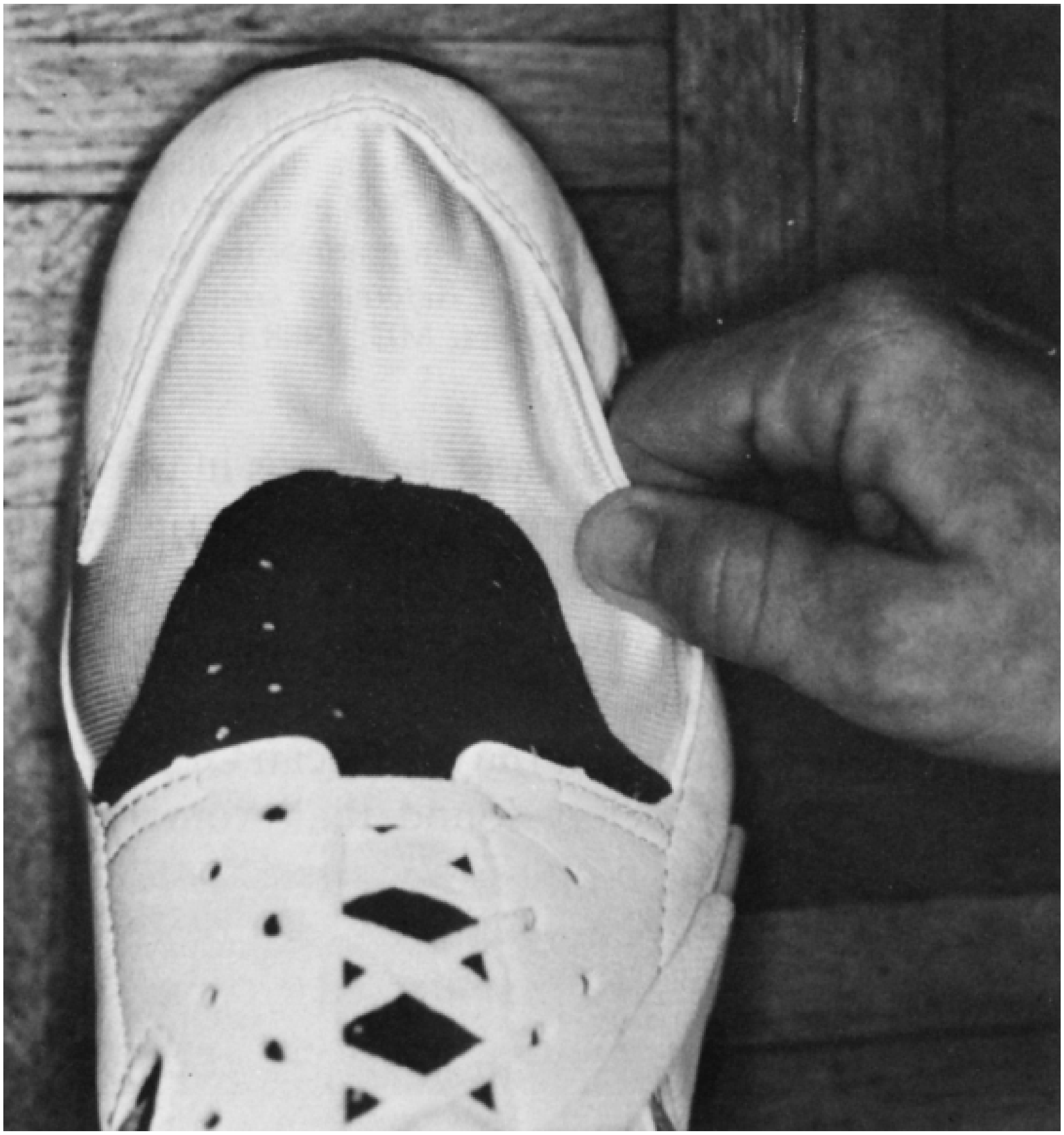

Figure 25B-32 Demonstration of the "pinch" test. The individual stands in the shoe bearing weight while the examiner pinches a small amount of material in the upper between the thumb and index finger across the forefoot of the shoe. *(Photograph by Thomas O. Clanton.)*

The final test is a determination of the flex point of the shoe in relation to the metatarsal break of the foot. If the shoe does not have the proper degree of flexibility in the appropriate location, one can expect problems (Fig. 25B-33). In the past, the flexibility test was one of the common tests used by *Runner's World* in their annual shoe survey. [9] The shoe was bent through a 40-degree range, and the force required to do this was measured with a strain gauge. It has been assumed that the less force required, the better, because this is force that must be generated by the runner. There is a fallacy with this assumption, however. It has become evident in shoes designed for artificial surfaces that the overly flexible shoe can predispose the wearer to sprains of the metatarsophalangeal joints, such as turf toe (see Chapter 25C for further details). [236] Furthermore, certain athletes may have underlying problems such as hallux rigidus or plantar fasciitis that are aggravated by the overly flexible shoe. Joseph, in a study from the 1930s, found that the average male needs only 30 degrees of flexibility in the first metatarsophalangeal joint for normal walking and that the stiffer soled shoe provided better support for the foot. [237] The findings documented in the earlier section on "Energy Return" also support increased stiffness in the shoe's sole. One can quickly realize that all the answers are not available on this aspect of comfort and fit. This is one of the many areas in which a great deal of individual variability exists in both objective and subjective factors.

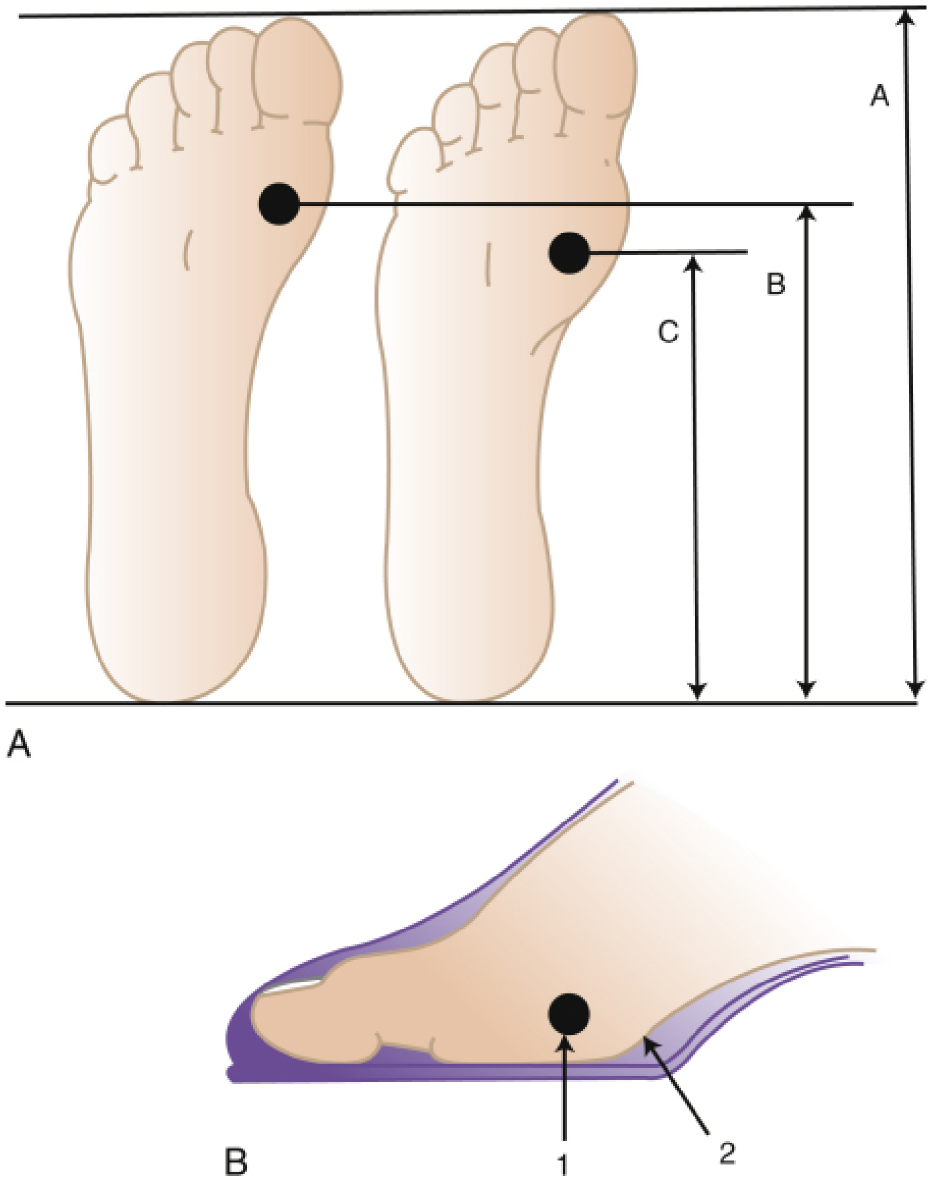

Figure 25B-33 Demonstration of differences in metatarsal break point seen in feet of the same length and how this affects shoe fit. **A,** Both feet have the same overall length (line A) but different heel-to-ball lengths (lines B and C). **B,** Improper fit of the heel-to-ball length can create functional problems in the shoe. Point 1 represents the center of rotation of the first metatarsophalangeal joint, and point 2 represents the flexion point of the shoe. *(From Prescription Footwear Association: Reference Guide and Directory to Pedorthic Practice. Columbia, Md, 1988.)*

A number of other considerations should be taken into account in the shoe selection process and are mentioned only briefly here. As noted earlier, the shape of the shoe is determined by the last, and this shape can be divided into either straight or curved forms (see Fig. 25B-12). The straight last provides greater support along the medial aspect of the foot and is better suited to the athlete who has a lower arch or who tends to overpronate. Cheskin also recommends the straight-lasted shoe for the athlete who participates in activities demanding slower and more controlled movements, whereas the curved last is better for faster movements. [22] The curved last is generally better for the individual who has an adducted foot or a cavus foot.

In purchasing shoes, one should try to mimic the conditions under which they will be worn as much as possible. Because feet tend to swell at the end of the day or after vigorous activity, this should be taken into consideration. In most individuals, one foot is larger than the other or has certain anatomic features that mandate greater emphasis in the fitting process. One generally uses a specific sock in specific shoes, and it is important to remember this in the fitting process. Also, one's shoe size does not remain static over the years. A recent survey by the Women's Footwear Council of the

American Orthopaedic Foot and Ankle Society found that women's feet increase in size in a relatively consistent fashion from the second to the sixth decade of life. [20] Even in the same manufacturer, there can be variations within the same labeled size for different shoes. Despite the fact that in the United States alone, there are an estimated 1.2 billion pairs of shoes sold annually by some 200,000 salespeople employed by more than 50,000 shoe stores or departments, fewer than one fourth of Americans can recall the last time they had their feet sized. It is estimated that nearly three fourths of Americans wear shoes that fit improperly, with the leading offenders being shoes that are too narrow or too short. [238] To help combat the seemingly bewildering task of finding a pair of shoes that fit well, the American Orthopaedic Foot and Ankle Society, in combination with the National Shoe Retailers Association and the Pedorthic Footwear Association, has summarized many of the points contained in the preceding paragraphs and compiled a brochure outlining the "10 Points of Proper Shoe Fit" (Table 25B-14).

TABLE 25B-14 -- Ten Points of Proper Shoe Fit

1.	Sizes vary among shoe brands and styles. Don't select shoes by the size marked inside the shoe. Judge the shoe by how it fits your foot.
2.	Select a shoe shape that conforms as nearly as possible to the shape of your foot.
3.	Have your feet measured regularly. The size of your feet changes as you grow older.
4.	Have *both* feet measured. Most people have one foot larger than the other. Fit to the largest foot.
5.	Fit at the end of the day when your feet are largest.
6.	Stand during the fitting process and check that there is adequate space (3⁄8 to ½ inch) for your longest toe at the end of each shoe.
7.	Make sure that ball of your foot fits comfortably into the widest part (ball pocket) of the shoe.
8.	Don't purchase shoes that feel too tight, expecting them to "stretch" to fit.
9.	Your heel should fit comfortably in the shoe with a minimal amount of slippage.
10.	Walk in the shoe to make sure it feels right! (Fashionable shoes CAN be comfortable!)

From American Orthopaedic Foot and Ankle Society, National Shoe Retailers Association, and Pedorthic Footwear Association: 10 Points of Proper Shoe Fit.

The quality of production is not always the same for every shoe even within the same product line. The purchaser should pay close attention to the construction of the shoe in the fitting process. There may be a bad seam or an improperly applied layer that will affect fit and comfort. The type of material used in the construction of the shoe upper is also of interest because it affects the conformability of the shoe as well as its breathability. The temperature and humidity perceived by the foot are related to this latter property and are a factor in the shoe's comfort. One should appreciate the knowledge required of the athletic equipment manager or skilled salesperson in selecting the correct shoe for individuals who have markedly varying feet and participate in numerous athletic endeavors. For this reason, it is helpful if the manager or salesperson has personal experience with athletic shoes as well as some book knowledge and apprenticeship training along with a desire for a satisfied athlete-customer.

It is quite common for those involved in the care of athletes, particularly runners, to be asked for suggestions about the best running shoe, tennis shoe, skating boot, and so on. From the foregoing discussion, it should be obvious that there is no one shoe that can fulfill all the criteria necessary to be the ideal shoe for all individuals. We have seen shoes used in sports progress from a rather simplistic design to a design that involves more computers and researchers than many fields of medicine. By the same token, the price has achieved similar emphasis, and we see teenagers getting summer jobs in order to purchase $150 basketball shoes, only to keep them in the closet for fear of having them stolen. [60] There is no question that a decision about which shoe to wear or to recommend is very important to an industry that pays six-figure salaries to college coaches and offers multimillion-dollar contracts to high-profile professional athletes. Nevertheless, it is this climate of commercialism and competitiveness that has produced dramatic achievements in the athletic shoewear industry during the past 20 years. No doubt even the least expensive shoes are a far cry from the technologically unsophisticated shoes worn by our forefathers. It is unnecessary to reach the conclusion that one must wear the shoe with the latest gimmick or the highest price tag to achieve one's maximal performance or to avoid serious injury. For the most part, all the well-known companies in the athletic shoe industry make a high-quality product and have high- and low-end options. In selecting a shoe, one must remember that price can be used only as a very general guide to the quality and functional usefulness of a shoe. Once a running shoe, for example, reaches the $50 level, it usually incorporates all the principal components that are critical to satisfactory performance in such a shoe. Also, the price of the shoe is directly proportional to the amount of advertising used to market that shoe. Interestingly, there is usually another shoe of virtually identical quality available at a lower price from the same manufacturer. The consumer should always consider that shoe manufacturers might provide misleading information to the consumer. For example, a 1991 survey by *Running* magazine asked manufacturers to provide information regarding the type of runner for whom their shoes were designed. Of 171 models of shoes, including spikes as well as running and general athletic shoes, 26% were purported to be for both pronators and supinators, 36% claimed an advantage for both the high-arched and flat-footed individual, and amazingly, two shoes in the survey claimed benefit for every distance, foot type, heel-strike pattern, and surface mentioned.[34]

SUMMARY

Knowledge of athletic shoes, pads, inlays, inserts, and orthoses has become important in the field of sports medicine for many reasons. This knowledge is essential not only from the standpoint of treating and preventing injuries but also to halt the propagation of misleading information and avoid unnecessary expense. Athletic shoewear is essential for the protection of the athlete’s foot, but this protection must extend to the athlete as a whole. With a brief historical perspective and knowledge of the construction of shoes and orthoses, individuals can better understand the factors involved in this protection. These factors include shock absorption, stability, friction and torque, and proper fit. Their specific contributions to athletic performance, comfort, and injury risk remain incompletely elucidated despite numerous laboratory and clinical studies. It is hoped that this chapter not only has stimulated increased awareness of the role of athletic shoes and orthoses in the field of sports medicine but also has pointed out the need for further research.

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SECTION C Ligament Injuries: 1. Ligament Injuries of the Foot and Ankle in Adult Athletes

Melissa D. Koenig

The foot and ankle serve as a constant interface with our environment. This unique collection of tissues, each with a variety of specialized functions, allows efficient, upright stance and locomotion. Injury to the foot and ankle ligaments results in varying degrees of impairment and associated disability. Athletic ability, regardless of competitive level, is dependent on foot and ankle function. Diagnosis and treatment of foot and ankle ligament injuries are dependent on a complete understanding of foot and ankle anatomy and biomechanics (Fig. 25C1-1). An excellent review of foot and ankle biomechanics is presented within the first section of this chapter (see Chapter 25A , Biomechanics). The diagnosis and treatment of common ligament injuries to the ankle and foot in the adult athlete are reviewed in the following sections.

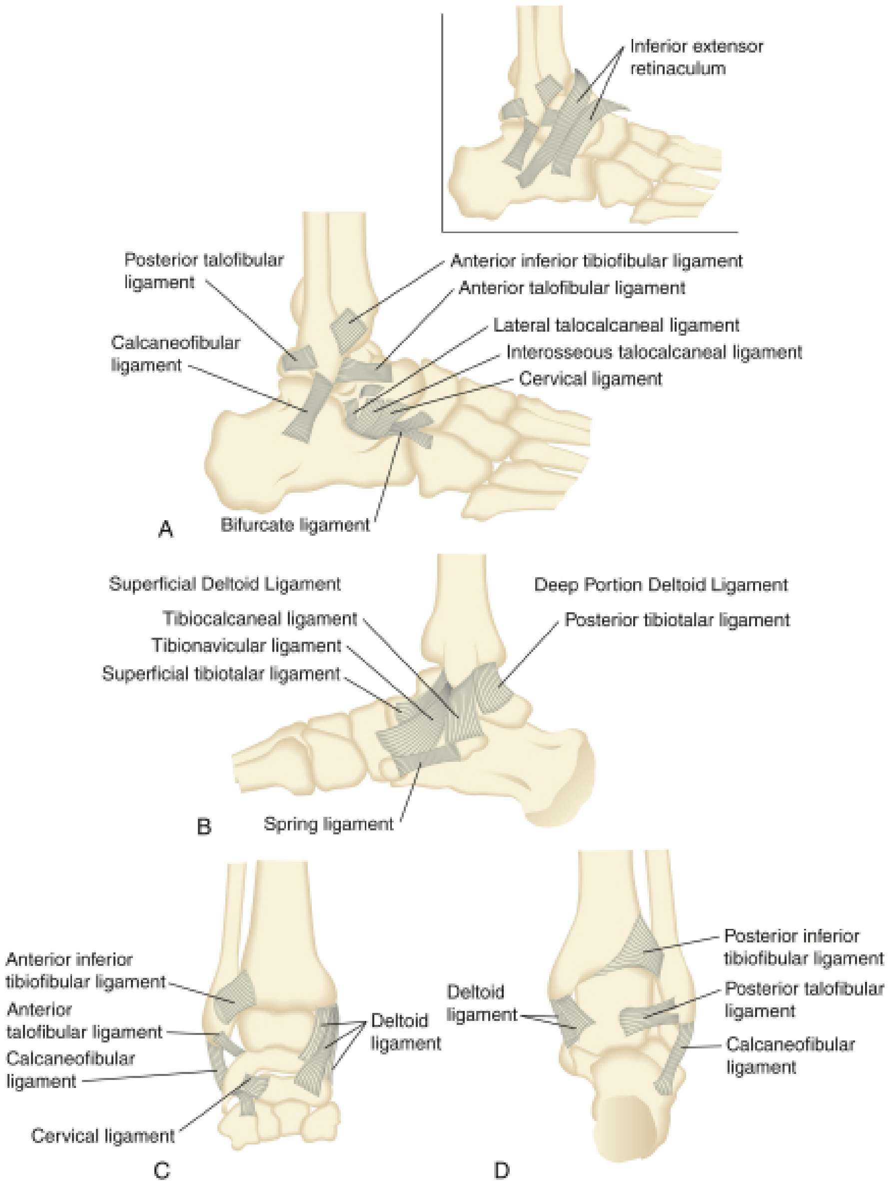

Figure 25C1-1 Compendium of the foot and ankle ligaments. **A,** Lateral view of the foot and ankle demonstrating the anterior talofibular ligament, calcaneofibular ligament, posterior talofibular ligament, anterior-inferior tibiofibular ligament, lateral talocalcaneal ligament, inferior extensor retinaculum, interosseous talocalcaneal ligament, cervical ligament, and bifurcate ligament. **B,** Medial view of the foot and ankle demonstrating the superficial deltoid ligament, including the tibionavicular, spring ligament, tibiocalcaneal, and superficial tibiotalar components. **C,** Anterior view of the ankle and hindfoot demonstrating the deltoid ligament with its superficial and deep components, the anterior-inferior tibiofibular ligament, the cervical ligament, the anterior talofibular ligament, and the calcaneofibular ligament. **D,** Posterior view of the ankle and hindfoot demonstrating the deltoid ligament with its superficial and deep components, the posterior-inferior tibiofibular ligament, the posterior talofibular ligament, and the calcaneofibular ligament.

INJURY TO THE ANKLE LIGAMENTS

In the following section, ankle ligament injuries are arbitrarily divided into *lateral ankle sprain, medial ankle sprain, ankle syndesmosis sprain,* and *dislocation of the ankle without fracture*. The division is artificial in that many ankle sprains represent a combination of ligament injuries. Ankle ligament injury associated with malleolar fracture is not discussed as a separate topic. An anatomic division of the ankle ligaments is presented in Table 25C1-1 for the purpose of completeness and discussion.

TABLE 25C1-1 -- Ankle Ligament Groups

Lateral ankle ligaments	Anterior talofibular ligament (ATFL)
	Calcaneofibular ligament (CFL)
	Posterior talofibular ligament (PTFL)
Medial ankle ligaments	Superficial deltoid (tibionavicular ligament, tibiospring ligament, and superficial tibiotalar ligament)
	Deep deltoid (deep anterior tibiotalar ligament and deep posterior tibiotalar ligament)
Ankle syndesmosis	Anterior-inferior tibiofibular ligament (AITFL), posterior-inferior tibiofibular ligament (PITFL), distal interosseous ligament (IOL)

Lateral Ankle Sprain

Lateral ankle sprain represents one of the most common injuries in the athletic population.[1] Inversion of the plantar flexed ankle is the accepted mechanism of injury for lateral ankle sprain (Fig. 25C1-2). Stretching or tearing of the lateral ankle ligaments leads to inversion instability, a condition that may present in an acute or chronic setting.

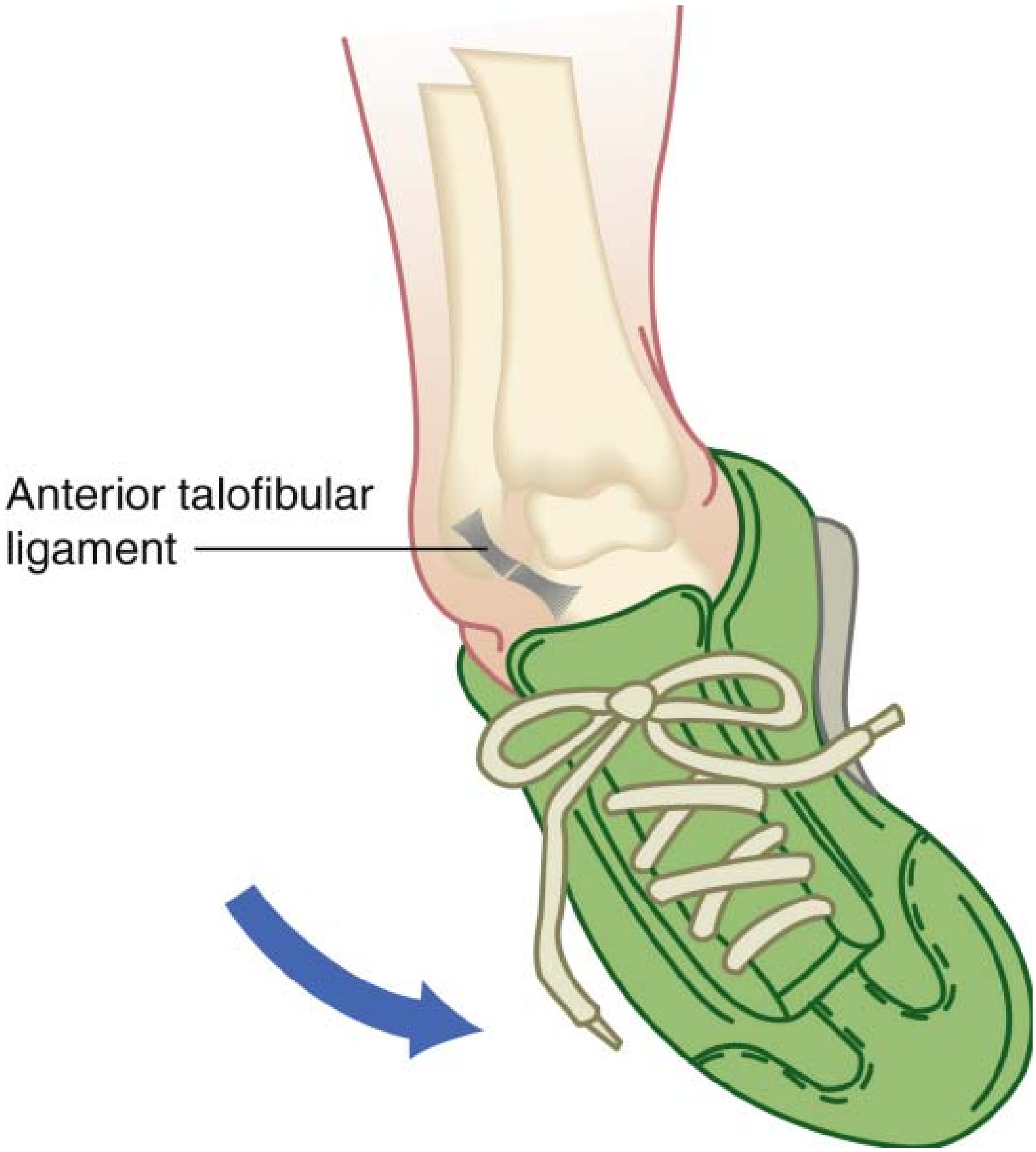

Figure 25C1-2 Inversion of the plantar flexed ankle is the mechanism of injury for lateral ankle sprain associated with a tear of the anterior talofibular ligament.

The highest incidence of injury is localized to the lateral ankle ligaments but may also include the subtalar joint. Among the lateral ankle ligaments, the most commonly injured structure is the anterior talofibular ligament (ATFL). Among cadets at West Point, one third sustained one or more inversion injuries during their 4-year placement. [1] Among high school students, ankle injuries are estimated to occur in 1 of every 17 participants per season; 85% of these injuries are ankle sprains. [2]

Gerber and associates prospectively evaluated cadets at West Point over a 2-month period. [3] Football, soccer, jogging, and basketball were the activities most often associated with ankle sprain. Among high school athletes, ankles sprains are most common among men's and women's basketball players, followed by participants in football and women's cross-country. [2] A varsity high school basketball survey revealed that 70% of players had a history of ankle sprain. [4] Ankle sprain is the most common soccer injury (17% of injuries in senior division men's soccer), with a cumulative incidence of 27% over an 8-year period. [5] Lateral ankle injuries were the injury most commonly associated with disruption of training among runners in one study. [6]

Commonly cited risk factors associated with lateral ankle injury include generalized ligamentous laxity, inappropriate shoewear, irregular playing surface, and cutting activity. Glick and colleagues reported that preexisting laxity of the lateral ankle ligaments, in the form of increased talar tilt on stress radiograph, is a significant risk factor. [7] Furthermore, Thacker and coworkers completed a review of the literature and determined that a history of previous lateral ankle sprain is the most commonly cited risk factor for ankle sprain.[8]

Although hypermobility, generalized joint laxity, and previous ligament injury should intuitively qualify as significant risk factors for lateral ankle injury, Baumhauer and associates published a contrary conclusion.[9] They completed a prospective study of joint laxity, foot and ankle alignment, ankle ligament stability, and isokinetic strength as risk factors for inversion ankle injuries. Among 145 college-aged athletes, 15 injuries were reported during a single intercollegiate season (lacrosse, soccer, and field hockey). No significant differences were found between the injured and uninjured groups with regard to the stated risk factors.

Relevant Anatomy

Normal Anatomy

The talus articulates with the tibia and fibula to form the ankle joint (talocrural joint). The clinical range of motion is variable but usually ranges from 0 to 10 degrees of dorsiflexion and 40 to 50 degrees of plantar flexion. The empirical axis of the joint is somewhat oblique such that plantar flexion and dorsiflexion produce concomitant internal and external rotation of the foot relative to the leg. The rotational movements are translated through the subtalar joint and the remainder of the foot to produce supination and pronation during the gait cycle.

Inman noted that the anterior margin of the talar dome is wider than the posterior margin by an average of 2.4 mm.[10] The implication of this differential width is the stability imparted to the ankle joint during ankle dorsiflexion, along with the relative instability associated with ankle plantar flexion.

The functional stability of the ankle is the product of its soft tissue support. The ankle capsule is reinforced by several groups of ligamentous structures. The lateral ligamentous complex includes the ATFL, the calcaneofibular ligament (CFL), and the posterior talofibular ligament (PTFL) (Fig. 25C1-3).

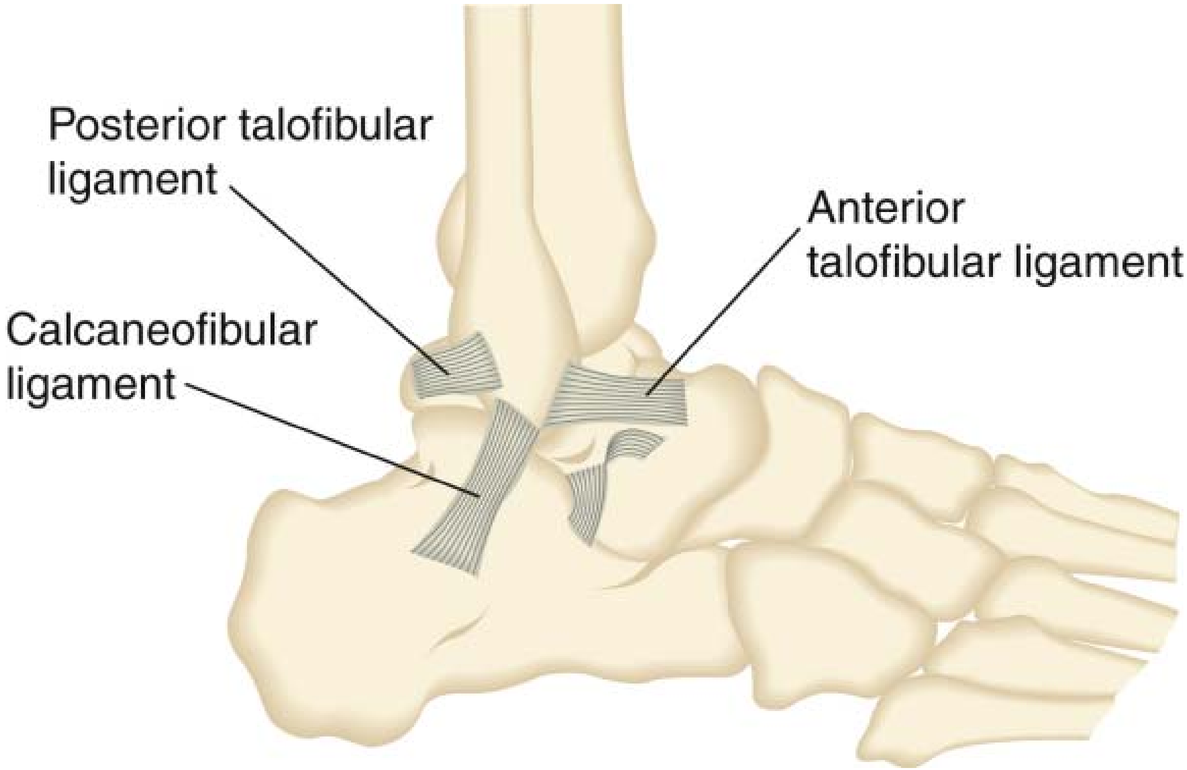

Figure 25C1-3 The lateral ligamentous complex of the ankle consists of the anterior talofibular ligament, calcaneofibular ligament, and posterior talofibular ligament.

The ATFL originates from the anterior aspect of the distal fibula and inserts onto the talar body just anterior to the articular facet. It measures 5 mm in width and 12 mm in length.[11] Its fibers blend with the anterior lateral capsule of the ankle.

The CFL originates from the anterior border of the distal lateral malleolus and courses medially, posteriorly, and inferiorly to its insertion on the calcaneus. Its fibers blend with the peroneal tendon sheath. Typically a cord-like structure 4 to 6 mm in diameter and 2 to 3 cm long, the CFL is directed 10 to 45 degrees posterior to the line of the longitudinal axis of the fibula. [11]

The PTFL originates from the posterior border of the fibula and inserts at the posterior lateral aspect of the talus; it is 6 mm in diameter and 9 mm in length. [11] The PTFL blends with the posterior ankle capsule.

The position of the talus relative to the long axis of the leg is important for determination of the function of the lateral ankle ligaments (Fig. 25C1-4). [10] At a position of neutral dorsiflexion, the ATFL is perpendicular to the axis of the tibia, and the CFL is oriented parallel to the tibia. [12] In this position, the CFL provides resistance to inversion stress or varus tilt of the talus. If, however, the talus is plantar flexed (the most common position for lateral ankle inversion injuries), the ATFL is parallel and the CFL is perpendicular to the axis of the tibia. Therefore, with the foot in the most common position for lateral ankle ligament injury, the ATFL is placed in the precarious situation of providing resistance to inversion stress. [13]

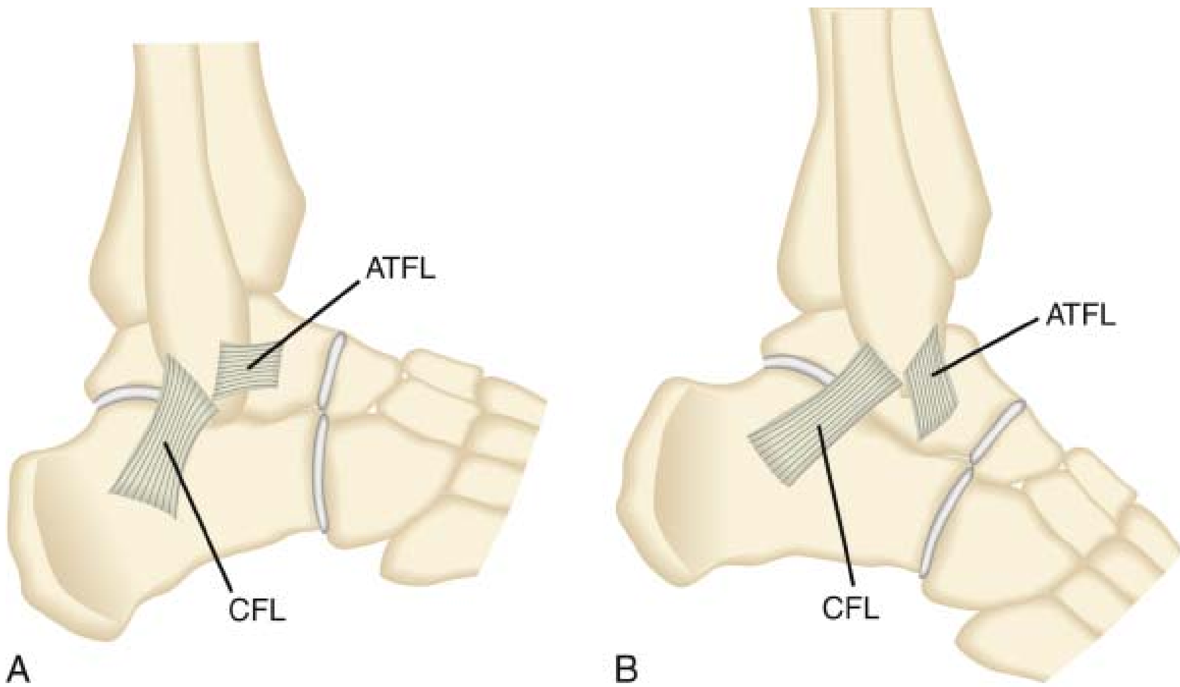

Figure 25C1-4 **A,** At a position of neutral dorsiflexion, the anterior talofibular ligament (ATFL) is perpendicular to the axis of the tibia, and the calcaneofibular ligament (CFL) is oriented parallel to the tibia. In this position, the CFL provides resistance to inversion stress or varus tilt of the talus. **B,** If, however, the talus is plantar flexed (the most common position for lateral ankle inversion injuries), the ATFL is parallel and the CFL is perpendicular to the axis of the tibia, and the ATFL provides resistance to inversion stress or varus tilt of the talus.

Colville and colleagues used 10 cadaveric ankles to measure strain in the lateral ankle ligaments with the ankle moving from dorsiflexion to plantar flexion. [14] The ATFL strain increased with increasing degrees of plantar flexion, internal rotation, and inversion. Conversely, the CFL strain increased with increasing degrees of dorsiflexion and internal rotation. The authors concluded that the ATFL and the CFL work in tandem to provide lateral ankle stability throughout the entire range of ankle motion.

Anatomy and Biomechanics

The biomechanical characteristics of the ankle ligaments are such that failure (rupture) is due to increasing load as opposed to twisting or shearing. [15] Isolated testing of the individual ankle ligaments demonstrates that the ATFL is the first to fail and the deep deltoid ligament is the last to fail. [15] The ATFL is considered the weakest lateral ankle ligament. [311] [312] [313] Lateral ankle ligament failure is typically midsubstance rupture or a talar avulsion. [15]

Isolated division of the three lateral ankle ligaments yields predictable results. [307] [314] [315] [316] [317] [318] [319] Division of the ATFL allows increased talar tilt with the ankle in a plantar flexed position, but not in a neutral dorsiflexion position. Division of the CFL allows increased subtalar motion in a neutral dorsiflexion position, but the ankle remains stable in a plantar flexed position. Division of both the ATFL and the CFL results in an unstable ankle. Division of the PTFL allows increased ankle dorsiflexion but does not impart lateral ankle instability. The stabilizing effect of each of the lateral ankle ligaments is dependent on the position of the talus at the time of the applied stress.

Stormont and coworkers further advanced the method of ligament testing through the addition of physiologic loads with fixed axial rotation to the sequential ligament sectioning protocol. [25] Inversion stability was provided by the CFL and the ATFL in the unloaded ankle and entirely by the articular surface in the loaded ankle. Eversion stability was provided by the deltoid in the unloaded ankle and entirely by the articular surface in the loaded ankle.

The stabilizing effect of the articular cartilage as described earlier (physiologic load and fixed axial rotation) does not represent the in vivo situation. Remember, normal gait and foot and ankle motion are associated with internal and external rotation of the tibia. [10] Kinematic experiments by Cass and Settles placed axial loads on the ankle while allowing axial rotation. [26] Computed tomography (CT) was used to evaluate the ankle subjected to an inversion stress. Interestingly, the talar tilt did not change with isolated section of the ATFL or the CFL. Division of both ATFL and the CFL produced an average of 20.6 degrees of talar tilt. The authors concluded that ankle joint stability is provided by the lateral ankle ligaments and not by the ankle articular surfaces.

Broström described the ligamentous lesions found during the surgical exploration of 105 recent ankle sprains. [27] The ATFL was the most commonly injured structure. The ATFL was completely torn as an isolated injury in 65 cases and as an associated injury in an additional 25 patients. The CFL was the second most commonly injured ligament; it was completely or partially torn as an associated injury in 23 patients. The most common combination of ligamentous injuries was a complete tear of the ATFL and a partial or complete tear of the CFL as described in 20 patients. Complete ligament tears were noted to occur with concomitant rupture of the adjacent joint capsule. Broström observed that with the talus in a reduced position, the torn ends of the capsule and ligament remained well apposed in most cases. Broström further noted that a tear of the CFL was always associated with a tear of the adjacent peroneal tendon sheath, an observation that is key to ankle arthrography (Table 25C1-2).

TABLE 25C1-2 -- Distribution of Ligament Injury after Inversion Injury

Study	Diagnostic Method	No.	ATFL (%)	ATFL + CFL (%)	ATFL + CFL + PTFL (%)	CFL (%)	AITFL (%)	Deltoid (%)
Broström, 1965 [40]	Arthrography	239	152 (64)	40 (17)		0 (0)	25 (10)	6 (2.5)
Brunner & Gaechter, 1991 [34]	Surgery	180	52 (29)	101 (56)		27 (15)		
Povacz et al, 1998 [68]	Surgery	73	29 (40)	42 (58)	2(3)			

AITFL, anterior-inferior tibiofibular ligament; ATFL, anterior talofibular ligament; CFL, calcaneofibular ligament; PTFL, posterior talofibular ligament.

The advent of magnetic resonance imaging (MRI) has allowed a more detailed evaluation of ligamentous injuries, beyond that provided and limited by surgical exposure. Tochigi and colleagues performed MRI on 24 patients with acute inversion injury of the ankle. [28] They detected 23 ATFL, 15 CFL, 11 PTFL, 8 deltoid ligament, 13 interosseous talocalcaneal ligament, and 12 cervical ligament injuries.

In addition to the loss of stability imparted to the ankle by ligament rupture, an interruption of normal neural processes has been documented. The disruption of capsular mechanoreceptors [29] and the subsequent loss of afferent nerve function and ankle motor coordination may further contribute to the development of chronic instability. [30] This loss of function is addressed directly by proprioceptive and coordinated motion rehabilitation.

The most significant problem associated with acute lateral ankle sprain is the predisposition to development of chronic instability. The natural history of untreated chronic lateral ankle instability is loss of function and progressive osteoarthritic changes around the ankle. [326] [327] [328]

Clinical Evaluation

Although rupture of the lateral ankle ligaments is appropriately considered when a plantar flexion–inversion injury is evaluated, it is imperative that the examination not be limited to these structures. An inversion foot or ankle injury is approached as a constellation of possible injuries, including ATFL sprain, CFL sprain, syndesmosis sprain, deltoid sprain, subtalar sprain (chronic insufficiency of the lateral hindfoot associated with subtalar instability isolated or combined with lateral ankle ligament instability has been shown to occur in up to two thirds of patients [34]), subtalar coalition, bifurcate ligament sprain, peroneal tendon instability, peroneal tendon tear, lateral malleolus fracture, talar dome osteochondral injury, anterior process of the calcaneus fracture, and fracture of the base of the fifth metatarsal.

Although classification systems abound, no single system is routinely used in the literature. Sprains can be considered from the perspective of graded ligament injury, as suggested by the American Medical Association [35] and O'Donoghue. [36] Injuries are graded based on stretch, partial tear, or complete rupture of the ligament, as is noted in Table 25C1-3 . Additional information with regard to associated ligamentous injuries is noted. Grading lateral ankle injuries is much more of a gestalt process than a scientific endeavor. It is not important for the physician to differentiate a grade I from a grade II injury, but the physician should be able to discern a grade I from a grade III injury, or an isolated ATFL injury from an ATFL injury associated with rupture of the syndesmosis.

TABLE 25C1-3 -- Grading System for Ankle Ligament Injury

Acute Grade	Anatomic Injury	Historical Findings	Examination Findings
I	Stretching of the ATFL	Inversion injury, subacute pain and swelling, continuous athletic activity	Mild swelling, mild ATFL tenderness, stable ankle
II	Partial tearing of the ATFL	Inversion injury, acute pain and swelling, inability to continue athletic activity, painful gait	Moderate swelling, moderate ATFL tenderness, stable ankle
III	Complete rupture of the ATFL	Inversion injury with associated "pop," acute severe pain and swelling, inability to walk	Severe swelling, severe ATFL tenderness, unstable ankle
Subclassification			
CFL injury	ATFL and CFL injury	Mechanism related to ankle dorsiflexion	Additional tenderness at CFL, increased varus tilt of the talar dome
Chronic instability	Persistent laxity at lateral ankle ligaments	Recurrent ankle sprains, "giving way," [30,75] apprehension and anxiety related to the ankle	Swelling and tenderness at lateral ankle ligaments associated with recurrent injury; ankle instability *despite* grade of injury
Medial ankle injury	Deltoid ligament—complete or partial disruption	Abduction or eversion mechanism, pain over the medial ankle	Swelling and tenderness over deltoid, valgus instability
Syndesmosis injury	AITFL, PITFL, and IOL injury	External rotation mechanism, pain over the ankle syndesmosis	Swelling and tenderness over syndesmosis, pain at syndesmosis with squeeze test [177] or forced external rotation [176]
Subtalar injury	CFL, interosseous talocalcaneal ligament, cervical ligament	Frequent "ankle sprains," sinus tarsi pain, difficulty on uneven ground	Increased subtalar range of motion, sinus tarsi tenderness

AITFL, anterior-inferior tibiofibular ligament; ATFL, anterior talofibular ligament; CFL, calcaneofibular ligament; IOL, distal interosseous ligament; PITFL, posterior-inferior tibiofibular ligament.

Jackson and associates have established a functional classification system that hinges on the ability of the patient to walk with no limp (mild), walk with a limp (moderate), or not walk (severe). [1] The degree of injury was related to a return to full activity in 8, 15, and 19 days for mild, moderate, and severe sprains, respectively.

History

Relevant historical information includes previous ankle injury, the mechanism of injury, the ability of the patient to continue to play or bear weight, and current symptoms. Severe lateral ankle sprains are associated with a history of inversion injury with a characteristic "pop." Acute pain and swelling develop quickly, and frequently the athlete is unable to continue playing.

Physical Examination

Examination of the patient includes evaluation of the entire extremity. Inspection of the leg, ankle, and foot may reveal swelling, ecchymosis, blister formation, or gross deformity. A vascular and sensory assessment is always performed. The region is palpated systematically with attention to pain over ligamentous, bony, or tendinous structures. Tenderness at the ATFL and the CFL is particularly important to note. Muscle testing is assessed for strength and pain during activation.

Motion around the foot and ankle is always assessed with the patient seated and relaxed. The knees are flexed and the feet allowed to fall into an equinus position. The leg is gently grasped while the heel is held in a neutral position and the ankle brought to a right angle or neutral dorsiflexion. From this position, maximal dorsiflexion and plantar flexion are observed in both passive and active modes and compared with the uninjured side.

Stress testing is a useful clinical tool that provides a portion of the diagnostic data needed for grading ankle sprains. Stress testing alone is not adequate for reproducible diagnosis of lateral ankle ligament injuries. [332] [333] Ankle stability is evaluated by several stress tests.

The *anterior drawer test* is used to demonstrate the integrity of the ATFL (Fig. 25C1-5). The patient is seated, and the flexed leg hangs off of the table. The examiner stabilizes the distal tibia with one hand while the other hand grasps the heel behind and pulls the foot forward to produce forward translation. The test is performed with the ankle in both neutral and plantar flexion positions. The results are compared with those of the contralateral ankle, and the test is repeated as required. A few millimeters of translation is normal. With a complete ATFL tear, the talus subluxates anteriorly and a dimple appears over the anterolateral joint due to suction. A portable ankle ligament arthrometer may be used in conjunction with manual testing to improve the accuracy and reliability of the test. [39]

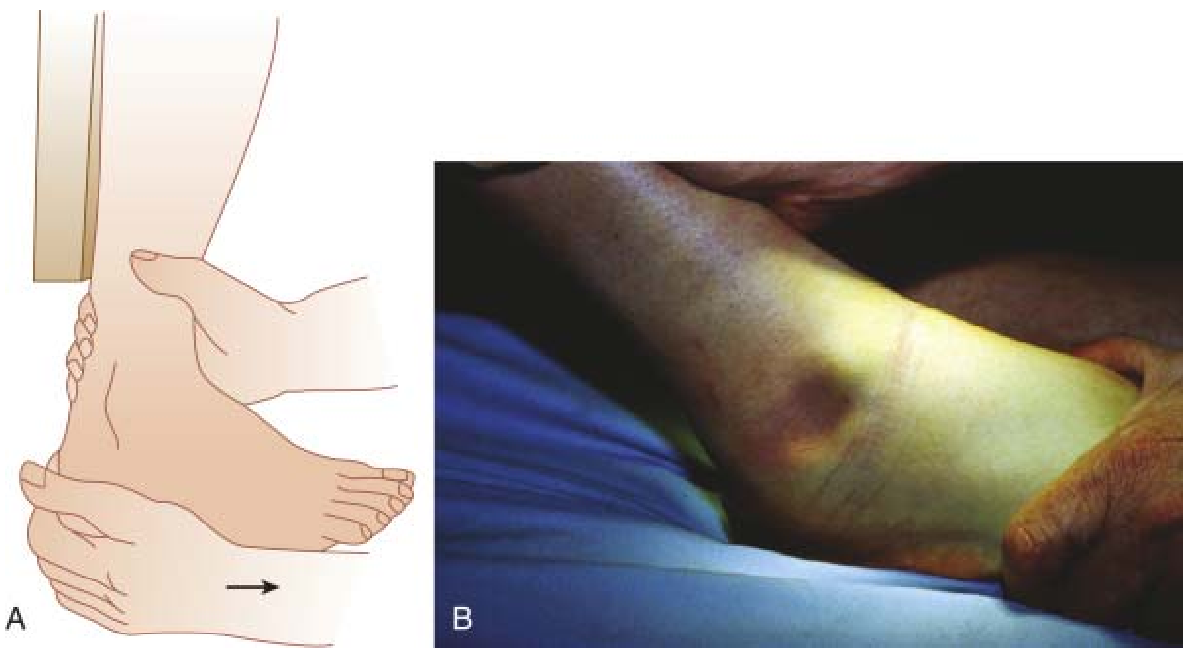

Figure 25C1-5 A and **B,** The anterior drawer test of the ankle. Note the skin dimple consistent with a positive test.

Testing is occasionally uncomfortable, particularly in the acute setting. False-negative results may be caused by involuntary guarding or pain response. Local anesthesia increases the accuracy of the anterior drawer test (when performed with a mechanical testing device). [37]

Broström noted that clinical instability was almost never present among arthrographically proven, acute, nonanesthetized ankle ligament ruptures. [40] After spinal anesthesia was established, all ankles with a proven ATFL rupture demonstrated a positive anterior drawer sign. Broström went on to report in a separate publication that the anterior drawer test without any form of anesthesia was useful in diagnosing persistent (chronic) tears of the ATFL. [41] The results of the anterior drawer test are also influenced by the thickness of the fat pad at the posterior calcaneal tuberosity and by ligamentous laxity. [42]

The *talar tilt test* is performed with the patient seated (Fig. 25C1-6). The leg is secured with the examiner's open hand, the heel is grasped from behind with the opposite hand, and a varus or inversion force is placed in an effort to produce talar tilt. The results are compared with those of the contralateral ankle, and the test is repeated as required. The test is performed with the ankle in both neutral and plantar flexion positions. Stressing the ankle in neutral dorsiflexion differentially tests the function of the CFL, whereas stressing a plantar flexed talus tests the ATFL. [12] Increased inversion of the calcaneus may represent ankle or subtalar instability. [34] Varus tilt to a limited degree is probably normal. [43]

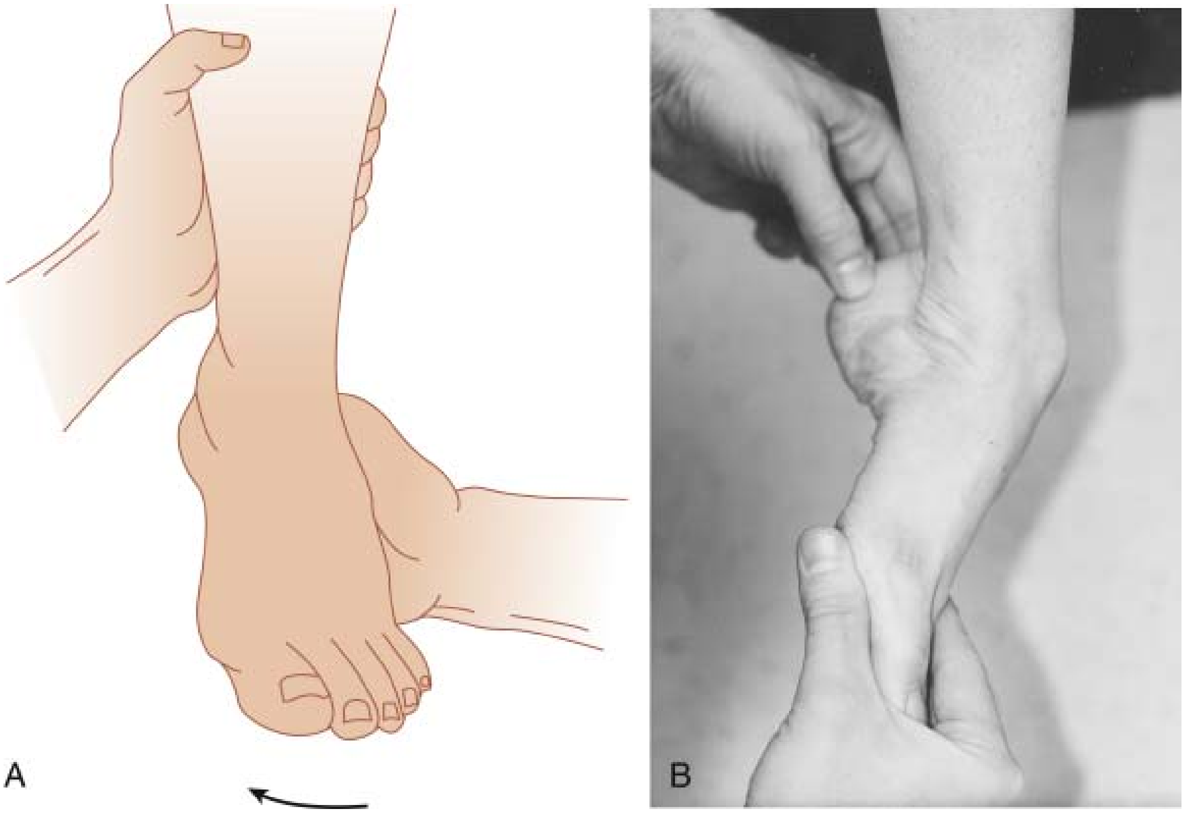

Figure 25C1-6 **A** and **B,** The talar tilt (inversion stress) test of the ankle.

The high incidence of bifurcate ligament sprain warrants a brief discussion with regard to its presentation. Broström noted clinical evidence of bifurcate ligament injury in 18.6% of patients with acute ankle sprains and 3.7% of patients with confirmed lateral ankle ligament ruptures. [40] Bifurcate ligament injury is characterized by diffuse lateral hindfoot and midfoot swelling with associated ecchymosis. Tenderness tends to localize to the course of the bifurcate ligament, which is distinct from the course of the ATFL. The ankle and midfoot remain stable. Pain is easily reproduced with forced inversion of the plantar flexed foot. Broström noted that the differentiation between lateral ankle ligament injury and bifurcate ligament injury was best achieved by eliciting indirect tenderness. [40] He suggested manipulation of the heel to produce lateral ankle pain and stabilization of the heel with simultaneous forced forefoot motion to produce bifurcate pain.

Imaging

Radiographs

Radiographs in the anteroposterior, mortise, and lateral projections are required for ankle evaluation. Weight-bearing radiographs better reproduce physiologic loading, but they are not always obtainable in the acute phase owing to pain.

The radiographs are evaluated with regard to malleolar fracture, physeal fracture, osteochondral fracture, and avulsion fracture. Alignment and translational abnormalities are also noted, particularly at the syndesmosis and the medial ankle joint space. (See "Ankle Syndesmosis Sprain" and "Medial Ankle Sprain," later.)

During surgical exploration of 60 chronic ankle sprains, Broström noted 5 cases (8%) with anterior lateral talar osteochondritis dissecans. [44] Anderson and Lecocq reported a 22% incidence of osteochondral lesions in a mixed series of 27 cases of single and recurrent lateral ankle injuries. [13] The lesions were located at the lateral talus in 5 patients and at the medial talus in 1 patient.

The presence of a subfibular ossicle may be indicative of acute or chronic injury to the ATFL, [45] or it may be a normal variant (os subfibulare). Broström noted that avulsion of bone fragments is an uncommon pathologic finding after an acute ankle sprain. [40] He further noted that patients who sustained an avulsion fracture were more likely to be older and female.

Stress Radiographs

The bilateral stress radiograph is used to quantify anterior talar translation and varus tilt of the talus. Stress radiographs are not routinely necessary for the evaluation of acute lateral ankle ligament injury. [296] [329] Accuracy is compromised by pain response, peroneal spasm, [46] variable stress technique, and lack of control data in the case of a previously injured contralateral ankle. The use of a local anesthetic injection may eliminate some of these variables and result in more accurate stress radiographs.

The talar tilt stress radiograph is an anteroposterior view of the ankle taken while an inversion force is applied. The stress can be performed manually or with commercially available devices that provide standardized and quantitative applied stress. The degree of tilt is determined by measuring the angular divergence between the distal tibial articular surface and the talar dome (Fig. 25C1-7). Stress radiographs have been described with the foot in dorsiflexion, neutral, and plantar flexion with the knee flexed or straight.

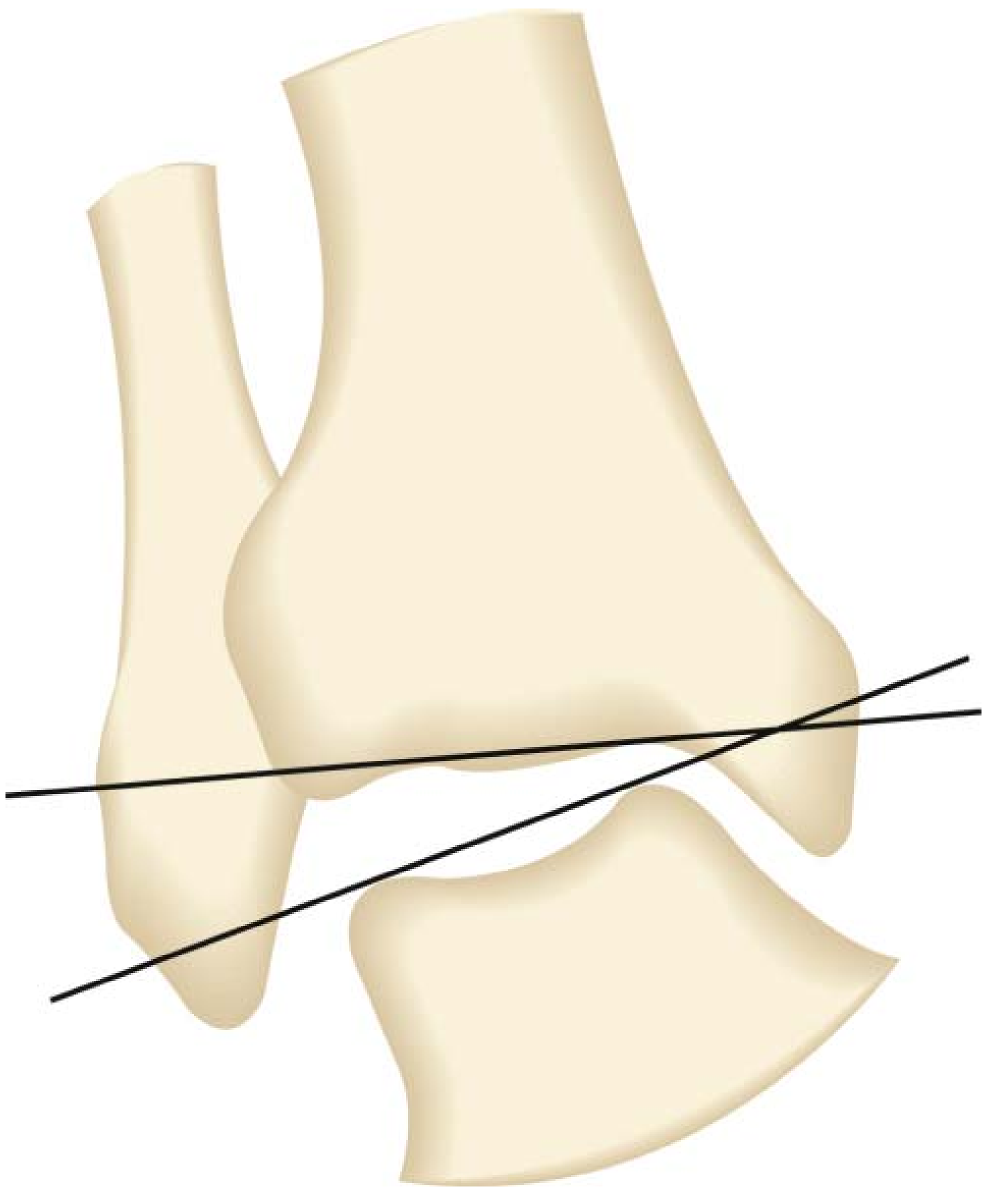

Figure 25C1-7 The talar tilt (inversion) stress radiograph. The talar tilt angle refers to the angle between two lines drawn to the tibial plafond and the talar dome.

The literature offers no consensus with regard to normal and pathologic findings in radiographic stress tests. Bonnin reported radiographic data demonstrating 4 degrees of varus tilt in 10% to 15% of noninjured ankles.[47] Hughes evaluated varus stress radiographs of both ankles in 90 injured and 90 noninjured patients and concluded that 6 degrees of increased talar tilt represents the transition from "normal to abnormal" talar tilt.[48]

Rubin and Whitten published data analyzing the range of talar tilt present in stress radiographs of 152 normal ankles.[38] About 56% of the noninjured ankles in their study had talar tilts of 3 to 23 degrees. However, only 2 ankles measured more than 20 degrees, and only 6 had more than 15 degrees of talar tilt. Seligson and associates used mechanical devices to obtain controlled stress radiographs of 25 functionally normal ankles.[49] The talar tilt in these asymptomatic ankles varied from 0 to 18 degrees. The anterior translation as seen on the lateral stress radiograph never exceeded 3 mm.

Cox and Hewes performed stress radiographs on 404 ankles of patients with no history of previous ankle injury.[50] Manual stress was applied to the plantar flexed ankle for an anteroposterior radiograph. No talar tilt was detected in 365 (90.3%) of the ankles, 1 to 5 degrees of talar tilt was detected in 32 ankles (7.9%), and greater than 5 degrees of talar tilt was detected in only 7 ankles (1.7%).

Glasgow and colleagues suggested the importance of a lateral stress radiograph.[21] They cited the prevalence of ATFL ruptures and the associated anterior instability. The *anterior drawer stress radiograph* is a lateral radiograph taken while an anterior displacement stress is applied to the ankle with the foot in gentle plantar flexion. The degree of translation is determined by measuring the shortest distance between the talar dome and the posterior margin of the tibial articular surface (Fig. 25C1-8).[51]

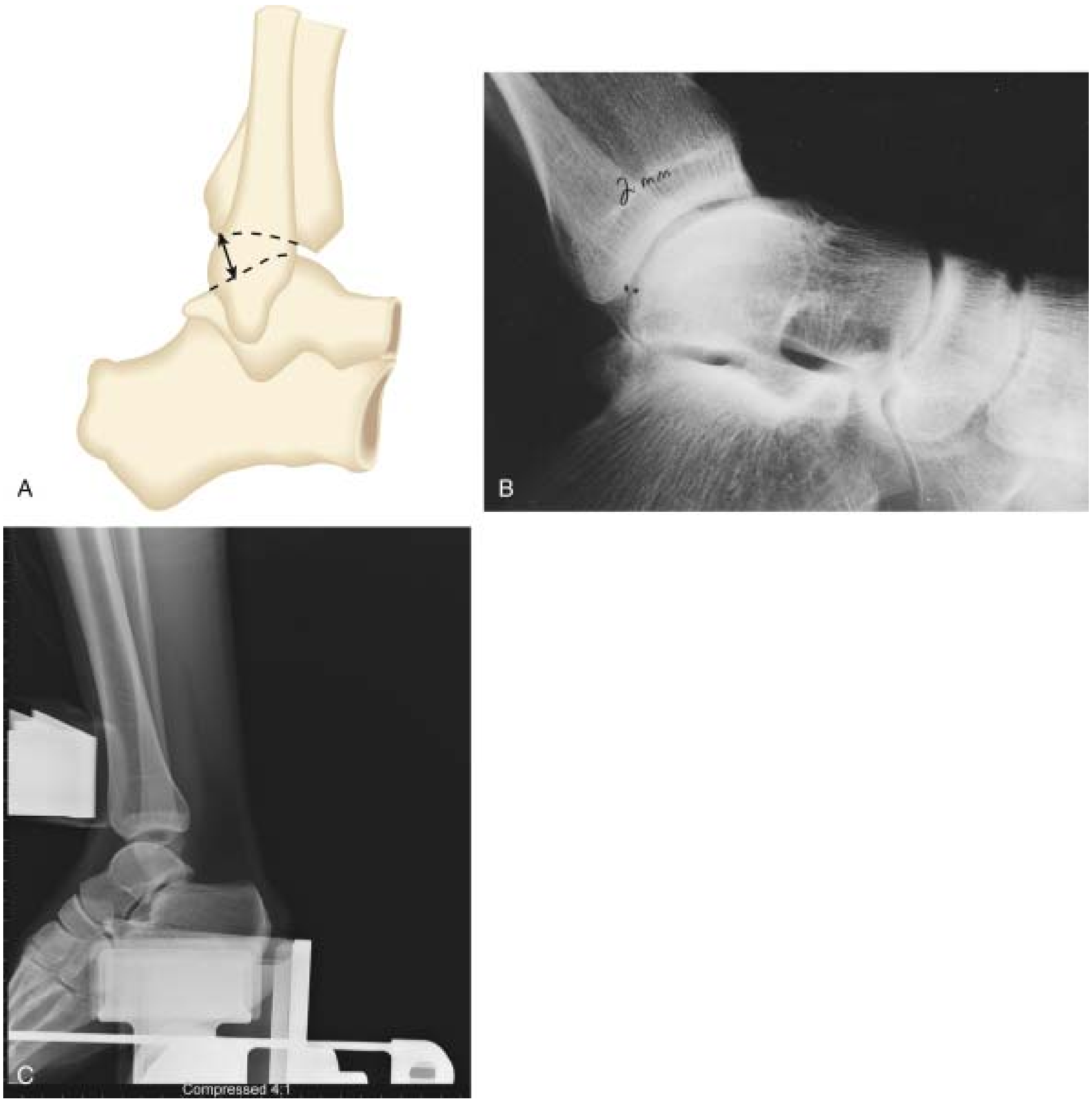

Figure 25C1-8 A, The anterior drawer stress radiograph. Anterior talar displacement (in millimeters) is recorded by measuring the shortest distance from the most posterior articular surface of the tibia to the talar dome. **B,** The anterior drawer stress radiograph with no anterior talar displacement, negative test. **C,** The anterior drawer stress radiograph with increased anterior talar displacement, positive test.

Numerous studies have found a range of translation between 2 and 9 mm, with most less than 4 mm. With more than 5 mm of anterior translation, most consider this a positive test consistent with ATFL rupture.

Other Imaging

Arthrography

The unpredictable effect of patient guarding during stress radiography is overcome by the use of ankle arthrography. [347] [348] This method is relatively simple. Contrast material is injected into the acutely injured ankle, preferably with fluoroscopic guidance, and radiographs are obtained in various projections with attention to areas of extravasation. A false-negative result from an early capsular seal is possible. The procedure has been used much less since the increased availability of MRI.

Broström and colleagues performed arthrography of 321 fresh ankle sprains. [54] Extra-articular leakage occurred in 239 cases (74%). Surgical exploration was performed in 99 of these cases and in an additional 6 cases that did not demonstrate leakage. The authors concluded that arthrography was useful within the first 7 days of an acute ankle sprain. Leakage of contrast into the peroneal tendon sheath correlated with tear of the CFL. Leakage in front of the lateral malleolus correlated with ATFL rupture. Leakage in front of the syndesmosis correlated with complete rupture of the syndesmosis. Leakage at the medial malleolus correlated with partial deltoid rupture. The presence of extra-articular contrast in the flexor hallucis longus (FHL) and flexor digitorum longus (FDL) tendon sheaths, as well as the posterior facet of the subtalar joint, was not diagnostic.

Peroneal sheath injection (tenography), as described by Black and associates, is useful in discerning injury of the CFL. [53] Contrast leakage from the sheath or passage into the ankle joint suggests CFL disruption.

Magnetic Resonance Imaging

MRI is a useful method for evaluation of acute, subacute, and chronic lateral ankle ligament injuries (Fig. 25C1-9). [55] Associated injuries to the talar dome, subchondral bone, and peroneal tendons, as well as the interosseous and cervical ligaments of the subtalar joint, are visualized. [28] MRI also reveals tarsal coalition.

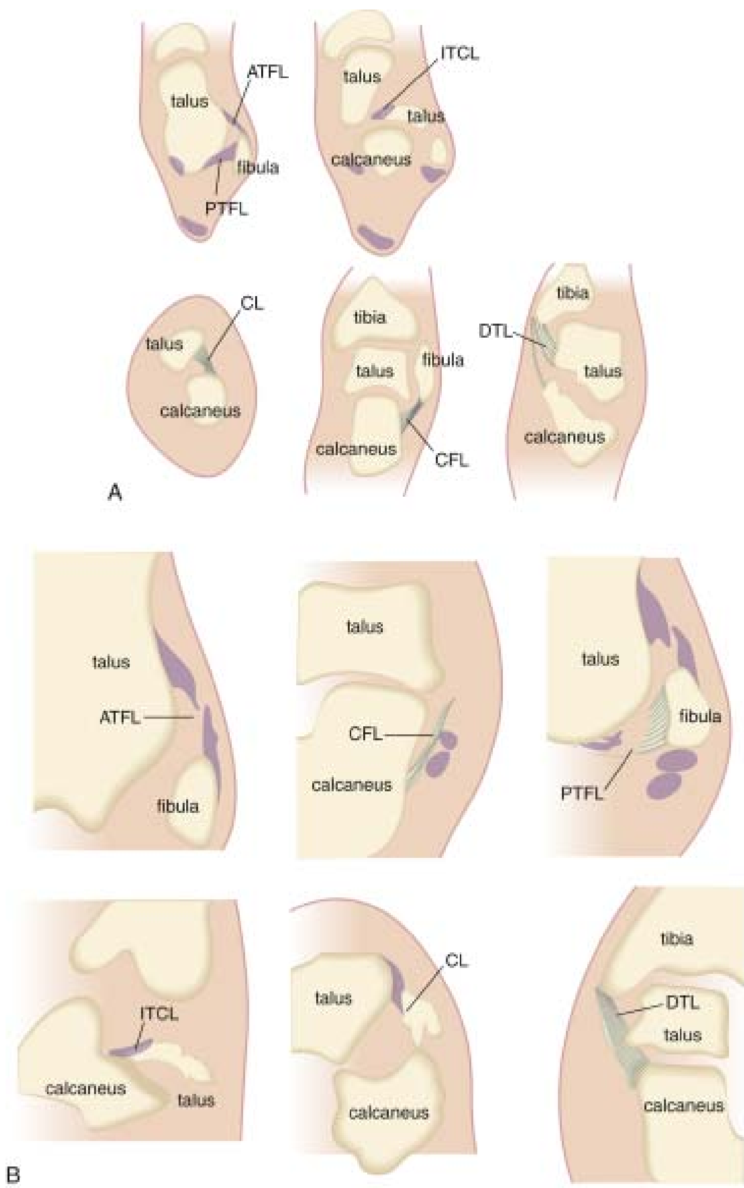

Figure 25C1-9 A, Magnetic resonance imaging key for the normal ankle. **B,** Magnetic resonance imaging key for the injured ankle. ATFL, anterior talofibular ligament; CFL, calcaneofibular ligament. *(Redrawn from Tochigi Y, Yoshinaga K, Wada Y, Moriya H: Acute inversion injury of the ankle: Magnetic resonance imaging and clinical outcomes. Foot Ankle Int 19:730-734, 1998.)*

Rijke and colleagues used a dedicated knee coil and axial images of the neutral and plantar flexed ankle to describe various ligament injuries. [55] Complete disruption, partial disruption, and laxity were all visualized. Additionally, hemorrhage and soft tissue swelling were indicative of an acute injury.

The high sensitivity associated with MRI mandates that images be carefully correlated with clinical findings. Its accuracy, lack of ionizing radiation, noninvasiveness, decreasing cost, and increasing availability suggest that MRI is the imaging modality of choice when a definitive diagnosis with objective documentation is important.

Therapeutic Options

Nonsurgical treatment is the primary choice of management for most lateral ankle sprains, no matter how severe. All acute injuries are treated with the RICE (*R*est, *I*ce, *C*ompression, *E*levation) method and protected weight-bearing. The amount of rest required after an acute ankle sprain is determined by several factors.

Many studies have reviewed the effect of cold application to the injured extremity. Cold therapy is an effective, inexpensive, and easy-to-use modality for the treatment of acute musculoskeletal injury. Appropriately applied cold therapy decreases both pain perception and the biochemical reactions that produce inflammation, and produces vasoconstriction with a concomitant reduction in soft tissue swelling and bleeding.

Hocutt and coworkers demonstrated the importance of using cold therapy (ice whirlpool or ice pack for 15 minutes 1 to 3 times a day) in the treatment of acute ankle sprains. [56] Furthermore, the group demonstrated a faster recovery associated with early cold therapy (within the initial 36 hours after an ankle sprain) compared with delayed cold therapy or early heat therapy.

Compression is typically provided in the form of an elastic bandage but may involve casting, splinting, pneumatic orthosis, or mechanical compression devices. [57]

Elevation of the ankle helps to reduce swelling and pain. Elevating the extremity above the level of the heart should be emphasized. Any decrease in height will reduce the pressure that edema must overcome before being mobilized out of the acutely swollen tissues surrounding the ankle.

Grade I and Grade II Lateral Ankle Ligament Injuries

Treatment of mild and moderate ankle sprains is symptomatic, with an emphasis on recovery of range of motion, strength, and coordination. The detailed rehabilitation program is outlined here. A nonrigid functional ankle brace, such as a lace-up brace, or a semirigid pneumatic ankle brace is used during all phases of recovery.

Grade III Lateral Ankle Ligament Injuries

Several reliable treatment methods are available for complete lateral ankle ligament injuries. Treatment methods include early mobilization, cast immobilization, and surgical repair.

Injection of anesthetics or corticosteroids into the acutely sprained ankle is never indicated. [296] [353] Aspiration of the joint hematoma and injection of hyaluronidase did not produce improved outcomes in one study. [59]

The ankle is supported by the use of a variety of methods, including a nonwalking cast, a walking cast, a removable cast boot, a semirigid pneumatic ankle brace, a nonrigid functional ankle brace, and various ankle taping methods. Various immobilization methods are summarized in Table 25C1-4 .

TABLE 25C1-4 -- Immobilization Methods for Foot and Ankle Injuries

Immobilization Method	Common Application	Advantage	Disadvantage
Short-leg nonwalking cast	Initial treatment for severe ankle and midfoot sprains; definitive treatment of stable syndesmosis and Lisfranc injuries	Excellent protection for all foot injuries and most ankle injuries; effective edema management	Poor rotational control for ankle syndesmosis; rapid deconditioning; inconvenient for dressing, showering, and sleeping
Short-leg walking cast	Initial treatment for severe ankle and moderate foot sprains	Excellent protection for most foot and ankle injuries; improved ability to bear weight; continuous rehabilitation provided;	Poor rotational control for the ankle syndesmosis; rapid deconditioning; inconvenient for dressing,

Immobilization Method	Common Application	Advantage	Disadvantage
		self-applied device	showering, and sleeping
Removable cast boot (3D Walker, Bledsoe Boot [Bledsoe Brace Systems, Grand Prairie, Tex], CAM boot)	Initial treatment for moderate ankle and foot sprains	Removable protection for ankle and foot injuries; improved ability to bear weight; continuous rehabilitation provided; self-applied device	No rotational control through ankle; poor edema control; athletic participation restricted
Semirigid pneumatic ankle brace (Air-Stirrup, Aircast, Summit, NJ)	Functional treatment for hindfoot and ankle injuries at various recovery phases, including acute ankle sprains,[51,58,63,66,72] prevention of recurrent ankle sprains[159,162-164]	Rigid support for hindfoot and ankle injuries—allows ankle range of motion, allows continued athletic participation, facilitates resolution of edema (air cell systems)[58]; self-applied device; device used within shoe; low cost	No rotational control through ankle; bulky within shoe
Nonrigid functional ankle brace (lace-up or Velcro closures)	Functional treatment for hindfoot and ankle injuries at various recovery phases, including chronic injuries	Nonrigid support for hindfoot and ankle injuries—allows ankle range of motion, allows continued athletic participation; self-applied device; device used within shoe	No rotational control through ankle
Ankle and foot taping	Functional treatment for foot and ankle injuries at various recovery phases, including chronic injuries	Custom-applied support for foot and ankle injuries; provides resistance to inversion,[64] provides biofeedback,[70] allows continued athletic participation	Rapid loosening with time-limited effectiveness[159]; requires trained personnel for application; high cost over the course of a season[158]

Cast Immobilization

Practically speaking, casting has a useful role when limited to the initial 2 to 3 weeks after acute injury. Application of a short leg cast allows a rapid return to work activities and early discontinuation of crutch walking. Published reports discuss casting periods as long as 6 weeks. Prolonged immobilization is not recommended. In fact, Jackson and colleagues suspect that the use of a cast for moderate and severe ankle sprains simply delays recovery for the number of days that the cast is used. [1]

Complete immobilization is provided with a short leg cast. The assumption is that casting allows the ruptured ends of the lateral ankle ligaments to heal in a near-anatomic position. Various cast positions have been advocated in an effort to reapproximate the torn ligament ends. Smith and Reischl suggested 5 to 15 degrees of dorsiflexion based on fresh cadaveric models. [299] [355] Slight eversion is also a common suggestion. [61] As soon as the swelling and pain remit, a functional rehabilitation program is instituted with a semirigid pneumatic ankle brace.

Drez and coworkers used a more protracted immobilization method to treat 39 patients with first-time, combined ATFL and CFL injuries. [61] All cases were evaluated by stress radiography before and after treatment. The protocol included 7 to 10 days in an everted splint and 6 weeks in an everted walking cast, followed by ankle rehabilitation and a 1-month delay in return to athletic activity. A 79.5% success rate was obtained as determined by a repeat talar tilt stress radiograph with 5 degrees or less angulation compared with the uninjured ankle.

Eiff and associates used a prospective study to compare early mobilization and immobilization for the treatment of first-time lateral ankle sprains. [62] The early mobilization group was treated with an elastic wrap for 48 hours followed by application of a semirigid pneumatic brace and a common rehabilitation program. The immobilization group was treated with a non–weight-bearing plaster splint for 10 days followed by a common rehabilitation program. Both methods produced excellent results at 1-year follow-up with low rates for residual symptoms (5%) and reinjury (8%).

A semirigid pneumatic ankle brace (Air-Stirrup, Aircast, Inc., Summit, NJ) has shown proven results with advantages that include cost-effective treatment, the capacity for independent rehabilitation, an early return to function, and predictable results (Fig. 25C1-10). [63] The semirigid orthosis is lined by opposing dual air cells; the system produces a milking action that actively reduces ankle edema. Several experimental studies have demonstrated the brace's effectiveness in reducing ankle inversion. [318] [359] [360]

Figure 25C1-10 **A,** Semirigid pneumatic ankle brace. **B,** Lace-up ankle brace.

Early Mobilization

Early mobilization, or functional treatment, is the current favored treatment method. Treatment and rehabilitation are directed without the use of rigid cast immobilization. After the acute pain and swelling remit (after about 48 hours), weight-bearing to tolerance is encouraged, and a rehabilitation program is instituted. This method specifically avoids immobilization, which many believe simply prolongs the recovery period. A semirigid pneumatic ankle brace, walking boot, or taping is used throughout the rehabilitation period.

Konradsen and colleagues used a prospective, randomized study to evaluate the effectiveness of early mobilization compared with total immobilization for complete lateral ankle ligament injuries. [51] Early mobilization allowed an earlier return to work and resumption of athletic activity. The 1-year follow-up was similar for both groups.

Klein and coworkers used a randomized study to evaluate the same methods. [66] Patients treated with a pneumatic ankle brace fared better than those treated with a short leg cast for 6 weeks. Stress radiographs did not significantly differ after treatment.

Sommer and Schreiber used a prospective, randomized study to compare plaster cast immobilization (for 3 weeks followed by a pneumatic ankle splint), early mobilization with a pneumatic ankle brace, and early mobilization with an Unna boot for the treatment of stress radiograph-documented lateral ankle ligament ruptures. [67] They reported lower direct cost and improved stability in the mobilization groups.

A recent study to evaluate the effectiveness of early mobilization was published by Povacz and associates in 1998. [68] The group performed a randomized prospective study of 146 adults with acute lateral ankle sprains diagnosed by clinical findings and stress radiographs. The treatment was either immediate operative repair followed by 6-week immobilization in a short leg cast or placement of an ankle orthosis for 6 weeks. Nonoperative treatment produced subjective and objective results comparable to those associated with operative treatment. The nonoperative treatment was also associated with a significantly shorter recovery period.

Surgical Repair

Surgical management of the acute unstable lateral ankle sprain remains controversial. Several authors have recommended primary surgical repair for severe injury or injury in the high-demand athlete (Fig. 25C1-11). [336] [364]

[365] [366] [367] Kaikkonen and colleagues reported excellent and good results at a 6- to 8-year follow-up after primary repair of acute lateral ligament rupture. [73] No operative complications occurred. Others have reported favorable, reproducible results with primary repair of acute ruptures. [306] [336] [369]

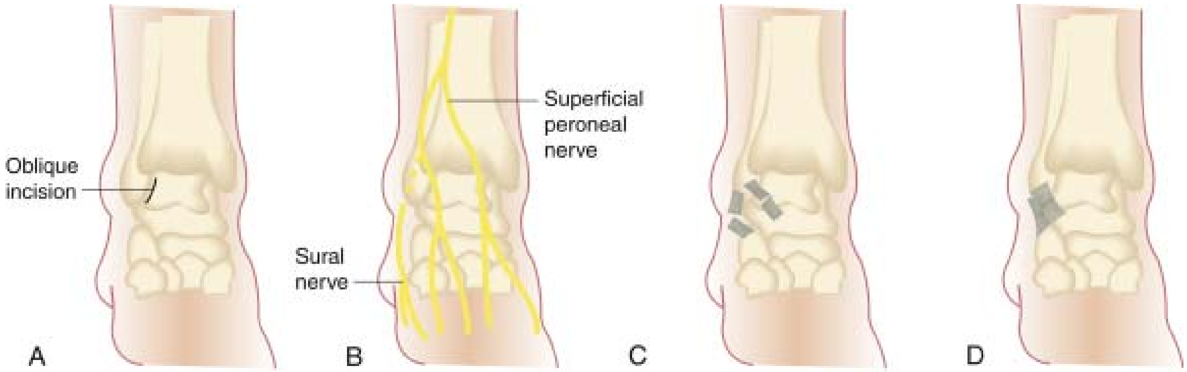

Figure 25C1-11 Surgical technique for primary lateral ankle ligament reconstruction. **A,** A short oblique incision is made at the anterior margin of the distal fibula. **B,** Blunt subcutaneous dissection is performed, with care taken to protect the superficial peroneal nerve and the sural nerve. **C,** The anterior lateral capsule is exposed, and the anterior talofibular ligament, calcaneofibular ligament, and capsular tears identified. **D,** The tears are approximated and repaired with absorbable suture.

Most physicians currently agree that the role of surgery in the acute setting is very small. Rare circumstances, such as open injuries, large avulsion fractures, and frank dislocation may warrant acute lateral ligament repair.

Some authors have reported on adverse outcomes with acute surgical treatment. Freeman compared strapping and early mobilization, immobilization with plaster for 6 weeks, and ligament repair with immobilization for the treatment of complete lateral ankle ligament ruptures. [75] The periods of disability were 12, 22, and 26 weeks for patients in the early mobilization, immobilization, and ligament repair groups, respectively. Ligament repair produced the greatest number of complaints at 1-year follow-up, including the only cases with residual loss of motion. Freeman suggested early mobilization as the treatment of choice for lateral ankle ligament ruptures.

Broström used a prospective study to compare the use of primary surgical repair followed by 3 weeks of plaster immobilization (95 cases), 3 weeks of plaster immobilization alone (82 cases), and strapping with early mobilization (104 cases). [41] Although surgical repair provided excellent results, including a low (3%) residual symptomatic instability rate, Broström went on to suggest that primary surgical repair should not be the routine treatment for acute ruptures. He cited the protracted postoperative recovery, the risk for infection, the risk for painful scar formation, and the success of late lateral ankle ligament reconstruction.

Evans and coworkers recognized the discrepancy in immobilization periods and performed a prospective, randomized trial with 3 weeks of immobilization in a plaster cast with or without surgical repair of the acute lateral ankle ligament rupture. [76] An independent 2-year follow-up concluded that surgical repair yielded similar radiographic results (stress radiographs) and slightly worse functional results. Twice as many patients in the operative group were forced to give up athletic activity. Loss of subtalar inversion and surgical complications were also cited as disadvantages of primary repair of acute injury.

Repair of acute injury produces results similar to those associated with delayed reconstruction. Because most patients respond to nonsurgical management, primary repair offers few advantages. Today, operative repair of the complete, acute lateral ankle ligament injury is very infrequent. [58] Most authors favor nonsurgical treatment for acute ruptures. [336] [370] [372] [373]

Technique: Primary Lateral Ankle Ligament Repair

1. The procedure is performed with the patient under general anesthesia. The patient is supine with a well-padded proximal tourniquet and a soft bump placed beneath the ipsilateral hemipelvis.
2. An image intensifier is used to obtain bilateral varus tilt and anterior drawer stress views. Brodén's stress views are also obtained, if indicated.
3. Arthroscopy of the ankle is performed if clinical presentation or imaging suggests intra-articular disease.

4. The extremity is exsanguinated, and the tourniquet is inflated. A bump is placed under the leg to prevent anterior translation of the talus.
5. A short oblique incision is made at the anterior margin of the distal fibula. This incision incorporates the lateral arthroscopy portal.
6. Blunt subcutaneous dissection is performed, with care taken to protect the superficial peroneal nerve and the sural nerve.
7. The anterior lateral capsule is exposed, and the ATFL, the CFL, and capsular tears are identified. The ankle joint is inspected for chondral or osteochondral fragments. The joint is copiously irrigated. The peroneal sheath is evaluated for tears.
8. The tears are approximated and repaired with absorbable suture. All sutures are placed and then tied from posterior to anterior with the ankle held in neutral dorsiflexion and slight eversion. Avulsion fractures are repaired directly to the fibula or talus.
9. The wound is closed in layers, and a short leg splint is applied. At 10 days, the incision is inspected, and a short leg weight-bearing cast or walking boot is applied. Four to 6 weeks after surgery, the patient is transitioned to an ankle brace. A guided rehabilitation program is instituted and monitored. Patients may actively dorsiflex and evert the foot with inversion limited to neutral. Progressive stretching, strengthening, and proprioception exercises are instituted 6 to 8 weeks after surgery. A semirigid pneumatic ankle brace is used for 6 months after surgery.

Chronic Lateral Ankle Ligament Instability

The chronic or recurrent lateral ankle sprain is associated with apprehension, discomfort, swelling, muscular weakness, tenderness, and loss of coordination. [325] [339] Instability may be overt or subtle and has been described as a “giving way” of the ankle. [30] Significant disability is noted, particularly if the patient runs on uneven or loose surfaces. Objective instability of the ankle joint is defined by patient symptoms and positive stress radiographic findings. The condition develops after acute injury in up to 20% of patients. [336] [372] [374]

A related condition is functional lateral ankle instability, defined by frequent sprains, difficulty running on uneven surfaces, and difficulty jumping and cutting. Freeman reported this sensation in 21 of 42 patients 1 year after initial lateral ankle ligament rupture. [80] Increased varus tilt on stress radiograph, was present in only 6 of these cases. Hansen and coworkers noted a similar lack of agreement between clinical symptoms and persistent talar tilt. [81] Brand and colleagues reported a 10% prevalence of functional lateral ankle instability among 1300 Naval Academy freshmen. [82] Functional instability may be related to previous ankle sprain, chronic lateral ankle instability, or peroneal weakness.

Rehabilitation without Surgery

Successful resolution of chronic lateral ankle instability is possible without surgical stabilization. [339] [378] [379] Patients with chronic, recurrent injuries are treated with a rehabilitation program, and their activity levels are reduced. Functional ankle bracing is continued throughout the treatment period. The key points of rehabilitation are motor strength (particularly of the peroneal muscles), proprioception, and coordination.

Surgical Treatment

Patients with chronic injuries that remain symptomatic after a supervised rehabilitation program are candidates for surgical management. Radiographic criteria include talar tilt greater than 15 degrees (or a side-to-side difference of more than 10 degrees) and anterior drawer translation greater than 5 mm (or a side-to-side difference of more than 3 mm). A multitude of procedures have been used for lateral ankle ligament reconstruction, many with reasonable success.

In 1932, Nilsonne described a procedure for stabilization of the lateral ankle. [85] He repaired a chronic CFL rupture and reinforced the repair with a peroneus brevis tenodesis. Evans [381] [382] [383] and Watson-Jones [379] [384] modified the tenodesis. A further modification in the form of an augmentation procedure was proposed by Elmslie [90] and refined by Chrisman and Snook. [91]

The *Evans procedure* is a transposition of the entire peroneus brevis tendon through the distal fibula (Fig. 25C1-12). Evans recognized the tenodesis resulted in the loss of subtalar inversion after this procedure. [87]

Figure 25C1-12 The Evans procedure.

The *Watson-Jones procedure* reconstructs the lateral ankle ligaments with a peroneus brevis tenodesis configured to replace the course of the ATFL (Fig. 25C1-13). [379] [384] The classic method uses the entire peroneus brevis tendon. [92] The procedure does not reproduce the anatomic orientation of the CFL. Because the tenodesis of the peroneus brevis lies at a right angle to the subtalar joint empirical axis, loss of subtalar motion is to be expected. [10]

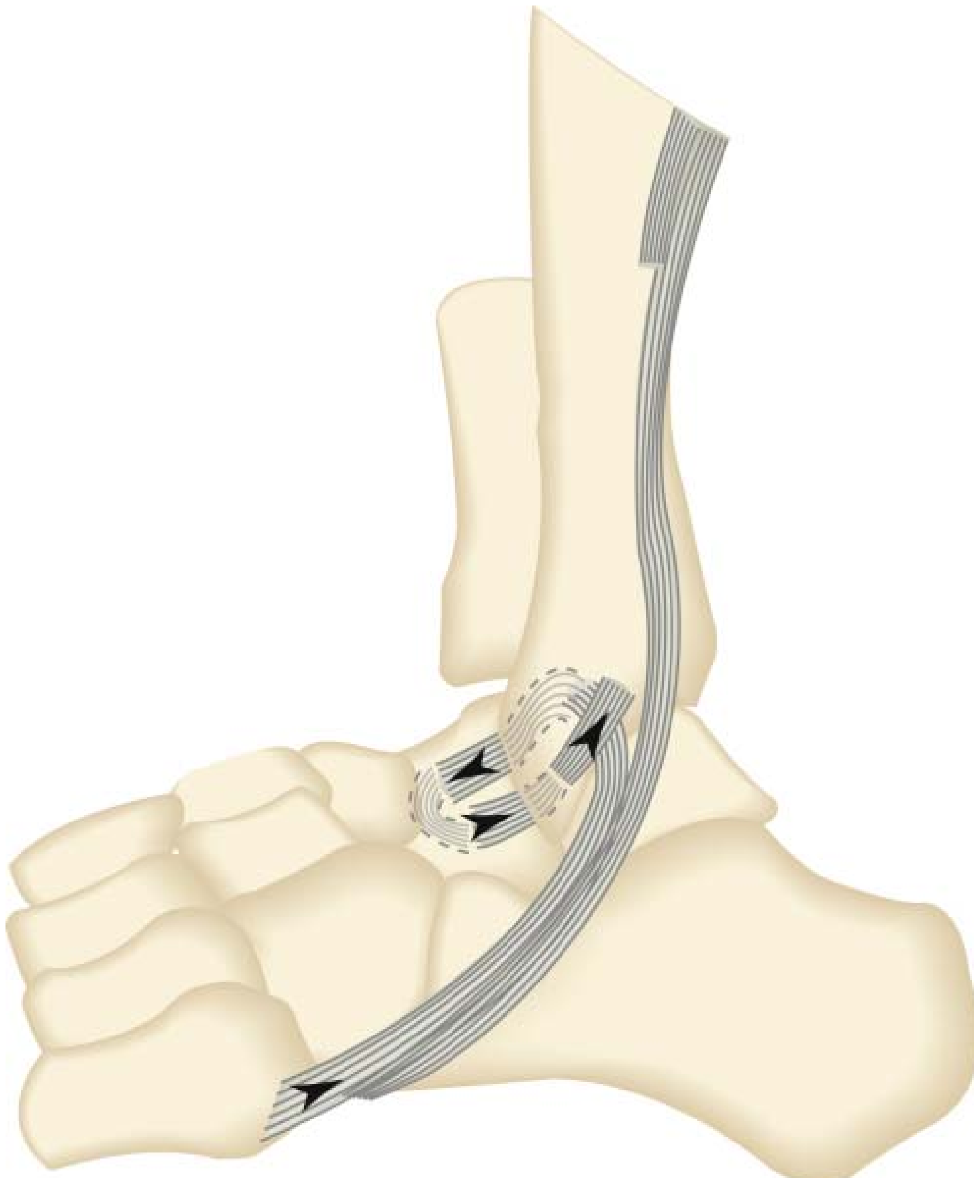

Figure 25C1-13 The Watson-Jones procedure.

The Watson-Jones procedure has been modified to spare the function of the peroneus brevis tendon with the use of a split peroneus longus tendon graft, [328] [388] a complete peroneus longus tendon graft, [94] a split peroneus brevis tendon graft, [328] [390] a plantaris tenodesis, [96] a free plantaris tendon graft, [329] [392] and a split Achilles tenodesis. [393] [394]

Using the split peroneus longus modification of the Watson-Jones procedure, Barbari and associates reported excellent or improved outcomes in 39 of 42 ankles (Fig. 25C1-14). [93] Problematic issues include loss of ankle dorsiflexion and subtalar inversion.

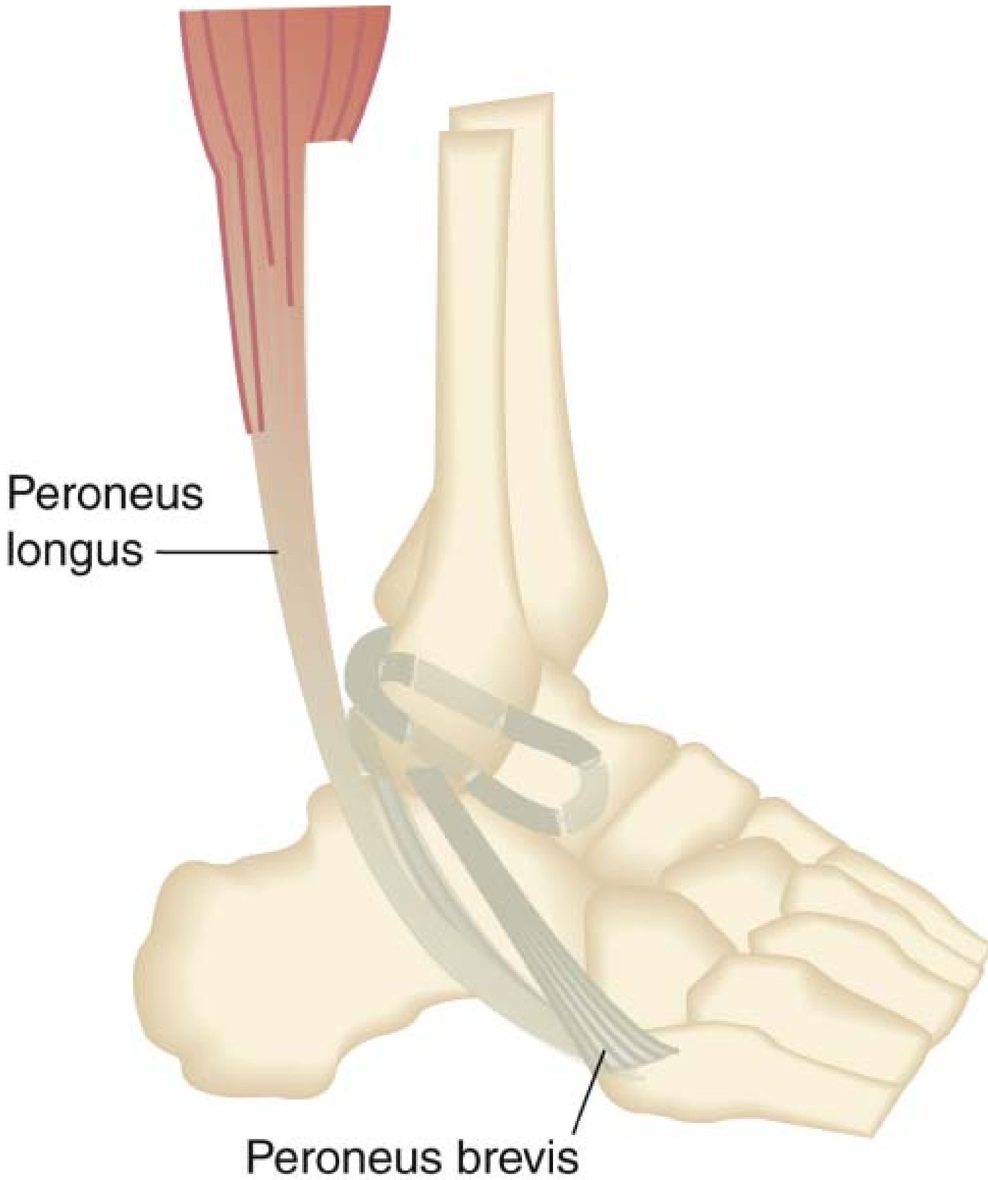

Figure 25C1-14 The Barbari modification of the Watson-Jones procedure. *(Redrawn from Barbari SG, Brevig K, Egge T: Reconstruction of the lateral ligamentous structures of the ankle with a modified Watson-Jones procedure. Foot Ankle 7:362-368, 1987.)*

Brunner and Gaechter completed a retrospective comparison of the split peroneus brevis modification with the free plantaris tendon graft modification of the Watson-Jones procedure. [34] This study demonstrated slightly more favorable results with the plantaris tendon graft. Increased patient satisfaction and fewer reoperations were noted. The free plantaris graft modification is a more anatomic approach to addressing lateral ligament failure, particularly of the CFL. This procedure preserves the function of the peroneus brevis while sacrificing the vestigial [100] plantaris.

In 1934, Elmslie described the use of a fascia lata graft passed through drill holes to re-create and augment the ATFL and the CFL. [90] The idea of re-creating the anatomic configuration of the ATFL and CFL was adopted by Windfeld. [101] He used the entire peroneus brevis to perform a modified Watson-Jones repair. The peroneus brevis tendon was passed through the fibula from front to back and sewn to the remnants of the ATFL at the talus and the CFL at the calcaneus. The procedure limited the graft to local tissue and attempted to reconstruct both lateral ligaments.

In 1969, Chrisman and Snook modified the Elmslie repair by using a split peroneus brevis tendon, instead of a strip of fascia lata, to reconstruct the ATFL and the CFL (Fig. 25C1-15). [91] The 2-year follow-up of seven patients confirmed a successful return to strenuous athletic activity in all participants. Although moderate loss of subtalar motion occurred, it was not problematic. In 1985, the same authors published a long-term follow-up (average follow-up, 10 years). [102] Of the 48 ankles evaluated, 43 had attained excellent and good outcomes. Of the 3 patients with fair and poor results, all had sustained subsequent severe trauma, and one had suffered from generalized ligamentous laxity. Other complications included sural nerve injuries and an asymptomatic loss of inversion.

Figure 25C1-15 The Chrisman-Snook procedure.

Riegler reported the 2-year follow-up of 11 young athletes after a Chrisman and Snook reconstruction. [103] Ten patients returned to their primary athletic activity. All patients lost inversion of the subtalar joint compared with the uninvolved side. Savastano and Lowe published similar results. [104] Among 10 ankles treated with a Chrisman and Snook reconstruction, 9 obtained satisfactory results despite limited subtalar inversion.

Colville and coworkers performed a biomechanical comparison of the Evans, Watson-Jones, and Chrisman and Snook reconstructions. [105] It was determined that each procedure stabilized the ankle but also produced limited subtalar inversion.

Colville and Grondel described an anatomic split peroneus brevis lateral ankle reconstruction. [106] This procedure reproduces the anatomic orientation of the ATFL and the CFL. The peroneus tendon function appears to remain intact; furthermore, the anatomic approach preserves subtalar function (80% of nonoperated hindfoot inversion). Mechanical stability was verified with stress radiographs in all 17 patients. Minor difficulties with aching and swelling persisted at the 42-month average follow-up. The authors advocate the technique for patients with inadequate local tissue for anatomic reconstruction, generalized hypermobility, and previously failed reconstructive techniques.

Paterson and associates described an anatomic reconstruction of the ATFL with a semitendinosus autograft.[107] The average 24-month follow-up suggests that the reconstruction is useful for instability not associated with the subtalar joint. Kin-Com dynamometer testing revealed no significant loss of knee flexion strength.

Anderson and Lecocq reported a successful delayed repair of the ATFL and CFL with simple plication.[13] Broström advocated a similar approach (Fig. 25C1-16).[44] His report of successful delayed lateral ankle ligament repair remains significant. Sixty patients with chronic symptoms after lateral ankle sprain were treated with end-to-end ligament repair or advancement of the torn end of the ATFL into the anterior border of the fibula. In 3 of these cases, the repair was fortified with a flap of the lateral talocalcaneal ligament. Average follow-up was 2.9 years. Forty-three patients were completely asymptomatic. Only 3 patients (5%) complained of severe or moderately severe symptoms. Forty-six of the 56 patients who were re-examined were “normal.” Abnormal findings included positive anterior drawer sign (4), impaired mobility (2), tenderness (5), and soft tissue induration (4). Broström’s technique has several advantages over tenodesis and augmentation methods. The technique accomplishes anatomic restoration and maintains joint mobility, it is relatively simple, and it is less morbid with regard to the use of peroneal tendon grafts and length of incision. Other authors have obtained similar good results.[108]

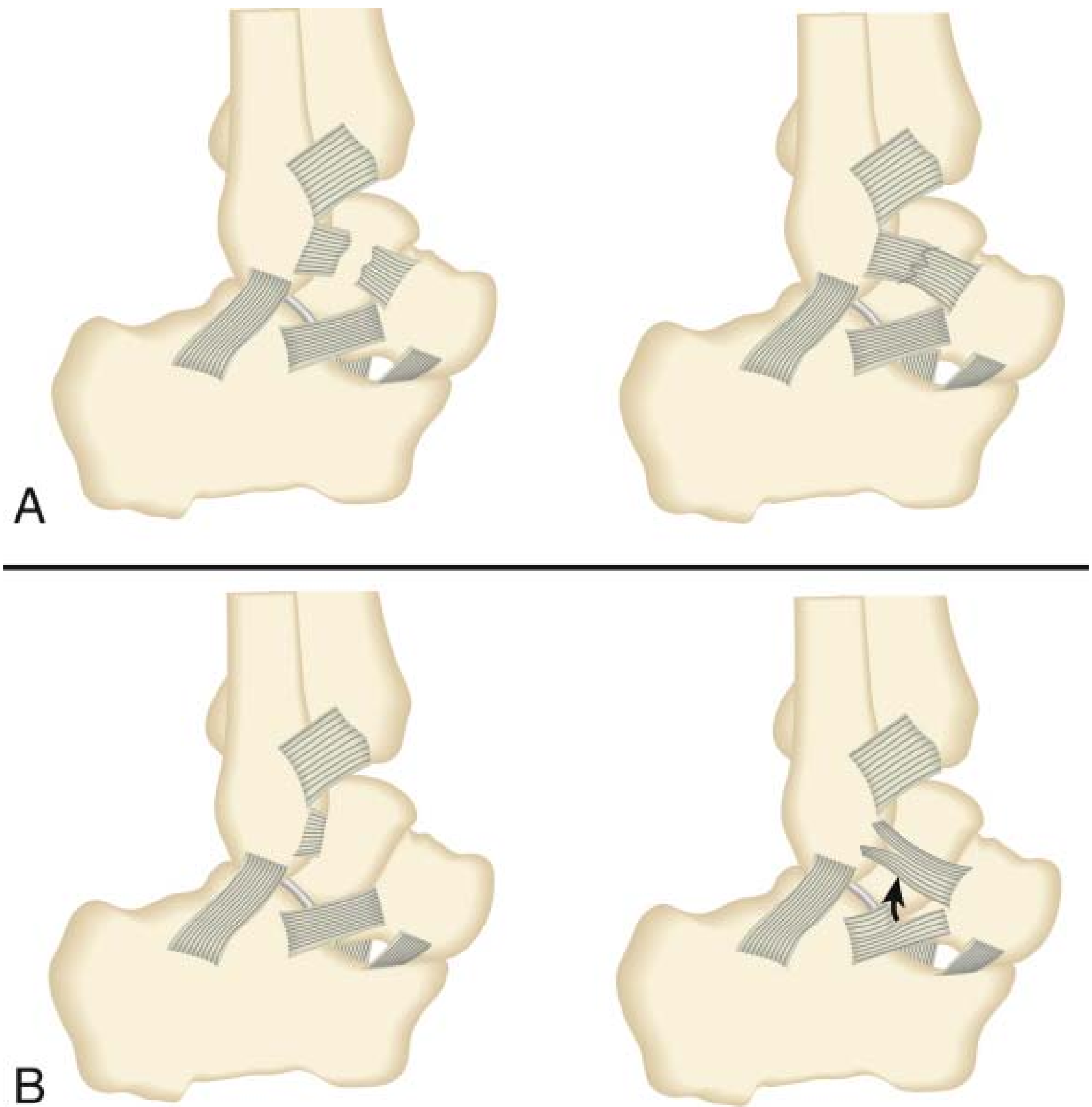

Figure 25C1-16 The repair of a chronic lateral ankle ligament rupture as described by Broström. **A,** Chronic anterior talofibular ligament (ATFL) rupture and direct repair. **B,** Chronic ATFL rupture with insufficient tissue for simple direct repair and reconstruction using ATFL repair with advancement of the flap of the lateral talocalcaneal ligament into the fibula. *(Redrawn from Broström L: Sprained ankles. VI. Surgical treatment of “chronic” ligament ruptures. Acta Chir Scand 132:551-565, 1966.)*

Gould and associates recommended modification of the Broström technique.[109] They removed the intervening scar from the ATFL, repaired the ligament, and supplemented the repair with both a flap from the lateral talocalcaneal ligament and an advancement of the inferior extensor retinaculum (Fig. 25C1-17). Late repair was performed on 50 patients. The anterior translation of the talus was reduced to 2 mm or less, and talar tilt to less than 12 degrees. All patients returned to athletic activity. A rating scale based on activity, stability, mobility, swelling, and overall satisfaction revealed that all patients scored between 8 and 10 of 10 possible points.

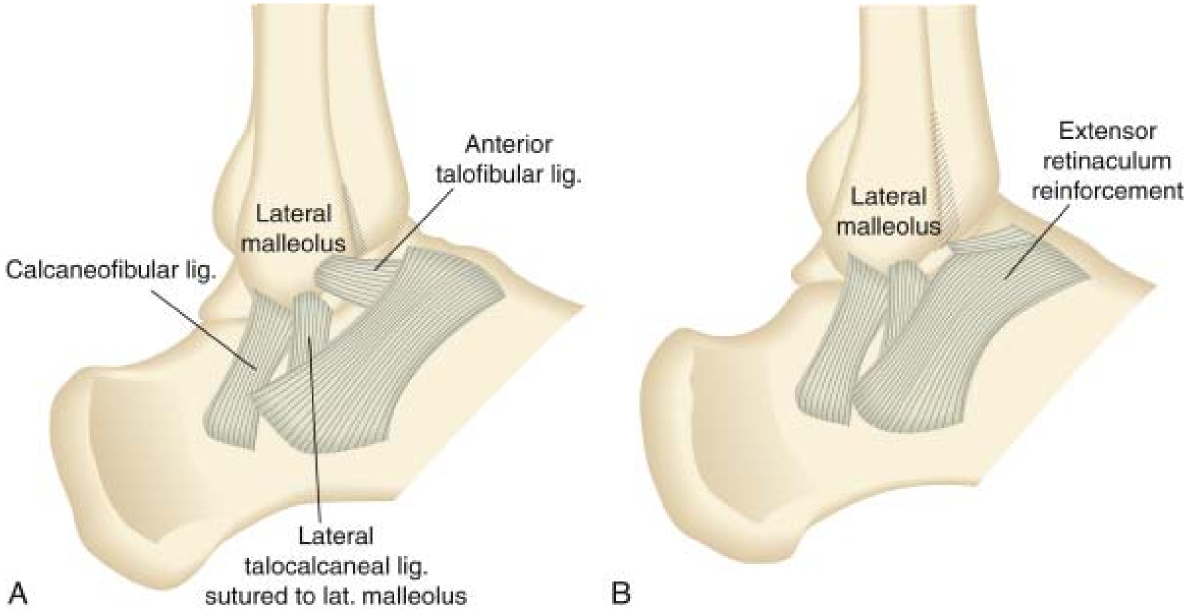

Figure 25C1-17 Gould modification of the Broström technique. After repair of the anterior talofibular or calcaneofibular ligament, reinforcements with the lateral talocalcaneal ligament (**A**) and extensor retinaculum (**B**) are made.

The inferior extensor retinaculum is composed of two layers, superficial and deep to the extensor tendons. Harper concluded that the superficial layer of the inferior extensor retinaculum remains a constant, substantial tissue, suitable for lateral ankle and subtalar joint reconstructions, as proposed by Gould and associates. [404] [405]

Biomechanical testing with cadaveric ankle has proved the efficacy of the Broström repair in restoring ankle stability while maintaining ankle and subtalar range of motion. [111] Others have used the Gould modification of the Broström repair with consistent reproducible stabilization and return to athletic activity. [112] Harper further modified the Gould modification by using a flap fashioned from the superficial layer of the inferior extensor retinaculum (Fig. 25C1-18). [113]

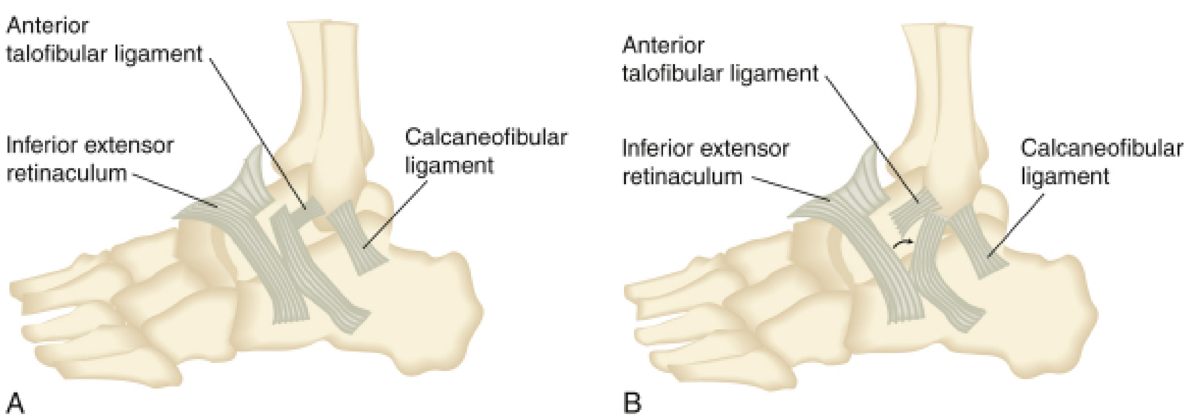

Figure 25C1-18 Harper modification of the Gould modification of the Broström technique. A flap from the inferior extensor retinaculum is mobilized and sutured to the lateral fibula. *(Redrawn from Harper MC: Modification of the Gould modification of the Broström ankle repair. Foot Ankle Int 19:788, 1998.)*

When a Broström repair is performed, identification of the torn ligament ends is often difficult, if not impossible. To bypass this difficulty, Karlsson and colleagues used division and imbrication of the attenuated ATFL and CFL. [114] They reported good to excellent results in 132 of 153 ankles available for follow-up (mean, 6 years). Reconstruction of both the ATFL and the CFL produced better results than did isolated reconstruction of the ATFL. Most patients with unsatisfactory results were noted to have generalized hypermobility, longstanding lateral ankle instability, or a history of previous ankle reconstruction.

Karlsson and coworkers performed 60 lateral ankle ligament reconstructions with a modification of the Broström repair. [115] These authors advanced the ATFL and the CFL into a 4 × 4-mm bone trough at the anterior aspect of the distal fibula. The remaining periosteal flap was repaired over the ATFL. Good or excellent results were obtained in 53 (88%) of the patients. Unsatisfactory results were associated with generalized ligamentous laxity or longstanding instability.

Despite the many surgical options, ankle stabilization is effective and produces good or excellent results in more than 91% of chronic lateral ankle ligament instability cases. [411] [412] Radiographic stability does not always correlate with clinical outcome. Patients with early signs of osteoarthritis may experience progressive arthrosis despite stabilization. Persistent instability leads to osteoarthritis of the ankle joint. [326] [327] [328]

Ankle arthroscopy at the time of lateral ankle ligament reconstruction provides the surgeon with an opportunity for a more thorough evaluation of the ankle joint. Komenda and Ferkel suggest the use of ankle arthroscopy before lateral ankle ligament stabilization for treatment of loose bodies, osteochondral lesions of the talus, and ankle pain unrelated to instability. [118] They also suggest the procedure for evaluation of ATFL integrity and suitability of local tissue for reconstruction.

Patients should be carefully evaluated for severe ankle or hindfoot cavovarus. A calcaneal osteotomy may need to be performed in conjunction with lateral ligament repair to prevent failure of the procedure.

Patients with failed lateral ankle reconstruction due to re-rupture are placed into rehabilitation and fitted with an orthosis. Repair of the acute injury is a reasonable alternative to functional treatment. Persistent instability can be treated with a repeat Broström repair with the Gould modification, or an anatomic reconstruction with local tissue or a free tendon graft. [119]

Technique: Lateral Ankle Ligament Reconstruction with a Gould Modification of the Broström Repair

1. The procedure is performed with the patient under general anesthesia. The patient is supine with a well-padded proximal tourniquet and a soft bump placed beneath the ipsilateral hemipelvis.
2. An image intensifier is used to obtain bilateral varus tilt and anterior drawer stress views. Brodén's stress views are also obtained, if indicated.
3. Arthroscopy of the ankle is performed if clinical presentation or imaging suggests intra-articular disease.
4. The extremity is exsanguinated, and the tourniquet is inflated. A bump is placed under the leg distally to prevent anterior translation of the talus.
5. A short oblique incision is made at the anterior margin of the distal fibula. The incision incorporates the lateral arthroscopy portal if used.
6. Blunt subcutaneous dissection is performed, with care taken to protect the superficial peroneal nerve and the sural nerve. The extensor retinaculum is identified and tagged.
7. The anterior lateral capsule is exposed, and the ATFL and the CFL are identified. The capsule is incised just distal to the origin of the ATFL and the CFL, and the ankle joint is inspected.
8. The anterior margin of the fibula is exposed by subperiosteal elevation of the proximal capsule. The anterior fibula is decorticated, and three to five tunnels are created with a small K-wire. The capsule with the ATFL and the CFL is secured with multiple nonabsorbable braided sutures. The sutures are passed through the tunnels, and the capsule is advanced onto the decorticated margin of the fibula with the ankle held in dorsiflexion and eversion. The lateral periosteal sleeve and the proximal capsule are repaired over the advanced ligament. This repair can also be performed with suture anchors in the fibula or by soft tissue imbrication.
9. The superficial layer of the extensor retinaculum is secured with suture and advanced to the anterior fibula. This provides significant subtalar stability.

10. The wound is closed in layers, and a short leg splint is applied in slight eversion. At 10 days, the incision is inspected, and a short leg weight-bearing cast is applied. Four weeks after surgery, a rehabilitation program is instituted and monitored. A removable cast boot or a semirigid pneumatic ankle brace is used for an additional 4 to 6 weeks. The repair is further protected during subsequent athletic activity with an ankle brace for 6 months after surgery.

Rehabilitation after Surgery

Tissue injury initiates a predictable and sequential series of events known as the *healing response*. The response is typically divided into three phases with arbitrary and overlapping time lines.[120] The initial phase is the inflammatory phase, which includes the first through the third day following injury. The second phase is a proliferative phase of tissue repair that extends from day 3 to day 20. The final phase is a remodeling phase that proceeds after day 9.

To a certain degree, rehabilitation follows the phases of the healing response in an effort to reduce the undesirable effects of inflammation (i.e., pain, swelling, loss of function) while simultaneously promoting tissue repair and functional recovery. For rehabilitation of the ankle, emphasis is placed throughout the protocol on ankle and subtalar flexibility, motor function, and coordination.[30] The ankle is supported by a semirigid pneumatic ankle brace. In addition to orthotic devices, an elastic sock is available for additional mobilization of edema (Fig. 25C1-19).

Figure 25C1-19 Elastic sock used for foot and ankle edema mobilization and control.

In the acute phase, the athlete's pain and inflammation are addressed with rest, cold therapy, and whirlpool. A trial of electrical stimulation may be considered. Ankle and subtalar joint passive and active range of motion are re-established with inversion limited to neutral for 6 weeks. Isometrics around the ankle and subtalar joints is initiated as pain allows. Weight-bearing to tolerance is encouraged.

Once the acute pain subsides, flexibility is addressed in all planes. An inclined board is a useful adjunct to gastrocnemius-soleus and Achilles stretching (Fig. 25C1-20). Strengthening is initiated with towel scrunches (Fig. 25C1-21), toe pick-up activities, manual resistive inversion and eversion, elastic bands (Fig. 25C1-22), seated toe and ankle dorsiflexion with progression to standing, and seated supination-pronation with progression to standing.

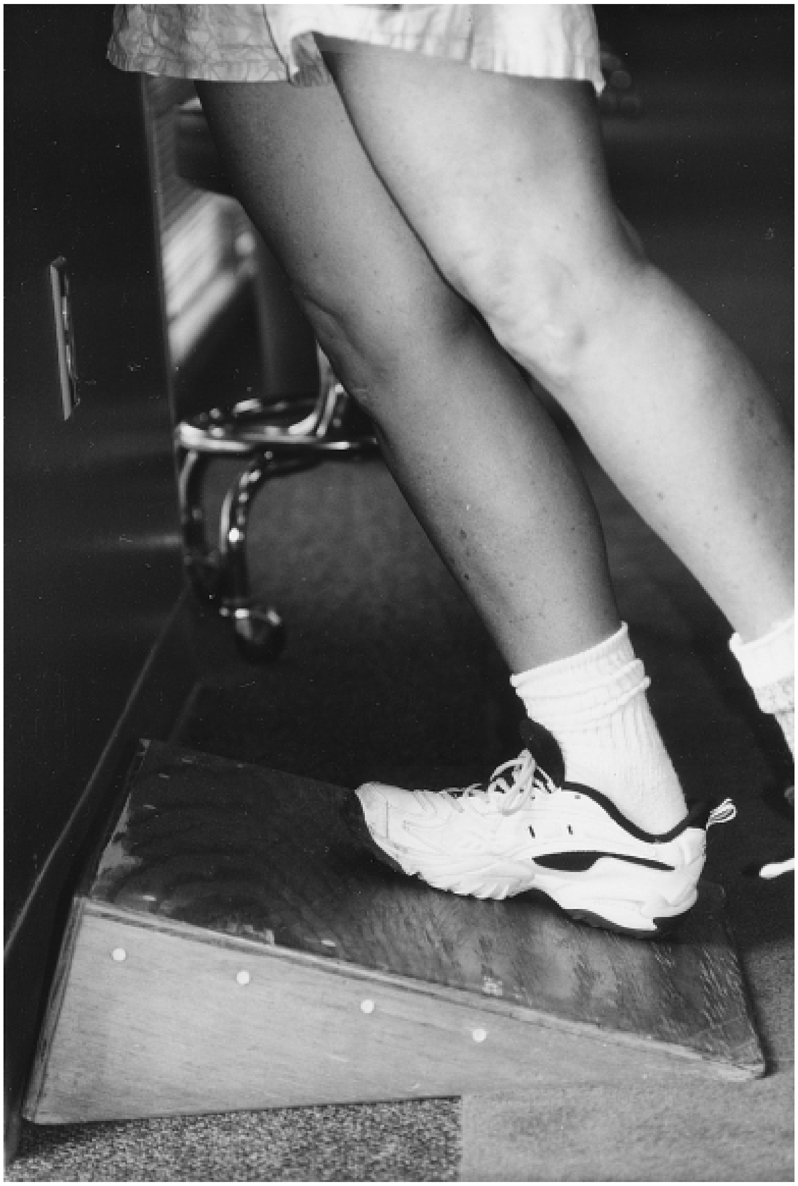

Figure 25C1-20 Gastrocnemius-soleus and Achilles stretching is facilitated with an inclined board.

Figure 25C1-21 Towel scrunches. The towel is gathered beneath the foot with active toe motion. The activity begins in a seated position and progresses to standing.

Figure 25C1-22 Elastic band exercises. The Thera-Band is posted on a table leg and is used to provide resistance as the foot is exercised. **A,** Elastic band exercises for resisted inversion. **B,** Elastic band exercises for resisted eversion.

Closed chain activities are gradually introduced, including one-leg balance and sport-specific activities on a trampoline, as well as use of the biomechanical ankle platform system (BAPS) (Fig. 25C1-23). Aerobic fitness is maintained with cross-training activities such as water running (Fig. 25C1-24) and cycling.

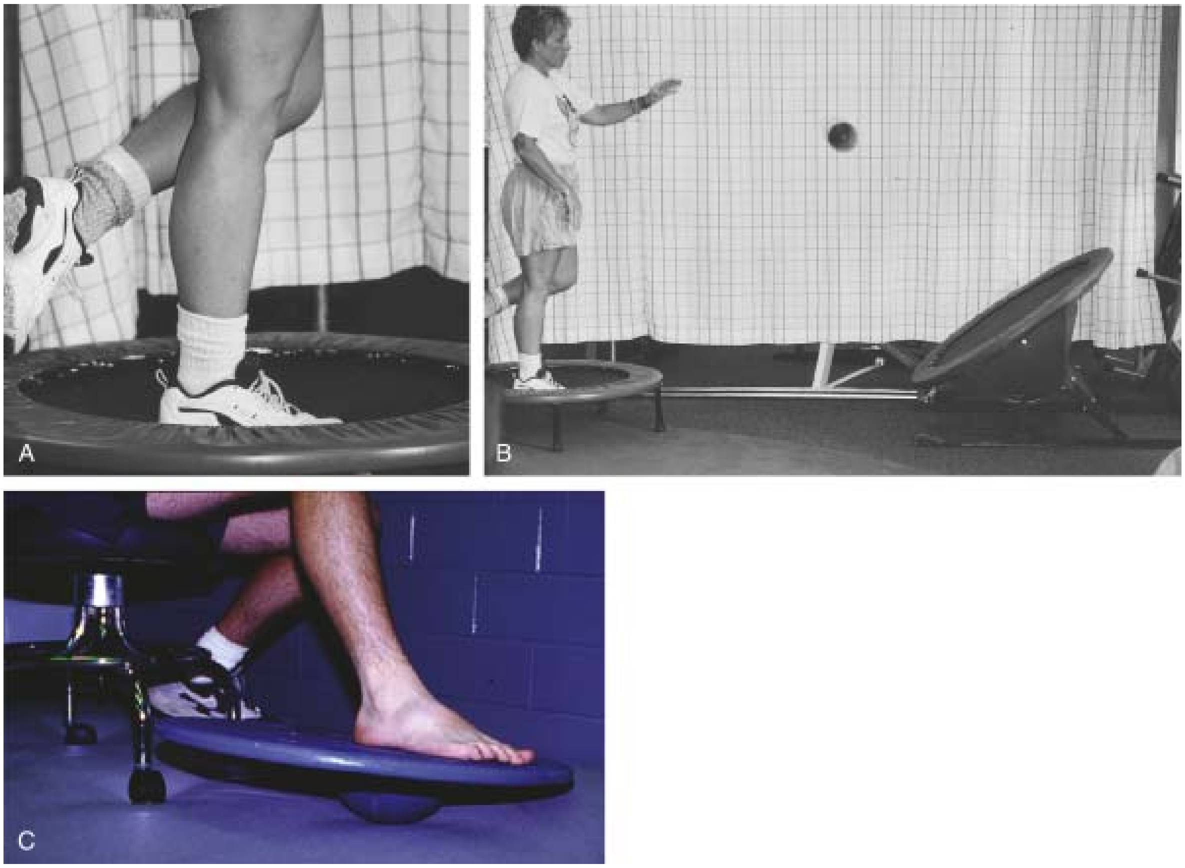

Figure 25C1-23 Closed chain activities. **A,** One-leg balance on a trampoline. **B,** Sport-specific activity (throwing) on a trampoline. **C,** Biomechanical ankle platform system.

Figure 25C1-24 Water running for cross-training after foot or ankle injury.

Heat therapy, such as the application of warm packs, is a useful modality before the therapy session. It reduces pain and spasms and thus facilitates increased range of motion. Cold therapy, compression, and elevation are used after each therapy session to reduce inflammation.

As the athlete returns to sport, protective bracing, range of motion, and strength activities are continued from the subacute phase. Walking and running activities are allowed to progress within the limits of a pain-free schedule. Once running activity is mastered, a monitored plyometric (cutting) program is introduced with progressive difficulty. Schedules are carefully controlled to avoid reinjury during these activities.

Associated Injuries

If a lateral ankle ligament injury is suspected either by the reported mechanism of injury (inversion mechanism) or by the initial physical findings (lateral ankle or hindfoot swelling), a multitude of diagnoses must be considered. These may present as isolated findings or as concomitant injuries. Evaluation begins with a detailed history and physical examination with particular attention to the ankle and hindfoot.

Osteochondral Lesion

Anderson and Lecocq reported a 22% incidence of osteochondral lesions in a mixed series of 27 cases of single and recurrent lateral ankle injuries. [13] These lesions were located at the lateral talus in five patients and at the medial talus in one patient.

Meyer and associates reported the successful use of CT in the evaluation of the chronically painful ankle after ankle sprain. [121] These scans demonstrated intra-articular or juxta-articular bony fragments in 13 of 31 patients. The fragments were located in the anterolateral or lateral aspect of the ankle or subtalar joints in 12 of the 13 patients.

Recurrent lateral ankle instability is associated with repetitive shear and compression forces across the ankle articular surface. Taga and colleagues used ankle arthroscopy to evaluate chondral lesions before lateral ankle ligament reconstruction in 31 patients. [122] Articular cartilage damage was seen in 89% of the acute ankle injuries and 95% of the chronic ankle injuries. They determined that cartilage damage most frequently was located at the anteromedial tibial articular surface. Furthermore, lesions tended to worsen with regard to depth of injury as the period of ankle instability lengthened. The location and severity of the cartilage damage as seen with the arthroscope correlated with clinical findings. The significance of the treated and untreated chondral lesions associated with a previously unstable ankle remains unknown.

Komenda and Ferkel also used ankle arthroscopy to evaluate ankles before lateral ankle ligament reconstruction for chronic instability in 54 patients.[118] Intra-articular disease, such as loose bodies, synovitis, and osteophytes, was seen in 93% of the ankles. Osteochondral lesions of the talus or chondromalacia, or both, were found in 25% of patients.

Options for treating osteochondral injuries include conservative and surgical approaches. Nonsurgical treatment includes rehabilitation, bracing, oral anti-inflammatory medications, and intra-articular steroid injection. When these treatments fail, surgery is considered. Various techniques, including arthroscopy with débridement, drilling, microfracture, internal fixation, and cartilage transfer, are used. Rehabilitation depends on the procedure performed.

Bone Bruise

With the advent of MRI, subtle injuries to the subchondral bone are easily imaged. Mink and Deutsch described a bone bruise as a traumatic, nonlinear marrow lesion localized to the subchondral bone, typically represented by a T1-weighted signal loss and a T2-weighted signal intensity.[123] The natural history of bone bruise is not completely understood and remains controversial. Isolated bone bruise subsequent to knee injury has a predictable and benign course.[124] Lahm and coworkers reported no arthroscopic changes (and no clinical sequelae) related to bone bruise associated with various knee injuries.[125] Conversely, Johnson and associates identified arthroscopic (e.g., softening, fissuring, and fracture) and histologic (e.g., chondrocyte and osteocyte necrosis) changes that suggested significant damage to the cartilage overlying bone bruise associated with anterior cruciate ligament disruption.[126]

Nishimura and colleagues and Labovitz and Schweitzer suggested that the bone bruise is an indicator of concomitant ligamentous injury to the ankle and that the pattern and location of the lesion correlate with a specific mechanism of injury.[422] [423] Alanen and coworkers used a prospective study to establish a 27% incidence of bone bruise (i.e., microtrabecular fracture) after ankle inversion injury.[129] Ninety-five patients with otherwise normal radiographs were imaged. No clinical significance was related to the occurrence of the bone bruise.

Anterior Lateral Ankle Impingement

Chronic anterior lateral ankle pain after an inversion ankle injury is a well-recognized entity. Bassett and associates described a distal fascicle of the anterior-inferior tibiofibular ligament; the structure was found in 10 of 11 cadaveric specimens[130] (Fig. 25C1-25). Impingement of the fascicle against the talar dome occurs with ankle dorsiflexion between 9 and 17 degrees. The clinical component of the study identified abrasion of the talar articular cartilage in five of the seven patients. Resection of the fascicle was curative and did not increase ankle instability.

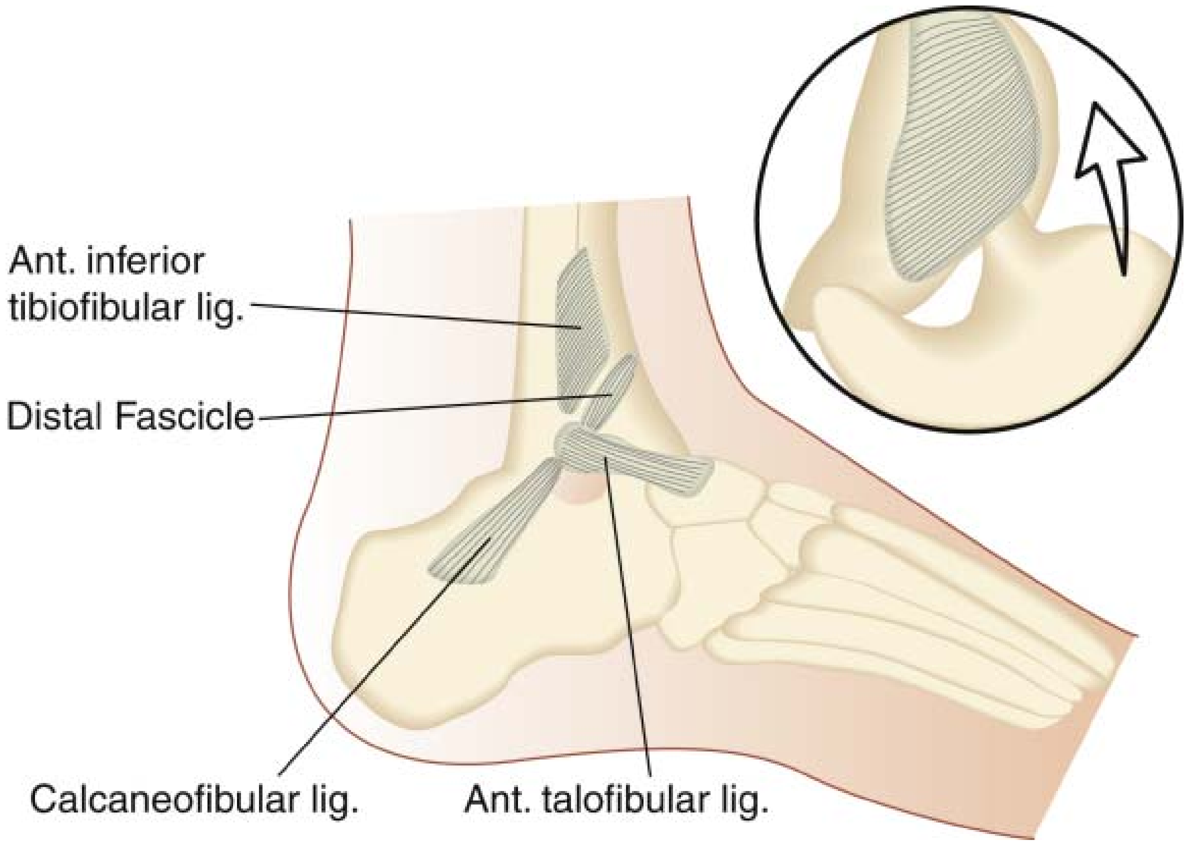

Figure 25C1-25 The distal fascicle of the anterior-inferior tibiofibular ligament is normally parallel and distal to the main ligament and separated from it by a fibrofatty septum. *Inset,:* After an inversion sprain of the ankle, the distal fascicle may impinge on the anterolateral aspect of the talus. *(Redrawn from Bassett FH, Gates HS, Billys JB, et al: Talar impingement by the anteroinferior tibiofibular ligament: A cause of chronic pain in the ankle after inversion sprain. J Bone Joint Surg Am 72:55-59, 1990.)*

A meniscoid lesion of the anterior lateral ankle has also been described (Fig. 25C1-26). [131] Hamilton reported finding entrapment of the capsule between the talus and the lateral malleolus during the surgical management of two of three acute, high-grade, lateral ankle sprains. [72] He speculated that the capsular interposition might provide the substrate for the classic meniscoid lesion.

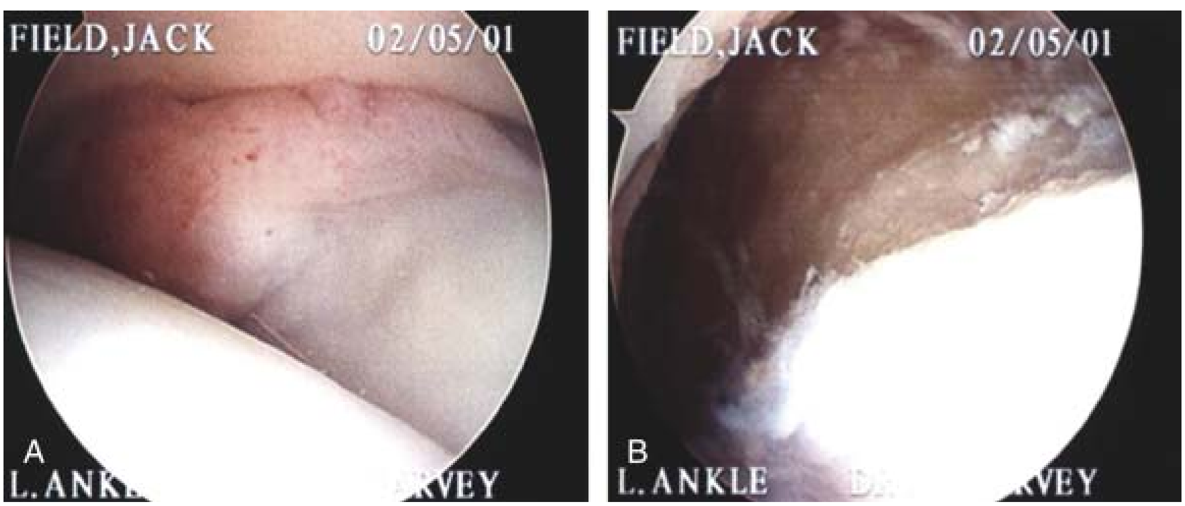

Figure 25C1-26 The "meniscoid lesion" associated with chronic anterior lateral ankle pain after lateral ankle ligament injury. **A,** Arthroscopic image of an anterior-lateral meniscoid lesion as seen from the medial portal. The lateral talofibular and talotibial joint space is occupied by the lesion. **B,** Arthroscopic image of the ankle after complete resection of the meniscoid lesion.

Chronic anterior lateral ankle pain is treated with an aggressive 6-week course of physical therapy and bracing to eliminate subtle instability. Oral anti-inflammatory and cortisone injection therapy may also be used. Patients who fail conservative management are treated with ankle arthroscopy and débridement of the anterolateral lesion (meniscoid lesion), if present.

Peroneal Tendon Instability

Peroneal tendon instability is an entity that may be associated with lateral ankle instability. The condition may be secondary to an inversion ankle injury. [13] It may produce functional ankle instability caused by the subluxation of the peroneal tendons. Chronic subluxation or frank dislocation of the peroneal tendons may also produce degenerative tears of the peroneus brevis tendon.

The peroneus brevis and longus muscles form individual tendons that pass behind the lateral malleolus to turn anteriorly toward their respective insertions at the base of the fifth metatarsal and the base of the first metatarsal. At the level of the lateral malleolus, the tendon of the peroneus brevis remains anterior to the peroneus longus. The tendons are retained within the peroneal groove by the superior peroneal retinaculum (SPR). [132] The SPR originates from the periosteum on the posterolateral ridge of the fibula. [133] The peroneal groove is a shallow bony groove [134] deepened by a fibrocartilaginous ridge.

The mechanism of injury for an acute dislocation is related to a sudden, forceful, passive dorsiflexion of the inverted foot combined with reflex contraction of the peroneal tendons. [430] [431] [432] As has been stated previously, chronic subluxation may also be related to chronic lateral ankle instability. [138] The injury produces a variety of pathologic features, which include elevation of the SPR off the lateral border of the fibula with concomitant dissection of the tendons beneath the lateral fibular periosteum, tear of the SPR, or fracture of the posterolateral margin of the fibula. The anatomic classification of peroneal tendon instability, as described by Oden, is based on the location of SPR disruption. [139]

Chronic subluxation of the peroneus brevis tendon onto the posterolateral border of the fibula has been implicated in the development of longitudinal tears of the peroneus brevis tendon. [435] [436] Longitudinal tear of the peroneal tendons has also been described after acute and chronic lateral ankle inversion injury. [437] [438] Acute peroneal tendon dislocation produces pain over the course of the peroneal tendons as well as along the lateral border of the fibula. The patient may recall a pop at the time of injury. Often, the patient is capable of providing a vivid description of dislocation. The tendon may or may not spontaneously reduce.

A careful examination of the acute injury confirms swelling and tenderness posterior to the lateral malleolus. [430] [431] [439] [440] [441] Active dorsiflexion of the foot from a plantar flexed and everted position may produce apprehension, subluxation, or dislocation (Fig. 25C1-27). Dislocation is not always actively elicited. Lateral ankle ligament stability is also assessed as part of a comprehensive examination.

Figure 25C1-27 A, Reduced peroneal tendons. **B,** Dislocated peroneal tendons as foot is dorsiflexed and everted.

As with all lateral ankle injuries, routine radiographs are obtained. Fracture of the posterolateral margin of the fibula is a rare finding but indicates SPR disruption. MRI is the best imaging modality to evaluate peroneal disease, including tenosynovitis, partial or complete peroneal tendon rupture, peroneal tendon subluxation or dislocation, integrity of the SPR, and the competency of lateral ankle ligaments and internal derangement of the ankle and subtalar joints.

A single, acute dislocation of the peroneal tendons is treated with an initial course of immobilization. A short leg cast is applied, and the patient is allowed to bear weight as tolerated. At 6 weeks, the stability of the peroneal tendons is verified. An ankle rehabilitation program is instituted with an emphasis on peroneal strength and proprioception. Casting is aborted if the patient detects peroneal instability within the cast.

Patients with recurrent or chronic dislocation do not respond to nonsurgical treatment methods. [431] [439] [441] [442] [443] [444] [445] [446] [447] For these patients, surgical reconstruction is required. Surgical treatment includes exploration, tendon repair or tenodesis, peroneal groove deepening, and superior peroneal retinaculum reconstruction, as dictated by surgical findings.

Technique: Deepening of the Peroneal Groove and Imbrication of the Superior Peroneal Retinaculum

1. The procedure is performed with the patient under general anesthesia. The patient is supine with a well-padded proximal tourniquet and a soft bump placed beneath the ipsilateral hemipelvis.
2. A curvilinear incision is made over the course of the peroneal tendons. The initial exposure can be limited to 4 cm, with most of the incision proximal to the tip of the lateral malleolus. Care is taken to protect the sural nerve at the distal aspect of the incision.
3. The SPR is exposed and instability of the peroneal tendons verified by manipulation of the foot and ankle. The SPR, identified as a thickening in the sheath, and synovial sheath are sharply divided in line with the posterior border of the lateral malleolus. Exposure is completed by systematic synovectomy and tenolysis of the peroneal tendons. The peroneus brevis muscle that lies within the peroneal groove is resected off of the tendon.

4. Partial longitudinal tears involving less than 50% of the peroneal tendons are débrided and repaired with running sutures.
5. A shallow peroneal groove is deepened through a three-sided, medially based osteoperiosteal flap. After a portion of the underlying cancellous bone is removed, the flap is replaced. Alternatively, the flap is resected and the underlying cancellous bone smoothed with bone wax.
6. The SPR is repaired and advanced onto the posterior lateral aspect of the fibula. This is typically accomplished through multiple drill holes using an absorbable suture.
7. The wound is closed in layers and a short leg splint applied in slight plantar flexion and eversion. At 10 days, the incision is inspected and a short leg weight-bearing cast applied for 4 weeks. Patients are then transitioned to a removable boot for 6 more weeks, and a rehabilitation program is instituted. An ankle brace is used for an additional 4 to 6 months. Activities are gradually resumed as the athlete regains full range of motion and strength.

Nerve Palsy

The superficial peroneal nerve is susceptible to tension injury after inversion ankle injury. Patients complain of numbness at the dorsal foot or pain of the fascial hiatus radiating distally. Careful physical examination confirms loss of sensation on the dorsal foot. Pain may be reproduced with percussion of the superficial peroneal nerve, particularly at the crural fascia hiatus, or with passive plantar flexion and inversion of the foot. Differential injection of local anesthetic is also used to establish the diagnosis. Treatment is symptomatic but must include conservative management for the ankle instability.

Johnston and Howell reported seven cases of superficial peroneal nerve neuropathy associated with inversion ankle injury. [153] Five of the patients were also diagnosed with reflex sympathetic dystrophy. Surgical exploration revealed several abnormalities, including a distal exit from the crural fascia, scar, anomalous nerve, and a vessel leash.

Nitz and colleagues performed electrodiagnostic testing on 60 consecutive patients with severe ankle sprains. [154] The group of patients with medial and lateral ligament injuries (30) included 5 patients with peroneal and 3 patients with posterior tibial injuries. The group of patients with medial, lateral, and syndesmosis ligament injuries (36) included 31 patients with peroneal and 30 patients with posterior tibial injuries. Traction injury to the peroneal and tibial nerves at the bifurcation of the sciatic nerve is one possible mechanism of injury.

Electrodiagnostic studies are used for documentation at presentation and follow-up. Sensory deficits do not require intervention. Motor deficits may require bracing with an ankle foot orthosis. Spontaneous recovery is typical.

Subtalar Sprain

Meyer and coworkers performed ankle stress radiographs and subtalar joint arthrography on 40 patients with acute inversion sprains. [155] They classified the injuries based on the extent of lateral ankle ligament and subtalar ligament injury. Thirty-two (80%) of the patients sustained injury to both the lateral ankle and the subtalar joint. Six patients with negative stress ankle radiographs had positive subtalar arthrograms.

Subtalar joint sprain and subtalar instability are discussed in detail in the following foot section.

Subtalar Coalition

Recurrent ankle sprain is not an uncommon presentation for the 10- to 14-year-old athlete with tarsal coalition. Lateral hindfoot pain, recurrent sprains, flatfoot, and decreased subtalar motion all point to talocalcaneal or calcaneonavicular coalition.

The entity is diagnosed with oblique radiographs, CT, or MRI. Treatment is symptomatic initially with orthoses. Persistent symptoms require excision or occasionally hindfoot arthrodesis.

Bifurcate Ligament Sprain

Injury to the bifurcate ligament or the anterior process of the calcaneus is associated with a plantar flexion–inversion mechanism (see Figs. 25C1-1, 25C1-48, and 25C1-49 [0240] [0475] [0480]). The injury is often associated with, or mistaken for, a concomitant lateral ankle sprain.

Diagnosis is clinical, but MRI confirmation may be obtained. Treatment is symptomatic with foot and ankle rehabilitation and is occasionally protracted owing to persistent symptoms. This injury is also reviewed in great detail in the foot section.

Tibiofibular Synostosis

Heterotopic ossification of the interosseous ligament occasionally is noted after disruption of the syndesmosis (Fig. 25C1-28). Whiteside and associates identified six patients, all professional athletes, with tibiofibular synostosis and a history of inversion and internal rotation injuries. [156] The authors' experiences suggest excision for cases of persistent pain and limited dorsiflexion. Recurrence occurred in two of three cases.

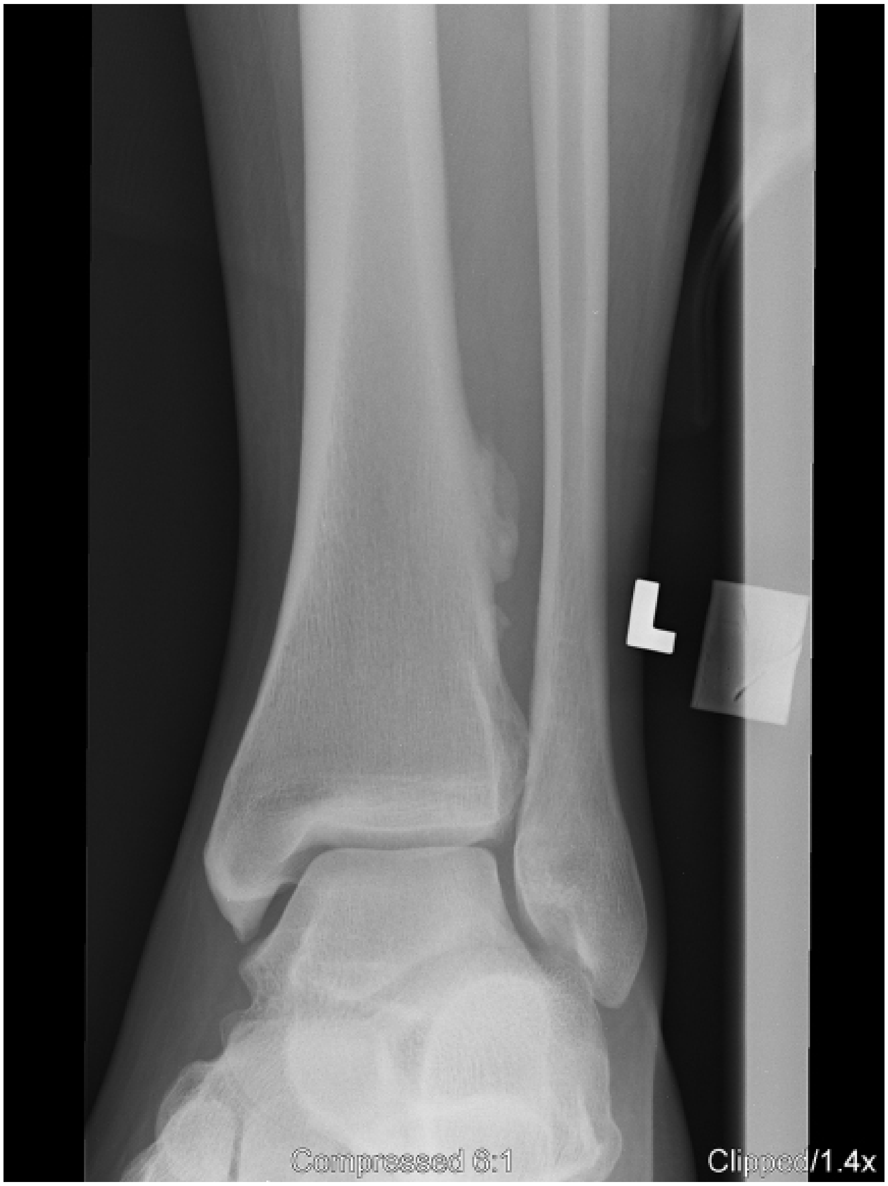

Figure 25C1-28 Ossification of the syndesmosis in a former collegiate football player.

In my experience, the presence of ossification of the interosseous ligament or complete synostosis does not appear to be associated with a poor outcome (among patients with syndesmosis injury, not isolated lateral ankle injury). Certainly, a poorly reduced syndesmosis with an associated synostosis presents a need for excision of the synostosis and anatomic reduction.

Fifth Metatarsal Base Fracture

The base of the fifth metatarsal is also susceptible to injury after a plantar flexion–inversion mechanism. The associated lateral hindfoot swelling and tenderness are distal and inferior to the ATFL. The diagnosis is confirmed with routine foot radiographs. Treatment is generally supportive with immobilization in a cast, walking boot, or stiff-soled shoe for 4 to 6 weeks.

Author's Preferred Method

Injury to the lateral ankle requires an individualized approach. Treatment must balance the needs of the athlete with the available professional support and facilities. High school, collegiate, and professional athletes are treated according to an accelerated schedule. These athletes are fortunate enough to have professional trainers for daily monitoring and treatment. Trainers and their staff are also able to provide ongoing ankle taping for practice and competition.

Acute grade I and grade II sprains are treated with a supervised physical therapy program. Medical anti-inflammatory therapy and cold therapy are used to reduce swelling and pain. Cast immobilization is avoided. A semirigid pneumatic ankle brace is used throughout the rehabilitation period and well into the return-to-sports phase.

High-grade (grade III) lateral ankle sprains are treated with a brief 3- to 7-day period of weight-bearing to tolerance in a removable boot or a cast. The most comprehensive approach avoids casting and focuses on a supervised physical therapy program, including modalities (e.g., cold therapy, compression, electrical stimulation) for swelling and pain control. A semirigid pneumatic ankle brace is used throughout the rehabilitation period and well into the return-to-sports phase, typically 4 to 6 months.

Chronic lateral ankle instability produces a variable amount of impairment. The treating physician should emphasize a comprehensive and well-supervised rehabilitation program to prevent recurrent instability. Until ankle stability is achieved, a nonrigid or semirigid ankle brace is used to support the ankle. If significant improvement is not demonstrated within 6 weeks, I use a Broström procedure as modified by Gould to reconstruct the ATFL and the CFL. Intraoperative imaging is used to obtain bilateral stress views of the ankles to document instability. Arthroscopy of the ankle is used before stabilization, if indicated by clinical examination (e.g., anterolateral tenderness, talar dome tenderness) or MRI findings (e.g., osteochondral lesion of the talus, loose body).

Return-to-Play Criteria

Recovery of function after a lateral ankle sprain follows a logical sequence of events. Once the initial pain and swelling subside, coordination and strengthening activities are emphasized. Gradually, the patient is able to return to walking, running, and cutting programs. I return patients to sport once they master sport-specific drills with minimal discomfort. A semirigid pneumatic ankle brace or taping is used to prevent recurrent injury.

Prevention of recurrence and new injuries has received much attention in the literature. Key components of prevention include strength conditioning, coordination, proprioception, stretching, and external support.

Taping of the ankle is perhaps the most widely used prophylactic method.[157] Rovere and colleagues published a retrospective analysis of taping, and the laced ankle stabilizer that confirmed that taping was much less effective in the prevention of new and recurrent ankle injuries among collegiate football players.[158] Taping appears to be limited by time-related loosening.[454][455] Glick and coworkers studied the effect of tape on six ankles with significant (>5 degrees) talar tilt.[7] Each subject performed 20 minutes of exercise. Only one of the six ankles remained firmly supported by the ankle tape.

Walsh and Blackburn acknowledge the time-related loosening associated with ankle taping.[161] They contend that the method continues to play a supportive role, perhaps limited to restricting the extremes of motion. They recommend taping against the skin and secondarily around the shoe (football). They emphasize that tape, in any amount or configuration, is not a substitute for rehabilitation. Although taping is inexpensive on a per-use basis, significant cost is associated with long-term application by trained personnel.[158]

Hamill and colleagues suggested the use of a semirigid pneumatic orthosis for the prevention of recurrent lateral ankle sprains; they cite a reduction in mediolateral excursion force and velocity in an experimental setting.[162] Decreased inversion, based on angular displacement, has also been demonstrated.[163]

Soccer players with and without a history of lateral ankle sprains were randomly assigned by Surve and associates to use a semirigid pneumatic ankle brace (Sport Stirrup, Aircast, Inc., Summit, NJ).[164] The orthosis reduced the incidence of lateral ankle sprains in the group with a history of previous ankle injury but not in the group without such a history.

Thacker and colleagues completed a review of 113 studies and concluded that supervised rehabilitation must be completed before resumption of running or practice.[8] Furthermore, these authors recommended the use of an orthosis for 6 months after a severe lateral ankle sprain.

A sobering study completed by Gerber and associates clearly illustrates the long-term disability associated with ankle sprain. [3] This prospective observational study of 104 West Point cadets confirmed that 95% of the cadets had returned to athletic activity by the 6-week follow-up. At the time of the 6-month follow-up, all cadets had returned to athletic activity, but 40% remained symptomatic. The persistent dysfunction was related neither to the grade of sprain nor the presence of joint laxity.

In summary, athletes return to sport after recovery of pain-free ankle motion, strength, and protective reflexes. The athlete must complete sport-specific drills with a high degree of confidence and comfort. Most important, the ankle is braced until functional and anatomic stability are achieved.

Medial Ankle Sprain

Isolated injury to the deltoid ligament is rare. Staples reviewed 110 cases of deltoid injury. [165] Deltoid rupture without associated ankle fracture was noted in only 10 cases. Of these 10 cases, 5 were isolated to the deltoid, 3 were associated with syndesmosis injury, and 2 were associated with anterior capsule injury.

Relevant Anatomy

The deltoid ligament consists of superficial and deep components (Fig. 25C1-29). [461] [462] The superficial deltoid ligament originates from the anterior portion of the medial malleolus and spreads out to insert on the navicular, talus, and calcaneus. The superficial deltoid includes four parts—the tibionavicular ligament, the tibiospring ligament, the tibiocalcaneal ligament, and the superficial tibiotalar ligament. The deep deltoid ligament includes two parts—the deep anterior tibiotalar ligament and the deep posterior tibiotalar ligament. It is a short, thick ligament that traverses from the intercollicular groove onto the medial talus and blends with the medial capsule of the ankle joint.

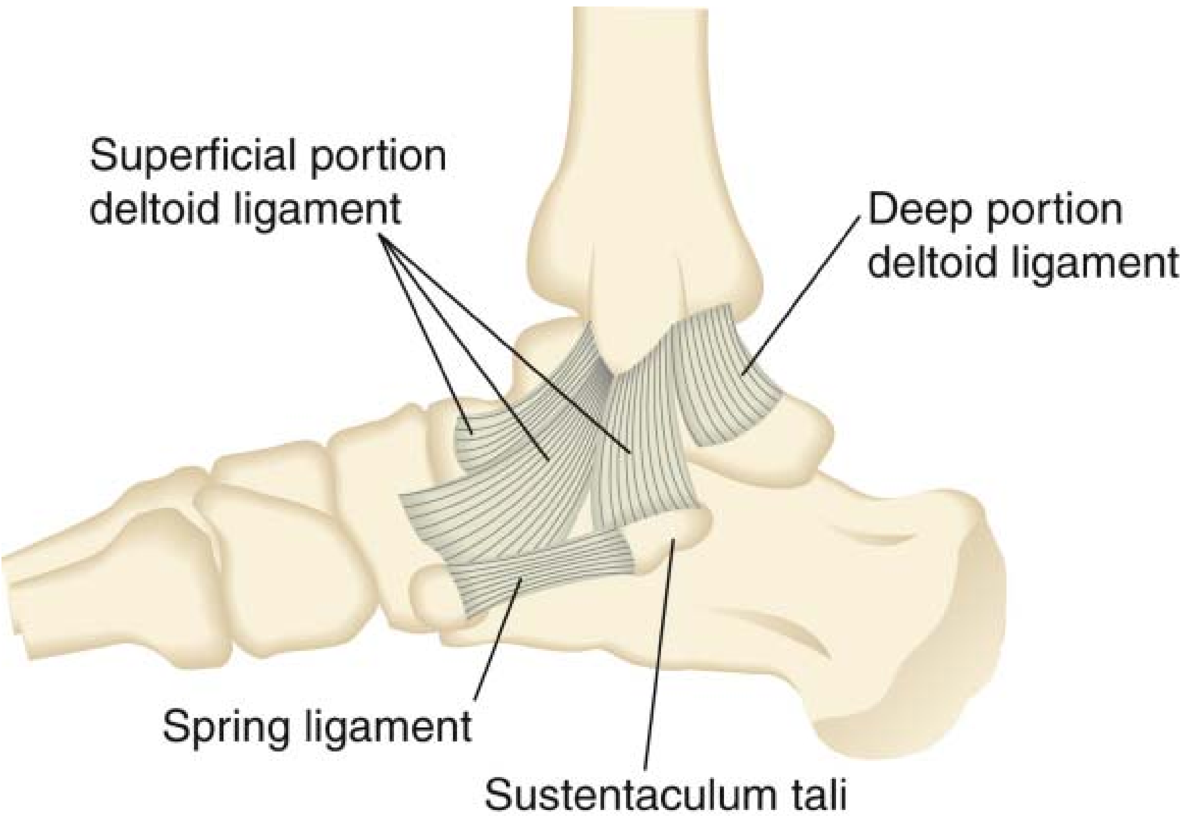

Figure 25C1-29 The superficial and deep layers of the deltoid ligament. *(Redrawn from Close JR: Some applications of the functional anatomy of the ankle joint. J Bone Joint Surg Am 38:761-781, 1956.)*

The biomechanical characteristics of the ankle ligaments are such that failure (rupture) is due to increasing load as opposed to twisting or shearing. [15] Isolated testing of the individual ankle ligaments demonstrates that the ATFL is the first to fail and the deep deltoid ligament is the last to fail. [15] The deltoid ligament functions to limit abduction. It is a strong structure that requires significant force to cause rupture.

Siegler and colleagues tested 20 fresh cadaveric ankles.[18] Based on increasing ultimate load, components of the deltoid were ordered from weakest to strongest as follows: the tibiocalcaneal ligament, the tibionavicular ligament, the tibiospring ligament, and the posterior tibiotalar ligament.

Close has demonstrated the importance of the medial ligaments in maintaining a normal medial clear space, that is, a normal intermalleolar distance.[166] He provided a detailed anatomic description but no data to support his conclusions.

Earll and coworkers conducted a cadaveric study to assess the importance of the deltoid ligament relative to talocrural contact and pressure.[168] Division of the tibiocalcaneal fibers of the superficial deltoid resulted in significant decreased contact area (maximum, 43%) and an associated increase in contact pressure (maximum, 30%). Division of the other components of the deltoid resulted in insignificant changes in joint contact.

Kjærsgaard-Andersen and associates used a cadaveric model to study the effect of isolated division of the tibiocalcaneal ligament.[169] They reported a maximal median increase in tibiotalocalcaneal abduction of 6.1 degrees and a corresponding maximal median increase in talocalcaneal abduction of 3.6 degrees. The authors concluded that the tibiocalcaneal ligament is an important stabilizer of the medial hindfoot.

Rasmussen and colleagues used a cadaveric model to study the effect of isolated division of the deltoid ligament with a device that allowed recording of rotatory movements in two planes.[170] The tibiocalcaneal ligament (superficial deltoid) and the intermediate tibiotalar ligament (deep deltoid) provided resistance to abduction of the talus. The deltoid also provided significant resistance to external rotation of the talus.

Clinical Evaluation

Isolated deltoid rupture is a rare injury, usually associated with a traumatic mechanism of injury. Most deltoid ruptures are associated with ankle fractures. Ankle fractures associated with a pure deltoid rupture typically involve the posterior deep tibiotalar ligament.[171]

Evaluation of the medial ankle sprain is designed to elicit information that allows classification of the injury. Various classifications have been described, but graded ligament injury, as suggested by the American Medical Association[35] and by O'Donoghue,[36] is sufficient. Injuries are graded based on stretch (grade I), partial tear (grade II), or complete rupture of the ligament (grade III). Additional information with regard to associated ligamentous injuries is noted.

History

Information relevant to previous ankle injury, the mechanism of injury, the ability of the patient to continue to play or walk, and current complaints represent the salient historical points. The patient frequently reports feeling a pop in the medial ankle with associated pain and swelling.

Physical Examination

It is imperative that the examination not be limited to the medial ankle ligaments. Inspection of the leg, ankle, and foot may reveal swelling, ecchymosis, blister formation, or gross deformity. A vascular and sensory assessment is performed, followed by palpation of the entire leg, ankle, and foot. Tenderness at the deltoid and surrounding area is particularly important to note. Attention is paid to symptoms in other areas that could indicate ankle fracture, lateral ligament sprain, syndesmotic injury, combined proximal fibular fracture (Maisonneuve pattern), or proximal tibiofibular joint injury.

Motion about the foot and ankle is determined with the patient seated and relaxed. The knees are flexed and the feet allowed to fall into an equinus position. Ankle and subtalar range of motion is documented and motor function graded.

Stress testing is a useful clinical and radiographic tool that provides a portion of the diagnostic data required for grading ankle sprains. The *anterior drawer test* and the *varus talar tilt test* are used to demonstrate the integrity of the ATFL and the CFL. A *valgus talar tilt test* is used to evaluate the integrity of the deltoid. The test is performed in both ankle neutral and plantar flexion positions. Valgus stress is applied to the talus through the hindfoot, and a comparison is made between injured and noninjured ankles.

Testing is occasionally uncomfortable, particularly in the acute setting. False-negative results may be caused by involuntary guarding or pain response. The ankle can be injected with local anesthetic to facilitate a better examination.

The chronic or recurrent medial ankle sprain is associated with functional or mechanical medial instability, apprehension, discomfort, swelling, and tenderness over the deltoid. This clinically manifests as a valgus and pronation deformity that many patients can correct by contracting the posterior tibialis muscle.

Imaging

Radiographs

Radiographs in the anteroposterior, mortise, and lateral projections are required for ankle evaluation. Weight-bearing radiographs are used in an effort to reproduce physiologic loading.

Radiographs are evaluated with regard to malleolar fracture, physeal fracture, osteochondral fracture, avulsion fracture, and deltoid ossification. Alignment and translation deformity are also inspected, particularly at the syndesmosis and the medial ankle joint space (Figs. 25C1-30 and 25C1-31 [0385] [0390]). An increased medial clear space suggests complete rupture of both components of the deltoid.[166] Radiographs are frequently normal with partial ligament injuries.

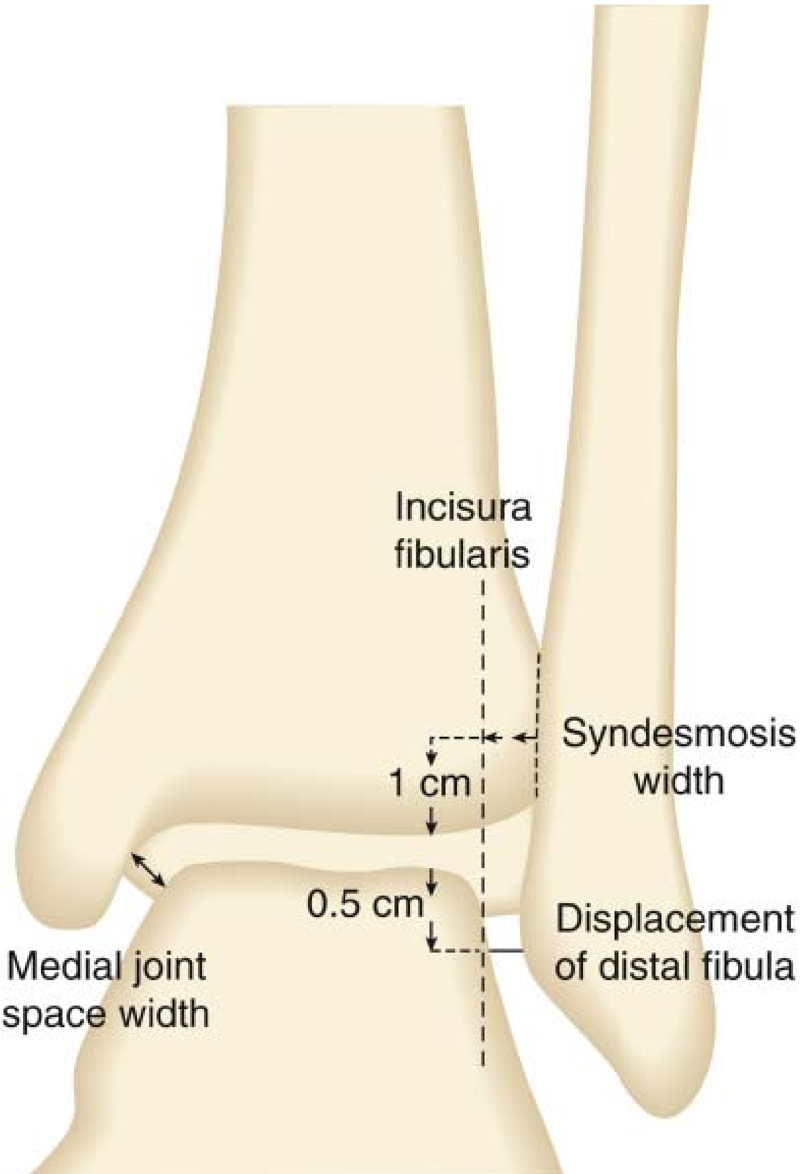

Figure 25C1-30 Techniques of measuring the lateral displacement of the lateral malleolus (mortise view) and the width of the syndesmosis (mortise view) and medial joint space (anteroposterior view). *(Redrawn from Harper MC: The deltoid ligament: An evaluation of need for surgical repair. Clin Orthop 226:156-168, 1988.)*

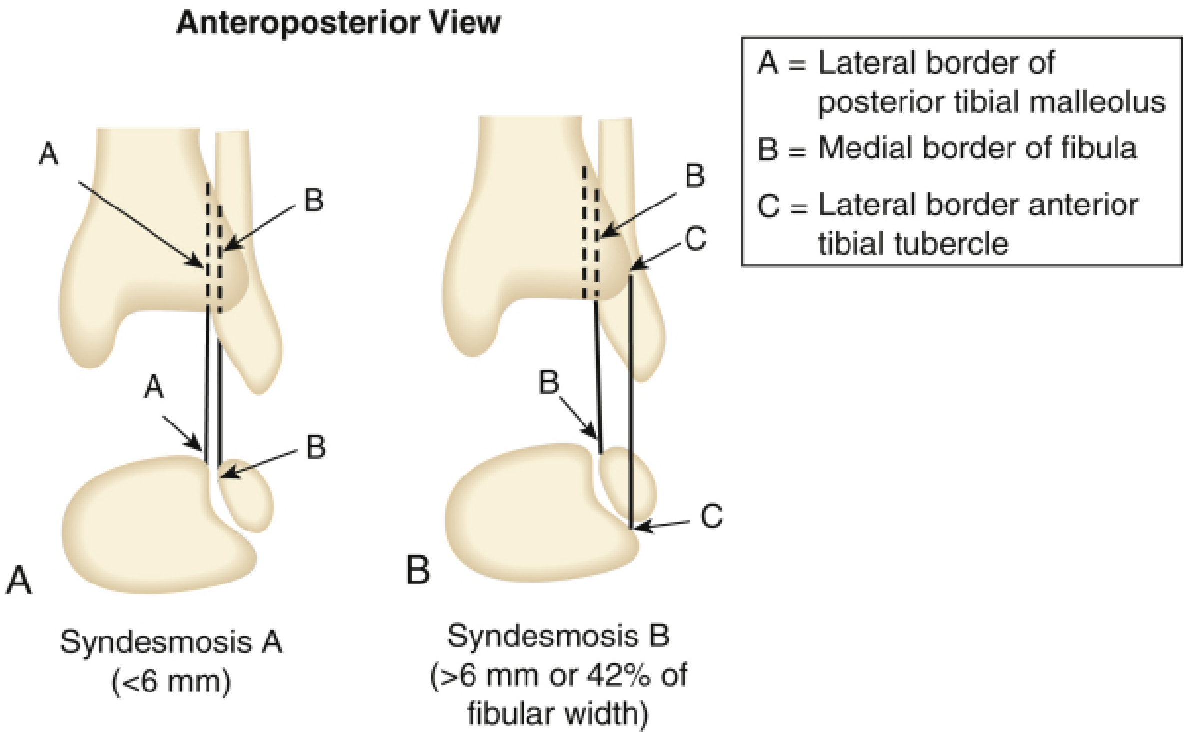

Figure 25C1-31 Syndesmotic radiographic criteria. **A,** The syndesmosis clear space as depicted on the anteroposterior view and by coronal section. The tibiofibular clear space is the distance between the lateral border of the posterior tibial malleolus (point A) and the medial border of the fibula (point B) on the anteroposterior radiograph. This space is normally less than 6 mm. [187] **B,** The syndesmosis overlap as seen on the anteroposterior view and by coronal section. The tibiofibular overlap is the distance between the medial border of the fibula (point B) and the lateral border of the anterior tibial prominence (point C) on the anteroposterior radiograph. This space is normally greater than 6 mm, or 42% of the fibular width. [187] *(Redrawn from Stiehl JB: Complex ankle fracture dislocations with syndesmosis diastasis. Orthop Rev 14:499-507, 1990.)*

Stress Radiographs

Valgus talocrural instability may occur after deltoid disruption. [172] The bilateral stress radiograph is used to quantify valgus tilt of the talus. The valgus tilt stress radiograph is similar to the clinical test but is performed during an anteroposterior radiograph. The degree of tilt is determined by measuring the angular divergence between the distal tibial articular surface and the talar dome.

Leith and associates performed valgus stress radiography on 32 previously uninjured patients. [173] This examination was performed on both ankles in a neutral position with and without valgus stress. The authors demonstrated that 91% of the ankles had less than 2 degrees and that the remaining 9% of the ankles had between 2 and 3 degrees of valgus tilt. They suggest that an ankle with a valgus tilt greater than 2 degrees has a high probability of deltoid injury.

The external rotation stress radiograph is also used to evaluate associated syndesmosis injury. This test is described in the section on “Ankle Syndesmosis Sprain.”

Arthrography

The unpredictable effect of patient guarding during stress radiography is overcome by the use of ankle arthrography. [347] [348] This method is relatively simple and can provide objective evidence of deltoid disruption. Contrast material is injected into the acutely injured ankle, preferably with fluoroscopic guidance, and radiographs are obtained in various projections with attention to areas of extravasation. A false-negative result from an early capsular seal is possible. The procedure has been largely replaced by the use of MRI.

Magnetic Resonance Imaging

MRI is a useful method for evaluation of acute, subacute, and chronic ankle ligament injuries. [55] Deltoid disruption, partial or complete, is demonstrated by fiber disruption, edema, and associated injuries to the surrounding soft tissue and bone (see Fig. 25C1-9). Visualization of the deltoid ligament requires careful attention to the position of the foot during the test. On coronal images, the tibionavicular and anterior tibiotalar components are best seen with the foot in plantar flexion, whereas the tibiocalcaneal and posterior tibiotalar portions are visualized with the foot in dorsiflexion. [174]

Therapeutic Options

The treatment of this injury depends on the severity of disruption and associated injuries. All acute injuries are treated with the RICE method followed by gentle range of motion and protected weight-bearing.

Many studies have reviewed the effect of cold application to the injured extremity. Cold therapy is an effective, inexpensive, and easy-to-use modality for the treatment of acute musculoskeletal injury. Appropriately applied cold therapy decreases pain perception, decreases the biochemical reactions that produce inflammation, and produces vasoconstriction with a concomitant reduction in soft tissue swelling and bleeding.

Compression is typically provided in the form of an elastic bandage but may also include casting, splinting, pneumatic orthosis, or mechanical compression devices. [57] Elevation of the ankle helps to reduce swelling and pain.

The ankle is supported by a variety of methods, including the use of a nonwalking cast, a walking cast, a removable cast boot, a semirigid pneumatic ankle brace, a nonrigid functional ankle brace, or various ankle taping methods. Various immobilization methods are summarized in Table 25C1-4 .

Grade I Medial Ankle Sprains

Treatment is symptomatic with an emphasis on recovery of range of motion, strength, and coordination. A structured ankle rehabilitation program remains an important part of treatment. A nonrigid functional ankle brace, such as a lace-up brace, or a semirigid pneumatic ankle brace is used during all phases of the recovery. Return to competition is usually delayed when compared with lateral ankle sprains.

Grade II and Grade III Medial Ankle Sprain

Complete immobilization is provided with a short leg walking cast or boot. Weight-bearing to tolerance is encouraged. The assumption is that immobilization allows the ruptured ends of the medial ankle ligaments to heal in a near-anatomic position. Immobilization is continued for 6 to 8 weeks depending on resolution of swelling, tenderness, and instability. A comprehensive ankle rehabilitation program is used along with a semirigid pneumatic orthosis for up to 6 months from the date of injury.

Surgical treatment is rarely necessary and is reserved for patients with a persistent valgus tilt or in whom the medial clear space is not reduced. [460] [470]

Technique: Repair of Medial Ankle Ligaments

1. A longitudinal incision is created over the medial malleolus and extended to the talonavicular joint.
2. Both the superficial and deep components of the deltoid ligament are inspected as are the adjacent posterior tibial and flexor digitorum tendons.
3. The ankle joint is inspected for articular surface lesions and loose bodies. This can be done arthroscopically before the open procedure if desired.
4. Anatomic repair of the deep fibers is completed before repair of the superficial fibers. Nonabsorbable sutures are used. In cases of complete avulsion from the medial malleolus, a suture anchor can be used.
5. The incision is closed in a layered fashion, and a splint is applied. At 10 days, the patient is placed into a short leg cast. Patients are not allowed to bear weight for 4 weeks after surgery. At that point, the patient is transitioned into a walking boot and allowed to progress to full weight-bearing over 4 weeks. An ankle rehabilitation program with emphasis on range of motion, strengthening, and proprioception is started. The athlete continues to wear an ankle brace during sports for 6 months after surgery.

Chronic Medial Ankle Sprain and Instability

Occasionally, a medial ankle sprain produces a chronic deltoid insufficiency or medial ankle pain. Difficulty with push-off may also be noted. Careful review of the initial treatment program may suggest the need for an aggressive and well-supervised rehabilitation program along with a semirigid pneumatic orthosis. If it is believed that the rehabilitation was adequate, further imaging with stress radiographs and MRI is obtained to better delineate the condition.

An ossicle within the anterior deltoid may produce medial symptoms without clinical instability. Symptomatic anterior deltoid ossicles are surgically débrided, followed by postdébridement valgus stress radiographs. Symptomatic medial ligament insufficiency is most easily treated with local repair and imbrication of the deltoid. Advancement of the deltoid to the medial malleolus may be facilitated through drill holes or with suture anchors. If the local tissues are inadequate for a direct repair, a reconstruction can be performed. Autograft or allograft material is placed from the tibia to the talus or navicular. This technique can be combined with spring ligament repair or reconstruction if that ligament is attenuated as well.

Rehabilitation

A detailed rehabilitation program is outlined in the section on lateral ankle ligament injury. Ankle and subtalar flexibility, motor function, and coordination [30] are emphasized throughout the protocol.

AUTHOR'S PREFERRED METHOD OF TREATMENT

Completely isolated deltoid injuries are rare. Occasionally, an athlete presents with a high-grade lesion with predominantly medial symptoms. In this instance, I prefer to use a short leg cast or walking boot for 4 to 6 weeks in an effort to promote anatomic healing of the deltoid fibers. A comprehensive, supervised physical therapy program then follows. An ankle brace is used throughout the rehabilitation period and well into the return to sports phase. Medical anti-inflammatory therapy is provided to reduce swelling and pain.

Chronic medial ankle instability is rare. The clinical and radiographic work-up must be meticulously done before a medial reconstruction is undertaken. Partial tears and ossicles associated with the anterior colliculus can be a common source of dysfunction. Isolated removal and local repair can provide relief.

Return-to-Play Criteria

Recovery follows a logical sequence of events. Once the initial pain and swelling subside, coordination and strengthening activities are emphasized. Gradually, the patient is able to return to walking, running, and cutting programs. Patients are returned to sport once they master sport-specific drills. A semirigid pneumatic ankle brace or taping accelerates the schedule.

Ankle Syndesmosis Sprain

Injury to the ankle syndesmosis, or a high ankle sprain, is most common in collision sports. [176] Injury to the syndesmosis results in more impairment to the athlete when compared with lateral ankle sprains. Boytim and colleagues reported 98 ankle injuries among the players of a professional football team over a 6-year period. [176] Twenty-eight significant lateral ankle sprains and 15 syndesmosis sprains were reported. The players with syndesmosis sprains missed more games and more practices and used more treatments than players with lateral ankle sprains.

Hopkinson and coworkers retrospectively reviewed 1344 ankle sprains that occurred over a 41-month period at the U.S. Military Academy. [177] Fifteen of these patients (1.1%) were diagnosed with syndesmosis sprain. A subsequent prospective study at the same institution revealed that syndesmosis sprains accounted for 17% of ankle sprains over a 2-month period. [3]

Relevant Anatomy

The interosseus membrane connects the tibia to the fibula. At the level of the ankle, three defined ligaments are present: the anterior-inferior tibiofibular ligament (AITFL), the posterior-inferior tibiofibular ligament (PITFL), and the interosseous ligament (IOL) (Fig. 25C1-32). The AITFL courses from the anterodistal fibula to the anterolateral (Tillaux-Chaput) tubercle of the tibia. The AITFL is the most commonly injured ligament in syndesmotic sprains and can result in symptomatic impingement in some cases.

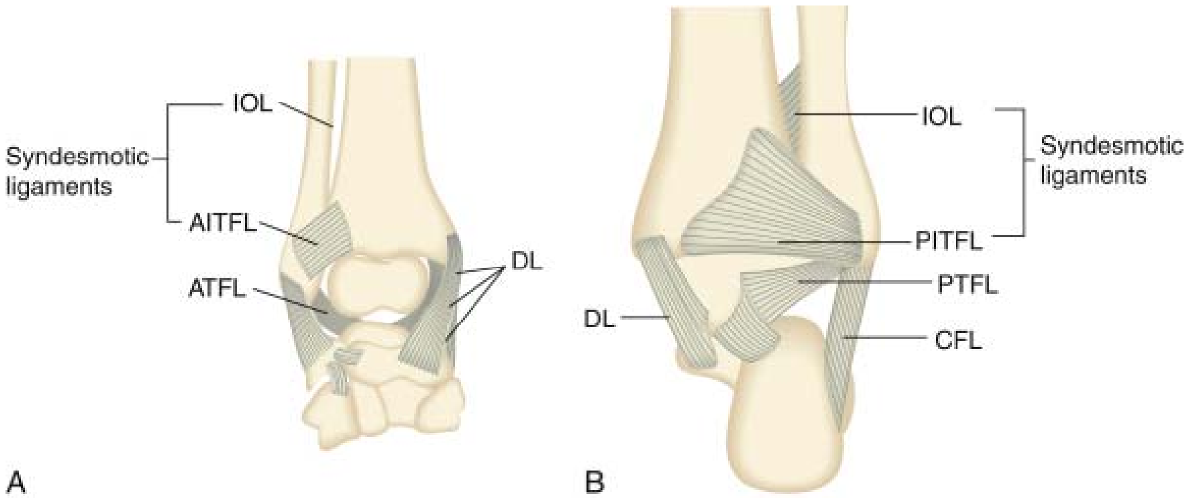

Figure 25C1-32 **A,** The tibiofibular syndesmosis from the front—anterior-inferior tibiofibular ligament (AITFL), anterior talofibular ligament (ATFL), and deltoid ligament (DL). **B,** The tibiofibular syndesmosis from the back—posterior inferior tibiofibular ligament (PITFL), distal interosseous ligament (IOL), posterior talofibular ligament (PTFL), calcaneofibular ligament (CFL), deltoid ligament (DL).

The PITFL is composed of the deep transverse tibiofibular ligament and a superficial portion. The two components form a strong yet elastic structure, which usually fails last in syndesmotic injury. The IOL connects the tibia to the fibula about 0.5 to 2.0 cm above the plafond. Proximally, it continues as the interosseus membrane, which provides little additional strength to the syndesmotic ligaments.

The syndesmosis, along with the deltoid ligament, maintains the critical anatomic relationship between the tibia and the talus. Ramsey and Hamilton used a carbon black transference technique to clearly demonstrate that lateral displacement of the talus results in an incremental decrease in contact area with each millimeter of translation.[178] The first millimeter of lateral translation produced an average 42% reduction in contact area between the tibia and the talus. Failure to reduce the ankle syndesmosis and the associated lateral talar translation greatly increases the risk for post-traumatic ankle arthritis (Fig. 25C1-33).

Figure 25C1-33 Post-traumatic ankle arthritis subsequent to an incompletely reduced ankle syndesmosis and lateral talar translation. Note the wide syndesmosis and medial joint space width, the narrow superior joint space, and the lateral subchondral cyst formation.

Anatomic dissection by Close revealed that division of the syndesmosis and of the interosseous ligament produces minimal widening of the intermalleolar distance.[166] Only after the deltoid ligaments are divided does the syndesmosis separate. His conclusion is that significant trauma to the ankle must occur for the ankle mortise to appear wide.

Broström described the ligamentous lesions found during the surgical exploration of 105 recent ankle sprains.[27] The ATFL was the most commonly injured structure. The ATFL was completely torn as an isolated injury in 65 cases and as an associated injury in an additional 25 patients. The CFL was the second most commonly injured ligament. It was completely or partially torn as an associated injury in 23 patients. The AITFL was completely torn in 6 cases. No incomplete ruptures were noted. Five of these injuries occurred at midsubstance, and 1 involved an avulsion fracture off the anterior tibia. The torn ends of the ligament remained well apposed. External rotation of the talus produced up to 5 mm of diastasis in 5 cases and 1 to 2 mm in the sixth case.

The mechanism of injury is thought to be external rotation, although researchers have been unable to reliably reproduce syndesmosis sprain without fracture. An external rotation force first disrupts the AITFL, followed by the IOL, but usually spares the PITFL. Increased force can lead to spiral fractures of the proximal fibula (Maisonneuve fracture).

In 1968, Lovell described the case of a 13-year-old tobogganer who sustained a forced external rotation injury to the ankle.[179] The patient presented with a fixed external rotation foot deformity that was associated with a posterior dislocation of the fibula perched behind the lateral tibia. Closed reduction and casting produced an excellent result.

Boytim and associates suggested two specific mechanisms of injury in the professional football player.[176] The first is direct force applied to the posterior leg of a downed player whose foot is in an externally rotated position. The second is an external rotation force at the knee while the foot is firmly planted.

Fritschy evaluated 10 world-class slalom skiers with syndesmosis injuries. [180] He speculated that a common mechanism of forced external rotation of the talus against the fibula produced all of their injuries. A retrospective review by Hopkinson and colleagues failed to establish a consistent mechanism of injury among athletes of different sports with syndesmosis sprain. [177]

Clinical Evaluation

Sprains can be considered from the simplistic perspective of graded ligament injury, as suggested by the American Medical Association [35] and by O'Donoghue. [36] Injuries are graded based on stretch (grade I), partial tear (grade II), or complete rupture (grade III) of the AITFL. Additional information with regard to associated ligamentous or bony injuries is noted.

Edwards and DeLee described a classification system for ankle diastasis without fracture (grade III sprain) based on the presence of radiographic diastasis with and without stress. [181] A *latent* syndesmosis injury appeared normal on an unstressed radiograph *and* abnormal or widened on external rotation stress mortise radiograph. A *frank* syndesmotic injury was seen as a widened syndesmosis on unstressed radiographs. The frank injuries were further divided into four types: type I, lateral fibular subluxation without plastic deformity of the fibula; type II, lateral fibular subluxation with plastic deformity of the fibula; type III, posterior subluxation of the fibula behind the lateral tibia; and type IV, superior dislocation of the talus between the tibia and fibula with diastasis and no fibula fracture.

History

Information relevant to previous ankle injury, the mechanism of injury, the ability of the patient to continue to play or walk, and current complaints represent the salient historical points. Syndesmosis injury is suggested by a mechanism of forced external rotation of the foot.

Physical Examination

The examination is systematic and includes careful palpation along the entire interosseous ligament and the fibula. Fracture of the fibula at all levels must be considered. Although dislocation of the proximal tibiofibular joint is rare, [182] it must also be considered when proximal leg symptoms are present. Local tenderness at the AITFL or along the interosseous ligament suggests a syndesmosis sprain. There may be tenderness over the deltoid ligament, usually with an associated abduction force at the time of injury.

Ankle range of motion is carefully assessed. Also, lateral ankle stability is determined with performance of the anterior drawer and talar tilt tests. One must never forget that pain out of proportion to the injury is a finding consistent with acute compartment syndrome.

Hopkinson and coworkers described a *squeeze test* used to identify syndesmosis ruptures at the time of initial presentation (Fig. 25C1-34). [177] The squeeze test was attributed to a former chief athletic trainer at the U.S. Military Academy. The test is performed by compression of the midleg from posterior lateral to anterior medial. Pain produced at the AITFL suggests injury to the same, as long as fracture, contusion, and compartment syndrome are not present. The authors retrospectively reviewed eight patients with syndesmosis sprains; all were noted to have a positive squeeze test at initial evaluation. A separate biomechanical analysis of the squeeze test demonstrated that the squeeze test produced reproducible separation of the fibula and tibia. [183]

Figure 25C1-34 The squeeze test. [177] Syndesmosis injury is suspected when compression of the midleg produces pain at the ankle syndesmosis.

Boytim and associates described an external rotation stress test (Fig. 25C1-35). [176] The patient is seated and relaxed with the hip and knee flexed and the foot and ankle held in a neutral position. The knee is maintained in a forward-facing position while a gentle but firm external rotation force is applied to the foot. Pain reproduced at the anterior syndesmosis is diagnostic of a syndesmosis injury.

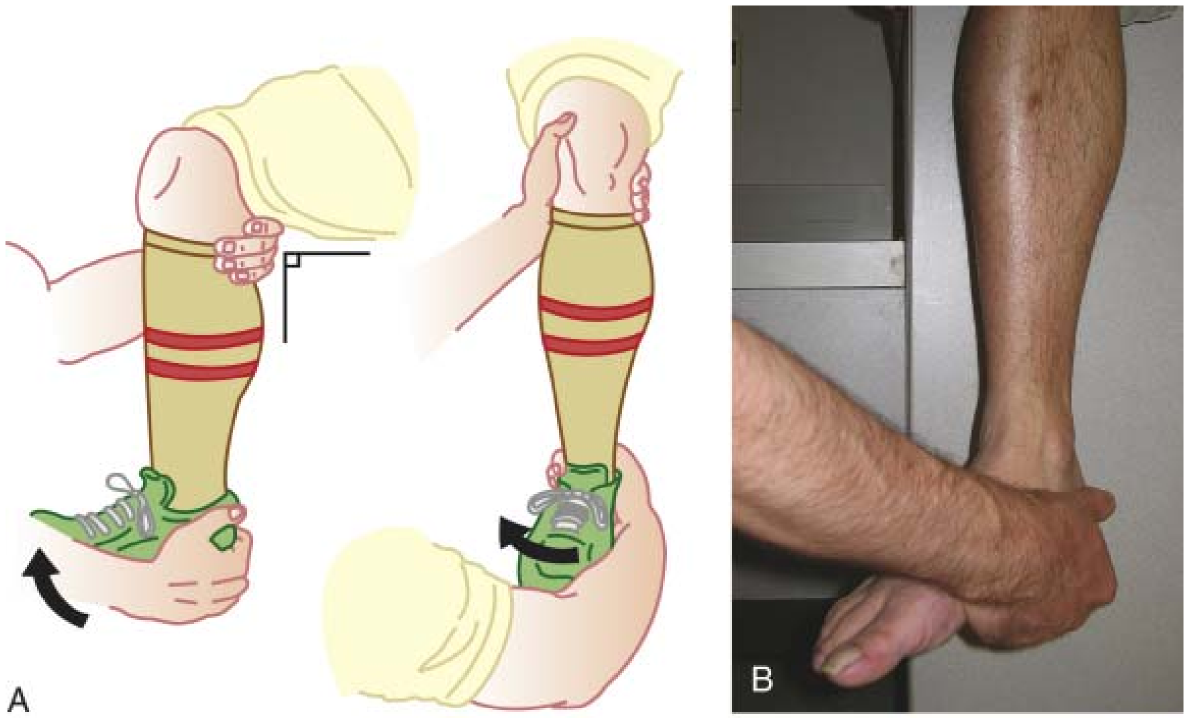

Figure 25C1-35 The external rotation stress test of the syndesmosis. *(**A**, Redrawn from Boytim MJ, Fischer DA, Neumann L: Syndesmotic ankle sprains. Am J Sports Med 19:294-298, 1991.)*

A secondary test is the direct eversion maneuver (Fig. 25C1-36). [184] The maneuver is accomplished with the patient in a seated and relaxed position. The examiner gently secures the leg and foot as a direct eversion or abduction force is applied across the ankle. Increased translation compared with the contralateral ankle is a positive result.

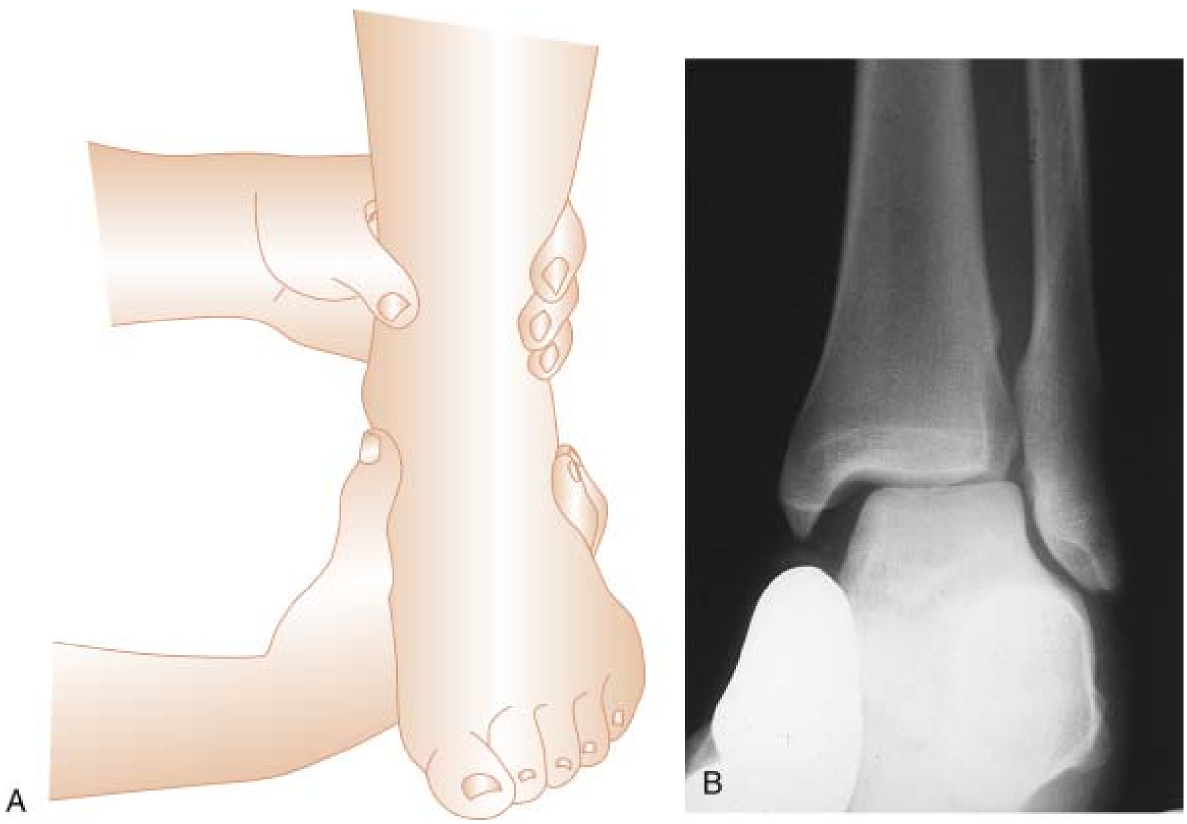

Figure 25C1-36 **A,** The direct eversion maneuver is accomplished with the patient in a seated and relaxed position. The examiner gently secures the leg and foot as a direct eversion or abduction force is applied across the ankle. Increased translation compared with the contralateral ankle is a positive result. **B,** A positive stress radiograph showing increased translation. *(**A,** Redrawn from Stiehl JB: Complex ankle fracture dislocations with syndesmotic diastasis. Orthop Rev 19:499-507, 1990.)*

Imaging

Radiographs

The radiographic examination includes weight-bearing views of the ankle, orthogonal views of the leg, and, when indicated, a computed tomographic scan of the syndesmosis (see Figs. 25C1-30 and 25C1-31 [0385] [0390]). [480] [481]

Harper and Keller used 12 cadaveric legs to establish radiographic criteria for a normal syndesmosis. [187] Plastic spacers with 1-mm increments were used to produce the diastases. With the use of standard anteroposterior and mortise radiographs, it was determined that the “clear space” on either view is normally less than 6 mm. This was in fact the most reliable parameter for evaluating the integrity of the syndesmosis. The normal overlap between the fibula and the anterior process of the tibia is greater than 6 mm, or 42% of the width of the fibula on the anteroposterior view, or greater than 1 mm on the mortise view.

Ostrum and colleagues used a more specific approach to the question of radiographic diastasis. [188] The authors included 40 female and 40 male normal volunteers to establish normal values. Their first finding was a distinct difference between the sexes. To bypass the sex variation, they suggested sex-specific absolute values or non–sex-specific ratios.

Late radiographs after a syndesmosis rupture may reveal varying degrees of interosseous ligament calcification. Hopkinson and associates retrospectively reviewed radiographs of 10 patients with syndesmosis sprains, as diagnosed by a positive squeeze test at initial evaluation. [177] Radiographs for nine of the patients demonstrated heterotopic calcification. At an average of 20 months after injury, all ankles were asymptomatic.

Taylor and colleagues reported finding heterotopic ossification on 11 of 22 follow-up radiographs after syndesmosis sprain sustained during football (diagnosed by tenderness at the syndesmosis). [189] No player developed frank synostosis. The lower incidence of heterotopic ossification, as compared with the Hopkinson report, may be related to the method of diagnosis (squeeze test versus local tenderness). Patients with heterotopic ossification experienced a delayed recovery (i.e., 32 days without heterotopic ossification, 43 days with heterotopic ossification). The authors suggested a correlation between severity of injury and the formation of heterotopic ossification. Patients with heterotopic ossification also were more likely to experience recurrent inversion ankle sprains.

Stress Radiographs

When routine radiographs are normal and there is a concern for a latent syndesmotic injury, stress radiographs are obtained. Radiographs are taken with application of an external rotation and abduction force through the ankle. Xenos and coworkers performed an experimental study with 25 cadaveric ankles. [190] Each ankle was tested under a constant external rotation torque. After sequential division of the syndesmosis and subsequent repair, the authors concluded that the lateral external rotation stress radiograph is superior to the mortise stress radiograph for the detection of syndesmosis injury.

Arthrography

Before the development of MRI, arthrography was commonly used to document injury to the syndesmosis. After injection of contrast material into the ankle, a positive test showed dye leakage more than 1 cm superior to the plafond. Broström and associates performed arthrography of 321 fresh ankle sprains. [54] Extra-articular leakage occurred in 239 cases (74%). Surgical exploration was performed in 99 of these cases and in an additional 6 cases that did not demonstrate leakage. The authors concluded that arthrography was useful within the first 7 days of an acute ankle sprain. Leakage of contrast into the peroneal tendon sheath correlated with tear of the CFL. Leakage in front of the lateral malleolus correlated with ATFL rupture. Leakage in front of the syndesmosis correlated with complete rupture of the syndesmosis. Leakage at the medial malleolus correlated with partial deltoid rupture.

Nuclear Imaging

Radionuclide imaging of the acute syndesmosis injury without fracture is not a common practice. Marymont and colleagues used radionuclide imaging 1 to 2 weeks after acute syndesmosis injury. Among 27 patients, the imaging was 100% sensitive and 71% specific. [191]

Computed Tomographic Scan

Computed tomographic scans are able to obtain images in the axial, sagittal, and coronal planes. These images can be reconstructed to create a detailed three-dimensional representation of the relationship of the tibia to the fibula. Ebraheim and coworkers demonstrated the superiority of CT over plan radiographs in detecting syndesmotic disruption. [192] They used a cadaveric model and plastic spacers to demonstrate the inability of routine radiographs and CT to identify 1-mm diastases at the syndesmosis. Computed tomographic scans identified 2- and 3-mm diastases. Routine radiographs failed to identify 2-mm and 50% of the 3-mm diastases.

Magnetic Resonance Imaging

MRI is a useful method for evaluation of syndesmosis injuries. Its accuracy, lack of ionizing radiation, noninvasiveness, decreasing cost, and increasing availability suggest that MRI is the imaging modality of choice for the ankle joint. Ligament discontinuity, wavy ligament contour, and the inability to image the ligament are all findings consistent with a syndesmosis injury. The high sensitivity associated with MRI mandates that images be carefully correlated with clinical findings (Fig. 25C1-37 A).

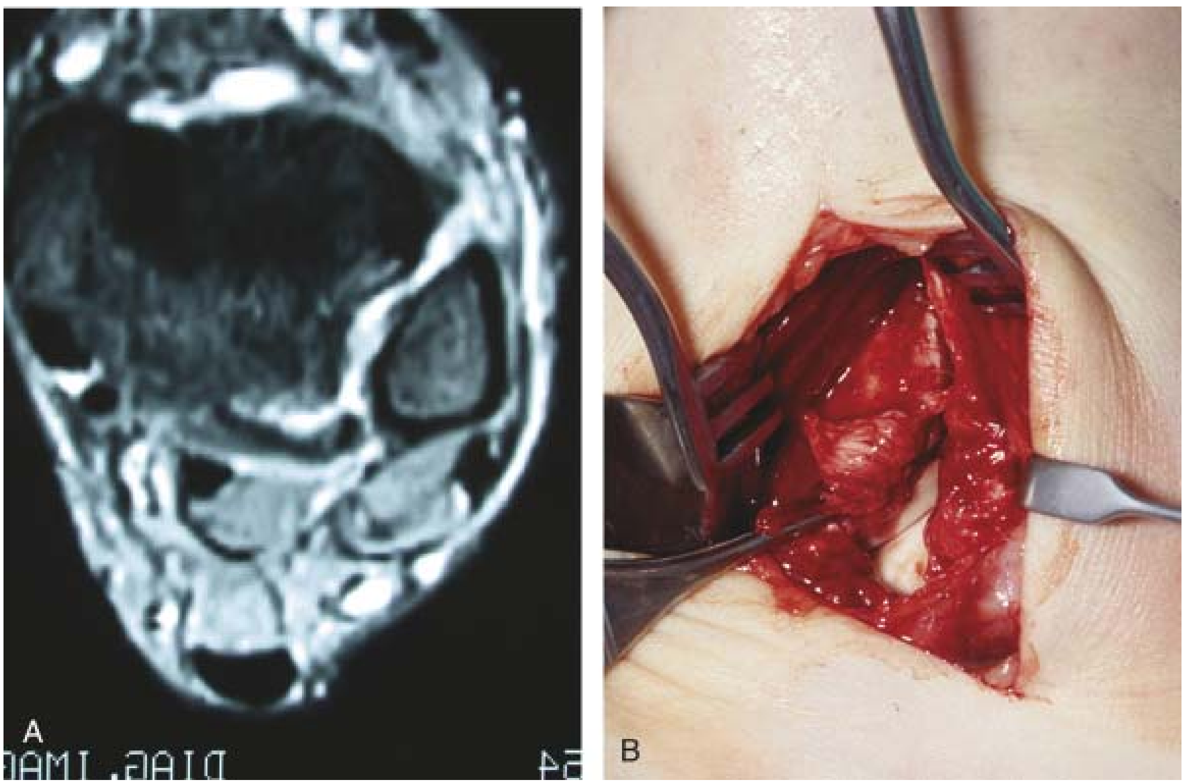

Figure 25C1-37 A, Magnetic resonance image demonstrating complete tear of AITFL and partial tear of PITFL. **B,** Open treatment of syndesmotic rupture.

Therapeutic Options

All acute injuries are treated with the RICE method and non–weight-bearing until definitive diagnosis is established.

Isolated Grade I and Grade II Syndesmosis Sprains

After the acute pain and swelling remit (within 72 hours), weight-bearing to tolerance is encouraged, and a rehabilitation program is instituted. Treatment is symptomatic with an emphasis on recovery of range of motion, strength, and coordination. A semirigid pneumatic ankle brace or taping is used throughout the rehabilitation period. Taping is applied with an effort to restrict or reduce external rotation. While facing the athlete, the trainer applies the tape from lateral to medial.

Gerber and coworkers prospectively evaluated 96 West Point cadets with acute ankle sprains.[3] Sixteen of these injuries were primarily syndesmosis sprains treated with early mobilization. Regardless of grade of injury, patients with syndesmosis sprains were most likely to experience an unacceptable outcome at 6 months' follow-up. The authors speculated that lack of accurate evaluation, underestimation of extent of injury, and incomplete rehabilitation may account for the poor results among patients with syndesmosis sprains.

Isolated Grade III Syndesmosis Injuries

Treatment of the complete syndesmosis disruption is based on displacement and stability. Latent injuries as described by Edwards and DeLee[181] may be treated with a protracted course of non–weight-bearing in a short leg cast or a removable cast boot. Non–weight-bearing status is maintained for 6 to 10 weeks depending on the resolution of local tenderness and pain with provocative maneuvers. Nonoperative treatment is recommended only for isolated, stable injuries. If the injury is associated with medial bony or ligamentous injury, internal fixation is suggested.

Frank injuries with a displaced or widened syndesmosis require closed reduction and internal fixation or open reduction and internal fixation (Fig. 25C1-38). The adequacy of the reduction is based on radiographs of the noninjured ankle. Based on the evaluation of 34 ankle fractures, Leeds and Ehrlich concluded that the poorly reduced tibiofibular diastasis predisposed the patient to a poor outcome and osteoarthrosis.[193] Reduction of the syndesmosis to within 2 mm of the contralateral side correlated with good subjective and objective results at an average follow-up of 4 years.

Figure 25C1-38 Syndesmosis rupture. **A,** Anteroposterior injury radiograph. **B,** Internal fixation with fully threaded 4.5-mm screw. **C,** Anteroposterior radiograph after screw removal.

Published recommendations for syndesmosis screw removal vary from as early as 6 weeks[194] to nonremoval. Most authors maintain screws for a minimum of 12 weeks after surgery. Harper reported syndesmosis failure following removal of syndesmosis screw fixation 6 and 8 weeks after insertion.[195] The syndesmotic screw eventually loosens and does allow at least some motion at the distal tibiofibular joint. [490] [491] [492] Grath noted in his comprehensive treatise that the removal or nonremoval of a syndesmotic screw does not produce detrimental effects.[198]

Thordarson and associates acknowledged the disadvantages of permanent screw fixation to include prominent painful hardware, disruption of normal biomechanical relationships at the syndesmosis, screw fracture, need for a second operative procedure if removal is selected, stress shielding of bone, and interference with MRI and CT.[199] To obviate the need for permanent hardware, the group tested 4.5-mm polylactide (PLA) screws against 4.5-mm stainless steel screws in a cadaveric model. They concluded that the PLA screws were of sufficient strength to maintain fixation and allow healing of the syndesmosis.

Surgical Repair

Technique: Closed or Open Reduction and Internal Fixation of the Ankle Syndesmosis (see Fig 25C1-37B)

1. The procedure is performed with the patient under general anesthesia. The patient is supine with a well-padded proximal tourniquet and a soft bump placed beneath the ipsilateral hemipelvis.
2. An image intensifier is used to attempt closed reduction of the syndesmosis. If reduction is not satisfactory, an open reduction is performed.
3. The extremity is exsanguinated and the tourniquet inflated. A linear incision is placed over the tibiofibular space about 2 to 3 cm above the plafond. Care is taken to protect the lateral branch of the superficial peroneal nerve as it courses over the syndesmosis.
4. The syndesmosis is exposed, and tearing of the AITFL is noted. The tear is approximated and repaired with absorbable suture. Avulsion fractures are repaired directly to the fibula or tibia. Open reduction is accomplished by débridement of the distal tibiofibular articulation. Reduction is performed and held with a large forceps on the tibia and fibula.
5. If reduction of the medial joint space remains incomplete, a medial ankle arthrotomy and a deltoid ligament repair are performed. Sutures are placed but are not tied until the syndesmosis is satisfactorily reduced and stabilized.
6. The syndesmosis repair is then protected by placement of one or two 3.5- or 4.5-mm screws. These screws are placed 2 cm [200] above the ankle joint line with the ankle in a neutral dorsiflexion position. The fixation is directed from the relatively posterior fibula into the more anterior tibia. Three or four cortices are captured with the screw. A nonlag technique is used in an effort to prevent over-reduction of the mortise and subsequent loss of ankle dorsiflexion (see Fig. 25C1-38 B).
7. The wound is closed in layers and a short leg splint applied. At 10 days, the incision is inspected and the patient is transitioned to a short leg cast or removable boot. Patients are prevented from bearing weight for a minimum of 4 weeks. After 4 weeks, a rehabilitation program is instituted and monitored. Partial weight-bearing is allowed after 4 to 6 weeks and progressed to full by 8 to 12 weeks. The fixation is removed 3 months after surgery, before resumption of athletic activity is permitted. The patient continues for 6 months with an ankle brace during rehabilitation and sports activities.

Unusual Syndesmosis Injuries

Olerud reported a single case of posterior dislocation of the fibula (Edwards and DeLee type III) and subluxated talus associated with a violent supination and external rotation mechanism of injury. [201] This patient was successfully treated with primary open reduction and syndesmotic screw placement.

Edwards and DeLee used a fibular osteotomy to reduce lateral displacement of the fibula associated with plastic deformity of the fibula (Edwards and DeLee type II). [181] The procedure was successfully used on two patients with this unusual injury.

Chronic Syndesmosis Sprains

Little literature exists on how to optimally treat chronic syndesmosis injuries. Reconstruction requires scar débridement from the syndesmosis and ankle joint. Reduction is held with large clamps while a tendon graft is placed through drill holes in the fibula and tibia to recreate the AITFL. Trans-syndesmotic fixation is placed to protect the reconstruction, and the medial deltoid ligament is repaired or reconstructed.

Some authors have advocated arthroscopic treatment of chronic syndesmotic injuries. [202] Patients who underwent resection of the torn portion of the interosseus ligament and chondroplasty reported improvement in postoperative pain, swelling, stiffness, and activity level. The authors also reported normalization of the external rotation stress test. Although the authors did not perform transsyndesmotic fixation, it can be done in conjunction with this procedure to stabilize the syndesmosis.

Katznelson and coworkers used a distal tibiofibular arthrodesis to treat chronic syndesmosis ruptures. [203] All five ruptures were the sequelae of injuries initially diagnosed as lateral ankle ligament sprains. All five patients obtained excellent results at the time of final follow-up. The report did not discuss return to athletic activity, nor was the time to follow-up noted.

Rehabilitation

Treatment of patients with low-grade syndesmosis sprains is symptomatic, with bracing and a rehabilitation program as outlined previously. Rehabilitation for postoperative syndesmosis injuries is effectively delayed by 4 to 6 weeks. Non–weight-bearing status is maintained for 6 to 8 weeks, followed by progression to full weight-bearing by 12 weeks.

Ankle and subtalar flexibility, motor function, and coordination [30] are emphasized throughout the protocol. The ankle is supported with an ankle brace during later rehabilitation and return to sports for 6 months postoperatively. In addition to the ankle brace, an elastic sock is available for mobilization of edema (see Fig. 25C1-19).

AUTHOR'S PREFERRED METHOD OF TREATMENT

I treat acute syndesmosis injury *not* associated with fracture with the RICE method and non–weight-bearing until the definitive diagnosis is established. Isolated grade I or II syndesmosis injuries are allowed to begin weight-bearing to tolerance after the acute pain and swelling remit. Treatment is symptomatic with an emphasis on recovery of range of motion, strength, and coordination. Patients frequently benefit from the support of a walking boot shortly after injury. They are later transitioned into a supportive ankle brace.

I use the Edwards and DeLee classification system for grade III sprains. [181] This system is based on the presence of radiographic diastasis with and without stress. A *latent* syndesmosis injury appears normal on an unstressed radiograph *and* abnormal or widened on external rotation stress mortise radiograph. A *frank* injury is seen as a widened syndesmosis on unstressed radiographs. Patients with latent injuries do not need surgery if the reduction of the fibula is anatomic on CT or MRI. Patients are not allowed to bear weight for at least 4 to 6 weeks. Sequential radiographs are used to monitor alignment. Patients are transitioned to a walking boot and are allowed to progressively start bearing weight after 6 weeks. Frank injuries require closed or open reduction and screw fixation. Rehabilitation is similar to that described for stable latent injuries. Screw fixation is removed 3 to 4 months postoperatively before resumption of full athletic activity is permitted.

Return-to-Play Criteria

Recovery follows a logical sequence of events. Once the initial pain and swelling subside, coordination and strengthening activities are emphasized. Gradually, the patient is able to return to walking, running, and cutting programs. Patients are returned to sport once they master sport-specific drills. A semirigid pneumatic ankle brace is maintained during this time.

Fritschy evaluated 10 world-class slalom skiers with syndesmosis injuries. [180] The unexpected finding was that all skiers returned to their original level of competition, but the time to return ranged between 18 months and 12 years.

Gerber and colleagues completed a prospective observational study of 96 West Point cadets with ankle sprains, including 16 syndesmosis sprains. [3] All patients were treated with a functional rehabilitation program. At the time of the 6-week and 6-month follow-up examinations, grade I syndesmosis sprains were associated with worse outcomes compared with all ankle sprains, including grade II and III syndesmosis sprains.

Ankle Dislocation without Fracture

Dislocation of the ankle joint is typically associated with major or minor bony injuries. Dislocation without associated fracture is rare.

Relevant Anatomy

The ankle joint ligamentous support is described in detail earlier. The stability of the ankle joint is such that ankle dislocation without associated malleolar fracture is uncommon.

Wilson and coworkers reviewed the literature and found 14 cases of ankle dislocation without fracture. [204] Most of these injuries were associated with falls or direct trauma. The authors further described two cases. The first was a posterior dislocation of the ankle and the second an upward dislocation of the ankle associated with a wide diastasis of the distal tibiofibular joint.

Clinical Evaluation

History

Dislocation of the ankle joint with or without fracture produces dramatic pain and deformity. The patient may report spontaneous reduction, or reduction on the field may be noted by the patient or a trainer. Reduction produces significant relief of pain. Information relevant to previous ankle and subtalar injury, the mechanism of injury, and current complaints represent the salient historical points.

Physical Examination

Examination of the patient includes evaluation of the entire extremity. Inspection of the leg, ankle, and foot may reveal swelling, ecchymosis, blister formation, or gross deformity. A vascular and sensory assessment is performed, followed by a palpation of the entire leg, ankle, and foot.

Imaging

Radiographs

Radiographs of a dislocated ankle are typically dramatic (Fig. 25C1-39). Three standard views are reviewed, and a lack of talocrural continuity is diagnostic for an ankle dislocation. Radiographs taken after a formal reduction or after spontaneous reduction are less dramatic but nonetheless must be obtained and carefully scrutinized for malleolar fractures, talar fractures, and osteochondral fractures (Fig. 25C1-40). Subsequent radiographs are obtained to ensure anatomic reduction and to monitor heterotopic ossification.

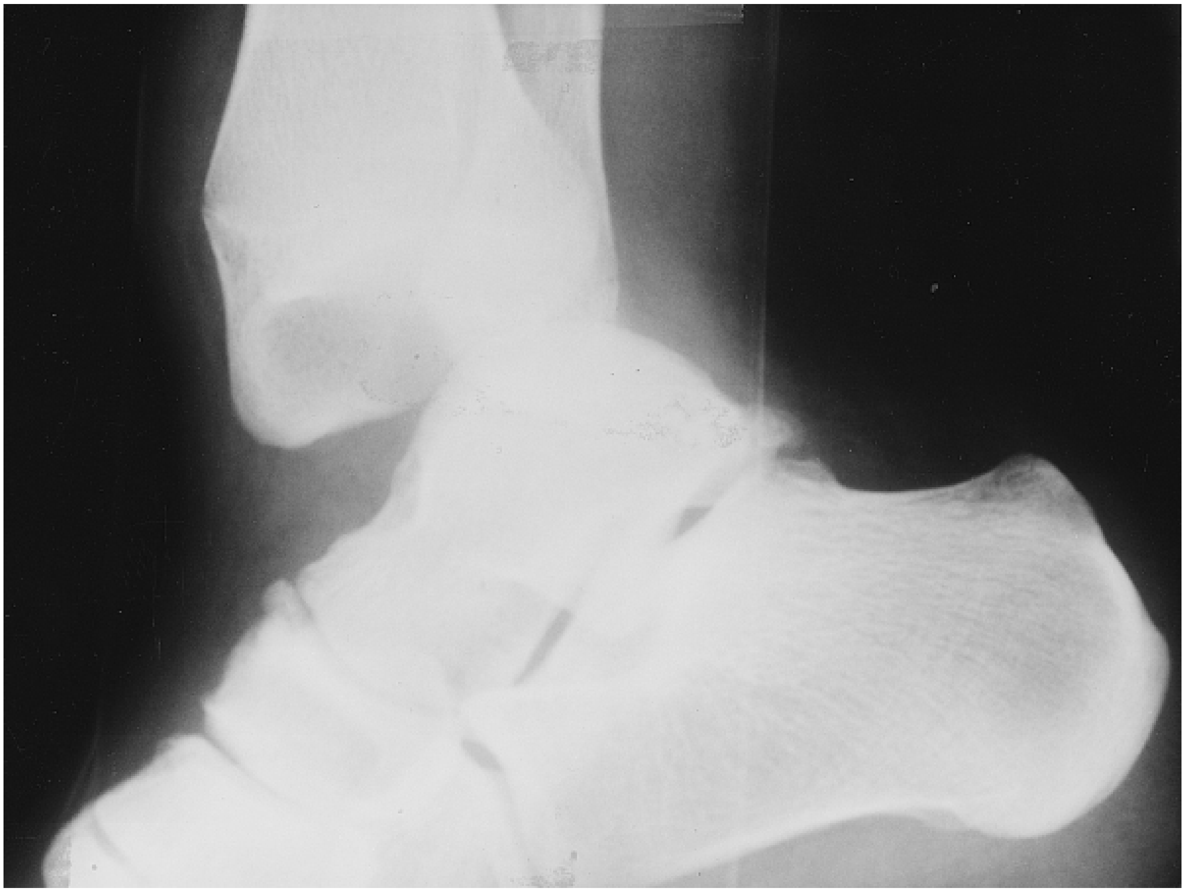

Figure 25C1-39 Ankle dislocation without fracture.

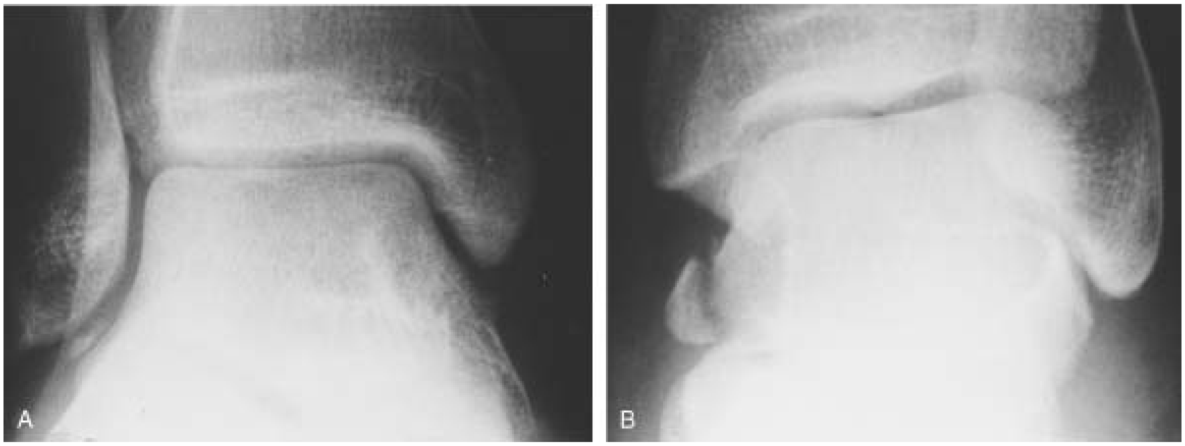

Figure 25C1-40 **A,** Ankle dislocation with fracture at the tip of the lateral malleolus. **B,** Ankle dislocation with fracture at the medial tuberosity of the talus (different case).

Magnetic Resonance Imaging

MRI is useful for delineation of ligamentous injury after ankle dislocation. The ligament remnants are occasionally imaged and may suggest continued nonoperative management. Osteochondral injury is also assessed.

Therapeutic Options

This injury is usually identified on the field. Reduction brings about tremendous pain relief and reduces ongoing swelling and damage to nerves, vessels, and articular cartilage.

Once the patient presents to the hospital, a complete examination is performed before reduction. General anesthesia is recommended, but early reduction with intravenous sedation may be possible. [204] If anatomic reduction is not possible, open reduction is required. If at the time of reduction, the ankle remains unstable, internal or external fixation is required. A tibiofibular diastasis associated with a syndesmosis rupture must also be reduced and stabilized (see previous section).

Olerud reported a single case of a posterior dislocation of the fibula and subluxated talus in association with a violent supination and external rotation mechanism of injury. [201] This patient was successfully treated with primary open reduction and syndesmotic screw placement.

Rehabilitation

The rehabilitation phase begins as soon as the ankle is deemed stable. The program focuses on reducing swelling and re-establishing range of motion. After completing rehabilitation, the athlete is gradually returned to progressive activity. An ankle brace is used to provide additional support and biofeedback.

AUTHOR'S PREFERRED METHOD OF TREATMENT

After radiographic documentation is obtained, a closed reduction is performed with the patient under conscious sedation or general anesthesia. The stability of the reduction is ideally simultaneously determined. Immobilization and complete radiographic studies, including CT or MRI, are obtained after reduction. The length of immobilization is determined by postreduction stability. Ideally, a non–weight-bearing cast is used for 4 weeks. This is followed by application of a removable cast boot for an additional 4 to 6 weeks of weight-bearing to tolerance and initial rehabilitation. A semirigid pneumatic orthosis is used for up to 6 months after the injury. Emphasis is placed on reducing swelling and improving range of motion, strength, and proprioception.

Return-to-Play Criteria

The patient must be prepared for an unpredictable return to competitive activity. Many pitfalls await the patient. The most significant is persistent stiffness. Return to play is allowed only after range of motion has been re-established and local tenderness and swelling have resolved.

INJURY TO THE FOOT LIGAMENTS

In the following section, *subtalar sprain, subtalar dislocation, bifurcate sprain,* and *Lisfranc sprain* are reviewed. First metatarsophalangeal joint sprain, also called *turf toe,* is reviewed in a separate section within this chapter (see Chapter 25H).

The incidence of injury to the foot ligaments is certainly lower than that to the ankle ligaments. The cause of injury is reviewed in great detail in a separate section within this chapter (see Chapter 25J).

Subtalar Sprain

Recently, more attention has been directed at the subtalar joint as a source of pathology. Subtalar joint injury varies from a mild subtalar sprain to a complete subtalar and talonavicular dislocation without fracture. Subtalar joint sprain is most commonly associated with a lateral ankle sprain. The injury less frequently presents as an isolated entity that produces persistent pain and instability after inversion injury to the foot and ankle. [205]

Relevant Anatomy and Biomechanics

A review of hindfoot bony anatomy and function facilitates understanding of the ligamentous anatomy in this region. The talus articulates with the calcaneus through the subtalar joint, which is composed of anterior, middle, and posterior facets. The most important of these articulations is the posterior facet. [206] The oblique empirical joint axis of the posterior facet converts rotatory movement from the leg to the foot. Internal rotation of the leg produces (by means of the oblique empirical axis) eversion of the calcaneus, which, in turn, places the foot in a supple configuration (pronation). External rotation of the leg produces the opposite effect of calcaneal inversion and increased foot rigidity (supination). Failure of the hindfoot to move through this natural motion reduces the effectiveness of the foot as a mechanical shock absorber and as a rigid lever for propulsion.

Subtalar motion is a complex three-plane motion that includes motion in the sagittal, frontal, and axial planes. [502] [503] [504] The clinical range of motion as described by Sarrafian includes 25 to 30 degrees of inversion and 5 to 10 degrees of eversion. [210] The determination of clinical range of motion is imperfect at best owing to the obliquity of the joint, its association with other joints, and the soft tissues surrounding the joint. Pearce and Buckley found a threefold overestimation of the clinical range of motion compared with the motion determined by CT. [211]

Stability of the subtalar joint is accomplished through bony configuration and ligamentous orientation. Bony stability is greatest with the calcaneus everted, a position that allows the greatest degree of posterior facet contact and congruity. [210]

Lateral subtalar joint stability is provided by a group of lateral ligamentous structures. Harper reviewed the anatomic literature and compiled data from 10 cadaveric dissections. [110] Based on Harper's work, the lateral ligaments of the subtalar joint are divided into three layers—superficial, intermediate, and deep (Figs. 25C1-41 and 25C1-42 [0440] [0445]; Table 25C1-5). The inferior extensor retinaculum is composed of two layers—superficial and deep to the extensor tendons. Harper concluded that the superficial layer of the inferior extensor retinaculum remains a constant, substantial tissue, suitable for lateral ankle and subtalar joint reconstructions, as proposed by Gould and associates. [109]

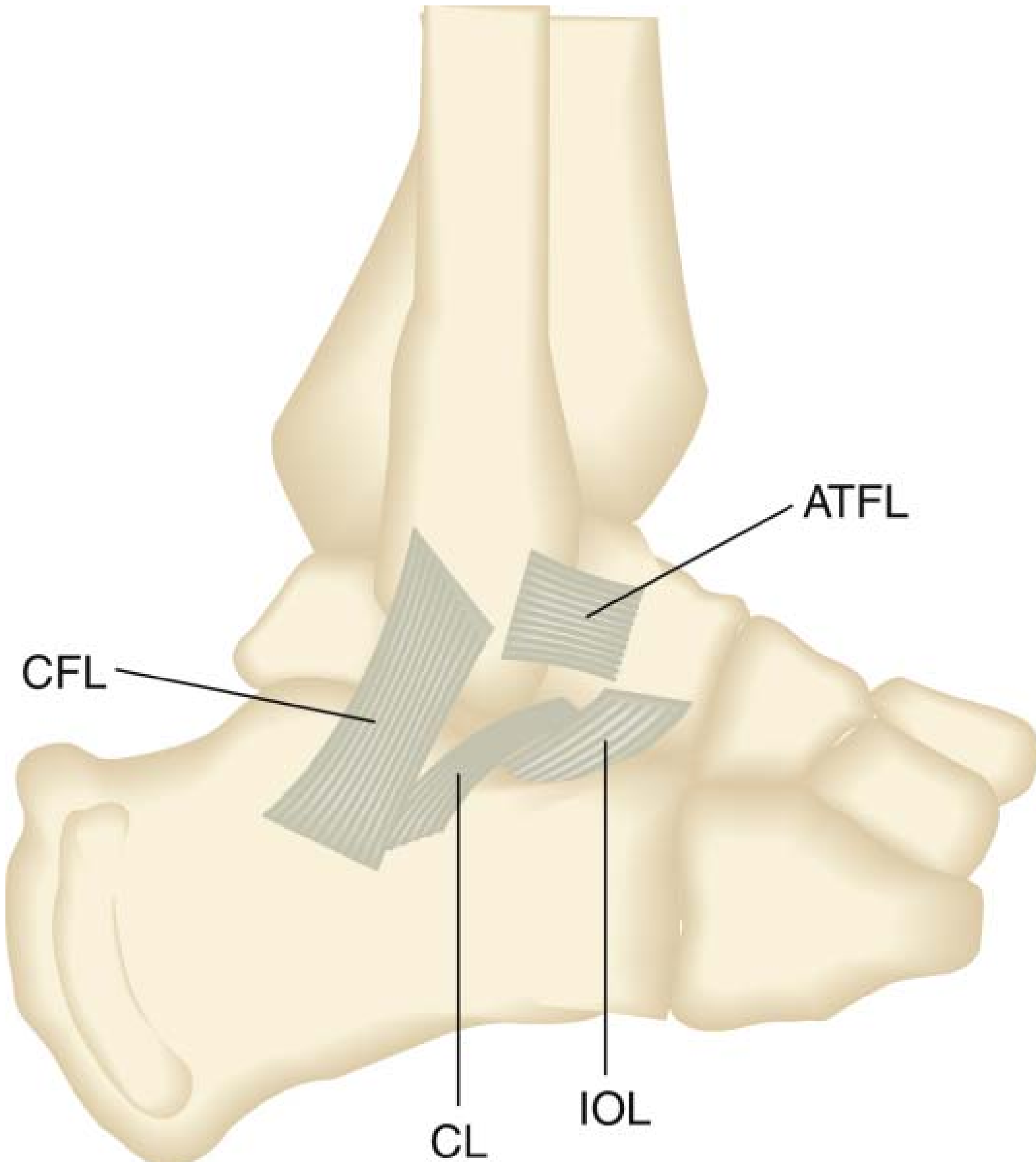

Figure 25C1-41 Anterolateral ligamentous structures of the subtalar joint—anterior talofibular ligament (ATFL), calcaneofibular ligament (CFL), cervical ligament (CL), and interosseous talocalcaneal ligament (IOL). *(Redrawn from Meyer JM, Garcia J, Hoffmeyer P, Fritschy D: The subtalar sprain: A roentgenographic study. Clin Orthop 226:169-173, 1988.)*

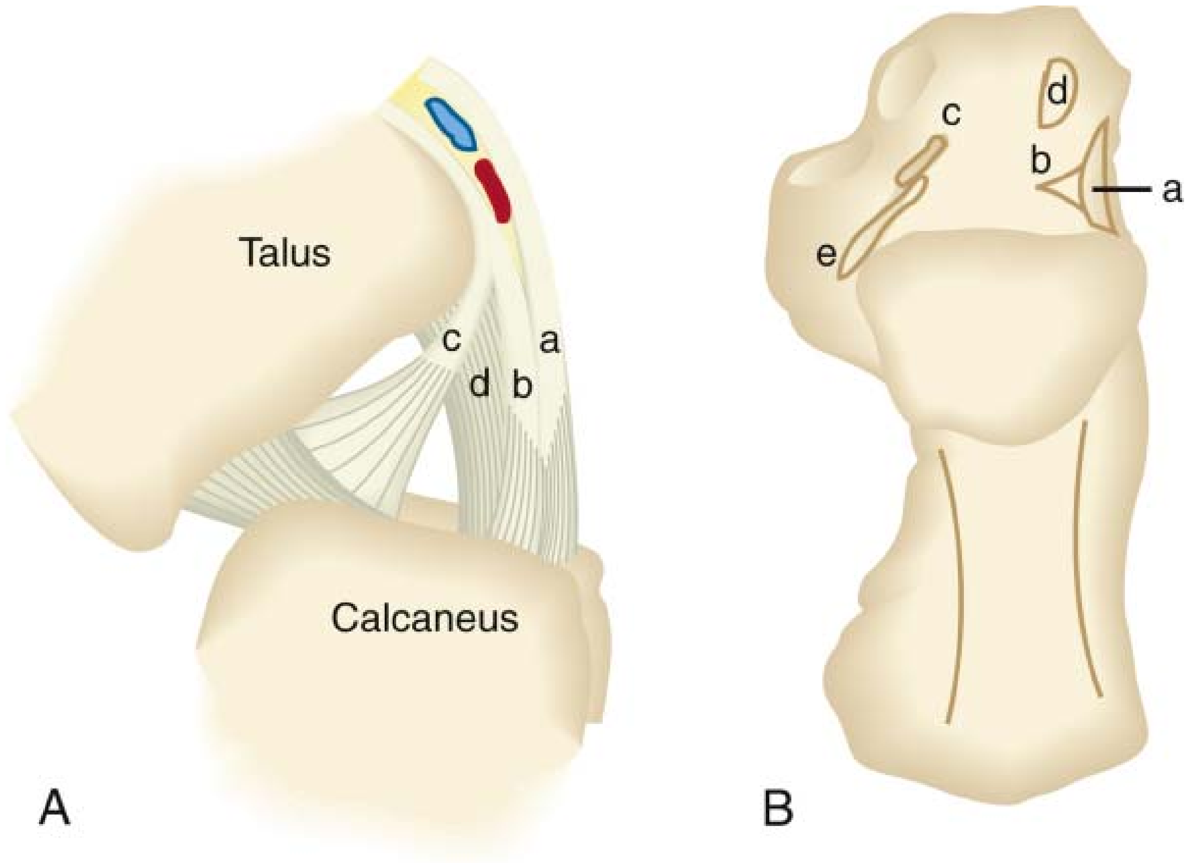

Figure 25C1-42 **A,** Ligaments of the sinus tarsi: a, lateral retinacular root; b, intermediate retinacular root; c, medial retinacular root; d, cervical ligament; e, interosseous ligament. **B,** Calcaneal attachments of the ligaments of the sinus tarsi. *(Redrawn from Harper MC: Lateral ligamentous support of the subtalar joint. Foot Ankle 11:354-358, 1991.)*

TABLE 25C1-5 -- Lateral Ligamentous Support of the Sinus Tarsi

Superficial layer	Lateral root of the inferior extensor retinaculum
	Lateral talocalcaneal ligament
	Calcaneofibular ligament
Intermediate layer	Intermediate root of the inferior extensor retinaculum
	Cervical ligament
Deep layer	Medial root of the inferior extensor retinaculum
	Interosseous talocalcaneal ligament

From Harper MC: Lateral ligamentous support of the subtalar joint. Foot Ankle 11:354-358, 1991.

Magnusson highlighted the importance of the talocalcaneal interosseous ligament, the ankle ligaments, and the medial and lateral malleoli for the restriction of extreme supination and pronation. [212] Smith, after empirically determining the axis of the subtalar joint, demonstrated that the cervical ligament limits inversion and the interosseous ligament limits eversion of the calcaneus across the subtalar joint. [213]

In 1968, Laurin and colleagues completed anatomic studies that demonstrated the importance of the CFL in maintaining subtalar stability. [23] This group sequentially divided the ATFL and the CFL (along with the lateral talocalcaneal ligament) before taking stress radiographs. Isolated division of the ATFL produced primarily talocrural instability, and isolated division of the CFL produced primarily talocalcaneal instability (lateral joint opening). The group further speculated that the mechanism of subtalar joint sprain (isolated CFL injury) is forced inversion of the foot below a dorsiflexed ankle.

Chrisman and Snook also used a cadaveric model to demonstrate that subtalar instability occurs after section of both the CFL and part of the lateral talocalcaneal complex. [91]

Kjærsgaard-Andersen and coworkers, also using a cadaveric model, obtained measurements continuously through the ankle range of motion with a constant adduction force across the tibiotalocalcaneal joint complex. [214] Isolated division of the CFL resulted in increased hindfoot adduction through the talocalcaneal joint, as opposed to the talocrural joint. This incremental difference was maximal at 5 degrees of ankle dorsiflexion. The clinical application, as suggested by the authors, is to place the ankle in slight dorsiflexion during stress testing of the ankle and subtalar joints.

A second cadaveric study by Kjærsgaard-Andersen and associates revealed that the CFL provides significant rotatory stability to the talocalcaneal joint. [215] External rotation after isolated division of the CFL increased up to 5.4 degrees at the tibiotalocalcaneal complex and up to 2.9 degrees at the talocalcaneal complex. The authors concluded that the CFL is the primary restraint to hindfoot external rotation.

Heilman and colleagues used 10 fresh cadaveric ankles to demonstrate that the CFL tightens with supination and dorsiflexion. [216] Selective division of the CFL produced 5 degrees of lateral opening across the posterior facet of the subtalar joint. Division of the lateral subtalar capsule added no further instability to the joint. Finally, division of the interosseous ligament completely destabilized the joint, leading to dislocation (Fig. 25C1-43).

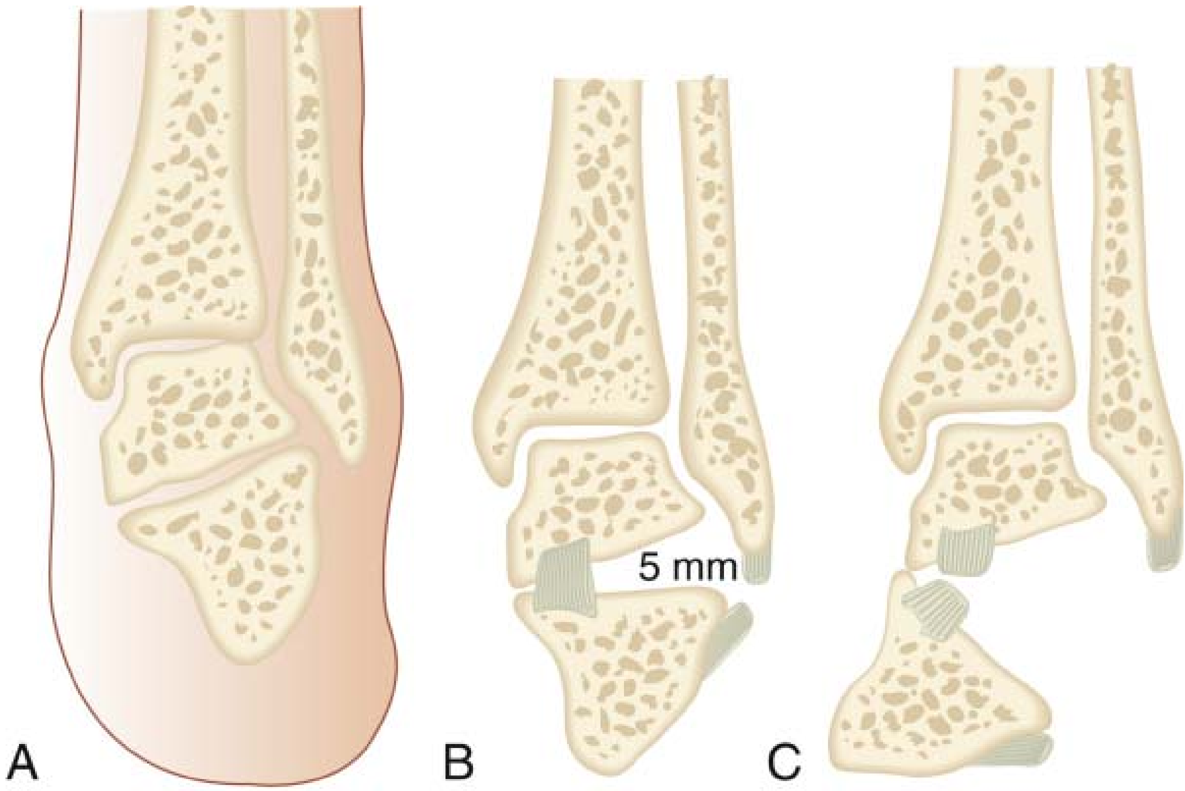

Figure 25C1-43 Stability of the subtalar joint after serial ligament sectioning. **A,** Brodén's view of the subtalar joint, intact ligaments. **B,** Brodén's view of the subtalar joint, after sectioning of the calcaneofibular ligament (CFL). **C,** Brodén's view of the subtalar joint, after sectioning of the CFL, capsule, and interosseous ligament. *(Redrawn from Heilmen AE, Braly G, Bishop JO, et al: An anatomic study of subtalar instability. Foot Ankle 10:224-228, 1990.)*

A third experimental study by Kjærsgaard-Andersen and coworkers demonstrated that isolated division of the cervical or interosseous ligament resulted in relatively minor increases in three-plane joint motion. [208] These authors concluded that the resulting instability was significant despite the small angular changes. Furthermore, injury to either ligament may be related to the sinus tarsi syndrome or talocalcaneal instability.

Knudson and associates used a cadaveric model to specifically study the effect of interosseous ligament division. [209] Measurements taken before and after interosseous ligament sectioning suggested that the interosseous ligament provides significant subtalar joint support, particularly in supination.

The exact cause of subtalar instability remains unsolved. Several factors, including local bony and ligamentous anatomy, contribute to this entity. Treating this condition requires an understanding of the anatomy and biomechanics of this region.

Clinical Evaluation

As has been stated previously, sprains are frequently evaluated by graded ligament injury, as suggested by the American Medical Association [35] and by O'Donoghue. [36] Acute injuries are graded based on stretch (grade I), partial tear (grade II), or complete rupture (grade III) of the subtalar capsule or supporting ligaments, including the CFL, the interosseus ligament, and the cervical ligament. A grade III tear is suggested by a clinical history of severe deformity or swelling, examination consistent with gross subtalar instability, or MRI demonstrating ligamentous disruption. The clinical evaluation provides additional information with regard to associated ligamentous injuries, especially of the lateral ankle.

History

History alone is usually insufficient to distinguish between subtalar and lateral ankle instability. Information relevant to previous ankle and subtalar injury, the mechanism of injury, the ability of the patient to continue to play or walk, and current complaints are important to obtain. Severe subtalar sprains are associated with a history of inversion injury with a characteristic pop; acute pain, swelling, or deformity; and the inability to continue activity. After the acute event, patients may report recurrent instability with walking, running, and sports. They frequently report difficulty on uneven surfaces. Lateral pain in the region of the sinus tarsi may be present.

Physical Examination

The differential diagnosis of an inversion foot or ankle injury includes ATFL sprain, CFL sprain, syndesmosis sprain, deltoid sprain, subtalar sprain, subtalar coalition, bifurcate ligament sprain, peroneal tendon instability, peroneal tendon tear, lateral malleolus fracture, talar dome osteochondral injury, anterior calcaneus process fracture, and fracture of the base of the fifth metatarsal. Chronic insufficiency of the lateral hindfoot associated with subtalar instability isolated or combined with lateral ankle ligament instability has been shown to occur in up to two thirds of patients. [34]

Examination of the patient includes evaluation of the entire extremity. Inspection of the leg, ankle, and foot may reveal swelling, ecchymosis, blister formation, or gross deformity. A vascular and sensory assessment is documented, followed by a palpation of the entire leg, ankle, and foot. Tenderness at the sinus tarsi, the ATFL, and the CFL is particularly important to note.

Clinical examination of the subtalar joint is difficult to complete owing to the complex nature of subtalar motion and its association with leg, ankle, and foot motion. [217] Evaluation of motion is determined by gently grasping the leg while the ankle is held at a right angle or in a neutral dorsiflexion position. With the widest part of the talus engaged into the ankle mortise, adduction of the heel is more likely to represent motion through the talocalcaneal joint rather than the talocrural joint. From a neutral position (heel vertical), maximal passive inversion and eversion are measured. An alternative method is placement of the empirical subtalar joint axis horizontal with the ground by allowing the foot to fall into 45 degrees of equinus, or by examining the patient prone with the knee flexed 135 degrees (Fig. 25C1-44). [10] Gross clinical instability is consistent with a grade III sprain.

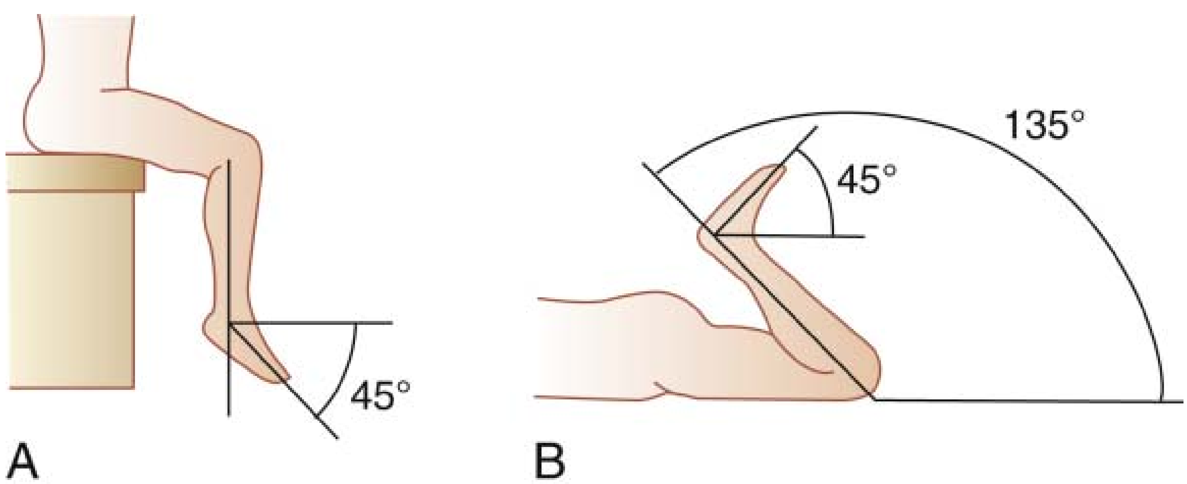

Figure 25C1-44 Clinical examination of the subtalar joint is facilitated by placement of the empirical subtalar joint axis horizontal with the ground. **A,** Place the foot in 45 degrees of equinus. **B,** Examine the patient prone with the knee flexed 135 degrees. *(Redrawn from Inman VT: The Joints of the Ankle. Baltimore, Williams & Wilkins, 1976, p 108.)*

Imaging

Radiographs

Radiographic demonstration of the subtalar joint is difficult. [218] Brodén described an oblique view of the foot designed to produce tangential images of the posterior facet of the subtalar joint (Fig. 25C1-45). [219] The view is obtained with the foot in 45 degrees of internal rotation and the beam centered on the sinus tarsi and angled posteriorly (by 10, 20, 30, or 40 degrees). The images are carefully inspected with attention to small fractures, nonconcentric joint alignment, loose body, and arthrosis.

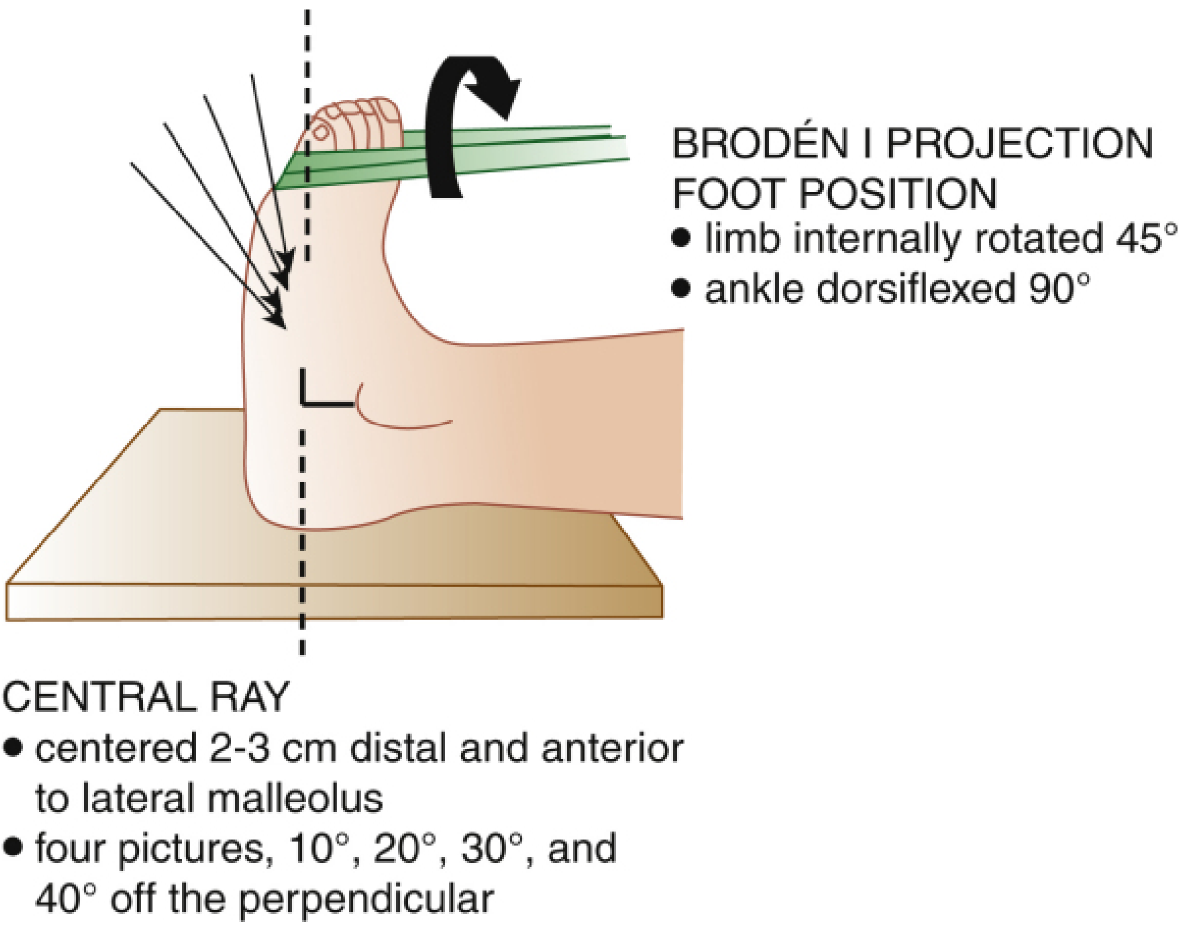

Figure 25C1-45 Brodén's oblique view of the foot designed to produce tangential images of the posterior facet of the subtalar joint. The view is obtained with the foot in 45 degrees of internal rotation and the beam centered on the sinus tarsi and angled posteriorly (10, 20, 30, or 40 degrees).

Stress Radiographs

Several methods for stress radiography of the subtalar joint have been described. Stress radiography of the ankle joint to quantify the extent of concomitant ankle instability is recommended and described previously.

The ankle *talar tilt test* is dependent on the contralateral ankle for control measurements. Varus tilt of the ankle, to a limited degree, is probably normal. Increased inversion of the calcaneus may represent talocrural (ankle) or talocalcaneal (subtalar) instability. [34] Radiographic data suggest that 4 degrees of varus tilt occurs in 10% to 15% of noninjured ankles.[47] Varus stress radiographs of both ankles in 90 injured and 90 normal ankles revealed that 6 degrees of increased tilt represents the transition from “normal to abnormal” tilt.[48]

The *subtalar varus tilt test* is performed with the ankle in a neutral position.[214] A Brodén view of the posterior facet of the subtalar joint is obtained as a varus stress is applied to the subtalar joint. Angular divergence or lateral opening at the posterior facet is compared with the contralateral foot for quantification of instability.

A relatively new method is the forced manual dorsiflexion-supination stress lateral radiograph described by Ishii and colleagues.[220] The true lateral ankle radiograph is used to establish the position of the lateral talar process relative to the posterior facet of the calcaneus. Clinical and cadaveric experiments indicate that this method is useful for detecting subtalar instability.

Stress radiographs can be performed in the office with a mini C-arm or in the operating room. A 40-degree Brodén view is obtained with inversion stress applied to both the calcaneus and the fifth metatarsal head laterally while the medial distal tibia is stabilized. Heilman and associates described a 5-mm separation between the talus and the calcaneus as indicative of subtalar instability.[221] The reliability of the stress Brodén view has been challenged by other authors. Harper completed a review of ankle and subtalar stress radiographs in 14 injured extremities, the contralateral noninjured extremities, and 18 additional asymptomatic extremities.[222] Lateral opening of the subtalar joint, as seen on the Brodén stress view, did *not* significantly differ between the injured and the uninjured contralateral extremity. Furthermore, results from the asymptomatic extremities revealed subtalar articular divergence between 0 and 20 degrees, with an average of 9 degrees. Similar findings using fluoroscopic methods have also been reported.[223]

Arthrography

Subtalar arthrography for evaluation of acute injury to the CFL or the interosseous ligament remains a useful technique. Meyer and coworkers performed ankle stress radiographs and subtalar joint arthrography in 40 patients with acute inversion sprains.[155] They classified the injuries based on the extent of lateral ankle ligament and subtalar ligament injury. Thirty-two (80%) of the patients sustained injury to both the lateral ankle and the subtalar joint. Six patients with negative stress ankle radiographs had positive subtalar arthrograms.

Sugimoto and associates recommend using an image intensifier to obtain anteroposterior, lateral, and 45-degree oblique views of the subtalar joint.[224] Extravasation of contrast into the peroneal sheath or ankle joint suggests CFL rupture; leakage into the sinus tarsi suggests interosseous ligament or anterior capsule of the posterior facet joint rupture. The authors also noted that this method is not limited by the lack of availability and the cost associated with MRI.

Computed Tomography

The inability of the stress Brodén view to provide useful screening for subtalar instability was confirmed by van Hellemondt and colleagues.[225] Using helical CT, the authors demonstrated no significant difference in subtalar tilt between a group of patients with suspected subtalar instability and the contralateral asymptomatic extremities. The authors suspect that translation produces the tilt seen on routine stress radiographs, as opposed to true divergence of the talus and calcaneus. It is interesting to note that CT did isolate four cases of fibrous middle facet coalition and a large calcaneal cyst. Finely cut CT is also useful in the evaluation of high-grade sprains to rule out associated fractures.

Magnetic Resonance Imaging

MRI is useful for the diagnosis of acute and chronic ankle and subtalar ligamentous injury[28] and their sequelae (see Fig. 25C1-9). With proper imaging, MRI can identify injuries to the cervical and interosseus ligaments. High sensitivity mandates that images be carefully correlated with clinical findings. MRI is also useful for identification of tarsal coalition.

Therapeutic Options

All acute injuries are treated with the RICE method, as detailed earlier. Patients with high-grade injuries are immobilized and protected from bearing weight until the acute symptoms resolve.

Grade I and Grade II Subtalar Sprains

Low-grade sprains are treated with early mobilization. Patients are allowed to bear weight to tolerance, and a rehabilitation program is instituted. Treatment is symptomatic, with an emphasis on recovery of foot and ankle range of motion, strength, and proprioception. A removable fracture boot is used during the initial recovery period, followed by use of a semirigid pneumatic ankle brace.

Grade III Subtalar Sprains

High-grade injuries, as determined by clinical findings, are treated with 3 weeks of immobilization in a short leg cast. Patients may bear weight as tolerated. Once clinical stability is achieved, a comprehensive foot and ankle rehabilitation program is instituted. A removable fracture boot is used during the second 3-week period, followed by the use of a semirigid pneumatic ankle brace.

Chronic Subtalar Instability

The Broström anatomic ligament reconstruction with the Gould modification reliably addresses lateral ankle and subtalar instability. The lateral capsular imbrication or advancement must extend to include the CFL as well as the ATFL. Imbrication of the extensor retinaculum to the lateral fibula effectively limits excessive subtalar motion.

Historical procedures using tendon graft or transfer to reconstruct the lateral ligaments also stabilize the subtalar joint. Elmslie's original technique using a piece of fascia lata to reconstruct the lateral ankle also constrained the subtalar joint. Chrisman and Snook's technique split the peroneal brevis and rerouted it to stabilize the ankle and subtalar joints. This nonanatomic reconstruction can overly constrain the subtalar joint. Newer techniques attempt to reconstruct the joints in a more anatomic manner.

Kato demonstrated that conservative management designed to prevent anterior translation of the calcaneus relative to the talus was effective for most patients.[226] Fourteen patients underwent reconstruction of the interosseus ligament with an Achilles tendon graft augmented by cervical and lateral ligament reconstruction. Because the reconstruction was performed near the center of rotation, subtalar motion was not limited (the publication did not provide data to substantiate this finding). All patients reported excellent outcomes.

Schon and coworkers reviewed several tendon transfers used for lateral ankle stabilization that also have utility for subtalar stabilization.[227] Stabilization with a tendon transfer is suggested in cases of severe injury, generalized ligamentous laxity, and previous failed reconstruction. Interosseous ligament reconstruction, as described by Schon, has the advantage of an anatomic reconstruction (Fig. 25C1-46). The anatomic configuration ensures preservation of ankle and subtalar motion. The authors recommend its use for mild subtalar instability.

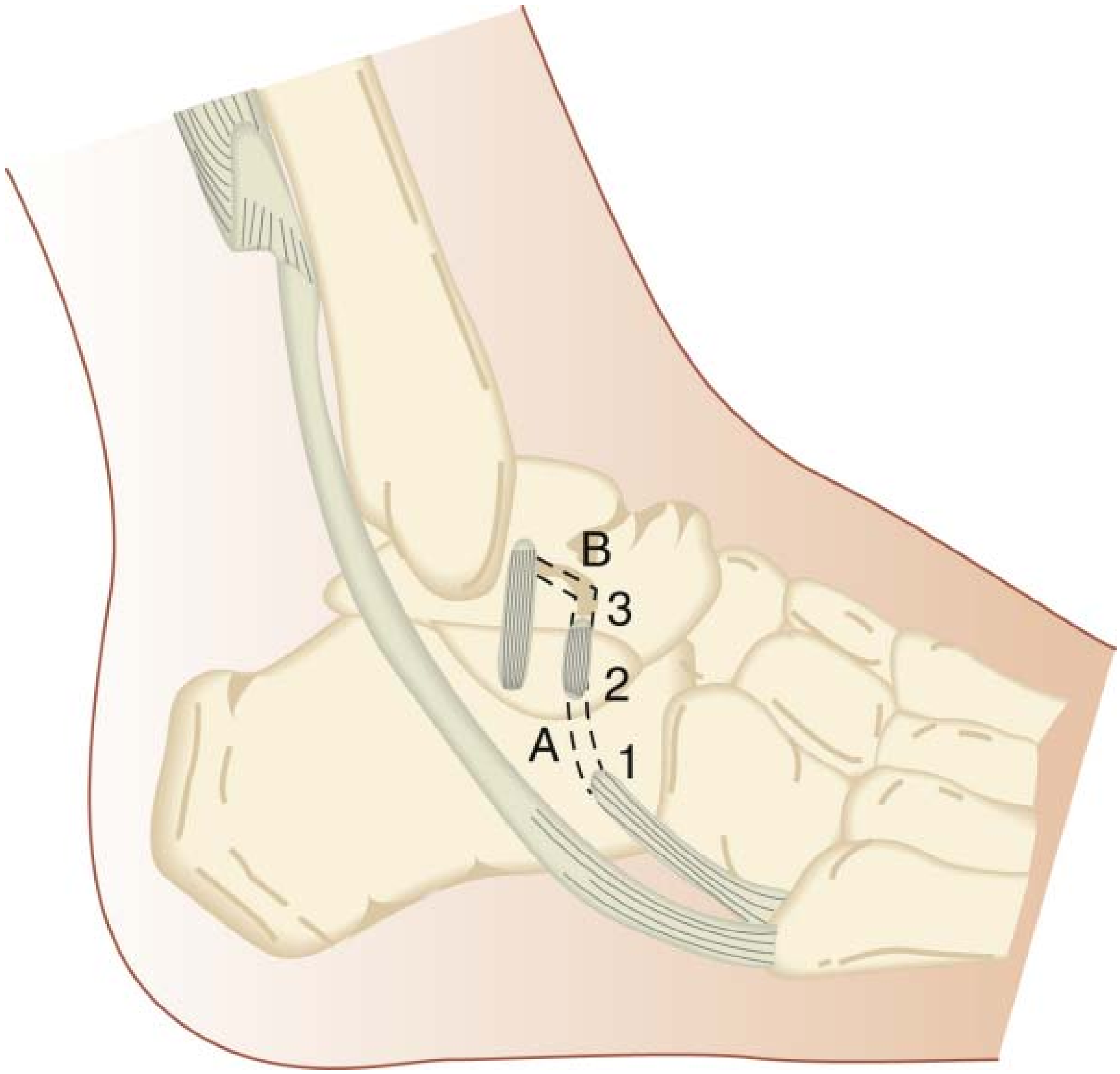

Figure 25C1-46 Interosseous ligament reconstruction as described by Schon has the advantage of an anatomic reconstruction. *(Redrawn from Schon LC, Clanton TO, Baxter DE: Reconstruction of subtalar instability: A review. Foot Ankle 11:324, 1991.)*

Rehabilitation

With nonoperative treatment, tissue injury initiates a predictable and sequential series of events known as the *healing response*. This response is typically divided into three phases with arbitrary and overlapping time lines.[120] The initial phase is the inflammatory phase and includes the first through third days after injury. The second phase is a proliferative phase of tissue repair that extends from day 3 to day 20. The final phase is a remodeling phase that proceeds after day 9.

To a certain degree, rehabilitation follows the phases of the healing response in an effort to reduce the undesirable effects of inflammation (e.g., pain, swelling, loss of function) while simultaneously promoting tissue repair and functional recovery. The emphasis of rehabilitation is placed on ankle, subtalar, midfoot, and forefoot flexibility, motor function, and coordination.[30] The hindfoot is supported by a functional ankle brace or various taping methods. An elastic sock is available for additional mobilization of edema (see Fig. 25C1-19).

The pain and inflammation associated with the first few days following subtalar joint sprain are addressed with rest, cold therapy, and whirlpool. A trial of electrical stimulation may be considered for nonbony injuries. Foot and ankle passive and active range of motion are re-established. Isometrics may be initiated as pain allows.

Once the acute pain subsides, flexibility is addressed in all planes. An inclined board facilitates stretching the gastrocnemius-soleus complex (see Fig. 25C1-20). Strengthening is initiated with towel scrunches (see Fig. 25C1-21), toe pick-up activities, manual resistive inversion and eversion, elastic bands (see Fig. 25C1-22), seated toe and ankle dorsiflexion with progression to standing, and seated supination-pronation with progression to standing.

Closed chain activities are gradually introduced, including one-leg balance, sports-specific activities on a trampoline, and use of the BAPS (see Fig. 25C1-23). Aerobic fitness is maintained with cross-training activities such as water running (see Fig. 25C1-24) and cycling.

Heat therapy, such as the application of warm packs, is a useful modality before the therapy session. It reduces pain and spasms and thus facilitates increased range of motion. Cold therapy, compression, and elevation are used after each therapy session to reduce inflammation.

Patients are allowed to progress to walking and running activities within the limits of a pain-free schedule. Once running activity is mastered, a monitored plyometric program is introduced with progression of difficulty. Schedules are carefully controlled to avoid reinjury.

Associated Injury

Sinus Tarsi Syndrome

O'Connor described a sinus tarsi syndrome as persistent pain at the sinus tarsi that follows a lateral ankle sprain.[228] Others have noted the syndrome to follow as a sequela of subtalar instability.[208] Taillard and associates noted that, in addition to pain over the sinus tarsi, patients with sinus tarsi syndrome complain of lateral ankle instability (Table 25C1-6).[229] The cause of the condition is poorly understood and few diagnostic criteria exist.

TABLE 25C1-6 -- Clinical Signs of Sinus Tarsi Syndrome

History of foot or ankle inversion sprain	Yes
Subjective instability	Yes
Tenderness	Localized to the sinus tarsi
Ankle stability	Yes/no
Anesthetic injection to the sinus tarsi	Temporary symptomatic improvement
Radiographic findings	Normal
Arthrography (subtalar)	Loss of sinus tarsi filling
Magnetic resonance imaging	Rupture of interosseous ligament or cervical ligament; bone edema, soft tissue swelling, or fibrosis at sinus tarsi
Laboratory testing (rule out systemic inflammatory process)	

Data from references 155, 228, and 229.

O'Connor speculated that the condition was related to fat pad scarring.[228] The diagnosis was made in 45 patients, 14 of whom were treated with sinus tarsi débridement, a procedure that included resection of the fat pad and the superficial ligaments at the floor of the sinus tarsi. Complete relief was noted in 9 patients and partial relief in the remaining 5.

Brown performed the O'Connor operation on 11 patients with sinus tarsi syndrome.[230] Ten patients reported significant relief. A single patient with early signs of talar osteoarthritis did not improve.

Meyer and Lagier presented four cases of sinus tarsi syndrome, all successfully treated by curettage of the sinus tarsi contents.[231] Histologic findings from the small series indicated fibrous scar formation consistent with a traumatic cause. Two of the patients were treated with simultaneous subtalar arthrodesis, and one was treated with repair of a ruptured CFL.

Parisien and Vangsness described the use of subtalar joint arthroscopy for evaluation of the posterior facet and its anterior recess.[232] Parisien used this technique to rule out intra-articular disease, to perform biopsy of synovium, and to release periarticular adhesions in three patients with post-traumatic hindfoot pain.[233]

A diagnostic approach to chronic sinus tarsi pain includes routine and stress radiographs to rule out ankle and subtalar instability as well as MRI to rule out contiguous arthrosis and ganglion formation. Local injection of anesthetic and corticosteroid is most important for diagnosis and initial empirical treatment. Ankle bracing and foot and ankle rehabilitation are used as adjunctive treatments. Occasionally, sinus tarsi exploration and débridement are performed either by open method or arthroscopically.

When indicated, open débridement begins with an incision over the sinus tarsi. Care is taken to avoid the lateral branch of the superficial peroneal nerve. The inferior extensor retinaculum is reflected distally along with the extensor digitorum brevis muscle. The capsule of the subtalar joint is incised. The joint is inspected for chondral injury and osteophytes, which are addressed as needed. Fibrofatty tissue is resected from the sinus tarsi, but the ligaments are kept intact. The wound is closed in a layered fashion with attention to reattachment of the extensor digitorum brevis. Patients are placed in a splint postoperatively. After 10 days, the patient is placed into a weight-bearing short-leg cast for 4 more weeks.

AUTHOR'S PREFERRED METHOD OF TREATMENT

Low-grade subtalar sprains are treated with a semirigid pneumatic orthosis, weight-bearing to tolerance, and early rehabilitation of the foot. Most of these injuries are associated with a lateral ankle sprain, and treatment is dictated by the ankle injury. High-grade injuries, as suggested by a clinical history of severe deformity or swelling, examination consistent with gross subtalar instability, or MRI demonstrating ligamentous disruption, are placed in non–weight-bearing short leg casts for 3 weeks. This is followed by application of a removable cast boot for an additional 3 weeks of bearing weight to tolerance. A comprehensive rehabilitation program follows, with an emphasis placed on reducing swelling and improving range of motion, strength, and proprioception.

Return-to-Play Criteria

Recovery follows a logical sequence of events. Once the initial pain and swelling subside, coordination and strengthening activities are emphasized. Gradually, the patient is able to return to walking, running, and cutting programs. A semirigid pneumatic brace (see Fig. 25C1-10) or taping accelerates the schedule. Patients are returned to sport once they have mastered sport-specific drills.

Subtalar Dislocation

Subtalar joint dislocation varies from isolated subtalar and talonavicular dislocation to dislocation associated with talar, calcaneal, or navicular fractures. The injury is unusual during athletic participation; it most commonly is associated with high-energy mechanisms.

Grantham reported five case of medial subtalar dislocation, all with an inversion mechanism of injury. [234] Four of these patients sustained the injury while playing basketball. Dendrinos and coworkers reported a single case of subtalar dislocation without fracture in a professional basketball player. [235] DeLee and Curtis identified 17 subtalar dislocations in a 7-year period. [236] The direction of the dislocation was anterior, lateral, and medial, for 1, 4, and 12 patients, respectively. Four of the medial dislocations were associated with an inversion mechanism of injury; each patient attained full subtalar range of motion and was asymptomatic at the last follow-up. Avascular necrosis did not occur.

Christiensen and associates noted arthrosis in each of 17 patients with a fracture-dislocation of the subtalar joint and 6 of 13 patients with isolated dislocation of the subtalar joint. [237]

Relevant Anatomy

A review of hindfoot bony and ligamentous anatomy is presented in the preceding section (see "Subtalar Sprain"). The subtalar joint is stable in an everted position. This suggests that lateral dislocations are more likely to be associated with higher degrees of trauma, fractures, and less favorable outcomes. [512] [532] [533]

Clinical Evaluation

History

Patients with subtalar joint dislocation present with a history of acute and severe pain associated with obvious hindfoot deformity.

Physical Examination

After trauma protocols are followed, the extremity is carefully examined (Fig. 25C1-47). Open injuries are documented along with the neurologic and vascular status of the extremity.

Figure 25C1-47 Subtalar dislocation.

Imaging

Radiographs

Routine radiographs of the foot and ankle in orthogonal views confirm the preliminary diagnosis. Repeat radiographs with anteroposterior, oblique, Brodén,[219] and lateral projections, as well as CT, are required before definitive and complete diagnosis is determined.

Therapeutic Options

Subtalar dislocation without fracture is treated expediently with closed reduction. Reduction is required for decompression of neurovascular structures and should not be delayed without appropriate reason. General anesthesia is probably more predictable and is less likely to produce associated injury to the foot. The knee is flexed in an effort to relax the gastrocnemius muscle.[217] Relocation is performed by accentuation and then reversal of the deformity along with traction applied through the foot.

As has been noted earlier, repeat and complete radiographs and CT are required for verification of subtalar joint reduction and assessment of associated fractures. After reduction of isolated subtalar joint dislocations is performed, the joint is usually stable. With a stable joint, immobilization for 4 to 8 weeks is indicated. DeLee and Curtis suggest 3 weeks of immobilization, immediate toe range of motion, and early subtalar range of motion. Recurrent dislocation did not occur in any of the 17 subtalar dislocations in the series.[236]

Dendrinos and colleagues reported a single case of subtalar dislocation without fracture in a professional basketball player.[235] The patient was treated with closed reduction. After 5 years of continued professional play, the same foot suffered an almost identical subtalar dislocation. The recurrence was attributed to coincidence.

Rehabilitation

After a period of immobilization, a comprehensive foot and ankle rehabilitation program (as described earlier in the section, "Subtalar Sprain") is instituted for maximal recovery of hindfoot function. Continued subtalar support is provided by a semirigid pneumatic orthosis.

AUTHORS'S PREFERRED METHOD OF TREATMENT

Subtalar dislocations, both related and unrelated to athletic activity, warrant significant attention at the outset. Historical information to determine the mechanism of injury is paramount. Trauma protocols are followed as indicated. After radiographic documentation is obtained, a closed reduction is performed with the patient under conscious sedation or general anesthesia. The stability of the reduction is assessed at the time of reduction. Complete radiographic studies, including CT, are obtained after reduction. The length of immobilization is determined by postreduction stability. A stable reduction is immobilized initially in a non–weight-bearing cast for 2 weeks. This is followed by application of another short leg cast or removable fracture boot for an additional 4 to 6 weeks. Patients are encouraged to bear weight as tolerated. Initial rehabilitation emphasizes swelling reduction and improved range of motion, strength, and proprioception.

Return-to-Play Criteria

After completing short-term rehabilitation, the athlete is gradually returned to progressive activity. An ankle brace is used to provide additional support and biofeedback. Return to play is allowed only after local tenderness and swelling have resolved.

Bifurcate Sprain

Injuries to the bifurcate ligament typically result from forceful inversion and plantar flexion of the foot. This results in either bifurcate ligament sprain or an avulsion fracture of the anterior process of the calcaneus.

Broström noted clinical evidence of bifurcate ligament injury in 18.6% of patients with acute ankle sprains and 3.7% of patients with confirmed lateral ankle ligament ruptures.[40] Backmann and Johnson considered bifurcate ligament rupture to be a common injury.[239] Søndergaard reported a 24% incidence of bifurcate or talonavicular sprain among patients presenting with an acute ankle or foot inversion sprain.[240] An additional 9% of patients were noted to have a combination of bifurcate and talonavicular ligament and lateral talocrural ligament injuries.

Relevant Anatomy

The bifurcate ligament is a short, stout ligament that originates from the anterior process of the calcaneus, divides into two arms, and inserts onto the navicular and cuboid (Fig. 25C1-48 ; see Fig. 25C1-1).[206] The origin is contiguous with the superior aspect of the calcaneal facet of the calcaneocuboid joint. The origin of the ligament is routinely visualized during the lateral approach to the sinus tarsi for triple arthrodesis. The ligament is distal and anterior to the inferior tip of the fibula, and superior and proximal to the base of the fifth metatarsal.

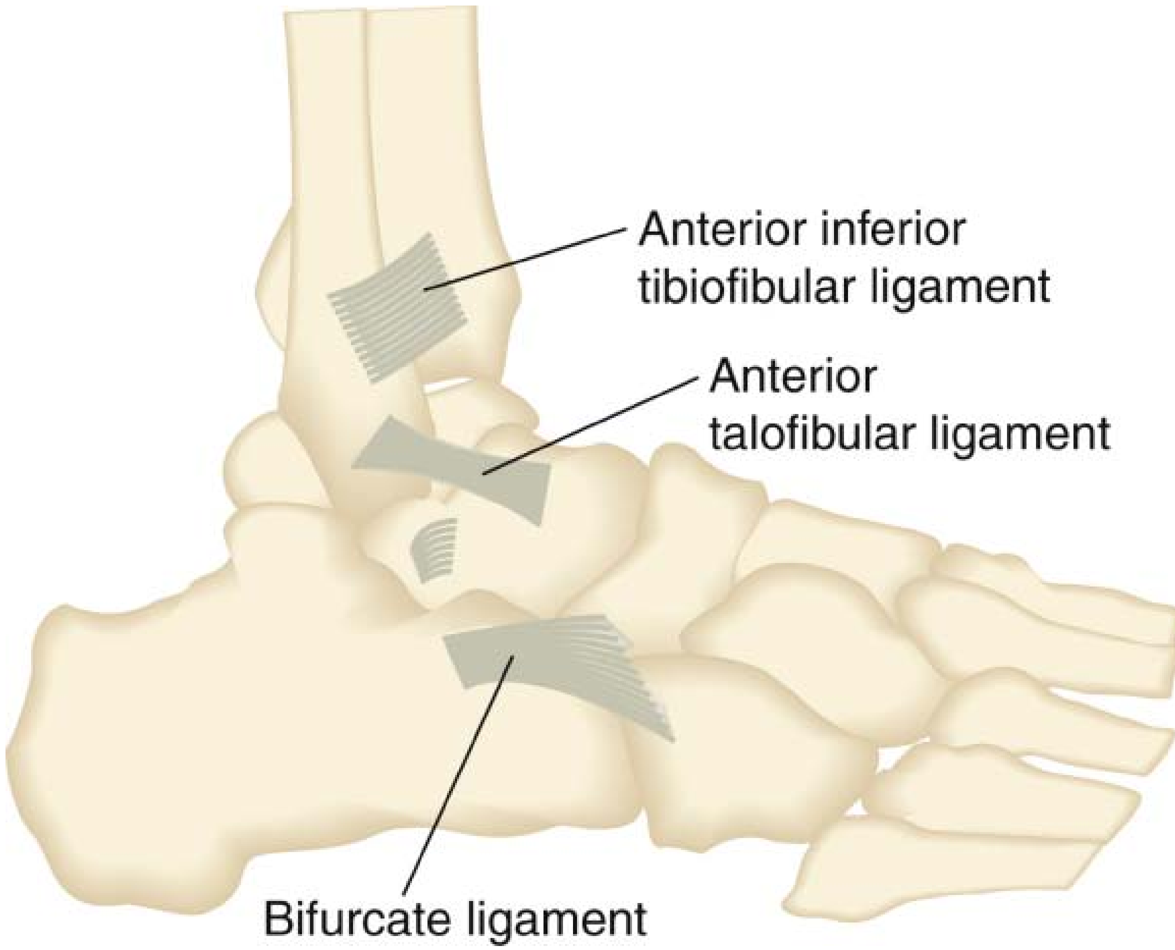

Figure 25C1-48 Schematic drawing demonstrating the bifurcate ligament.

Clinical Evaluation

Sprains can be classified according to the perspective of graded ligament injury, as suggested by the American Medical Association[35] and by O'Donoghue.[36] Acute injuries are graded based on stretch (grade I), partial tear (grade II), or complete rupture (grade III) of the bifurcate ligament. Additional information with regard to associated ligamentous injuries, especially at the lateral ankle, is noted.

History

A bifurcate ligament sprain or an avulsion fracture of the anterior process of the calcaneus must be considered when a patient presents with a suspected ankle sprain. The plantar flexion inversion mechanism associated with injury to the ATFL is the same mechanism as that responsible for the bifurcate ligament sprain. The patient often recalls a pop or snap followed by swelling and ecchymosis. The patient's ability to continue play after the acute injury is variable.

Physical Examination

Physical examination confirms diffuse lateral hindfoot and midfoot swelling with associated ecchymosis. Tenderness tends to localize to the course of the bifurcate ligament, an area that is distinct from the course of the ATFL. The ankle and midfoot remain stable. Pain is easily reproduced with forced inversion of the plantar flexed foot. Broström noted that the differentiation between lateral ankle ligament injury and bifurcate ligament injury was best achieved by eliciting indirect tenderness.[40] He suggested manipulation of the heel to produce lateral ankle pain and stabilization of the heel with simultaneous forced forefoot motion to produce bifurcate pain.

Imaging

Radiographs

Routine radiographs, including anteroposterior, lateral, and oblique views of the foot and ankle, are obtained. A pure bifurcate ligament sprain is not associated with bony injury; however, an avulsion fracture of the anterior process of the calcaneus is confirmed with the lateral radiograph (Fig. 25C1-49). The size of the fragment may vary from a fine calcified body to a significant portion of the anterior process and the contiguous calcaneocuboid facet.

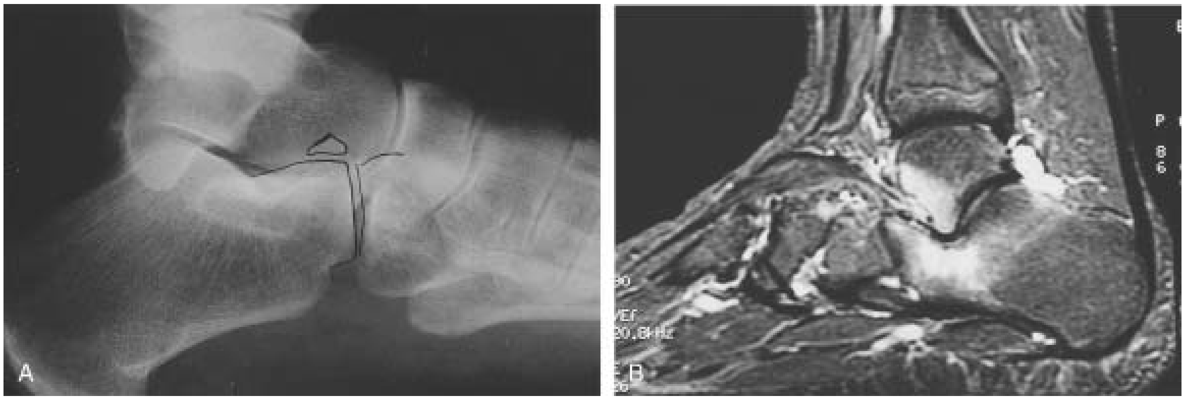

Figure 25C1-49 A, Avulsion fracture of the anterior process of the calcaneus. The triangular fragment is either intra-articular or extra-articular and varies in size. **B,** Magnetic resonance image demonstrating increased signal within the bifurcate ligament consistent with sprain.

Computed Tomography

Computed tomography is the preferred method for assessing avulsion fractures, but it is not particularly useful for evaluating an isolated bifurcate ligament sprain.

Magnetic Resonance Imaging

Isolated bifurcate ligament injury is not routinely imaged by MRI. A sprain is confirmed by edema within or adjacent to the ligament as well as by increased marrow signal at the anterior process of the calcaneus.

Therapeutic Options

Acute bifurcate sprains are treated with the RICE method followed by gentle range of motion and protected weight-bearing. The hindfoot is supported by the use of a variety of methods, including a splint, a walking cast, a removable boot, a functional ankle brace, or various taping methods.

Chronic bifurcate sprains are treated with a range of motion program and reduced activity levels. Intralesional and intra-articular (calcaneocuboid) steroids are placed, under fluoroscopic guidance, with the patient either in the sports medicine practitioner’s office or in a radiology suite.

Operative treatment is rare for bifurcate ligament injuries. Large, displaced, intra-articular anterior process fractures are treated with open reduction and internal fixation through a sinus tarsi approach. The extensor digitorum brevis muscle is elevated and the fragment reduced and provisionally pinned. Fixation is accomplished with a small or mini-fragment screw. For symptomatic nonunions, the same approach is used, with the addition of local bone graft. Unfortunately, most bony nonunions are not amenable to open reduction with internal fixation owing to their small size. Excision is a reasonable option, but it does not yield the immediate relief that both patient and surgeon expect. Therefore, excision of a symptomatic nonunion is considered only after 6 to 12 months of rehabilitation.

Rehabilitation

Emphasis throughout the protocol is placed on rehabilitation of the foot and ankle and on subtalar flexibility, motor function, and coordination.[30] The foot is supported by a functional ankle brace or various taping methods. An elastic sock is available for additional mobilization of edema (see Fig. 25C1-19).

In the acute phase, the athlete’s pain and inflammation are addressed with rest, cold therapy, and whirlpool. A trial of electrical stimulation may be considered for nonbony injuries. Foot and ankle passive and active range of motion are re-established. Isometrics may be initiated as pain allows.

Once the acute pain subsides, flexibility is addressed in all planes. An inclined board is a useful adjunct to gastrocnemius-soleus and Achilles stretching (see Fig. 25C1-20). Strengthening is initiated with towel scrunches (see Fig. 25C1-21), toe pick-up activities, manual resistive inversion and eversion, elastic bands (see Fig. 25C1-22), seated toe and ankle dorsiflexion with progression to standing, and seated supination-pronation with progression to standing.

Closed chain activities are gradually introduced (see Fig. 25C1-23), including one-leg balance, sport-specific activities on a trampoline, and use of the BAPS. Aerobic fitness is maintained with cross-training activities such as water running (see Fig. 25C1-24) and cycling.

Heat therapy, such as the application of warm packs, is a useful modality before the therapy session. It reduces pain and spasms and thus facilitates increased range of motion. Cold therapy, compression, and elevation are used after each therapy session to reduce inflammation.

As patients prepare to return to sports, walking and running activities are progressed within the limits of a pain-free schedule. Once running activity is mastered, a monitored, plyometric and cutting program is introduced. Schedules are carefully controlled to avoid reinjury.

Søndergaard and coworkers reviewed the results of treatment for 162 midtarsal sprains (bifurcate ligament or talonavicular ligament, or both) and 161 talocrural sprains and concluded that the two injuries produce similar outcomes.[240] Both groups returned to preinjury athletic activity at an average of 21 days.

AUTHORS' PREFERRED METHOD

Bifurcate ligament sprains are initially treated with a short removable fracture boot. Patients are allowed to bear weight as tolerated. Early rehabilitation of the foot and ankle follows. Occasionally, I place a less athletic patient in a short leg walking cast in an effort to improve pain control and allow increased levels of independent activity.

Injuries associated with small anterior process fractures are treated as severe sprains. Delayed union or nonunion is unlikely and is even less likely to remain symptomatic. Large, intra-articular fractures are treated with a short leg cast and weight-bearing to tolerance. Displaced fractures are treated surgically with internal fixation.

Chronic pain related to nonunion or malunion is treated conservatively for a minimum of 6 to 12 months. If a small fragment remains symptomatic, it is excised. Attempted union of larger fragments requires open treatment with internal fixation and bone grafting.

Return-to-Play Criteria

Recovery follows a logical sequence of events. Once the initial pain and swelling subside, coordination and strengthening activities are emphasized. Gradually, the patient is able to return to walking, running, and cutting programs. A protective brace or taping accelerates the schedule. Patients are returned to sport once they master sport-specific drills.

Lisfranc Sprain

Much of the literature discussing injury to the Lisfranc joint complex is in association with severe high-energy fracture-dislocations. The poor outcome among these patients is well known by orthopaedic surgeons. Subtle Lisfranc injuries can occur during athletic activity and can be challenging to diagnose.

Curtis and associates described 19 such injuries associated with athletic activity.[241] The most common activity was basketball, followed by running. Meyer noted 24 midfoot sprains among university football players between 1987 and 1991.[242] The incidence was calculated at 4% of football players per year. Shapiro and colleagues identified nine injuries to the Lisfranc ligament associated with collegiate gymnastics (four), collegiate football (three), collegiate pole vault (one), and recreational tennis (one).[243]

Relevant Anatomy

The midfoot is a stable configuration of five bones (navicular, cuboid, medial cuneiform, middle cuneiform, and lateral cuneiform) joined together in a complex system of multifaceted, relatively immobile joints (Fig. 25C1-50). The tarsometatarsal articulation between the midfoot and the metatarsals is known as the *Lisfranc joint.* The second metatarsal cuneiform joint is the most stable of the entire complex. Two factors contributing to the second metatarsal cuneiform joint stability include a recessed bony configuration (keystone) and a strong plantar ligament connecting the base of the second metatarsal to the medial cuneiform (Lisfranc's ligament).

Figure 25C1-50 The Lisfranc articulation with its ligamentous attachments. Note the recessed second tarsometatarsal joint and the Lisfranc ligament in place of the first-second intermetatarsal ligament.

A significant amount of force is required to produce fracture-dislocation. The mechanism of injury is either direct crushing or indirect loading of the fixed forefoot. [244] Wiley reviewed 20 cases of Lisfranc injury and identified direct and indirect mechanisms of injury. [245] The indirect mechanisms of injury were acute abduction of the forefoot (most common) and forced plantar flexion of the forefoot.

Forced dorsiflexion of the forefoot may occur as the result of landing from a jump, continued forward motion on a planted forefoot, a fall from a height, or a brake pedal injury. Curtis and coworkers described the mechanism of injury to include plantar flexion and rotation with or without abduction of the forefoot. [241] Meyer and associates determined a mechanism of injury from 16 football players with midfoot sprains. [242] Eight players reported an indirect twisting mechanism; six players reported contact to the heel of a plantar flexed forefoot; two players reported a crush injury to the dorsum of the foot. Shapiro and colleagues identified a consistent mechanism in nine athletes who sustained a Lisfranc injury. [243] Each athlete placed full weight onto the first ray with the foot in an externally rotated and pronated position.

The resulting injury includes tearing of a relatively weak dorsal capsular structure, tearing of the strong plantar ligament between the medial cuneiform and the base of the second metatarsal, and to a varying degree, fracture of chondral and bony structures on both sides of the joint. Subsequent to the displacement that occurs at the time of injury, the joint complex either returns to a nondisplaced state or remains displaced owing to the interposition of the capsule and osteochondral fragments.

Clinical Evaluation

Sprains can be classified according to ligament injury, as suggested by the American Medical Association [35] and by O'Donoghue. [36] Acute injuries are graded based on stretch (grade I), partial tear (grade II), or complete rupture (grade III) of the Lisfranc capsule and supporting ligaments, including the Lisfranc ligament. Stable injuries, including grade I and II sprains, are not associated with displacement or deformity. Unstable injuries, grade III sprains, vary between nondisplaced injuries and frank fracture-dislocations.

Nunley and Vertullo have proposed a classification system for athletic midfoot injuries. Stage I represents a ligament sprain with no diastasis or loss of arch height on a weight-bearing radiograph. Stage II injuries have diastasis of 1 to 5 mm between the first and second metatarsals without arch height loss. Stage III injuries are associated with diastasis and loss of arch height as defined by a decrease or reversal in the distance between the plantar medial cuneiform base and the fifth metatarsal base on a weight-bearing lateral foot radiograph. [246]

History

Typically, the athlete can recall a specific mechanism of injury. The injury is associated with a pop or snap followed by pain, swelling, and ecchymosis localized to the midfoot. Some patients are able to bear weight with pain, but are unable to run, jump, or continue to play after the acute injury. After a severe injury, weight-bearing is very painful and unlikely. Pain localized to the midfoot should raise suspicion for a subtle Lisfranc injury.

Physical Examination

Physical examination confirms diffuse midfoot swelling with associated ecchymosis. Sensory and vascular examination with particular attention to the deep peroneal nerve and the dorsalis pedis artery is documented.

Stability is tested in the sagittal plane (dorsiflexion and plantar flexion) by securing the midfoot with one hand and grasping the first metatarsal with the other hand. A dorsiflexion force is applied; when compared with the opposite midfoot, pain and increased mobility are abnormal findings. Frontal plane stability is demonstrated by applying an adduction or abduction force across the Lisfranc joint. Myerson and colleagues described a passive pronation-abduction test to evaluate the stability of the joint complex (Fig. 25C1-51). [542] [543] [544] This maneuver elicits pain and reproduces the patient's symptoms.

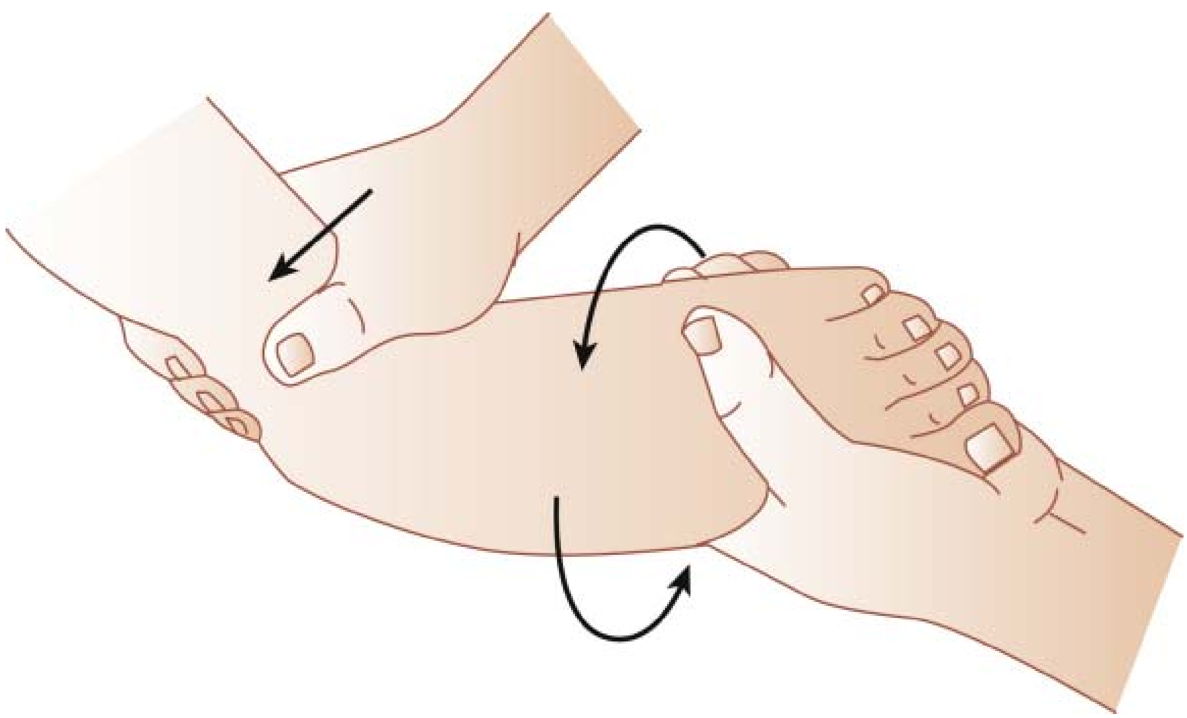

Figure 25C1-51 Frontal plane stability at the Lisfranc joint is demonstrated by application of a passive pronation-abduction. *(Redrawn from Komenda GA, Meyerson MS, Biddinger KR: Results of arthrodesis of the tarsometatarsal joints after traumatic injury. J Bone Joint Surg Am 78:1668, 1996.)*

Imaging

Radiographs

The bilateral standing anteroposterior radiograph provides critical information used to help classify the injury. Additionally, weight-bearing lateral and oblique views of the foot are obtained. A diastasis between the bases of the first and second metatarsals suggests an unstable injury. A small fragment of bone, called the *fleck sign*, represents an avulsion fracture of the Lisfranc ligament from the base of the second metatarsal (Fig. 25C1-52). The medial border of the second metatarsal should be parallel to the medial edge of the middle cuneiform on the anteroposterior view. The medial border of the fourth metatarsal should align with the medial border of the cuboid on the oblique radiograph. Dorsal displacement of the metatarsal bases is best assessed on the weight-bearing lateral projection.

Figure 25C1-52 A diastasis and occasionally a bone fragment between the first and second metatarsal bases suggest injury to the Lisfranc joint. **A,** Lisfranc injury as seen on routine standing radiograph. **B,** Lisfranc injury as seen on computed tomography.

Faciszewski and associates reviewed 15 cases of subtle injury of the Lisfranc joint and determined that flattening of the longitudinal arch correlated with a poor outcome, whereas persistent diastasis up to 5 mm did not correlate with a poor outcome. [250] The authors also studied 20 normal volunteers. With the use of the standing lateral radiograph, the distance between the plantar aspect of the medial cuneiform was related to the plantar aspect of the fifth metatarsal base. In all normal subjects, the medial cuneiform was higher than the fifth metatarsal base.

Stress Radiographs

In cases with strong clinical signs and normal radiographs, stress films can help elicit subtle widening. [251] The procedure can be performed in the office after injection of local anesthetic into the region or in the operating room with the patient under general anesthesia. Both pronation-abduction and supination-adduction forces are applied to the forefoot, and widening or instability of the tarsometatarsal articulation is noted.

Computed Tomography

Further imaging with CT allows for a more detailed analysis. Alignment, displacement, and subtle osseous injury are best evaluated with fine-cut CT (see Fig. 25C1-52).

Magnetic Resonance Imaging

MRI is occasionally used to identify the hemorrhage and edema associated with acute ligamentous injury or to differentiate complete and partial tears. [252]

Therapeutic Options

All acute injuries are treated with the RICE method, as described earlier. Patients are immobilized and prevented from bearing weight until a definitive diagnosis is established. Subsequent treatment is predicated on the stability of the Lisfranc complex.

Grade I Lisfranc Sprain

These injuries are immobilized in a short leg cast and kept from bearing weight for 4 to 6 weeks. On verification of stability by clinical and radiographic examination, patients are transitioned to a walking boot, and weight-bearing to tolerance is encouraged. A rehabilitation program is instituted, with an emphasis on recovery of foot and ankle range of motion, strength, and coordination. As healing progresses, the boot is discontinued, and a steel or fiber carbon shoe insert is used to reduce bending forces at the midfoot. Supportive tape or custom-molded orthotics may decrease the time before return to athletic activity. If pain persists or the patient is unable to return to competition, operative management is considered.

Grade II Lisfranc Sprain

As previously described, this group includes injuries with 2 to 5 mm of displacement. There is some disagreement about whether these injuries can be addressed through closed reduction and internal fixation instead of open treatment. Those that favor this technique use large reduction forceps and fluoroscopy to guide reduction before screw placement. Other authors advocate open treatment described next.

Grade III Lisfranc Sprain

Unstable nondisplaced injuries, as verified by radiographs and CT, are treated with open reduction and internal fixation. Weight-bearing is delayed for 8 to 12 weeks after surgery. Routine screw removal remains controversial, but generally does not occur until 12 to 16 weeks after surgery.

Meyer and colleagues reported no untoward effect associated with small, persistent diastasis between the first and second metatarsal bases after a midfoot sprain in a collegiate football player. [242]

Aitken and Poulson [244] and Brunet and Wiley [253] found that persistent subluxation or malalignment caused little functional disability. Others contend that anatomic reduction and stable fixation remain paramount. [546] [549] [550] [551] [552] [553]

Nondisplaced injury or anatomic reduction does not prevent poor outcome. Resch and Stenström completed a review of 45 consecutive tarsometatarsal injuries, with an average 5-year follow-up. [257] They noted that 3 of the 11 patients with nondisplaced tarsometatarsal injuries (<2 mm displacement on initial radiographs) reported persistent pain and poor functional outcome.

Displaced grade III injuries require open reduction and internal fixation with the use of screws or plates. K-wire fixation does not provide sufficient fixation for these injuries. The injury pattern dictates the hardware configuration. Fully threaded 4-mm screws or larger are preferred, but partially threaded screws can be used as long as they are not placed in a lag manner. Weight-bearing is delayed for 8 to 10 weeks. The hardware is removed 3 to 4 months after surgery, before the patient's return to full athletic activity.

Technique: Lisfranc Open Reduction with Internal Fixation (Fig. 25C1-53)

1. The procedure is performed with the patient under general anesthesia. The patient is supine with a well-padded bump placed beneath the ipsilateral hemipelvis.
2. The procedure can be performed with or without tourniquet.
3. A 5- to 6-cm longitudinal incision is created starting 2 cm proximal to Lisfranc's joint and carried distally over the first intermetatarsal space.

4. Blunt subcutaneous dissection is performed. The extensor retinaculum is identified and divided. The extensor tendons are carefully retracted laterally. The deep peroneal nerve and dorsalis pedis artery are mobilized and protected.
5. The capsule and the periosteum over the second metatarsal base are inspected and divided as required for exposure of the second tarsometatarsal joint. The interspace between the first and second metatarsal bases is also inspected. Hematoma, capsule, cartilage, and bone fragments are débrided. An anatomic reduction is obtained with manual pressure and held with bone reduction forceps.
6. The anatomically reduced midfoot is temporarily fixed with K-wires. Permanent fixation is carried out with 4- or 4.5-mm screws. The second metatarsal base must be anatomically and rigidly fixed to the medial and middle cuneiforms. If the first metatarsal–medial cuneiform or medial–middle cuneiform articulations are disrupted, they must be stabilized as well.
7. The periosteum and the capsule are repaired with absorbable sutures.
8. The wound is closed in layers and a short leg splint applied. At 10 days, the incision is inspected and a short leg non–weight-bearing cast applied. Alternatively, a removable fracture boot can be used, and a rehabilitation program is instituted once the soft tissues have healed. Patients are not allowed to bear weight for 6 to 8 weeks after surgery. After that, patients are allowed to bear partial weight with crutches. Over 4 to 6 weeks, the patients progress to bear full weight.
9. A comprehensive foot rehabilitation program is continued. The timing and necessity of screw removal remains controversial but generally occurs between 12 and 16 weeks after surgery. Screw retention does not prevent the athlete from training or competing. The foot is protected with a stiff-soled shoe and rigid orthosis as the athlete returns to competition.

Figure 25C1-53 A, Percutaneous Lisfranc reduction. **B,** Open reduction of a Lisfranc dislocation. **C.** Screw fixation of a Lisfranc dislocation.

Delayed-onset arthrosis after missed injuries or even appropriately treated injuries may occur. For advanced arthritic conditions, arthrodesis (fusion) of the midfoot may be required.

Rehabilitation

Ankle, hindfoot, and forefoot flexibility, motor function, and coordination[30] are emphasized throughout the protocol. The foot is supported by tape, a steel or fiber carbon shoe insert, or a custom-molded orthotic device to reduce bending forces at the midfoot. An elastic sock is available for additional mobilization of edema (see Fig. 25C1-19).

Early in rehabilitation, the athlete's pain and inflammation are addressed with rest, cold therapy, contrast bath, and whirlpool. A trial of electrical stimulation may be considered. Foot and ankle passive and active range of motion are re-established. Isometrics may be initiated as pain allows. Weight-bearing is protected for 6 to 8 weeks in grade I and II injuries. Weight-bearing after grade III injury is delayed for at least 8 weeks postoperatively.

Once the acute pain subsides, flexibility is addressed in all planes. Joint mobilization is used at the midfoot as well as in other affected joints. An inclined board is a useful adjunct to gastroc-soleus and Achilles stretching (see Fig. 25C1-20). Strengthening is initiated with towel scrunches (see Fig. 25C1-21), toe pick-up activities, manual resistive inversion and eversion, elastic bands (see Fig. 25C1-22), seated toe and ankle dorsiflexion with progression to standing, and seated supination-pronation with progression to standing.

Closed chain activities are gradually introduced, including one-leg balance, sport-specific activities on a trampoline, and use of the BAPS (see Fig. 25C1-23). Aerobic fitness is maintained with cross-training activities such as water running (see Fig. 25C1-24) and cycling.

Heat therapy, such as the application of warm packs, is a useful modality before the therapy session. It reduces pain and spasms and thus facilitates increased range of motion. Cold therapy, compression, and elevation are used after each therapy session to reduce inflammation.

After patients are able to bear full weight, walking and running activities are allowed to progress within the limits of a pain-free schedule. Once running activity is mastered, a progressively difficult monitored plyometric and cutting program is introduced. Schedules are carefully controlled to avoid reinjury.

AUTHORS' PREFERRED METHOD

I prefer to evaluate all acute-grade Lisfranc sprains with weight-bearing radiographs. If the physical examination or radiograph suggests instability (grade III sprain), a computed tomographic scan is obtained. Occasionally, MRI is obtained to assess a partial injury to the Lisfranc ligament.

Stable injuries are treated with a comprehensive rehabilitation program, with emphasis on recovery of foot and ankle range of motion, strength, and coordination. A non–weight-bearing short leg cast is used during the initial recovery period, followed by a walking boot and, finally, a carbon fiber shoe insert or a custom orthotic.

Unstable nondisplaced injuries, as verified by radiographs and CT, are treated with either closed or open reduction and screw fixation. Weight-bearing is delayed for 8 to 10 weeks.

Displaced or angulated injuries are treated surgically with open reduction and screw fixation. Weight-bearing is delayed for 8 to 10 weeks, and the hardware is removed 4 months after surgery.

Return-to-Play Criteria

Recovery follows a logical sequence of events. Once the initial pain and swelling subside, coordination and strengthening activities are emphasized. Gradually, the patient is able to return to walking, running, and cutting programs. Patients are returned to sport once they master sport-specific drills. A custom orthotic device or taping supports the midfoot during the transition back to competition.

Prolonged convalescence is the rule for the Lisfranc sprain. Persistent stiffness at the midfoot, forefoot, and hindfoot is common after surgery. Shapiro and coworkers identified nine Lisfranc injuries that occurred during athletic activity. [243] Seven players were treated with casting and non–weight-bearing for 4 to 6 weeks. Each player returned to athletic activity, but average time for return to competitive activity was 4 months.

Depending on the severity of the injury, return to previous performance level is guarded. Curtis and associates noted that 3 of 19 patients who sustained a Lisfranc injury during athletic activity were unable to return to sport. [241] Two of the patients were forced to modify their athletic activity. The average time to return to athletic activity was 4.1 months.

CRITICAL POINTS

- Lateral ankle sprains occur frequently in athletes. Remember to evaluate patients for other sources of lateral ankle pain.
- Subtalar instability can occur independently or together with lateral ankle instability.
- In patients with recurrent instability, the modified Broström reconstruction works well to address lateral ankle and subtalar instability.
- Medial ankle and syndesmotic sprains have a longer rehabilitation and return-to-play time.
- With suspected injury to Lisfranc's joint, a weight-bearing anteroposterior radiograph of both feet should be obtained.

- Prognosis for return to competition after Lisfranc injury requiring surgical management is guarded compared with other injuries.

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SECTION C Ligament Injuries: 2. Ligament Injuries of the Foot and Ankle in the Pediatric Athlete

J. Andy Sullivan

Most of the injuries that occur in the ankle and foot of the pediatric athlete are not unique to athletic participation but occur normally during childhood. Some, however, occur with greater frequency in the athlete. The conditions covered in this chapter occur in childhood and may present in the athlete, raising the question of whether the athlete should be allowed to participate in sports activities.

VARIATIONS OF NORMAL ANATOMY

Tarsal Coalition

Tarsal coalition is a bony or fibrous or cartilaginous connection of two or more of the tarsal bones. The cause is unknown, but it has been established that the condition results from failure of differentiation and segmentation of the primitive mesenchyme. [1] The overall incidence is 1% to 3%. [563] [564] [565] The most common coalitions are the calcaneonavicular and talocalcaneal types. The first is bilateral in about 60% of cases, and the second is bilateral in about 50%. [564] [565] [566] More than one type of coalition can exist in one foot. In a review of 60 cases of tarsal coalition Clarke found that 6 of 30 patients had multiple coalitions in the same foot. [5] The exact mode of inheritance is unknown, but it is postulated to be autosomal dominant with variable penetrance. [2]

Most patients seek medical care during early adolescence, at a time when the coalition is ossifying. The pain is vague in nature and insidious in onset. There may be a history of precipitating trauma. Sports participation or running over uneven ground may accentuate the pain. The pain is thought to be due to microfractures in the coalition. [6] Physical findings include pain on palpation over the subtalar joint, limited subtalar motion, and at times pes planus and ankle valgus. The peroneal muscles may be tight and resist inversion, but true muscle spasm occurs rarely (Fig. 25C2-1). Any condition that injures the subtalar joint can produce similar symptoms.

Figure 25C2-1 This patient had a tarsal coalition in the left foot. Note that the foot is held in an everted position. Attempted inversion caused pain and resistance.

The clinical diagnosis can be confirmed by radiographic imaging. Plain radiographs, especially the 45-degree oblique view (Fig. 25C2-2), usually demonstrate the calcaneonavicular coalition and other less common coalitions, such as the calcaneocuboid. The talocalcaneal coalition is difficult to visualize on plain radiographs, but secondary changes, which may suggest the need for other studies (Fig. 25C2-3), include beaking and shortening of the talar neck, a middle subtalar facet that cannot be seen, elongation of the lateral process of the calcaneus, and ball-and-socket ankle joint (see Fig. 25C2-3). Computed tomography (CT), which is now the method of choice for the diagnosis of tarsal coalitions not identified on plain films, is comparable in cost and radiation dosage to the other studies. It is technically simple to perform, noninvasive, and accurate. CT provides precise delineation of the anatomy if surgical resection is contemplated. Three-dimensional reconstruction may aid in clearly defining the coalition and planning surgery. CT may also demonstrate the presence of more than one coalition, which is important for treatment planning (see Fig. 25C2-2).

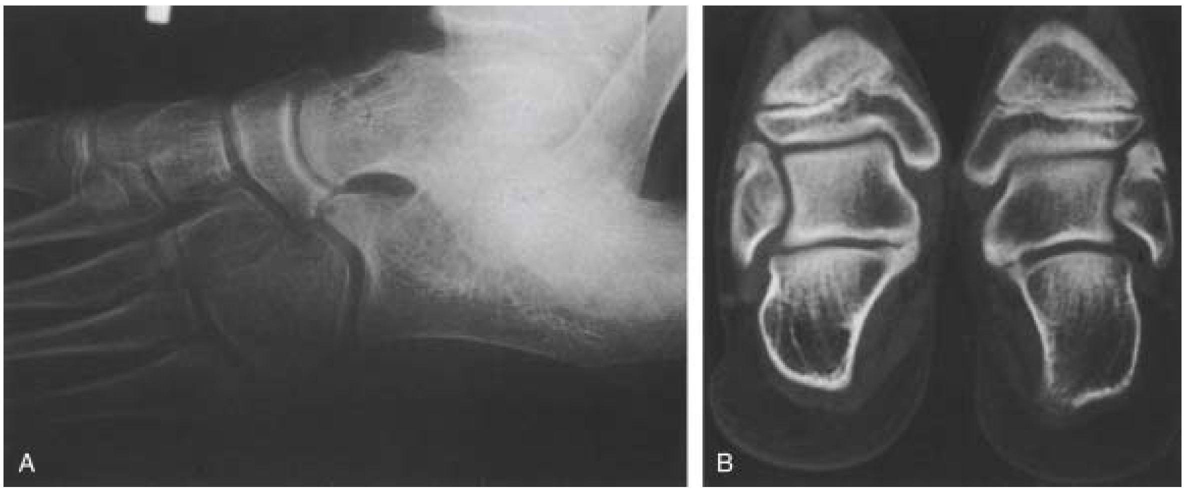

Figure 25C2-2 Plain radiography (**A**) and computed tomographic scan (**B**) showing calcaneonavicular and talocalcaneal coalitions, which occurred bilaterally in this patient. The patient presented with a painful, rigid foot and was having difficulty playing tennis.

Figure 25C2-3 Lateral radiograph of the foot. Note the beaking of the talus and the widening of the talonavicular joint. The subtalar joint is narrowed.

El Rassi and colleagues [7] reviewed 19 patients with symptoms and signs consistent with tarsal coalition and normal imaging studies. They used technetium-99m scintigraphy and found slightly increased uptake in the middle facet. They obtained good or fair results in all patients after resection of hypervascular capsule and synovium, which had produced arthrofibrosis.

Magnetic resonance imaging (MRI), which also can be used, provides the advantage of demonstrating fibrous coalitions. [8] The initial treatment of these conditions should include conservative measures aimed at relieving the pain. These measures are empirical and include casting and the use of various shoe inserts and orthotics. The main indication for surgical resection is persistent pain. For calcaneonavicular coalition, resection of the bar with interposition of the extensor digitorum brevis is usually associated with good results. Studies by Cowell [2] and by Jayakumar and Cowell [4] indicated that 23 of 26 feet treated in this manner became symptom free.

Talocalcaneal coalition (TCC) is more difficult to recognize, and its surgical management is less certain. [9] Before the advent of CT, the diagnosis was often confirmed at the time of surgery. Jayakumar and Cowell [4] reported that up to one third of their patients responded to conservative treatment, and they believed that there were few indications for resection. This conclusion was based on evidence showing that family studies indicated that many adults with tarsal coalition were asymptomatic. The surgical alternatives include resection of the bar with interposition of fat or tendon, calcaneal osteotomy, and triple arthrodesis.

Scranton [10] reviewed 14 patients with 23 symptomatic talocalcaneal coalitions. Five feet (3 patients) were treated successfully with casts. Four feet were treated with triple arthrodesis. Eight patients with 13 coalitions that had been resected had a good result. The review was done at a mean of 3.9 years after surgery. In Scranton's series, about half of the joint surface was removed in some patients. In the series of Swiontkowski and associates, [11] 10 patients were treated for TCC—four by resection of the bar, and the remainder by some type of arthrodesis. This article stressed that the talar beak is not a true degenerative sign and therefore is not a contraindication to resection of the bar. Olney and Asher [12] evaluated nine patients with persistent pain from 10 middle facet TCCs who were treated by resection of the bar and autogenous fat graft. At an average follow-up of 42 months, the results in five were rated excellent, three good, one fair, and one poor. In one patient who underwent reoperation, the fat graft was replaced by fibrous tissue.

Luhmann and Schoenecker used CT to evaluate TCC in 25 feet. They quantified heel valgus and the size of the coalition relative to the posterior facet. [13] The ratio of mean TCC cross-sectional area to the posterior facet was 53.4%. Mean hindfoot valgus was 17.8 degrees. Statistical analysis determined a significant association between TCC greater than 50% the size of the posterior facet and poor outcome ($P = .014$). Heel valgus greater than 21 degrees was also associated with poor outcome ($P = .014$). There were good results, however, in some patients with TCC greater than 50% and in those with heel valgus greater than 21 degrees. These authors advocate using the CT information for preoperative discussion with patients and families.

Comfort and Johnson reviewed resection of 20 TCCs at an average of 29 months of follow-up. [14] They found good or excellent results with resections involving less than one third of the total joint surface. They did not find increasing age to be a contradiction to the procedure.

The management of these coalitions is still controversial and awaits the results of larger series with longer follow-up. Patients with persistent symptoms who do not have degenerative findings have the option of continued conservative care, resection of the coalition, or arthrodesis. Talar beaking is not necessarily a degenerative sign. Severe malalignment of the foot is a contraindication to resection alone. A sliding osteotomy or medial closing wedge as described by Cain and Hyman [15] can be used to realign the foot. The size of the coalition that can be resected is unknown, and the question of whether interpositional material is beneficial remains unanswered.

Adolescent Bunion

The etiology of adolescent bunion is unknown. Fifty to sixty percent of patients have a positive family history. [16] Patients with this condition have an increased intermetatarsal angle (the angle between the first and second metatarsals, normally 10 degrees) and an increased first metatarsal–phalangeal angle (normally 20 degrees). Many also have a relaxed flatfoot and a long first metatarsal ray. None of these conditions is known to be the cause of adolescent bunion. Shoewear has been implicated, but because bunions occur in cultures in which shoes are not worn, this theory seems unlikely.

These patients complain of pain, prominence, and difficulty associated with shoewear. On examination, lateral deviation of the toe is found, along with a medial prominence and a wide forefoot. The bursa that is a prominent part of the adult deformity may be present but is usually less impressive. Arthritis and decreased range of motion are also less common. The patient should be evaluated with anteroposterior and lateral weight-bearing radiographs. The joint space is usually maintained. The sesamoids may be laterally displaced in advanced cases. The medial eminence of the metatarsal head is prominent, and a sagittal groove may be present medially.

Children should be treated nonsurgically whenever possible. Alteration or stretching of shoewear may alleviate symptoms. Although some series have claimed a success rate of 80% to 95%, this high rate of success has not been the universal experience. Factors implicated in these complications included failure to correct the abnormal deviation of the first metatarsal, failure to correct the soft tissues, too early weight-bearing, inadequate immobilization, and osteotomy performed distal to the open physis. Patients with a hypermobile flatfoot or a long first ray also seemed more prone to recurrence.

Indications for surgery include pain that is not responsive to conservative measures and severe deformity. The goals of surgery should include realignment of the first ray and of the metatarsophalangeal joint, cosmesis, and prevention of arthritis. Discussion of the types of surgical procedures used is beyond the scope of this chapter. In general, the most common procedures are distal soft tissue realignment, a distal osteotomy such as Mitchell's or chevron, or a proximal realignment osteotomy combined with soft tissue procedures. Arthrodesis and resection arthroplasty have no place in surgery for neuromuscularly normal children.

Accessory Navicular

Numerous accessory ossicles can occur in the foot, and one needs to be aware of these to avoid confusion with acute fracture. The most common are the os trigonum posterior to the talus and the os vesalianum at the base of the fifth metatarsal. The accessory navicular is a separate ossification center of the navicular. It may be completely separate or joined by a synchondrosis. It may also present as a large or cornuate prominence on the medial side of the navicular (Fig. 25C2-4). We now know that only a small slip of the tendon inserts into this ossicle and that these patients are no more likely to have flatfoot than are those with a normal navicular. [17]

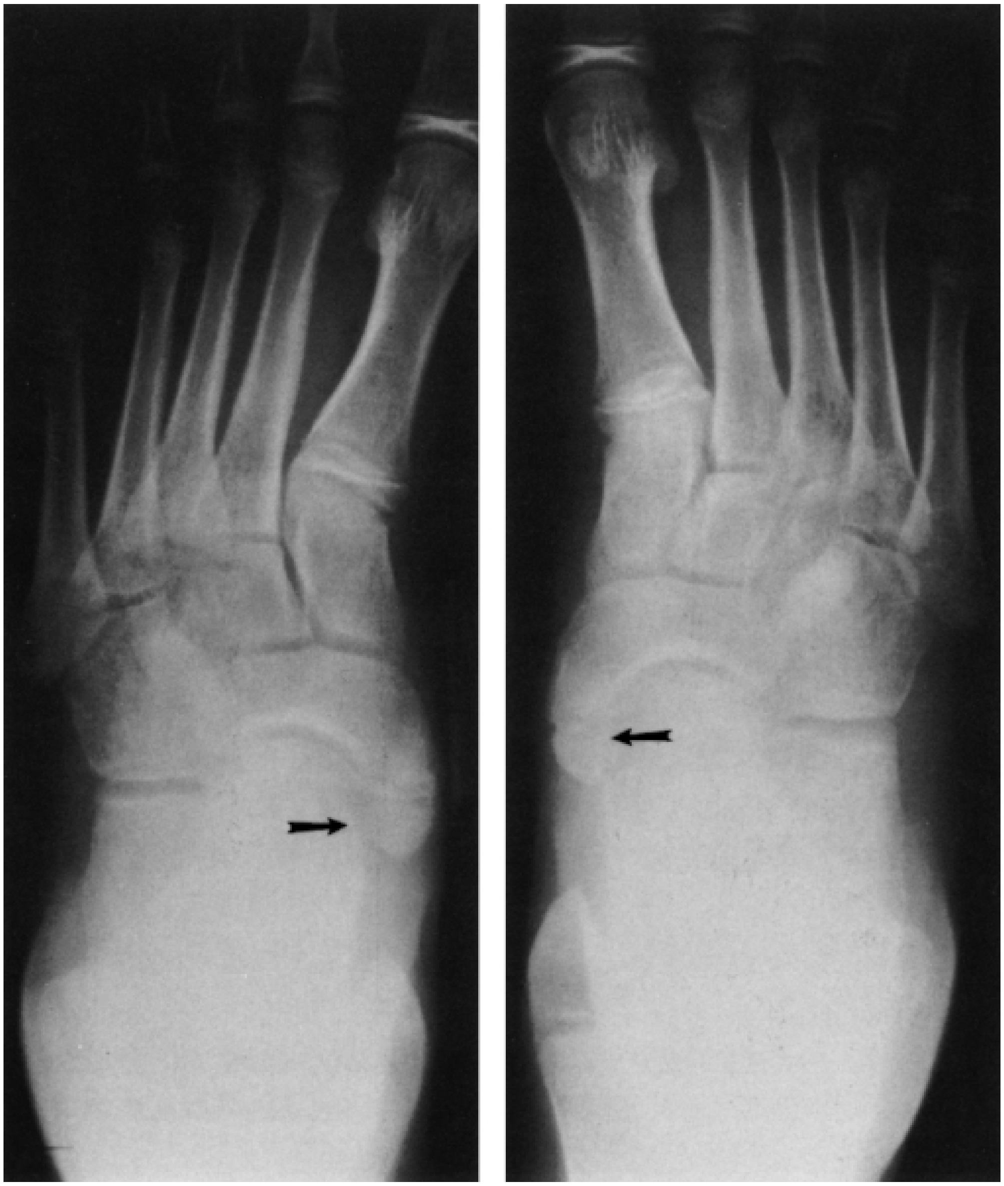

Figure 25C2-4 Large bilateral cornua of prominent accessory naviculars. These are joined by a synchondrosis to the navicular.

Many of these patients are asymptomatic. Symptomatic patients experience pain directly over the prominence, usually from shoewear over the prominence. If the shoes are stretched or altered over this prominence, symptoms may be relieved. Other patients experience pain when the posterior tibial tendon is stretched or placed under tension. In those with persistent pain, simple excision of the ossicle without rerouting of the tendon is usually successful. [579] [580]

Cavus Foot

Cavus is defined as an increase in the height of the longitudinal arch of the foot. A variety of other modifiers, such as cavovarus and calcaneocavus, are used to further describe the position of the heel. Often, the patient has claw toes or hammer toes and metatarsal head calluses. The presenting complaint can be pain or abnormal wear of the shoe. A cavus foot is usually the result of muscle imbalance that is caused by an underlying neurologic disorder. The patient should undergo a meticulous neurologic examination so that evidence of disorders such as Charcot-Marie-Tooth disease, spinal dysraphism, or a spinal tumor can be detected. Initial radiographic evaluation should include weight-bearing views of the feet and at least an anteroposterior view of the entire spine in search of an occult spinal anomaly.

Miscellaneous Foot Abnormalities

A variety of other bony variations may produce pain. “Pump bumps,” which are soft tissue bursae associated with prominent calcaneal tuberosities, are irritated by the heel counters of some shoes. Treatment consists of altering the shoewear or padding the heel. Bunionettes are similar bursae that occur over the lateral aspect of the fifth metatarsal head. Stretching the shoe is usually curative. Only rarely is removal of the bony prominence or osteotomy of the fifth ray necessary. A dorsal bunion can occur over the first metatarsal head. In most instances, it is a result of weakness or loss of function in the peroneus longus, which is responsible for depressing the first metatarsal. Treatment must be individualized to balance the overpull of the dorsiflexors of the first ray and may require tendon transfer, osteotomy, or arthrodesis.

SOFT TISSUE INJURIES

The ligaments of the ankle insert on the epiphyses distal to the physeal line (Fig. 25C2-5). Because the physis is the weakest link in this bone-tendon-bone interface, it is usually the part that gives way when significant force is applied to the ankle. Serious ankle sprains are unusual in the skeletally immature athlete. Physeal fractures that do occur are discussed in the next section. Minor ankle sprains do occur and are diagnosed by a history of inversion or eversion strain with findings of tenderness over the anterior talofibular or deltoid ligament. Treatment is accomplished by the usual conservative means of rest, ice, compression, elevation, and immobilization. Formal rehabilitation is rarely necessary but may be beneficial for the competitive athlete. Continued pain or disability should provoke a search for other, more serious injury.

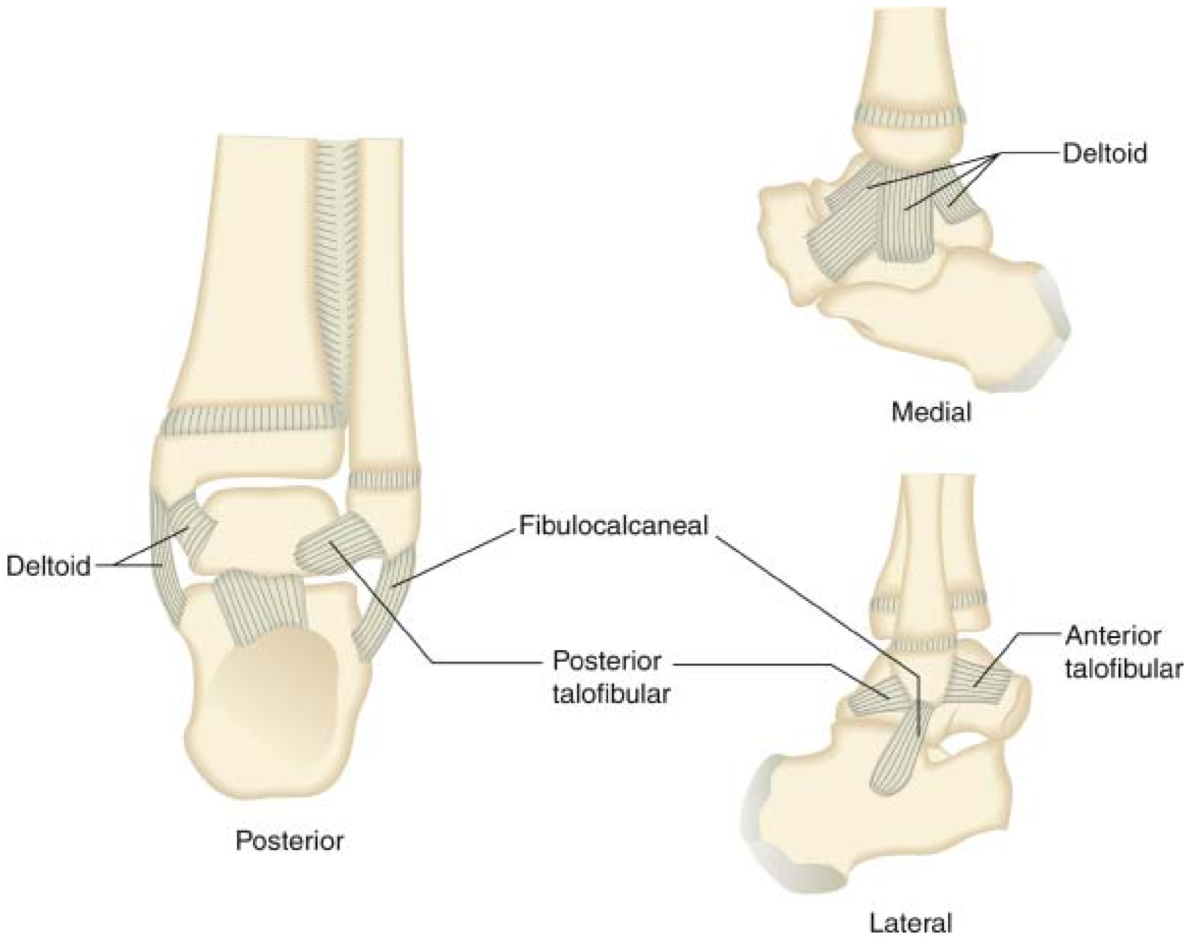

Figure 25C2-5 The ligaments of the ankle in posterior, medial, and lateral views.

Recurrent subluxation of peroneal tendons can occur in the adolescent athlete. Usually, a history of injury is followed by recurrent episodes of a snapping sensation and pain. The subluxation can be provoked by forceful dorsiflexion with the foot everted. In patients whose symptoms are sufficiently severe, surgical correction may be indicated. Surgical alternatives include deepening the groove on the fibula, creating a bony block, and reconstructing the superior peroneal retinaculum. The first two are rarely useful in treatment of the pediatric athlete because the physis is still open. Poll and Duijfjes reviewed nine patients aged 15 to 45 years (average age, 25 years) who underwent reconstruction with the posterior calcaneofibular ligament attached to a bone block. [19] Results were said to be good.

Contusions on the foot are treated in the same way as those on any other area. Blisters are a frequent problem and require alleviation of the stress, which is usually provided by a new shoe, and protection until healing occurs. Tinea pedis (athlete's foot) usually responds to a regimen of antifungal medication, along with education about the need to change socks frequently and to use antifungal powders.

FRACTURES AROUND THE ANKLE

Multiple systems have been proposed to classify ankle injuries. All these classification systems take into account the position of the foot at the time of injury and the force applied. The Salter-Harris classification[20] is based on the mechanism of injury and the pathoanatomy of the fracture pattern through the physis, as is interpreted on plain radiographs. The authors described types I through V. Type V, a crush injury to the physis, is difficult to recognize on plain radiographs and is not discussed in this chapter (Fig. 25C2-6).

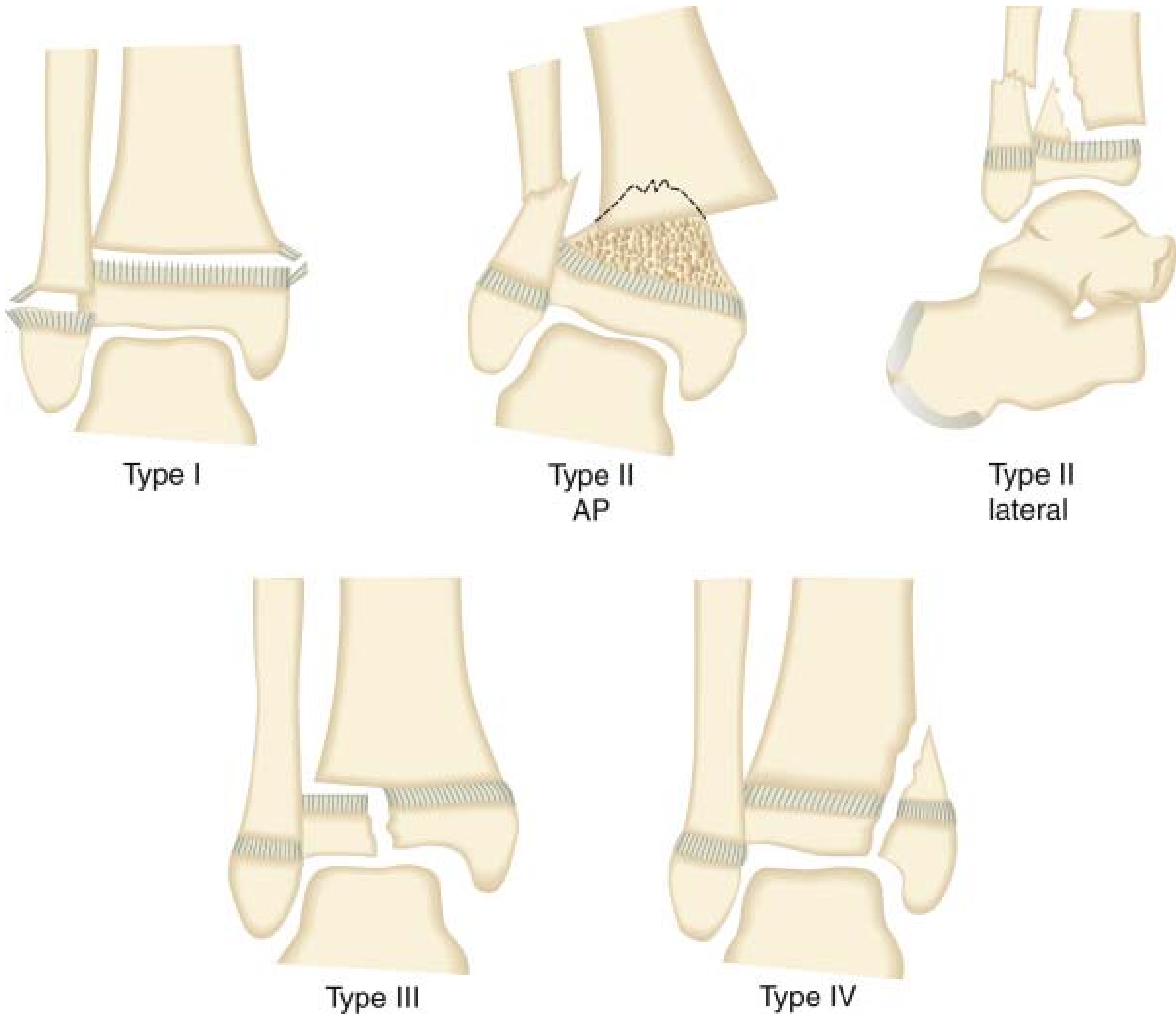

Figure 25C2-6 Fracture patterns of the distal tibial and fibular physes classified by the Salter-Harris system.

In the skeletally immature patient, the tibiotalar joint surface is rarely disturbed. The injury pattern changes in adolescence as the physis begins to close. The outcome of the injury depends on the type of physeal injury and its management. Tension injuries usually produce Salter-Harris I and II injuries of the physis. Compression forces can produce Salter-Harris III and IV injuries.

CT and MRI are valuable techniques in assessing and classifying some fractures because they allow more accurate evaluation of the fragments. Carey and associates performed a study of plain films and MRI in 14 patients with acute injuries and reached the following conclusions.[21] Direct visualization of cartilage afforded by MRI improved evaluation of growth plate injury in each case. MRI changed Salter-Harris classification or staging in 2 of 9 patients with fractures visualized on conventional radiographs, allowed the detection of radiographically occult fractures in 5 of 14 cases, and resulted in a physical change in management for 5 of the 14 patients studied. MRI has an important role in the evaluation of acute pediatric growth plate injury, particularly when diagnostic uncertainty persists after the evaluation of conventional radiographs. MRI allows detection of occult fractures, may alter Salter-Harris staging, and may lead to a change in patient management.

Rohmiller and colleagues have recently used the Lauge-Hansen system to look at the mechanism of injury in Salter-Harris II distal tibial fractures. [22] This is discussed later.

Clinical Evaluation

The mechanism of injury and the time elapsed since the accident should be noted. The neurovascular status of the foot should be carefully documented. The amount of swelling and the status of the skin are important. Gentle examination should be carried out to seek areas of point tenderness, especially over the physis. This examination may be more useful than radiographs in the diagnosis of Salter-Harris I injuries of the fibula.

Radiographs should always include three views of the ankle. Only in this manner will the physician be able to see some fractures and determine whether there is disruption of the ankle mortise. Plain tomography and CT may be indicated in the juvenile fracture of Tillaux and in triplane fractures, which are discussed later. Because treatment and prognosis depend on the Salter-Harris classification, the fractures are discussed according to fracture type.

Salter-Harris Type I

Rohmiller and colleagues combined Salter-Harris I and II fractures of the distal tibia and found an incidence of premature physeal closure (PPC) of 38%. [22] No distinction was made between type I and II. Salter-Harris I fractures of the fibula are common and may be missed entirely or misdiagnosed as a sprain. The characteristic history of an external rotation force in a patient who presents with localized tenderness and swelling directly over the distal fibular physis is diagnostic. The radiograph shows only localized swelling and widening of the fibular physis. Many of these injuries probably go unrecognized and untreated.

Salter-Harris Type II

This injury is uncommon or is infrequently recognized in the fibula. Salter-Harris II injury of the distal tibia combined with a fracture of the distal fibula is one of the most common injuries of the ankle, accounting for 47.3% of cases in the series compiled by Peterson and Cass. [23]

Rohmiller and colleagues studied 91 Salter I and II fractures. [22] Treatment options include no reduction and cast, closed reduction and cast, closed reduction and percutaneous pins and cast, and open reduction with internal fixation. They found a 39.6% incidence of PPC. Using the Lauge-Hansen classification system, they found a significant increased incidence of PPC in pronation-abduction injuries (54%) compared with supination–external rotation injuries (35%). The most important determinant of PPC was the amount of fracture displacement following reduction. In some cases, periosteum is trapped in the fracture site medially, blocking reduction. They thought that operative treatment may decrease the incidence of PPC. They recommended less than 2 mm of displacement in a child with 2 years of growth remaining to decrease the risk for PPC.

Traditional treatment consists of a long leg bent-knee cast for 2 to 3 weeks followed by a short leg walking cast for 4 weeks. Dugan and coworkers reviewed 56 patients with this injury who were treated with a long leg weight-bearing cast for 4 weeks. [24] There were no nonunions and no angular deformities. There was one case of clinically insignificant premature closure of the growth plate. This appears to be the treatment of choice because it allows early healing, low morbidity, and rapid rehabilitation.

Salter-Harris Type III

In adolescents, this injury is also known as the *juvenile fracture of Tillaux*. The distal tibial physis closes first in the central region, and then from the medial side toward the fibula. An external rotation force applied to the partially closed physis applies traction on the physis through the anterior talofibular ligament. This avulses a fragment of the lateral physis, which remains attached to the ligament (Fig. 25C2-7). Closed reduction under anesthesia should be attempted. The injury can be treated closed if the fragment is not displaced more than 2 mm, or if it can be reduced closed and percutaneously fixed. Most of these injuries require open reduction and fixation of the fragment with a pin or cancellous screw. Fractures of the medial malleolus can be either type III or IV injuries. If displaced less than 2 mm, they may be treated closed. This treatment should consist initially of a long leg non–weight-bearing cast for 3 weeks followed by a short leg walking cast for 3 weeks. These injuries are the most unpredictable of ankle epiphyseal injuries. Near-anatomic reduction must be obtained. [25]

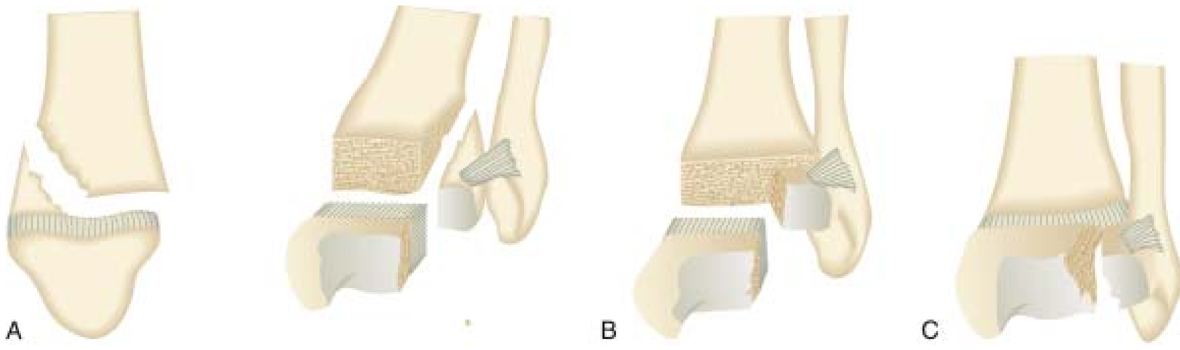

Figure 25C2-7 **A** and **B,** The triplane fracture can consist of two or more fragments. **C,** The juvenile fracture of Tillaux.

Salter-Harris Type IV

This group includes some of the medial malleolar fractures and the triplane fractures. The triplane fracture, first described by Marmor, is so named because the fracture lines extend from the physis into the transverse, sagittal, and coronal planes (see Fig. 25C2-7). [588] [589] [590] [591] This type of fracture may be mistaken for a Salter-Harris II injury if the radiographs are not carefully scrutinized.

Many authors have described this fracture and have argued about the number of fragments involved. [588] [590] [591] Most of these studies were based on plain radiography. Figure 25C2-7 illustrates the possibilities. In the two-part fracture, the main fragment is the tibial shaft, including the medial malleolus and a portion of the medial epiphysis. The second fragment is the remaining epiphysis, which is attached to the fibula. In the three-part injury, the third fragment is usually an anterior free epiphyseal fragment. Brown and colleagues studied 51 children with tibial triplane fractures. [30] By evaluating them with CT with multiplanar reconstructions, these authors have used the best radiographic evaluation possible to define the number of fragments. The classic two-fragment type with medial epiphyseal extension was most frequent (33 of 51). All three-fragment types (8 of 51) had a separate anterolateral fragment. Extension to the medial malleolus was common (12 of 51). None of the four reported fracture types involving anteromedial extension was seen.

Karrholm reviewed the literature on this injury. [31] Triplane fracture made up 7% of physeal injuries in girls and 15% in boys. Of the injuries, 35% were treated closed without manipulation, 30% by manipulation and casting, and 35% by open reduction and internal fixation.

If this injury can be reduced to within 2 mm, it may be treated closed. In the series of Cooperman and associates, 13 of 15 fractures were treated closed, [26] and in the series of Dias and Giergerich, 5 of 8 were treated in this way. [27] In the series by Ertl and colleagues, residual displacement of more than 2 mm was associated with a high incidence of late symptoms. [28] Obtaining a reduction of less than 2 mm by either closed or open means did not ensure an excellent result. Poor results may be related to damage done to the articular surface, or to the amount of displacement. Fractures outside the weight-bearing area did not show this tendency toward poor results.

Evaluation of the adequacy of reduction in this injury is difficult, and because most authors recommend manipulation under general anesthesia, the only radiographic means of diagnosis available is plain radiography. The author's preferred method is manipulation by internal rotation of the foot under sedation, which usually occurs in the emergency room. If there is any question about the adequacy of reduction on plain radiographs, CT or plain tomography is used to evaluate the articular surface and the reduction (Fig. 25C2-8); if displacement is greater than 2 mm, open reduction with internal fixation is carried out. This may require two incisions. The first is an anterolateral incision, which allows identification of the anterolateral fragment. Usually, it is first necessary to reduce and fix the posterior fragment. If this cannot be done closed, a second posteromedial approach is used to reduce the fragment under direct vision. Fixation is by cannulated screws, cancellous screws, or pins. These injuries require 6 weeks in a cast.

Figure 25C2-8 **A** and **B,** The position attained after manipulation of a severely displaced triplane fracture. The position was not acceptable, so further imaging was not necessary. **C,** The position achieved by open reduction. **D,** The position that is retained after hardware removal.

Prediction of Outcome

The prognosis for an ankle fracture in a skeletally immature patient depends on the following factors:

1. Mechanism of injury
2. Salter-Harris classification
3. Quality of reduction
4. State of skeletal maturity
5. Amount of displacement
6. Miscellaneous modifiers (open fracture, vascular injury, infection, systemic illness, interposed periosteum)

Spiegel and colleagues retrospectively studied a series of closed distal tibial physeal injuries. [29] One hundred eighty-four patients (of 237) were followed for an average of 28 months. The authors looked specifically at the complications of angular deformity of greater than 5 degrees and shortening of more than 1 cm, joint incongruity, or asymmetric closure of the physis. These complications appeared to correlate with the Salter-Harris type, the amount of displacement or comminution, and the adequacy of reduction. The patients were divided into the groups shown in Table 25C2-1 .

TABLE 25C2-1 -- Complications of Ankle Fractures

Group	Complication Rate (%)	Salter-Harris Group and Bone Involved
Low risk (89 patients)	6.7	Type I and II of fibula, I of tibia, III and IV with displacement of less than 2 mm, epiphyseal avulsion injuries
High risk (28 patients)	32	Types III, IV, and V of tibia Tillaux and triplane
Unpredictable (66 patients)	16.7	Type II of the tibia

The overall complication rate was 14.1% for 184 patients. Salter-Harris II injuries of the tibia appeared to be the least predictable because the incidence of complications remained about the same, regardless of the amount of displacement. Displacement is not always mentioned as one of the factors involved in prediction of outcome, but it is intuitive that greater displacement implies greater force, with more likely damage to the articular cartilage, the circulation, and the soft tissues important in healing. Karrholm thought the good results were based on the adequacy of the reduction. [31]

Near-anatomic reduction of type II injuries of the tibia is desirable. Gruber and associates have show in an animal model that interposed periosteum in an intact physis produces a spectrum of changes at the tissue level and a small but statistically significant leg-length discrepancy compared with fracture alone. [32] Because this is a spectrum, it may explain the unpredictable nature of these injuries and support the need to remove interposed periosteum (Fig. 25C2-9). Residual displacement after attempted closed reduction may be secondary to this interposition.

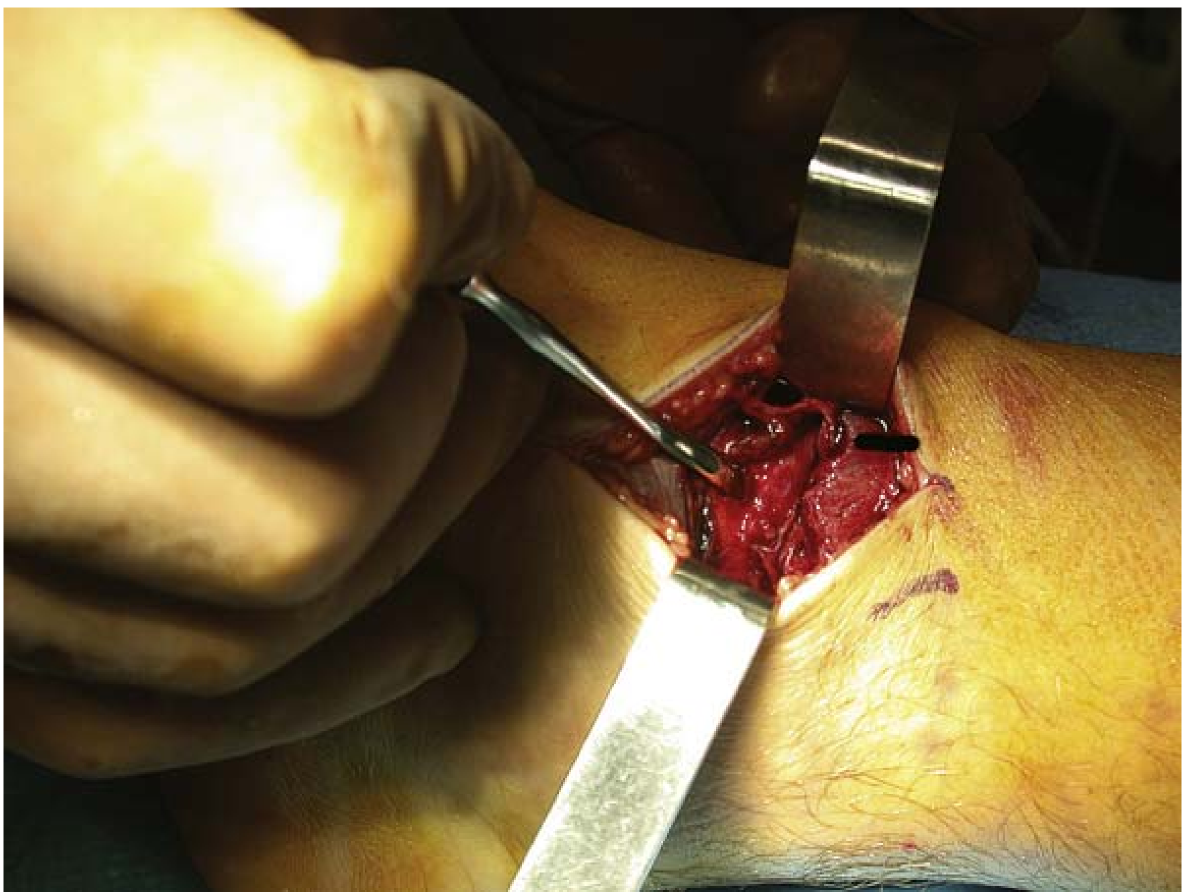

Figure 25C2-9 The Freer elevator is on the metaphyseal periosteum that is inverted into the physeal fracture site. The *black mark* is on the epiphyseal fragment.

The patient in Figure 25C2-10 A was treated by closed manipulation and casting. He had residual medial displacement (see Fig. 25C2-10 B and C). Follow-up radiographs show the development of a defect in the medial cortex. He subsequently developed a valgus ankle deformity (see Fig. 25C2-10 D), which would indicate the medial physis and suspected interposed periosteum grew more than the lateral side. He required a varus tibial osteotomy and physeal closure. Although I have seen patients who developed normal growth despite the medial cortical defect, the unpredictability and high frequency in the Rohmiller article support a near-anatomic reduction with less than 2 mm of displacement in a child with more than 2 years of growth remaining.[22] These injuries must be followed until the patient attains skeletal maturity or a normal growth pattern is ensured because some will go on to premature closure and angular deformity.[25]

Figure 25C2-10 **A,** Anteroposterior (AP) radiograph on the day of injury of this Salter-Harris II tibial fracture. **B,** Postreduction AP radiograph. **C,** AP radiograph at 4 months. **D,** AP radiograph at 2 years.

The juvenile fracture of Tillaux and the triplane fracture result from incomplete closure of the physis. Because growth is nearing an end, angular deformity and shortening are uncommon. In these patients, the tibiotalar joint surface is disturbed and must be restored to as near normal as possible to prevent incongruity and subsequent traumatic arthritis. In the series by Cooperman and colleagues,[26] triplane fractures were reduced with the patient under general anesthesia by internally rotating the foot. The adequacy of reduction was determined by plain tomography. Dias and Giergerich had nine Tillaux and triplane fractures that were followed for an average of 18 months, and all did well.[27]

Peterson and Cass reviewed all Salter-Harris IV distal tibial injuries seen at the Mayo Clinic, paying particular attention to injuries of the medial malleolus.[23] Nine of 18 of these injuries went on to premature physeal closure sufficient to require additional surgery for physeal bar resection, angular deformity, or leg-length discrepancy. Thirteen of these patients received their care at the Mayo Clinic, and of these, 11 were closed injuries. Six patients were treated by closed reduction and a short leg cast. Five had open reduction and internal fixation. Five additional patients in the study had been referred to the clinic because of complications of a closed injury that had been treated closed. They concluded that oblique radiographs are necessary to ensure an accurate diagnosis and to confirm the adequacy of reduction. Some injuries that resemble type III injuries are actually type IV. The authors also found that partial arrest that results in angular deformity was more common than complete arrest. They concluded that there are three patterns of medial malleolar injury and that type IV injuries constitute the most common and most dangerous pattern because they usually occur in a patient who has remaining growth potential (Fig. 25C2-11). They also concluded that the medial malleolus requires anatomic reduction, which often necessitates open reduction and internal fixation.

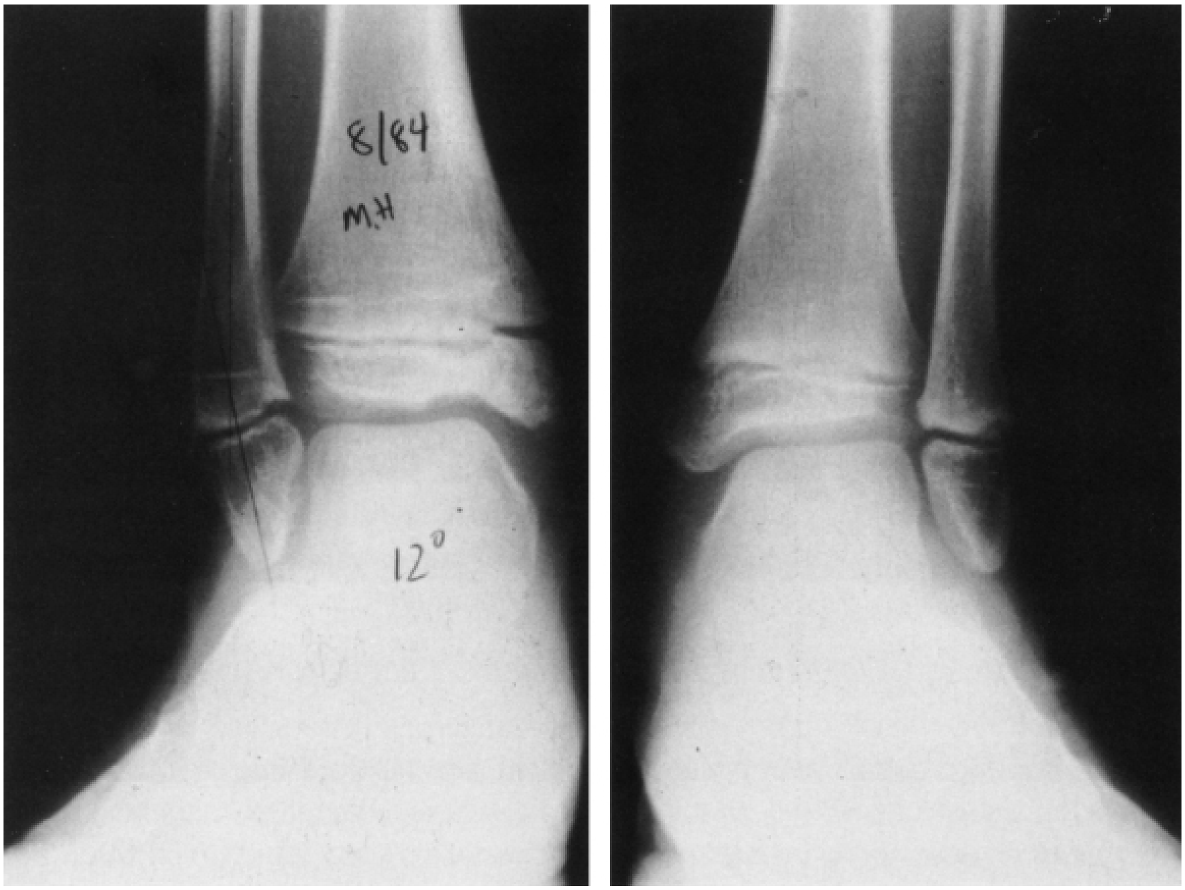

Figure 25C2-11 This patient sustained a Salter-Harris IV medial malleolar fracture, which was treated closed. The patient was referred 6 months after injury, at which time she had trouble remembering which ankle had been injured. These radiographs were taken 18 months after the injury. Resection of a bony bridge and interposition were required. She resumed growth, and the fibular angular deformity had been corrected. Note the irregular Harris growth arrest lines.

In any patient with an open physis, it is preferable to avoid crossing the physis with a fixation device. This goal can usually be achieved by placing smooth pins from metaphysis to metaphysis, or from epiphysis to epiphysis. At times, crossing the physis cannot be avoided. Smooth pins can be used, and care should be taken that they do not cross within the physis. Patients need to be followed to skeletal maturity, or until one is certain that a normal growth pattern is occurring. An asymmetric Harris growth arrest line may be the earliest clue to an abnormal growth pattern (see Fig. 25C2-11).

FRACTURES IN THE FOOT

Fractures of the foot resulting from sports are unusual in children. Fractures of the metatarsals can result from direct trauma (Fig. 25C2-12). These can be treated by immobilization in a short leg walking cast. The most controversial fracture in the foot may be an avulsion injury at the base of the fifth metatarsal.

Figure 25C2-12 A, This patient sustained fractures of the lateral four metatarsal necks when he caught his foot on a base while sliding. **B,** This patient fractured his first metatarsal when he was stepped on during a football game.

Fractures of the fifth metatarsal in children can be divided into distal physeal fractures, fractures of the proximal diaphysis, and avulsion fractures of the apophysis. The fifth metatarsal has its epiphysis distally and an apophysis proximally. The tendon of the peroneus brevis is inserted into the apophysis. With inversion stress, the apophysis can be avulsed. Findings include tenderness at the base of the fifth metatarsal and radiographic confirmation of widening of the apophysis. Treatment should be symptomatic with compression and partial weight-bearing until the pain subsides. Crutches and an elastic bandage may be sufficient. Two to three weeks in a short leg cast also yields good results.

Fracture of the proximal diaphysis of the fifth metatarsal (Jones' fracture) is less common in skeletally immature patients and usually occurs in the 15- to 20-year age range. [33] When such fractures do occur, a trial of immobilization in a short leg walking cast is indicated because many acute fractures do heal. Even fractures with delayed union may heal if they are treated conservatively. [34] Early operative intervention in highly competitive athletes has been advocated by some, but others have shown that each patient needs to be treated individually because some of these fractures will heal if treated conservatively, allowing early return to athletics. [595] [596] [597] Early operative intervention in the pediatric athlete is rarely if ever indicated. Patients with established nonunion require operative treatment that includes reopening of the medullary canal, bone grafting, and internal fixation (Fig. 25C2-13).

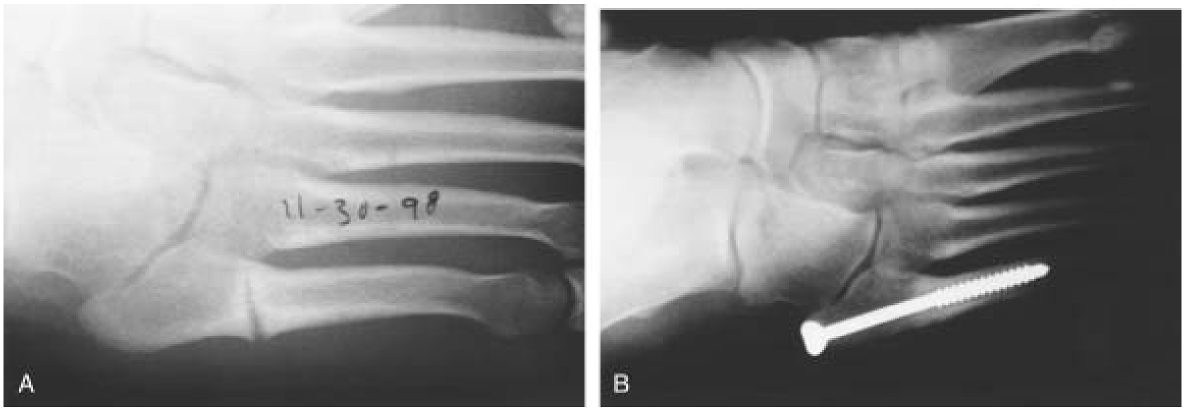

Figure 25C2-13 **A,** This patient sustained a fracture of the diaphysis of the fifth metatarsal. This is the radiograph 2 months after treatment with a cast. The metatarsal was tender to palpation, and the patient walked with a limp. **B,** This radiograph was taken after treatment with internal fixation and local bone graft from the calcaneus.

Fractures of the toes are unusual in sports. Most phalangeal fractures can be treated by buddy taping them to the adjacent toe, wearing appropriate shoes for a few weeks, and avoiding sports until the toe is asymptomatic. Articular fractures are even rarer. The only one that may merit consideration of operative management is an intra-articular physeal fracture of the great toe. These should be reduced to as near-anatomic alignment as possible by whatever means necessary (Fig. 25C2-14).

Figure 25C2-14 This intra-articular fracture was treated by closed reduction and percutaneous pinning.

Stress fractures are less common among children than in adults but cannot be entirely dismissed. Some children participate in marathons and other sporting events that can result in stress fractures. Basketball, soccer, and other team sports have tournaments that may require considerable running. The stress fracture shown in Figure 25C2-15 resulted from a tournament and was thought to be a sprain.

Figure 25C2-15 This patient presented with localized tenderness just above the ankle. After being injured in a basketball tournament, she continued to play with a presumptive diagnosis of a sprained ankle. The initial radiographs (**A** and **B**) showed periosteal elevation. Follow-up radiographs (**C** and **D**) taken after 2 weeks of short leg walking cast treatment illustrate new bone formation.

Yngve found 131 pediatric stress fractures in 23 references in the literature. [36] Two reports of the 131 documented metatarsal fractures—two of the tarsal navicular, and one of the medial sesamoid. The primary training error was too much too soon. Other factors that should be considered are a change in training surface or equipment (shoes) and a sudden change in intensity of training (tournaments). Diagnosis depends on an appropriate history, a high index of suspicion, and the presence of localized tenderness. The differential diagnosis includes contusion, tendinitis, and sprains.

The initial radiograph may be normal but should be diagnostic in half of cases. One should look for cortical thickening or a translucent fracture line. A bone scan may be diagnostic at this stage and may be particularly helpful if the diagnosis is in question and one wishes to avoid immobilization. A bone scan may be indicated when the diagnosis is in doubt and the athlete wishes to return to play. MRI has also been used to identify occult stress fractures. Conversely, immobilization for 2 weeks in a cast is usually diagnostic in that pain is relieved; repeat radiographs are then positive, making a bone scan unnecessary. Although some of these fractures heal without a cast, the athlete should be immobilized for protection from himself or herself as well as from well-meaning parents and coaches.

Osteochondral Lesions of the Talus

The term *osteochondritis dissecans* (OCD) has been used to describe lesions in the dome of the talus. These lesions have been attributed to a vascular insult, but trauma has been implicated, especially in lateral lesions. [599] [600] [601] Berndt and Harty developed a classification system based on the amount of damage and the degree of displacement involved. [37] This system, modified slightly, is still in use.

Stage I: Localized trabecular compression

Stage II: Incompletely separated fragment

Stage IIA: Formation of a subchondral cyst

Stage III: Undetached, undisplaced fragment

Stage IV: Displaced or inverted fragment

Most series consist predominantly of adults; however, 21 of 29 patients studied by Canale and Belding experienced onset of symptoms during the second decade. [38] CT and MRI can reveal lesions not seen on plain films and further make staging more accurate. These techniques may reveal that this disorder is more common among adolescents than was previously suspected. Proper treatment depends on identification of the lesion and accurate staging.

Canale and Belding recommended that nonoperative treatment by immobilization of all stage I, stage II, and medial stage III (Berndt and Harty classification) lesions would result in a high percentage of good clinical results and delayed development of arthrosis. [38] Persistent symptoms after conservative treatment were an indication for operative treatment by excision and curettage. They further recommended that all stage III lateral lesions and all stage IV lesions be treated by immediate excision and curettage of the lesion. Anderson and associates recommended immobilization for 6 weeks for patients with stage I and II fractures, but they cautioned that these patients need to be followed for a prolonged time so that delayed development of arthrosis can be detected. [38a] Operative treatment was recommended for stage IIA, III, and IV lesions.

Letts and colleagues reviewed 24 children treated since 1983 for OCD of either the medial or lateral dome of the talus (2 were bilateral). [39] Average age at presentation was 13 years, 4 months. Nonoperative treatment included activity restriction, physiotherapy, and immobilization. Surgical intervention was required in 15 (58%) ankles. Surgical treatment included arthroscopy (1), arthrotomy and drilling of the defect (9), drilling with excision of the lesion (3), excision of the lesion (2), and pinning of the fragment (1). Most recent follow-up revealed resolution or decreased symptoms in 25 (96%) and no change in 1. MRI was useful in preoperative assessment in 6 cases. In this series, there was a slight female preponderance (58%). Higuera and coworkers reported good to excellent results in 94.8% of children. [39a]

Osteochondroses in the Foot

The osteochondroses are a group of conditions of unknown origin. Suggested causes have included endocrinopathies, vascular phenomena, infection, and trauma. [40] Many of these conditions are now known to represent radiographic variations of normal ossification of the epiphysis. Most are named for the person or persons who originally described them. They all include a pattern of clinical symptoms coupled with a radiograph that suggests that the epiphysis or apophysis is undergoing necrosis. In the foot, the most commonly described conditions are Kohler's syndrome and Freiberg's infarction.

Kohler's syndrome is a clinical syndrome consisting of pain in the midfoot coupled with a finding of localized tenderness over the navicular. Radiographs demonstrate increased density and narrowing of the tarsal navicular. Irregular ossification in this bone may be the rule rather than the exception, so the existence of this condition is in question (Fig. 25C2-16). Williams and Cowell reviewed a series of patients with the following findings. [41] Thirty percent of males and 20% of females demonstrated irregular ossification in the tarsal navicular. Most patients appeared to respond to 6 weeks of immobilization in a cast. All 23 patients eventually became asymptomatic, and the navicular became normal. The authors believed that patients treated in a cast became asymptomatic sooner than did those treated with shoe inserts. Regardless of treatment, no long-term problems were associated with this condition, again raising the question of whether it is a distinct pathologic condition.

Figure 25C2-16 This patient presented with undisplaced fractures of the metatarsals. The condensed, narrowed appearance of the navicular is the same as that seen in patients with Kohler's syndrome, but it was an incidental finding in this patient.

Freiberg's infarction is a condition of condensation and collapse of the metatarsal head and articular surface. It commonly occurs during the second decade of life while the epiphysis is still present. [42] It is of unknown cause and is more common among females. Many causes have been proposed, but repetitive trauma probably plays a role. The lesion occurs most commonly in the second or third metatarsal (Fig. 25C2-17). [40] These are the longest and least mobile of the metatarsals. The patient presents with pain on weight-bearing and has localized tenderness over the metatarsal head. Radiographs reveal collapse of the articular surface (see Fig. 25C2-17). Conservative treatment with a cast or orthotic device that minimizes weight-bearing over the involved head is often successful in relieving the pain. Surgical treatment consisting of removal of loose bodies or bone grafting has been reported for persistent symptoms. A dorsiflexion osteotomy to relieve weight-bearing has also been reported to work well. Removal of the metatarsal head should be avoided because this results in transfer of weight-bearing to the adjacent metatarsal heads. Prosthetic replacement has also been tried but is not indicated in children. In most instances, the disease runs its course, and the head reossifies within 2 to 3 years.

Figure 25C2-17 This anteroposterior radiograph of the foot shows irregularity and collapse of the second metatarsal head.

Sever's disease is a term used to refer to a nonarticular osteochondrosis or a traction apophysitis (Fig. 25C2-18). The real question is whether a distinct syndrome exists and, if it does, whether the apophysis has anything to do with it. The calcaneal apophysis appears and develops in the 5- to 12-year age range and is typically irregular. Often, a child with heel pain and an irregular apophysis has the same radiographic finding in the opposite asymptomatic heel. These children are usually in the 9- to 12-year age range and are active in sports. They may have a tight heel cord. The calcaneus serves as the insertion of the powerful gastrocnemius-soleus muscle and the origin of the plantar fascia. Traction or overuse can strain these structures, producing pain. Stretching may be beneficial. Symptomatic treatment by avoidance of the offending exercise is usually curative. Shock-absorbing inserts or a heel cup may be advantageous. A heel lift to relieve some of the pull of the gastrocnemius-soleus, or at times an arch support for a child with a high arch, may give symptomatic relief as well. Heel cord stretching exercises may be tried. One must carefully search for the point of maximal tenderness and seek its cause rather than implicating an irregular apophysis, which is probably not the source of the pain. The exact time frame for resolution of symptoms in children with heel pain is unknown and at times can be vexing. If I feel that the child has followed the previous conservative measures and is no better after 2 to 3 months, I proceed with further work-up, such as a bone scan and other studies, to seek more occult sources of the pain.

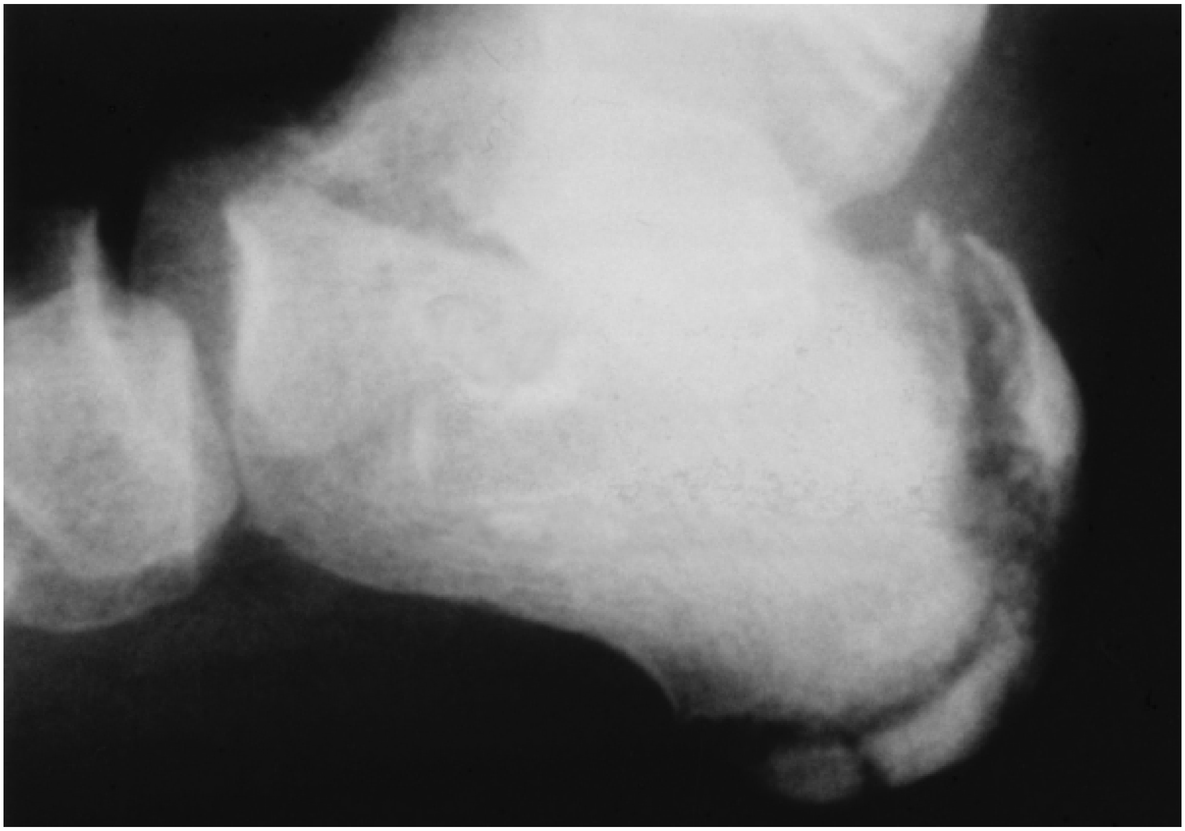

Figure 25C2-18 Lateral radiograph of the calcaneus. Note the irregularity, especially in the superior part of the apophysis. Fragmentation and increased density are common occurrences in the calcaneal apophysis.

SYSTEMIC ILLNESS

Systemic illness can present with foot pain and must be remembered in the athlete as well as in other children. Rheumatoid arthritis or hemophilia can involve the subtalar joint. Osteomyelitis can involve the foot but is unusual unless there is a history of a puncture wound. Acute lymphocytic leukemia is a great masquerader and can infiltrate the bones of the foot. Although they are rare, these sorts of diseases must be considered. One cannot develop tunnel vision and believe that all pain in an athlete is of traumatic origin.

SHOES AND ORTHOTICS

The athletic shoe business is a lucrative one, as is shown by the intense marketing, endorsement, and competition for the introduction of new technology and an edge in the marketplace. Little scientific evidence supports the hype associated with shoe sales. Most often, the advertisements depict current sports heroes wearing shoes from the high end of the price scale, and they tell us little about the shoes themselves. Athletic shoes should fit adequately in both width and length. The models and range of widths available are more limited for children. The material should be reasonably soft. Too often, children's athletic shoes are made of stiff, unyielding, synthetic material and are poorly padded around the heel counter. Multisport shoes with small-diameter, evenly spaced cleats that distribute weight-bearing more evenly are preferable to cleated or studded shoes. Padding over the heel counter and ankle may increase comfort. Most shoes now come with a built-in arch support that has little scientific basis but may give some support to children with a well-developed arch. Those with a flatter foot may actually find it necessary to remove the pad.

Orthotics is another area of controversy. An asymptomatic flexible flatfoot should be left alone. There is no evidence to support the idea that an orthotic will bring about any structural change in such a foot. A painful flatfoot should prompt a thorough search for its cause, such as a tarsal coalition. Orthotics may be tried in a patient with aching feet or shins and a flexible flatfoot. Heel cups may be beneficial in the symptomatic treatment of heel pain. There is little if any scientific information available about the use of sports orthotics in children.

CRITICAL POINTS

- Patients presenting with cavus deformity of the foot must have a thorough neurologic examination and workup.
- Serious ankle sprains are rare in the skeletally immature patient. The physes are the weak link and usually give way before the ligaments.
- One must have knowledge of the factors involved in predictions of outcome in ankle fractures in skeletally immature patients in order to choose treatment modalities and obtain informed consent.
- Salter Harris II fractures of the tibia associated with an abduction mechanism of injury must be reduced to near anatomic position.
- Patients with Salter Harris III and IV injuries require restoration of normal joint and physeal alignment and congruity.

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SECTION D Tendon Injuries of the Foot and Ankle

Geoffrey S. Baer,
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Although the Achilles tendon has been the focus of most reports on tendon injuries of the foot and ankle, athletes sustain many disabling injuries that involve other tendons (e.g., anterior and posterior tibial, flexor hallucis longus [FHL]) in this anatomic region. The frequency of tendon injuries around the foot and ankle has been recorded in only a limited number of studies. [606] [607] [608] [609] [610] In 1959, Anzel and colleagues reviewed 1014 cases of muscle and tendon disruptions treated at the Mayo Clinic and found that 143 (14%) of the injuries occurred in the lower extremity. [1] Of these 143 injuries, 34 (24%) involved the Achilles tendon or triceps surae, 10 (7%) the anterior tibial, 3 (2%) the posterior tibial, 3 (2%) the FHL, and 2 (1%) the peroneus longus or brevis tendon. Although earlier studies [611] [612] [613] did not provide specific numbers or percentages, they did state that muscles and tendons are torn in the following order of frequency: triceps surae, quadriceps, biceps, triceps, and rectus abdominis.

Most of these studies emphasize the rarity of injuries to the tendons of the foot and ankle, and several suggest that these injuries, if untreated, cause little functional impairment. [614] [615] [616] [617] [618] Other studies emphasize that disruptions of the tendons of the foot and ankle, particularly those that occur in the fibro-osseous tunnels, are subject to the same complications characterizing tendon injuries in other anatomic regions. [607] [608] [610] [619] [620] [621] [622] [623] [624] [625]

These latter studies conclude that the same principles of treatment well established in injuries of the wrist and hand should be applied to tendon injuries of the foot and ankle if maximal function is to be regained.

In this chapter, the issues related to operative versus nonoperative treatment of injuries to the Achilles, anterior and posterior tibial, FHL, and peroneal tendons are addressed, and pertinent demographic data (age, sex, sport, and so forth) relative to an athlete's predisposition to injure the aforementioned structures are delineated.

INJURIES DEFINED

Before beginning a discussion of specific tendon injuries, it seems germane to define the terms used to describe and classify these conditions. The dictionary definition of *tendinitis* (also spelled tendonitis) is “inflammation of tendon” and that of *tenosynovitis* (or tenovaginitis) is “inflammation of a tendon and its sheath.” It is apparent that these definitions are too generic when one considers that many large tendons, such as the Achilles, do not have a true synovial sheath and are composed of dense fibrous connective tissue that has little vascularity. [21] Thus, these tendons lack the extrinsic vascularity that would predispose them to an inflammatory process. They are, however, encased by a *peritendineum*—a vascular, hypercellular, peritendinous connective tissue that has visceral and parietal layers, and septa that run between the fibers of the tendon. It is this peritendinous tissue that has the potential for an inflammatory response, and thus inflammation of tendons that lack a synovial sheath (e.g., Achilles) is more accurately described as either *peritendinitis* [627] [628] [629] or *paratendinitis*. [617] [630] The latter term appears to be less descriptive because studies have shown that in cases of “pure peritendinitis” the underlying tendon is entirely normal. [628] [629] It has been documented, however, that tendon ruptures often are preceded by recurrent episodes of peritendinitis. [627] [629] [631] In these cases, an inflammatory process occurs in the peritendinous tissues and degenerative changes also occur in the tendon itself. Puddu and colleagues defined this situation as a “peritendinitis with a tendinosis.” [24] Tendon ruptures also occur with no previous history of peritendinitis. [629] [631] [632] Surgical specimens from these cases demonstrate microscopic evidence of degeneration of the tendon but no associated peritendinitis. [629] [632] Thus, we agree with others who suggest that the term *tendinosis* best describes these latter pathologic changes in the tendon. [627] [628] [629]

ANTERIOR TIBIAL TENDON INJURIES

Disruptions of the anterior tibial tendon are uncommon and most often are the result of lacerations or open injuries. [606] [608] [610] [628] [633] [634] [635] Anagnostakos and colleagues [31] identified 111 previous reports of anterior tibial tendon disruptions in their review of the literature and report of 3 additional cases. Anzel and colleagues reviewed 1014 cases of muscle and tendon injuries treated at the Mayo Clinic and found that only 10 (9 of which were open injuries) involved the anterior tibial tendon. [1] In contrast, Hovelius and Palmgren [29] and Simonet and Sim [30] reported on a total of seven boot-top tendon lacerations in ice hockey players. In Hovelius and Palmgren's series of two cases, the anterior tibial, extensor hallucis, and extensor digitorum tendons were all cut, as were the deep portion of the peroneal nerve, the anterior tibial artery, and the saphenous vein. [29] Similar structures were found lacerated in Simonet and Sim's series of five cases.[30] In their series, the diagnosis of the tendon lacerations was missed by the initial treating physician in two of the five cases. They concluded that the lack of the apparent seriousness of the skin laceration contributes to this error. They recommended that all boot-top lacerations be thoroughly explored.[30] Both reports stressed that this injury could be prevented if players wore their skate tongues in the up position, laced the skate to the top, and put the tongue under the shin guard. [634] [635] In both series, none of the injured hockey players were wearing the tongue of the skate in the up position.Closed disruptions are uncommon, with only a limited number reported to date in the literature, [610] [614] [618] [619] [623] [624] [625] [633] [636] [637] [638] [639] [640] [641] [642] [643] [644] and only six of these injuries occurred in athletes: three during cross-country skiing, [638] [643] [645] one during alpine skiing,[41] two during fencing.[31] The alpine injury occurred in a 45-year-old man who fell while downhill skiing,[41] and the others occurred in a 72-year-old retired physician who caught the tip of his ski while cross-country skiing,[40] a 40-year-old lawyer who was attempting the skating technique for the first time while cross-country skiing,[33] and a 68-year-old man who was attempting cross-country skiing for the first time and fell, which led to forced plantar flexion of his foot.[38] Both fencing injuries occurred during a lunge maneuver with both athletes (67 and 53 years old) feeling acute sharp pain over the anterior ankle.[31] Additionally, Hamilton and Ford[42] reported a case of a longitudinal tear within the substance of the tibialis anterior tendon in a 60-year-old woman who had a painful mass and swelling over her anterior ankle. These reports indicate that diagnosis of a closed disruption often is delayed because the inability of the patient to dorsiflex the foot forcefully is interpreted as an acute neurologic problem. [610] [639] Attention to the details of the history of onset and a thorough physical examination of the foot and ankle, however, will delineate the problem.

Pertinent Anatomy

The main extensor muscles of the foot and ankle are the tibialis anterior, extensor hallucis longus, extensor digitorum longus, and peroneus tertius. At the level of the ankle joint, the tendons of the tibialis anterior, extensor hallucis longus, and extensor digitorum longus are immediately adjacent to the distal tibia and pass under the superior extensor retinaculum (transverse crural ligament) and the inferior extensor retinaculum (cruciate crural ligament) in tight, fibro-osseous canals. Each of these tendons not only has its own compartment but also has a separate synovial sheath. The tibialis anterior tendon, a subject of this chapter, passes behind the superior retinaculum and lies within the most medial compartment of the inferior extensor retinaculum. After crossing the front of the ankle joint, it inserts into the medial and plantar aspects of the medial cuneiform bone and the adjacent base of the first metatarsal. At least seven insertion types for the tibialis anterior tendon have been identified. Musial[43] described four types of insertion, all with two slips, with a wide insertion on the medial cuneiform and a narrow insertion onto the first metatarsal (type 1, 56.6%), two slips of equal width inserting on the medial cuneiform and first metatarsal (type 2, 37.7%), principal insertion onto medial cuneiform with accessory slips to the first metatarsal (type 3, 4.1%), and a wide insertion onto the first metatarsal and a narrow one onto the medial cuneiform (type 4, 1.6%). Arthornthurasook and Gaew-Im[44] identified an additional configuration where there was only a single slip inserting onto the medial cuneiform (15.9%). Brenner[45] found similar insertion sites with differing frequencies but also found two cases of tendons inserting only onto the first metatarsal. Luchansky and Paz[46] identified an additional variant with the primary tendon inserting on both the medial cuneiform and first metatarsal with a second tendon originating from the primary tendon near the cuneiform and inserting onto the first metatarsal. Anagnostakos and colleagues identified similar insertions for the tibialis anterior tendon, with 68% of tendons inserting into both the medial cuneiform and first metatarsal.[31] The tibialis anterior (the largest anterior muscle of the leg) is the primary dorsiflexor of the foot, and because of the tendon's medial site of insertion, it also adducts and inverts (supinates) the foot. The tendon, however, is not a very powerful adductor owing to its proximal insertion on the foot.

The blood supply of the tibialis anterior tendon may be a causal factor in spontaneous rupture of the tendon. One study, which only used injection techniques, found that the anterior tibial tendon had a complete blood supply without any evidence of an avascular zone.[47] A more recent study that employed histochemical methods and injection techniques demonstrated an avascular zone at the usual site of rupture, which is 5 to 30 mm from the site of insertion of the tendon.[48] Specifically, they found that the blood supply of the proximal part of the tendon arises from the anterior tibial artery. Distally, the tendon is supplied by branches of the medial tarsal arteries. The branches of both arteries reach the tendon through vincula from the posterior side. Distribution of the blood vessels within the tendon, however, is not homogeneous. The avascular zone is located at the level of the superior and inferior retinaculum. This is the site where the tendon changes its direction of traction and where closed disruptions usually occur.[48]

Clinical Evaluation

In general, closed disruptions of the anterior tibial tendon occur in individuals older than 45 years of age, and surgical specimens have documented that degeneration of the tendon often precedes the disruption. [610] [618] [624] [625] [638] [639] The rupture is usually the result of minor trauma (such as stumbling over a door jamb or a stone), resulting in forceful plantar flexion of the foot. Degenerative tears after corticosteroid injections also have been reported. [636] [638] [654] [655] Other reported causes of spontaneous rupture include systemic lupus erythematosus,[51] hyperparathyroidism,[52] chronic acidosis of lead nephropathy,[53] diabetes mellitus, [659] [660] [661] rheumatoid arthritis, [623] [662] [663] psoriasis, [59] and gouty tophi. [60] With this injury, an individual usually experiences a transient twinge of pain in the anterior aspect of the ankle but is able to continue his or her usual activities. The injury usually does not produce discoloration or swelling, and thus it ultimately is manifested only by a painless, flatfooted (slapping) gait. Because of the lack of awareness of this rare condition, late diagnosis of this injury occurs in most cases. [625] [637] [638] The differential diagnosis for this condition often includes peroneal nerve palsy, compartment syndrome, lumbar disk herniation, tendinitis, insertional tendinopathy, and neoplasm. [31] In one series, time to diagnosis averaged 2 1⁄2 months (range, 0 to 8 months). [33] Most individuals seek medical evaluation only when they or others notice the abnormal gait pattern, often many weeks after the injury occurred.

On physical examination, it will be noted that the patient stands with the foot everted and that there is a loss of dorsiflexion when the patient attempts to walk on the heels. The degree of footdrop is variable and depends on the amount of time that has elapsed since the injury. Within weeks of the rupture, the proximal end of the tendon will become reattached to the surrounding structures and will provide variable power for dorsiflexion of the ankle. On examination of the ankle, however, one will find a visible and palpable defect in the tendon just distal to the transverse retinacular (cruciate crural) ligament, or in the case of tenosynovitis or a longitudinal tear, a mass may be palpated. [647] [666] On active dorsiflexion of the foot, one may feel the tendon curl up and produce a mass in the anterior leg. This mass disappears with relaxation of the muscle. If there is a question about the cause of the footdrop, electrodiagnostic evaluation will rule out other causes (e.g., peroneal nerve palsy) for this abnormality. Standard anteroposterior and lateral radiographs of the ankle should be obtained to rule out an avulsion fracture at the tendon's insertion on the navicular bone. [624] [667] Although diagnosis can usually be established by physical examination, sonographic studies will demonstrate discontinuity of the tendon fibers separated by a hypoechoic defect. [49] Magnetic resonance imaging (MRI) has been advocated for diagnosis of both partial and complete anterior tibial tendon ruptures when clinical history and physical findings are confusing. [645] [654] [668]

Treatment Options

Treatment of *open* injuries and lacerations is surgical and consists of end-to-end repair with nonabsorbable sutures. [607] [608] [610] [633] [634] [635] Closed ruptures have been treated successfully with both operative [607] [610] [619] [623] [624] [633] [636] [638] [640] [641] and nonoperative regimens. [614] [669] Moskowitz reported on two patients treated with orthotics (two men, 69 and 70 years of age) and found that 1 year after injury they both walked with an almost normal gait, experiencing only slight difficulty in walking on their heels. [13] He concluded that surgical repair "hardly seemed indicated" in elderly patients. Markarian and coworkers [18] found no difference in outcome in a comparison of operative and nonoperative treatment in a group of 16 patients but recommended surgical repair in acute cases and in the delayed management of young, high-demand patients.

Many authors, however, have concluded that surgical repair is the best treatment for acute (≤4 weeks) and subacute (≤3 months) injuries because it prevents the complications of nonoperative treatment, which include a flatfooted gait, decreased ankle motion, heel cord contractures, and a flat, pronated foot. [607] [610] [614] [619] [623] [624] [625] [633] [638] [640] [641] These studies document that restoration of normal function is more rapid with surgical repair of the tendon. Acute injuries are best treated with primary, end-to-end repair of the tendon when possible, whereas tendon-lengthening procedures may be required for a larger defect. [623] [636] [638] [670] Wong [66] treated a chronic traumatic tear in which a 7-cm defect was bridged by harvesting a 7-cm free tendon graft from the proximal stump of the tibialis anterior and then attaching in an end-to-end fashion to bridge the defect. This same technique has been used to treat smaller defects and has the advantage of no donor site morbidity. [672] [673] After surgery, the ankle is immobilized (in dorsiflexion) in a short leg cast for 6 weeks; return to strenuous activity (including mountain climbing) ranges from 14 to 24 weeks. [610] [614] [624]

Appropriate treatment of avulsion fractures from the navicular bone is predicated on the amount of displacement. Scheller and colleagues, who stated that they have observed this injury in athletes, recommend closed treatment if displacement is less than 5 mm and open reduction and internal fixation if displacement is greater than 5 mm. [62]

Longitudinal split tears within the tibialis anterior tendon are rare injuries, and when conservative management has failed to provide relief, surgical intervention has been shown to be effective. Hamilton and Ford [42] reported on the surgical management of a 60-year-old woman with four to five longitudinal tears within the tendon just proximal to its insertion. The tears were treated with débridement and tubularization of the tendon with nonabsorbable 2-0 suture. The patient was treated in a splint for 2 weeks, returned to regular shoewear at 6 weeks, and by 3 months had painless, full range of motion and strength compared with the contralateral foot.

AUTHORS' PREFERRED METHOD

Our preferred method of treatment is predicated on the patient's age, level of activity, and the time elapsed from injury to evaluation. If the patient is not active and is in the sixth or seventh decade of life, the results of operative and nonoperative treatment are presented to the patient, and nonoperative treatment is recommended. In active individuals (regardless of age), operative treatment is recommended. We perform an end-to-end repair with a Krakow-type stitch of 2-0 nonabsorbable suture. The periphery of the tendon subsequently is sutured with a running absorbable 3-0 suture. If the tendon ends cannot be brought together, reconstruction of the tendon should be performed. Several reconstructive techniques have been advocated. [623] [625] [633] [641] [655] The technique using the tendon of the extensor hallucis longus as a graft, as described by Moberg, appears attractive. [19] To date, however, we have not had a case for which this or other reconstructive techniques were needed.

After end-to-end repair, the ankle is immobilized in a neutral position with either a cast or a removable plastic orthosis for 3 weeks. At that time, the orthosis is removed several times each day for active range of motion exercises. Six weeks after surgery, all immobilization is discontinued, and stretching exercises are initiated. Return of full function usually is achieved within 12 to 14 weeks of surgery.

POSTERIOR TIBIAL TENDON INJURIES

Injuries of the posterior tibial tendon include closed disruptions, anterior dislocations, and stenosing tenosynovitis. Isolated anterior dislocations are rare injuries and have been reported infrequently in the literature with mechanisms often including twisting the ankle while running or inverting the ankle during dismounts or ballet maneuvers when the foot becomes inverted and either dorsiflexed or plantar flexed with contraction of the posterior tibial tendon. [674] [675] [676] [677] In a large systematic review, Lohrer and Nauck [73] identified 61 cases of posterior tibial tendon dislocation in the English, French, and German literature. [677] [678] [679] [680] [681] [682] [683] [684] [685] [686] [687] [688] [689] [690] [691] [692] [693] [694] [695] [696] [697] [698] [699] [700] [701] [702] [703] [704] [705] [706] [707] [708] [709] [710] [711] [712] They found that more than 75% of the dislocations were caused by trauma, and when specified, 58% had occurred with sporting activities. The injury was more prevalent in males, and the average age was 33 years. At least 50% of the patients were misdiagnosed initially with ankle sprains or posterior tibial tendon dysfunction. Patients often complain of persistent pain, swelling, and tenderness over the medial malleolus following an inversion injury. In 58% of the patients, a cord-like structure could be felt passing over the medial malleolus. The patients have often undergone conservative care, including periods of immobilization and physical therapy for suspected ankle sprains, and may have imaging studies in which tendon subluxation or dislocation was not present or was not recognized. Lohrer and Nauck identified 10 cases [680] [684] [685] [686] in which the patients were treated conservatively, whereas results were reported for only four patients, with three excellent results and one fair result. Surgery was required in the other cases, with retinacular reconstruction in 42.9%, primary repair of the retinaculum in 32.7%, and groove deepening in 18.4% of patients. Patients were typically immobilized in either a cast or walking boot for an average of 4.8 ± 1.9 weeks, and full weight-bearing was achieved an average of 3.6 ± 2.4 weeks after surgery. Excellent results were identified in 80%, good in 12%, and fair in 8% of the patients at final follow-up.

The frequency of closed disruptions has only recently been appreciated. [621] [622] [713] In 1983, Johnson reported the results of 11 surgical repairs of ruptured posterior tibial tendons that were performed over a 3-year period. [16] In 1985, Mann and Thompson [109] reported on 17 cases that were treated over a 4-year period and noted that the results of only 50 operatively treated disruptions had been previously reported. [616] [620] [621] [715] [716] [717] [718] In 1991, Woods and Leach reported on six athletic people, 20 to 50 years of age, who required surgery for partial (three cases) or complete ruptures of the posterior tibial tendon. [108] Holmes and Mann reported on 67 patients diagnosed with rupture of the posterior tibial tendon. [114] They divided their 67 patients into older (>50 years of age) and younger (<50 years of age) groups. The profile of the older group was significantly different in that 60% of the cases were associated with hypertension, diabetes, and obesity. Younger patients had no significant association with the aforementioned problems, but 5 of these 14 patients had significant trauma to the foot. [114]

All the aforementioned studies stressed that closed disruptions often are not diagnosed because the signs and symptoms, although pathognomonic, are not recognized. In Mann and Thompson's series of patients, all had seen one or more physicians (in only two was the proper diagnosis made), and the average delay in diagnosis was 43 months (range, 1 month to 12 years). Average time to diagnosis in Johnson's series of patients was 19 months; he noted that 10 of his 11 patients had had previous medical evaluations, but in no instance was disruption of the posterior tibial tendon recognized. [16] Woods and Leach noted that the delay in diagnosis in five of their six athletes (range, 6 to 18 months) adversely affected the results of operative treatment. [108] They concluded that the key to successful treatment, particularly in athletic individuals, is to make the diagnosis early, before deformity occurs.

Pertinent Anatomy

The posterior tibial tendon is formed in the distal third of the leg by the convergence of the large muscle units that arise more proximally from the posterior surfaces of the tibia and fibula and the adjacent interosseous membrane. At the level of the medial malleolus, the posterior tibial tendon is the most anterior structure, located immediately adjacent to the posterior surface of the malleolus. The other structures, in order from anterior to posterior, are the flexor digitorum, the posterior tibial artery, vein, and nerve, and the FHL (one mnemonic for remembering the order of these structures is "Tom, Dick, and A Very Nervous Harry"). In this region the structure of the posterior tibial tendon changes to a more fibrocartilaginous structure with a change in cellularity and extracellular matrix components. [115] The ability of this altered structure to resist repetitive microtrauma may make this area more vulnerable to spontaneous rupture. [115] Histologic analysis of spontaneously ruptured tendons has not shown normal tendon structure; instead, degenerative tendinosis, mucoid degeneration, tendolipomatosis, and calcifying tendinopathy have been identified. [721] [722]

The posterior tibial tendon has numerous insertion sites on the plantar aspect of the foot. These include the inferior aspect of the navicular bone, all three cuneiform bones, and the bases of the second, third, and fourth metacarpals. [723] [724] The incidence of an accessory navicular or os tibial externum, a sesamoid bone invested in the posterior tibial tendon near its insertion, is 10% to 20%. [723] [724] [725] If present, it may be a small accessory bone within the posterior tibial tendon without attachment to the navicular (type I); an accessory navicular with a synchondrosis (type II); or a cornuate bony, navicular tuberosity (type III). [118] The tibialis posterior is the main dynamic stabilizer of the hindfoot against valgus (eversion) forces, owing to its multiple insertions, which are variously taut at different positions of foot function, and to the size and strength of its muscle. Disruption of the tendon, therefore, results in elongation of the ligaments of the hind and midfoot and a painful flatfoot deformity.

Variations in the vascular anatomy of the flexor tendons also may play a role in the frequency and location of injury. In a recent study, Frey and colleagues used injection techniques to examine the intrinsic vascularity of the posterior tibial and flexor digitorum longus tendons in 28 cadaveric limbs. [121] In all 28 specimens, they found that the posterior tibial tendon had a zone of hypovascularity in its midportion. The zone started about 4 cm from the tendon's insertion on the medial tubercle of the navicular bone and ran proximally for an average of 14 mm. Their dissections confirmed that there was no mesotenon present in this region and that the visceral layer of synovial tissue was present but hypovascular. Although this zone of hypovascularity may predispose the tendon to degenerative processes, it is proximal to the most common site of rupture of this tendon (1.0 to 1.5 cm from its insertion into the navicular) reported in the literature. [620] [621] [622] In contrast, the midportion of the flexor digitorum longus tendon has ample vascularity throughout its length, owing to a longitudinal system of vessels arising from the proximal and distal arteries. [121]

Clinical Evaluation

The hallmark of a posterior tibial tendon disruption is the progressive deformity of the foot. Patients usually present with a painful flatfoot and note that they cannot walk "normally," have difficulty going up and down stairs, and have lost control of their foot. Disruptions most commonly occur in women older than 40 years of age, and many patients cannot recall any significant previous acute trauma. [16] Most patients do, however, have a history of antecedent tenosynovitis [108] and state that the problem began after twisting the foot, slipping off a curb, or stepping in a hole. [17] Most do not seek emergent care because their pain, located medially between the tip of the malleolus and the navicular bone, is not incapacitating. [621] [622] [713]

Salient physical findings include valgus of the hindfoot, abduction of the forefoot, a positive single-heel rise test, a too-many-toes sign, and loss of function on manual testing of the tendon. The concurrent development of abnormal hindfoot valgus and forefoot abduction produces the flatfoot deformity. These deformities are best evaluated by observing and comparing the posterior aspects of both feet with the patient standing. Patients with tendon ruptures have either a unilateral flatfoot or, in those who had previous flat feet, a relatively flatter foot on the involved side. Excessive forefoot abduction also can be suspected from posterior observation when more toes are visible lateral to the patient's heel on the involved side. This finding is called the *too-many-toes sign* (Fig. 25D-1).

Figure 25D-1 The too-many-toes sign. More toes are visible lateral to the heel on the patient's right side.

The best method for evaluating loss of posterior tibial function is the single-heel rise test. [16] In this test, the patient is instructed to stand on one leg with the knee extended and to rise onto the ball of the foot and the tips of the toes. The test is positive if the hindfoot (heel) on the involved side fails to invert and assume a stable (varus) position (Fig. 25D-2). The patient also will have great difficulty in raising the heel off the floor because gastrocnemius muscle function is compromised when there is no posterior tibialis to bring the heel into a locked, stable varus position. [16] Manual testing of the strength of the posterior tibial tendon can be misleading because of the inversion strength of the tibialis anterior. This latter muscle and its tendon effectively resist eversion of the foot when the foot is in full inversion. Thus, loss of posterior tibial function is best assessed by everting the heel and placing the forefoot in full abduction. The patient is then asked to invert the foot. The presence of posterior tibial weakness will be apparent with this maneuver, particularly if one compares it with the strength of the opposite foot.

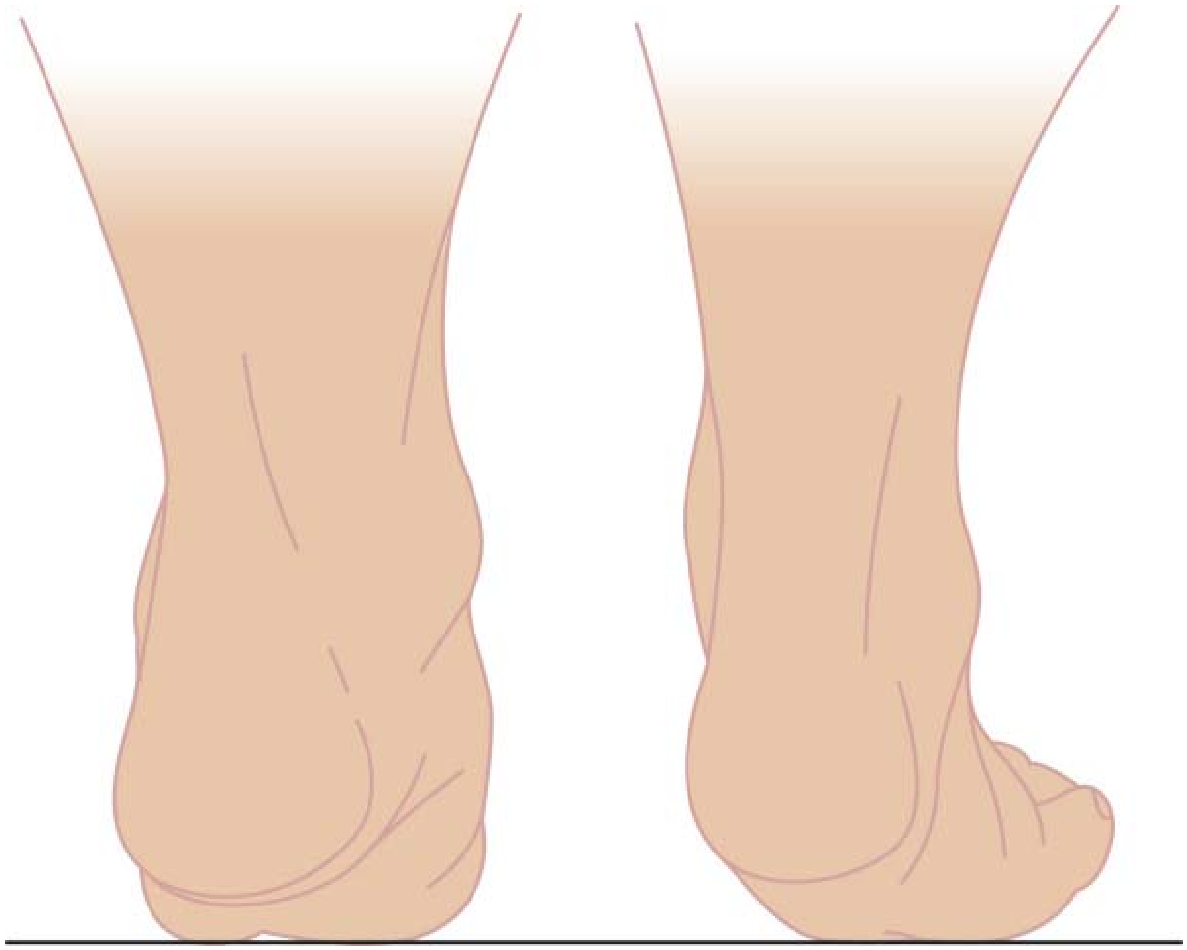

Figure 25D-2 Single-heel rise test. The right heel does not assume a stable (varus) position (as seen on the left) when the patient attempts to stand on the toes.

Standard weight-bearing anteroposterior and lateral radiographs show variable changes depending on the time elapsed since the injury. Although several studies state that standard radiographs are not useful in making a diagnosis of posterior tibial tendon disruption, [626] [622] [727] Johnson concluded that standard films usually show pronounced changes. [16] Specifically, an increase in the talocalcaneal angle and inferior subluxation of the talus at the talonavicular articulation are evident on the lateral view. On the anteroposterior view, subluxation of the forefoot at the talonavicular articulation and an increased talocalcaneal angle are evident. [16] In Mann's study, the lateral talar-first metatarsal angle averaged 22 degrees. [17] This angle is 0 degrees in normal feet, 1 to 15 degrees in mild flatfoot deformities, and more than 15 degrees in severe flatfoot deformities. [123] Ancillary studies such as computed tomography (CT) and MRI are rarely indicated. CT scans should be obtained in feet with rigid deformities to rule out tarsal coalitions. Although MRI demonstrates the site or sites of tendon disease, it is not required to establish the diagnosis of posterior tibial tendon disruption.

Treatment Options

Nonoperative treatment of posterior tibial tendon disruption is appropriate in patients who have low activity levels, are in their 60s or 70s, have life-limiting medical problems, or have minimal discomfort from their acquired deformity. If the injury is diagnosed acutely, immobilization of 4 weeks' duration in a short leg cast that is well molded along the longitudinal arch is recommended. [616] [613] [620] [621] In subacute cases, the use of an orthosis (a flexible leather arch support with a 3⁄16-inch heel wedge) is appropriate if the patient's only complaint is that he or she is wearing out the medial counter of his or her shoe because the results of later surgery will not be compromised if the foot is flexible. [620] [622] In most patients, however, the prognosis for an unrepaired posterior tibial tendon is poor because of the unopposed pull of the peroneus brevus tendon and loss of hindfoot inversion and the inevitable development of a painful flatfoot. [621] [729] Goldner and colleagues [15] reported on three patients with untreated posterior tibial tendon lacerations, all of whom reported progressive loss of the arch and weakness with increased physical activity. Thus, early surgical repair or reconstruction is the best treatment, particularly in athletic individuals. [713] [730] [731]

Appropriate surgical treatment of a posterior tibial tendon disruption is predicated on the site and extent of the tear, the flexibility (duration) of the foot deformity, and the expectations of the patient. Based on these factors, there are four distinct surgical procedures: (1) end-to-end suture, (2) reattachment or advancement of the tendon to its primary site of insertion on the navicular bone, (3) reconstruction of the tendon, and (4) a triple or limited arthrodesis.

Primary end-to-end repair often is not possible owing to retraction of the proximal segment of the tendon. [616] [622] [718] In Johnson's series of 11 patients, primary repairs were performed successfully in three cases, [16] but in other series of patients (Mann and Thompson [17 patients], [17] Wren [12 patients], [113] Kettelkamp and Alexander [3 patients] [11]), end-to-end suture was not possible, even when surgery was performed within 7 days of the injury. [11]

Advancement of the tendon and tendon transfers are indicated when the posterior tibial tendon is found to be within its sheath but is partially torn and lengthened, or is ruptured within 3 cm of its distal insertion. In the latter situation, advancement of the posterior tibial tendon and anchoring the tendon through a vertical drill hole in the navicular bone may be indicated. One must ascertain, however, that the proximal part of the tendon moves freely within the tendon sheath, particularly at the medial malleolus, and that there is good elasticity of the muscle-tendon unit.

Tendon transfers are often indicated even when the proximal end of the tendon is retrievable because in most cases there are more proximal segments of degenerated tendon. [620] [621] [622] [715] [718] In these cases, resection of the degenerated segments usually does not allow an end-to-end repair of the tendon. Some studies have found that tendon transfers are not necessary for the aforementioned problem and have bridged the gap and reconstituted the function of the posterior tibial tendon with Z-plasty lengthening or turn-down techniques. [616] [732] Because of the short excursion of the posterior tibial tendon, however, a Z-plasty lengthening of more than 1 to 2 cm will cause a functional deficit. [116]

The flexor digitorum longus is most often used in tendon transfers because of its size, strength, and location. [620] [621] [622] [713] [715] [718] When the flexor digitorum longus tendon is used, it is transected just distal to the medial malleolus, and the proximal segment of the tendon is placed within the posterior tibial tendon sheath and tenodesed to the distal end of the posterior tibial tendon. [17] The distal stump of the flexor digitorum longus is tenodesed to the adjacent FHL. Tenodesis of the more proximal segment of the flexor digitorum longus to the musculotendinous junction of the posterior tibial tendon is indicated only when the posterior tibial muscle has normal color and good elasticity. [620] [621] [622]

When scarring within the sheath of the posterior tibial tendon is too extensive to permit free motion of the transferred flexor digitorum longus, the latter tendon is left in its own sheath and is passed dorsally through a vertical drill hole in the navicular and sutured back on itself. The flexor digitorum longus tendon should be sutured under maximal tension while the foot is held in maximal inversion and plantar flexion.

Most studies recommend the use of the flexor digitorum longus as the graft because although it is only one third the diameter of the posterior tibial tendon, it is only slightly smaller than the diameter of the peroneus brevis. The peroneus brevis is the strongest everter of the foot, and in patients with posterior tibial tendon ruptures, its unopposed function results in the flatfoot deformity. [621] [622] [718] In contrast, Goldner and colleagues recommend transferring the FHL when advancement or end-to-end suture of the posterior tibial tendon is not advisable. [15] They note that the key to success of this transfer is to section the FHL tendon proximal to its attachment to the flexor digitorum so that the flexor digitorum will provide flexion of the big toe and prevent hyperextension of the distal phalanx. Other authors do not use the FHL because they believe it plays an important role in push-off and in stabilization of the longitudinal arch of the foot. [621] [622] [718]

Tendon transfers are not appropriate when there are severe fixed deformities of the foot and secondary degenerative changes in the talonavicular or talocalcaneal joints. In these cases, tendon transfers will not correct the deformity or alleviate the patient's pain, and a limited or triple arthrodesis is indicated.

A triple arthrodesis should be performed when there are degenerative changes in the talonavicular and calcaneocuboid joints and fixed medial-inferior rotation of the head and neck of the talus. A single talocalcaneal arthrodesis is appropriate when the talonavicular and calcaneocuboid joints do not have secondary degenerative changes. [621] [622] If the primary deformity is abduction of the forefoot, and the foot is flexible and can be placed in its normal position, a talonavicular and calcaneocuboid fusion (double arthrodesis) can be performed and will produce the same result as a triple arthrodesis. [16] In Johnson's series of 11 patients, two patients with fixed deformities were treated by single (subtalar) arthrodeses, [16] and in Mann and Thompson's 17 patients, one had a double (calcaneocuboid and talonavicular) arthrodesis after an unsuccessful flexor digitorum longus tendon transfer. [17]

AUTHORS' PREFERRED METHOD

To date, we have not found it necessary to perform tendon transfers in the limited number of patients we have treated with posterior tibial tendon disruptions. In these few cases, posterior tibial function has been restored either by end-to-end suture or by advancing and reattaching the tendon to the navicular bone as described earlier. One should always be prepared, however, to perform tendon transfers when one operates to restore posterior tibial tendon function because, as noted previously, the surgical findings indicate which procedure is most appropriate.

POSTERIOR TIBIAL TENDINITIS

Based on our own experience and a review of the literature, it is apparent that tenosynovitis of the posterior tibial tendon is far more common than a total disruption of the tendon. [667] [717] [733] [734] [735] [736] Patients with this problem typically are women (in their fourth to sixth decades of life) who have occupations that require long periods of standing. In general, onset of symptoms is gradual, and the pain is aggravated by prolonged walking and standing. Posterior tibial tendinitis is often an initiating event in the development of adult-acquired flatfoot deformity. There is a paucity of literature that addresses this problem in younger individuals, however. Goldner had three patients younger than 20 years of age with tibialis posterior dysfunction in his series. In all three patients, trauma was the cause of their dysfunction. [132] In nonathletic individuals, the pain from their posterior tibial tendinitis is not disabling and has been present for several months before the patient comes in for evaluation.

The problem is less common in younger patients. The incidence has been reported as 2.3% to 3.6% in runners presenting to sports medicine clinics. [738] [739] In athletic people, the pain is debilitating because they are unable to run properly or perform any athletic activity requiring a strong push-off action. [713] [733] The pain, which is usually localized to the posterior tibial tendon in the vicinity of the medial malleolus, increases with prolonged and strenuous activity.

Clinical Evaluation

Physical examination reveals medial ankle swelling and, in later stages, flattening of the arch of the involved foot. Palpation along the course of the posterior tibial tendon causes pain, and the site of maximal tenderness usually is located behind the medial malleolus. The integrity of the posterior tibial tendon can be confirmed by the functional tests outlined earlier, but there will be relative weakness of the tendon compared with the contralateral side owing to pain. Specifically, lack of heel inversion when the patient attempts a single-leg toe raise indicates rupture of the posterior tibialis tendon. In contrast, patients with tendinitis demonstrate heel inversion with this test but are unable to perform the single-leg toe raise 10 or more times owing to posterior tibialis pain. In addition to tenderness, swelling is usually found along the lower part of the tendon, and there may be palpable crepitus over the tendon on active movement. Active inversion and passive eversion of the foot usually reproduce the patient's pain.

Radiographic evaluation of these patients should include standard, weight-bearing anteroposterior, and lateral views of the foot, as well as anteroposterior, lateral, and mortise views of the ankle. The relationship between the bones of the foot, including the longitudinal arch (a 0 to 10 degrees lateral talometatarsal angle), is normal in posterior tibial tendinitis.

MRI, [740] [741] [742] [743] ultrasonography, [744] [745] [746] and scintigraphy [142] have been advocated for evaluation of posterior tibial tendinitis. Several studies have documented that MRI is more effective for determining abnormalities within the posterior tibial tendon in cases of tendinitis than ultrasound or tenography. [740] [741] [743] Other studies found that sonographic evaluations had greater accuracy (94% versus 66%) and sensitivity (100% versus 23%) than MRI for demonstrating posterior tibial tendon abnormalities, [744] [745] [746] and may demonstrate tendon ruptures (two cases) not diagnosed by MRI. [140] The exact role of these modalities will be defined only through prospective studies that demonstrate that the information gained from sonographic or MRI evaluation altered the patients' final outcome.

Posterior tibial tendinitis is usually evident by clinical evaluation, but in our practice we obtain an MRI to rule out osteochondral fractures of the talar dome, navicular stress fractures, and accessory navicular injuries. MRI is also useful to evaluate the posterior tibial tendon for skip areas and to determine the extent of internal tendinous degeneration when operative intervention is necessary for treatment of this problem. Tendon degeneration found at surgery is often much greater than that expected based on clinical evaluation.

In the evaluation of posterior tibial tendinitis, it is also important to evaluate for the development of adult-acquired flatfoot deformity. Adult-acquired flatfoot deformity has been described as occurring in four stages. [143] In stage I, the patient may or may not have flatfoot deformity but will present with tenosynovitis or tendinosis. [621] [714] [748] [749] Flatfoot deformity then develops as the posterior tibial tendon fails, and subsequent ligament failure (often involving the spring ligament supporting the talonavicular joint) occurs. Once the talonavicular joint fails and begins to subluxate, the interosseous ligament becomes involved, and the subtalar joint begins to subluxate. The talar head continues to migrate medially and plantar, leading to further ligamentous failure and progression of deformities at the naviculocuneiform and tarsometatarsal joints and failure along the entire medial arch. In stage II disease, the flatfoot deformity can be passively corrected with inversion of the talonavicular joint and correction of heel alignment. Radiographs of stage II disease will show uncovering of the talar head and forefoot abduction. In stage III disease, the flatfoot deformity is no longer correctable. Lateral pain may develop as subtalar subluxation advances and the hindfoot progresses into further valgus deformity. [749] [750] With stage IV disease, lateral talar tilt is noted at the tibial-talar joint. [146]

Treatment Options

Initial treatment of this condition is immobilization for 2 to 6 weeks in a short leg cast or rigid plastic boot, along with anti-inflammatories, which in most cases relieve the pain. [667] [752] [753] In several earlier studies, local steroid injections into the synovial sheath were administered to patients who did not respond to nonoperative (supportive casts and anti-inflammatory medications) treatment. [735] [736] [754] These injections, however, have been associated with tendon rupture. [715] [717] [719] In Trevino and associates' series of eight operative cases, three of the five tendons that received local steroid injections before surgery were partially or completely ruptured. [112] Based on our current knowledge of the deleterious effects of steroids on the strength of tendons, we feel that steroid injections are never indicated for treatment of this problem.

Operative treatment of this condition was required in 12 of the 52 patients reported by Williams [131] and also in other smaller series of patients. In most cases, the operation performed included a release of the tendon sheath, excision [734] [735] [736] of the scar tissue, and a partial synovectomy. At surgery, the tendon had a normal appearance and was not opened. The reported results of these procedures were good in 19 of 20 patients. [713] [734] [735] [755] Crates and Richardson reported their results on seven patients who had failed at least 6 weeks of cast immobilization and were available for follow-up at 10.9 months after tenosynovectomy and débridement of partial tendon tears (3 patients). [150a] Three and one-half months after surgery, 6 of 7 patients were completely pain free and could perform a single-heel rise test. The single failure had had persistent symptoms for 12 months before surgery and significant intrasubstance degeneration of the tendon at the time of surgery and later required lateral column lengthening and tendon transfer for progressive disease. Some authors noted the frequent presence of a bulbous distal enlargement of the tendon causing triggering and recommended resection and thinning of the tendon down to the approximate diameter of the noninvolved proximal and distal portions. [717] [735] [736] We have not observed this phenomenon but have found that patients with posterior tibial tendinitis often have an intact, normal-appearing tendon, even when areas of intrinsic degeneration are present (Fig. 25D-3 **A**).

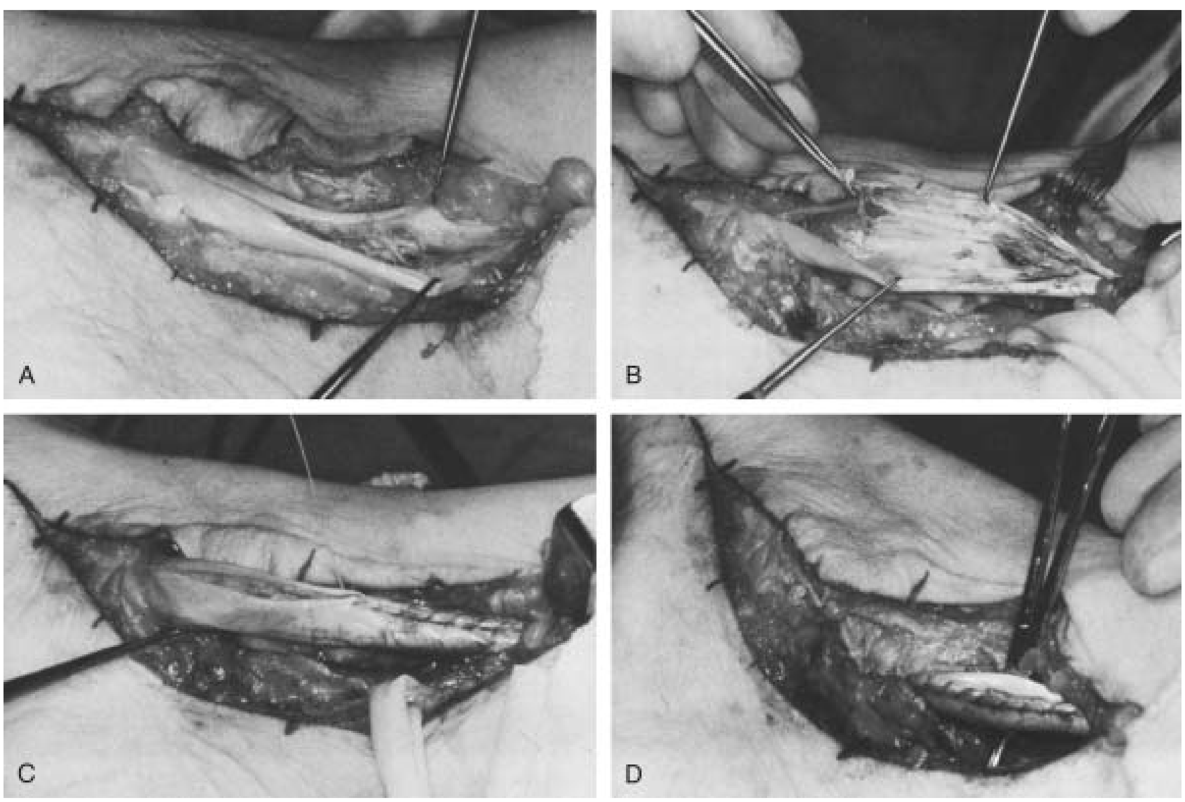

Figure 25D-3 Posterior tibial tendinitis. **A,** The surface of the tendon appears normal, but intrinsic degeneration is apparent when the tendon is opened. **B,** The degenerated central segment of tendon was excised, but the peripheral fibers were intact. **C,** The periphery of the tendon was closed with a running absorbable 2-0 suture. **D,** The retinaculum was closed at the medial malleolus to prevent subsequent subluxation of the tendon.

Recently, tendoscopy has been advocated for treatment of posterior tibial tendon disorders. [756] [757] [758] In 1997, van Dijk and colleagues reported the results of posterior tibial tendon sheath endoscopy performed in 16 consecutive patients. [151] All patients had pain on palpation over the posterior tibial tendon, a positive tibial tendon resistance test, and local swelling. Indications for arthroscopy were diagnostic in 11, chronic tenosynovitis in 2, screw removal in 1, and posterior ankle arthrotomy for a loose body in 2. The results of surgery, performed through a two-portal technique, were good in 3 of 4 patients with adhesions resected, and in the 2 patients who had a tenosynovectomy and tendon sheath release performed. All of these patients were free of symptoms at 1-year follow-up. [151] Chow and colleagues [153] reported on 6 patients with stage I posterior tibial tendon dysfunction treated with tendoscopic débridement. No complications were reported, none of the patients progressed to stage II dysfunction, and the patients had smaller scars, less wound pain, and short hospital stays compared with patients with traditional open débridement.

AUTHORS' PREFERRED METHOD

The posterior tibial tendon is exposed through a curved medial incision from its origin to its musculotendinous junction. The tendon sheath is then divided longitudinally, and hypertrophic synovium, when present, is excised. The tendon is then explored throughout its length to detect unanticipated, partial, or complete ruptures. If disruption of the tendon is encountered, end-to-end suture, advancement, and reinsertion into the navicular, or tendon transfers (as outlined earlier), are performed as indicated. When the tendon is intact, we recommend opening and exploring areas of nodularity, areas where pain was localized preoperatively, and areas where the MRI has demonstrated abnormalities because, as noted earlier (see Fig. 25D-3 **A**), intrinsic degeneration may not be apparent on the surface. Subsequently, all degenerative tissue is removed with a curet or knife (see Fig. 25D-3 **B**), and after débridement, the tendon is closed with a running absorbable 2-0 suture (see Fig. 25D-3 **C**). Although several studies have concluded that releasing and not repairing the tendon sheath is appropriate, [734] [735] [736] subluxation of the posterior tibial tendon has been reported as a complication of tarsal tunnel decompressions. [154] Thus, the posterior tibial tendon sheath is routinely preserved, restored, or reconstructed at the medial malleolus to prevent this complication (see Fig. 25D-3 **D**). The wound is then closed with a running, subcuticular, nonabsorbable 3-0 Prolene suture, and the ankle is immobilized for 2 weeks to allow the incision to heal. At that juncture, immobilization is discontinued, the suture is removed, and the patient is instructed and supervised in gaining full range of motion, full strength, and full function. Relief of symptoms and return to full activity can be anticipated within 12 to 14 weeks of surgery.

FLEXOR HALLUCIS LONGUS INJURIES

There are a limited number of published reports about complete disruptions of the FHL tendon. [606] [615] [730] [760] [761] [762] [763] [764] [765] [766] Only eight cases of closed complete disruptions of the FHL tendon have been reported to date. [760] [761] [762] [763] [764] [765] [766] [767] Six of the eight individuals were injured during the following sports activities: diving from a board, marathon running, walking; and playing soccer. [760] [761] [762] [763] [765] [766] The average age of the eight individuals was 40 years (range, 27 to 54 years), and the sites of tendon injury were in the groove of the posterior process of the talus (one), under the sustentaculum tali (two), just distal to the knot of Henry (two), at the metatarsal head (one), and 0.5 cm proximal to the tendon's insertion on the great toe (two). All eight FHL tendon ruptures were repaired because of functional disability due to loss of great toe push-off. Six were repaired acutely, and two late, at 4 months and at 2 years. Although only three of the eight patients regained active interphalangeal (IP) joint flexion, all of the six athletes regained their preinjury level of performance. [760] [761] [762] [763] [765] [766] Postoperative stiffness of the IP joint did not cause any functional deficit. Boruta and Beauperthuy reported on three patients with longitudinal split tears within the FHL tendon just distal to the knot of Henry. [163] The patients were treated with release of the knot of Henry, débridement and repair of the longitudinal tears, and excision of the interconnections between the FHL and flexor digitorum longus tendons. All three patients obtained good long-term results.

The frequency and causes of FHL tendon lacerations are well chronicled in two studies. [615] [730] In 1977, Frenette and Jackson reported on 10 young athletes (median age, 11 years) with FHL tendon lacerations. [10] In 8 of their 10 patients, the laceration was caused when the athlete was running barefooted and stepped on a sharp object, usually broken glass. To find that number of cases, they surveyed 100 orthopaedic surgeons and did a 5-year review of the hospital records of four large hospitals. They found that three of the six primary repairs of this tendon resulted in no active plantar flexion of the IP joint of the great toe and concluded that repair of the FHL tendon was not essential for good push-off or balance in running sports. In the nonoperatively treated group, one athlete was a former Olympic high jumper and noted no change in jumping ability, whereas a second athlete went on to become the city 100-yard dash champion. However, one of the patients did develop a cock-up deformity that required an IP joint fusion and extensor hallucis longus transfer. [10] In contrast, Floyd and associates reported on 13 cases of open FHL tendon lacerations and found that of 12 tendons repaired (10 sutured primarily, 2 delayed), 9 retained active motion of the IP joint of the great toe. [125] Most of the reports on open or closed disruptions of the FHL tendon have concluded that an intact tendon is essential for good push-off and balance in running sports. [735] [760] [761] [762] [763] [764] [765] [766] [769]

Tendinitis of the FHL tendon is more common than complete disruption. Although cases of flexor hallucis tendinitis and tenosynovitis have been reported in tennis players and long-distance runners, [770] [771] [772] it is much more prevalent, and is the most common site of lower extremity tendinitis, in classical ballet dancers. [773] [774] [775] [776] [777] [778] [779] [780] Washington's survey study of musculoskeletal injuries in dancers puts the incidence of this problem in perspective. [176] Of 414 injuries reported by individual dancers, 55 involved the ankle joint, and 3 of these 55 (5.5%) were diagnosed as tendinitis. Hamilton and colleagues noted that tendinitis around the ankle is common in classical ballet dancers and that the tendon involved is almost always the FHL. [171] They stated that, in ballet, the FHL is the Achilles tendon of the foot when a dancer is *en pointe*. [776] [782] FHL tendinitis is more often seen in the left foot than the right because choreography more often calls for the dancer to turn to the right (clockwise). This requires the dancer to be *en pointe* on the left foot. Michelson and Dunn recently reported on 81 patients with FHL tenosynovitis. [178] The patient population consisted of 55 women and 26 men with an average age of 38.3 years and with 33% involved in athletic activities. All the patients had tenderness over the FHL, and 82% of the patients who had an MRI had evidence of synovitis. Nonoperative management consisted of a stretching program and short-term immobilization. Sixty-four percent of the patients treated nonoperatively had successful outcomes, whereas all 23 of the patients who underwent decompression and synovectomy had successful outcomes.

Pertinent Anatomy

The FHL arises from the lower two thirds of the posterior surface of the shaft of the fibula, and the FHL tendon inserts into the base of the distal phalanx of the great toe. In the sole of the foot, the FHL tendon is connected by a fibrous slip to the flexor digitorum longus tendon. This slip (see earlier) tethers the FHL tendon and prevents excessive retraction of the proximal segment after the FHL tendon is severed. In the great toe, the FHL tendon lies superficial to and between the two heads of the flexor hallucis brevis. Thus, it is relatively easy to find the proximal end of the FHL tendon when it is severed in this location. [10] At the level of the ankle joint, the FHL, posterior tibial, and flexor digitorum longus tendons pass under the flexor retinaculum (lancinate ligament), and septa from this strong fibrous retinaculum convert a series of bony grooves into fibro-osseous tunnels. The tendons also are enclosed in separate synovial sheaths, which are 8 cm long and extend proximal and distal to the aforementioned tunnels. The blood supply to the FHL tendon arises from the posterior tibial and medial plantar arteries. [179] Injection and immunohistochemical studies revealed two avascular zones that correlated with the sites of frequent tendon degeneration and rupture: (1) as the tendon passes behind the talus and (2) around the first metatarsal head. [179]

The constrictive nature of the fibro-osseous tunnels can cause "triggering" of a flexor tendon when partial tearing and healing of the tendon produce exuberant scar tissue. The most common tendon involved is the FHL, and the most common precipitating activity is classical ballet dancing. [773] [774] [775] [776] [777] [778] [779] [780] The FHL tendon is further constrained by bony grooves in the posterior surface of the tibia, the talus, and the sustentaculum tali. Stenosing tenosynovitis has also been reported at the level of the sesamoids, and surgical tenolysis at this level has provided successful relief of symptoms in patients who did not respond to conservative management. [785] [786]

Clinical Evaluation

In evaluating patients with lacerations to the FHL tendon, it is important to evaluate for associated injuries as well. In two studies, injury to the digital nerve was found in 46% and 80% of the patients with open laceration of the long flexor. [615] [730] The injury to the digital nerve may be identified with a thorough physical examination for sensory loss as well as with wound exploration. The function of the flexor hallucis brevis tendon should be evaluated (ability to flex the metatarsophalangeal joint of the great toe) and documented. Additionally, the wound should be explored when there is concern for an open joint as well.

Dancers with FHL tendinitis often note the insidious onset of pain at the posterior medial aspect of both ankles behind the malleolus and often seek evaluation only when triggering of the FHL tendon produces so much pain that they are unable to dance *en pointe*. [773] [776] [778] [779] [780] Sammarco and Miller reported on four ballet dancers who had triggering of the FHL; in two of these dancers, the hallux became locked in a flexed position. [174] They noted that the triggering becomes more severe over a period of several months, but pain is not an outstanding characteristic of this condition. [174] In Kolettis and associates' series of 13 female ballet dancers that had operative release because of isolated stenosing tendosynovitis, all had pain and tenderness over the medial aspect of the subtalar joint. [172] Their symptoms were exacerbated by jumping and attempts to perform *en pointe* work, and all 13 had lost the ability to stand *en pointe*. Crepitus was present in 6 patients, but triggering was present in only 3. None of their dancers had pain or tenderness in the posterolateral aspect of the ankle with forced passive plantar flexion, which would have suggested involvement of an os trigonum.

In contrast, Hamilton and associates reported on 37 dancers who had 41 operations for posterior ankle pain. In their series, only 9 operations were performed for isolated tendinitis. Of the remaining 32 ankles, 26 were operated on for tendinitis and posterior impingement and 6 for isolated posterior impingement. [171]

Physical examination of patients with triggering of the FHL tendon reveals that the hallux can be flexed with ease when the foot is in a neutral position. When the foot is brought into plantar flexion, however, the patient is unable to flex the hallux. On forcible active contraction of the FHL, however, a snap or pop is noted in the posterior medial region of the ankle, and the patient is then unable to extend the IP or metatarsophalangeal joints of the great toe. Subsequent passive extension of the IP joint produces a painless snap or pop posterior to the medial malleolus with subsequent freeing of motion in the great toe.

In dancers who have tendinitis without triggering, localized tenderness and swelling are present, occasionally with crepitus over the FHL tendon just posterior to the medial malleolus. [169] Hamilton stated that FHL tendinitis can be distinguished from tendinitis of other adjacent tendons by the following maneuver. The foot is placed in the pointe position, and the patient is asked to flex the great toe against mild resistance and then to flex toes two through five against resistance. Sequential palpation of the FHL and the flexor digitorum longus will reveal which tendon is locally tender and has the tendinitis. [775] [776]

Differential diagnosis of FHL tendinitis includes peroneal tendinitis, posterior impingement of the os trigonum, acute fracture of the lateral tubercle of the posterior process of the talus, Achilles tendinitis, bone spurs, and arthritic or osteochondrotic lesions of the talus. [775] [776] [787] Posterior impingement can be tested for by forcibly plantar flexing the ankle. When impingement is present, this maneuver usually produces pain in a posterolateral location. [776] [787] [788] Achilles tendinitis can be differentiated from FHL tendinitis by carefully ascertaining the location of tenderness. In Achilles tendinitis, the pain is over the sheath of the Achilles tendon, not over the fibrous tunnel of the FHL. Osteochondritis and arthritic changes of the talus and bone spurs emanating from the tibia or fibula can be excluded by obtaining standard radiographs of the ankle. Hamilton suggests that an additional lateral view of the ankle in the full pointe position should be obtained to evaluate the significance of bony exostoses. [170]

Treatment Options

Although Frenette and Jackson found that repair of FHL tendon lacerations was not essential for restoring good push-off in running sports, they concluded that, in young athletes, all FHL tendon lacerations should be explored, and they recommended the following protocol based on the operative findings. [10] If the tendon ends are easily identifiable, the tendon should be repaired using the same meticulous technique that one would use for flexor tendon lacerations in the hand. Repair is recommended because they found that adhesion formation was a problem, particularly when the laceration is at the level of the first metatarsal head. Patients treated in this manner were subsequently immobilized in a plaster cast and kept non–weight-bearing for 5 to 6 weeks.

Second, if there is concomitant injury to the flexor hallucis brevis and the proximal end of the longus cannot be located, the brevis should be repaired by suturing the proximal end of the distal segment of the FHL tendon to the brevis muscle to prevent the possibility of a hyperextension deformity. Preoperatively, one may be able to establish the integrity of the flexor hallucis brevis by asking the patient to flex the big toe. If active flexion of the metatarsophalangeal joint is present without active flexion of the IP joint, the flexor hallucis brevis is intact.

They also found that the first common digital nerve was divided when lacerations were located distal to the origin of the flexor brevis. In their series of patients, primary neurorrhaphy was attempted in four patients, but restoration of normal sensation was achieved only in one case. They concluded that nerve injuries often were associated with a hypersensitive scar but that the sensory deficit, although annoying, had little effect on total function. They recommended repair, however, of nerves during primary treatment if feasible or, if repair was not possible, resection of the proximal end of the nerve to prevent its entrapment in the scar and the formation of a neuroma in a weight-bearing area.

Scaduto and Cracchiolo recommend surgical exploration and repair of the FHL tendon following a laceration, although the patient should be informed that results of repair of the tendon led to normal active flexion of the IP joint in only 61% of patients. [164] The tendon should be repaired in an end-to-end fashion with a Kessler or Bunnell suture technique with 2-0 or 3-0 nonabsorbable suture. A short leg, non–weight-bearing cast, with the foot in mild equinus, extended to block great toe extension should be worn for 4 weeks. A neutral position weight-bearing cast should then be worn for an additional 2 weeks. Active flexion and extension of the hallux is permitted at 6 weeks, with gradual return to unlimited activity over the following 3 to 4 weeks.

In patients with closed FHL rupture, surgical repair or reconstruction has been recommended to alleviate pain and not necessarily restore IP joint flexion. With closed ruptures, it is recommended that an MRI scan or ultrasound be obtained to localize the disruption and to evaluate the amount of distraction. [164] Multiple surgical techniques have been described in the literature, and primary end-to-end repair is recommended when feasible. [767] [769] In cases in which significant distraction exists, techniques including a side-to-side repair of the proximal and distal stumps of the FHL tendon to the flexor digitorum longus tendon, [157] grafting of the defect with a tensor fasciae latae graft, [161] and a distal tenodesis of the distal FHL tendon to the flexor digitorum longus [162] have been used to restore some active IP joint flexion.

Treatment of FHL tendinitis is predicated on the stage at which it is diagnosed. In the acute phase, treatment includes anti-inflammatory medications and avoidance of activities that stress the FHL. Specifically, the individual may be allowed to continue dance workouts but should be instructed to avoid dancing *en pointe*. In most cases of tendinitis *without* triggering of the tendon, the pain will resolve within a matter of days to weeks. [774] [775] [776] [777] [778] Prevention of recurrences should focus on instructing the dancer to reduce the amount of turnout at the hips so that they are working directly over the foot, avoiding hard floors (a major contributing factor), and using firm, well-fitting toe shoes so that the foot is well supported and no additional strain is placed on the tendon. [775] [789]

When the tendinitis has been present for several months, a prolonged recovery period of up to 3 months should be anticipated. In these cases, a short period of immobilization (2 to 3 weeks) may be indicated. Chronic tendinitis is most often seen in relatively “tight” dancers, especially those with stiff feet and an incorrect pointe position. [775] [776]

Operative treatment should be considered when persistent synovitis or triggering of the FHL tendon prevents dancing *en pointe*. The primary procedure performed is release of the tendon sheath. Hamilton stated that this operation is rarely if ever indicated and observed that dancers often say that the presence of scar tissue from surgery is almost as incapacitating as the tendinitis before surgery. [775] [776]

In more recent reports, however, several authors have recommended exploration of the tendon and release of the tendon sheath in recalcitrant cases of flexor hallucis tendinitis. [763] [773] [774] [790] Garth explored the FHL in both ankles of a 21-year-old ballet dancer who was unable to return to dancing after 2 years of treatment including intermittent rest and anti-inflammatory medications. [169] At surgery, he found that both tendons were eroded beneath the retinaculum so that they were only 25% and 50% of their normal diameter. He used double strips of plantaris tendon to reinforce the FHL and released the flexor retinaculum. At follow-up 7 months after surgery, the patient had no pain and good FHL function but was not dancing *en pointe*. Cowell and colleagues explored the FHL tendon of an 18-year-old who had a 1-year history of tendinitis without triggering. [168] At surgery, he found calcified nodules within the tendon that did not allow the tendon to glide freely in its sheath. He released the tendon sheath and excised the calcific nodules. The patient was able to return to dancing *en pointe* 5 months after surgery.

Several studies have concluded that surgical treatment is indicated earlier in patients who have triggering of the FHL tendon compared with those who have pure tendinitis. [777] [779] [780] [791] Sammarco and Miller reported on two ballet dancers with bilateral triggering and locking of the FHL tendon. [174] All four tendons in these dancers were explored, and in every case, there was a fusiform enlargement of the tendon just distal to the flexor retinaculum at the medial malleolus. There were ruptures of the central fibers in the enlarged area of the tendon. These fibers, which were thickened and contracted, were encompassed by peripheral, intact fibers of the tendon. In 1984, Tudisco and Puddu reported similar surgical findings in a 25-year-old professional ballet dancer who had a 2-year history of bilateral triggering of his FHL tendon. [175] At surgery, the authors were able to demonstrate entrapment of the tendon within the flexor retinaculum by passively flexing the great toe.

In both of the aforementioned reports, release of the flexor retinaculum relieved the symptoms and allowed the tendon to move smoothly in its tract. Sammarco and Miller concluded that the active dancer requires at least 3 months of slow, progressive rehabilitation of the ankle before he or she will be able to return to dancing *en pointe*. [174] In contrast, Tudisco and Puddu’s patient, who began active range of motion at 3 days and walked unaided at 14 days, returned to dancing en pointe 40 days after surgery without problems. [175]

Operative treatment of dancers with tendinitis and posterior impingement or isolated posterior impingement requires individualizing the surgical approach. [776] [787] Hamilton stressed that a lateral approach to the ankle should only be used to treat isolated posterior impingement. [171] He advocated a medial approach for treatment of both FHL tendinitis and posterior impingement because he believed that the fibro-osseous tunnel of the FHL tendon cannot be released safely from the lateral side. [171] Marotta and Micheli recommended a similar approach. [182] They reported the results of 12 ballet dancers (15 ankles) that had excision of the os trigonum, through a lateral approach, for treatment of posterior impingement. There were two minor complications (one superficial wound infection and one transient tibial nerve neurapraxia). Both complications resolved without sequelae. At follow-up, 2 years after surgery, 8 (67%) still had occasional discomfort, but all 12 dancers returned to unrestricted dance activity. [182]

Recently, tendoscopy has been proposed as a possible treatment for FHL decompression. [792] [793] Keeling and Guyton performed a cadaveric study to evaluate the utility of FHL decompression at the posterolateral talar process. [189] They found that in 3 of 8 ankles, the tendon was injured during the release and that in no case was the sheath completely released. Additionally, the sural nerve and the lateral calcaneal branch of the sural nerve, as well as the first branch of the lateral plantar nerve, were closely associated to the tendon, and the portal locations placed these nerves at risk during endoscopic release. They thought that open release had a better reliability and less potential morbidity associated with it. [189] For more distal disease, Lui and Chow have shown that tendoscopy can be effective in the management of toe flexor tenosynovitis in a 2-year follow-up study of 3 patients with tenosynovitis and metatarsalgia. [188] No other reports in the literature could be identified to expand on these findings.

AUTHORS' PREFERRED METHOD

To date, the authors have not treated an athlete with a laceration of the FHL tendon; however, the operative protocol of Frenette and Jackson [10] and Scaduto and Cracchiolo [164] presented earlier appears sound and would be our approach to this injury.

Our preferred method of treatment of FHL tendinitis is based on the length of time the tendinitis has been present and whether there is triggering of the tendon. In the one professional ballet dancer treated by the senior author (JSK) for this problem, tendinitis of the FHL tendons of both ankles was present for several months before the onset of triggering of the tendons. In the left ankle, the triggering subsided after 3 weeks of rest—specifically, avoiding dancing *en pointe*—and the use of anti-inflammatory medications. In the right ankle, however, the triggering persisted and became so painful that the dancer was unable to rise onto or push off her right great toe. Thus, her right FHL was explored through a 5-cm incision that was located 2 cm distal to the tip of the medial malleolus (Fig. 25D-4). At surgery, it was evident that the peripheral fibers were intact and that there was a longitudinal tear and a bulbous enlargement of the tendon at the distal edge of the flexor retinaculum (see Fig. 25D-4 **A**). After release of the tendon sheath, the central degenerated fibers and scar tissue were excised, and the tendon was repaired with a running 2-0 absorbable suture (see Fig. 25D-4 **B**). The sheath was left open, and the tendon glided smoothly throughout a full range of motion of the great toe. The subcutaneous tissues and skin were closed with interrupted 2-0 absorbable sutures and a running 3-0 nonabsorbable Prolene suture, respectively. The ankle was immobilized for 2 weeks to facilitate wound healing, and floor exercises were initiated 1 week later. The patient was allowed to do a complete workout with the exception of going *en pointe* 8 weeks after surgery. Three months after surgery, she was able to resume *en pointe* positioning without fatigue or pain. At follow-up 1 year later, the patient was dancing *en pointe* without difficulty.

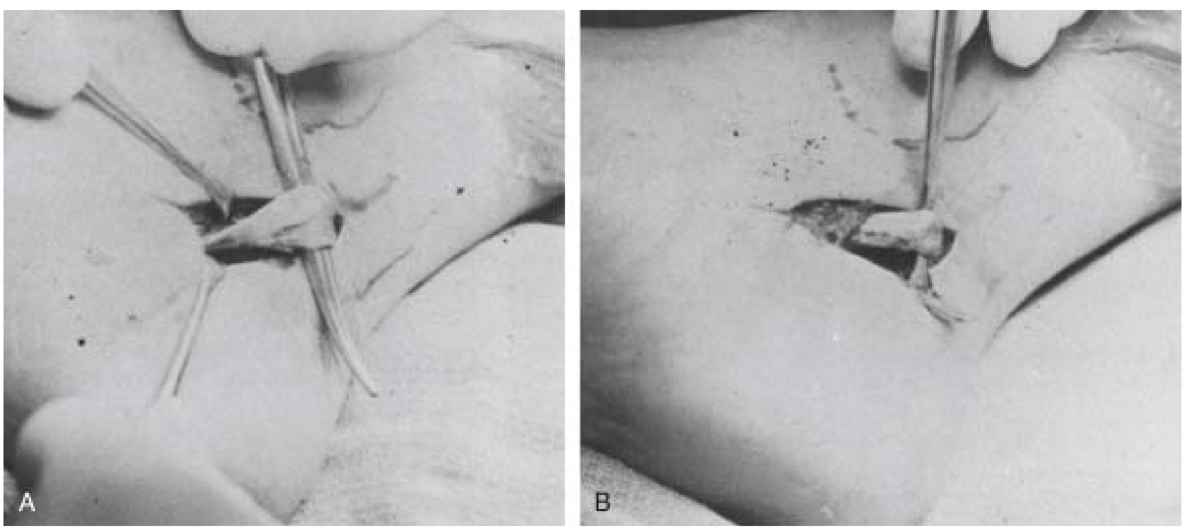

Figure 25D-4 Flexor hallucis tendinitis. At surgery, a longitudinal tear and a bulbous enlargement of the flexor hallucis longus tendon were found just distal to the flexor retinaculum (**A**). With passive flexion of the toe, the enlarged portion of tendon became entrapped within the retinaculum. The degenerated fibers and the central scar tissue were excised, and the tendon was repaired (**B**).

PERONEAL TENDON INJURIES

Peroneal tendon injuries include ruptures, tendinitis, and acute or chronic subluxations. Subcutaneous ruptures of the peroneus brevis tendon (PBT) occur more often than peroneus longus tendon (PLT) tears, but both are uncommon injuries. A limited number of cases of complete traumatic peroneus longus tendon ruptures have been reported in the literature. [640] [795] [796] [797] [798] [799] [800] [801] [802] [803] [804] Two involved soccer players, [797] [798] two occurred in runners, [799] [805] one occurred in a collegiate football player, [197] and one in a walker. [195] All six of these individuals twisted the ankle and felt a distinct “pop” at the lateral aspect of the injured ankle. Most of the cases occurred in older, less active individuals. One was attributed to overgrowth of the fibula after osteomyelitis of the tibia and a varus deformity of the foot, [191] and two occurred in middle-aged women (ages 48 and 58 years) who had twisted their ankles. [640] [795]

Disruption of the PLT due to a fracture of the os peroneum also has been reported. [806] [807] [808] [809] [810] [811] Most cases are the result of either repetitive inversion injuries or forced eversion of the ankle against resistance. Although PLT rupture with an associated os peroneum fracture is an uncommon occurrence, it should be included in the differential diagnosis of any patient with lateral ankle pain and instability and either of the aforementioned mechanisms of injury.

Although longitudinal and interstitial tears of the PBT have been well described in the literature, [800] [801] [812] [813] [814] [815] [816] [817] [818] [819] complete ruptures of the PBT are less common, with a limited number of cases reported in the literature. [800] [801] [802] [820] [821] [822] Most of the PBT tears occurred in combination with partial or complete PLT tears. [801] [822] [823] [824] One occurred in a young, healthy collegiate athlete, [197] two were discovered at the time of surgical treatment of the PLT tears, [800] [821] and another was found when the PBT was to be used for correction of chronic lateral ankle instability. [215]

Pertinent Anatomy

The PLT and PBT cross the ankle joint within a common fibro-osseous tunnel and synovial sheath. The tunnel is created laterally by the superior peroneal retinaculum; medially by the posterior talofibular, calcaneal fibular, and posterior inferior tibiofibular ligaments; and anteriorly by the posterior surface of the lateral malleolus. The superior peroneal retinaculum (SPR) is a strong fibrous band that blends with the periosteum of the lateral surface of the lateral malleolus and is rarely torn when subluxations or dislocations of the peroneal tendons occur. [220] Eckert and Davis, who described the retinaculum origin from the periosteum of the posterior ridge of the fibula, found no actual tears of the SPR in their 73 repairs for subluxating peroneal tendons. [220] Davis and colleagues delineated the different patterns of insertions and the relationship of the superior retinaculum to the peroneal tendons and the lateral ankle ligaments. [221] They observed that the SPR has at least one insertional band that parallels and inserts just lateral to the calcaneofibular ligament and that these two structures are at maximal length in ankle dorsiflexion. They concluded that inversion ankle sprains that cause calcaneofibular ligament ruptures may often produce concomitant SPR injuries. [220] Numerous studies have documented the relationship between lateral ankle instability and peroneal tendon disease. [800] [806] [809] [811] [813] [814] [816] [817] [818] [823]

The inferior peroneal retinaculum is continuous (anteriorly) with the fibers of the inferior extensor retinaculum and is attached posteriorly to the lateral surface of the calcaneus. Some retinacular fibers attach to the peroneal tubercle of the os calcis, forming a septum that creates separate compartments for the peroneus longus and brevis tendons. A thin vincula-like structure that runs between the PLT and PBT and is dorsally attached to the dorsolateral aspect of the fibula has also been described. [222] The peroneal tendons have a common synovial sheath until they separate under the inferior retinaculum. At that juncture, each tendon is enveloped by a distal progression of the common sheath. Proximal to the lateral malleolus, the PBT lies deep (medial) to that of the peroneus longus. Distal to the lateral malleolus, the PBT lies anterior to the peroneus longus. Knowledge of the changing spatial relationships of these tendons is important if one uses the PBT for lateral ankle reconstructions.

The PBT inserts into the tuberosity of the lateral aspect of the base of the fifth metatarsal. The PLT exits the posterior compartment of the inferior retinaculum, crosses the lateral surface of the cuboid bone, and traverses the plantar surface of the cuboid in a tunnel created by a groove in the bone and the lateral plantar ligament. The tendon inserts into the lateral side of the base of the first metatarsal and the medial cuneiform. There is an abrupt change in direction of the PLT at two points: first at the tip of the lateral malleolus and second at the distal (lateral) edge of the cuboid bone. In both of these locations, the tendon is thickened, and at the point of contact with the smooth surface of the edge of the cuboid, there usually is a sesamoid within the tendon composed of fibrocartilage or bone. The blood supply of the PLT and PBT comes from the peroneal artery, and microvascular studies have documented that there are no critical zones of hypovascularity within these tendons that may be attributed to rupture. [828] [829]

Relevant Biomechanics

Several studies have noted the critical role that subluxation of the tendons over the posterolateral edge of the fibula plays in the development of peroneal tendon tears. [813] [814] [816] [818] Sobel and associates, in their study of 15 fresh-frozen specimens, demonstrated that PBT splits were uniformly located at the sharp posterior lateral edge of the fibula. [213] They concluded that PBT tears were the result of either acute or repetitive mechanical trauma caused by subluxation of the tendon over the posterior corner of the lateral malleolus. Subsequent studies have supported this proposed mechanism of injury. [813] [816] [817]

Clinical Evaluation

Diagnosis of complete peroneal tendon disruptions is difficult and often is delayed. [640] [795] [797] [798] [799] [802] [805] In one soccer player, the diagnosis was not suspected and was discovered 10 hours after injury when an emergency fasciotomy was performed for an acute peroneal compartment syndrome. [192] The PLT was found to be avulsed from the muscle belly, and subsequently the entire muscle became necrotic and was excised. Seven months after the injury, function of the peroneus brevis and the anterior compartment muscles was normal, and the patient had returned to playing soccer. In the other case involving a soccer player, the individual sought treatment 8 months after his injury because of recurrent, painful swelling at the outer aspect of the heel. [193] At that time, he had full range of motion and full strength except for eversion, which was painful and weaker than normal. Ultimately, the tendon was explored because of his persistent tenosynovitis. At surgery, a bulbous enlargement and transverse tear of the tendon were discovered just below and behind the lateral malleolus. The enlarged portion of the tendon was excised, and the two ends of the peroneus longus were sutured to the adjacent tendon of the peroneus brevis. Two months after surgery, the patient had returned to full activity.

The two runners with this injury were initially treated for lateral ankle sprains. The diagnosis of a complete PLT tear was made 4 weeks after injury when both sought a second opinion because of persistent pain and difficulty walking. [799] [805] One collegiate football player was found to have complete ruptures of the PLT and PBT tendons after being treated for 2 years with taping and bracing for a lateral ankle sprain. [197]

In the two middle-aged women with this injury, the diagnosis of a PLT disruption was made 1 or more years after the injury. One was treated successfully with 3 weeks of immobilization, [35] and the other was treated with end-to-end repair of both the peroneus longus and brevis tendons. [190]

It is apparent from the reports cited earlier that rupture of the PLT is an uncommon injury and that diagnosis is delayed because the physical findings (pain and swelling about the lateral malleolus) usually imply a lateral ankle ligament injury. In addition, swelling often makes determination of point tenderness more difficult. Thus, one should look for increased hindfoot varus and test for pain with active eversion in all individuals with lateral ankle pain and tenderness proximally along the peroneal tendons.

Although fractures of the os peroneum without disruption of the tendon occur [791] [830] (and are the subject of a subsequent chapter), the association of this sesamoid fracture with PLT disruption warrants further emphasis. The os peroneum, which is present in 5% to 26% of individuals, is found within the PLT at the level of the plantar and lateral aspects of the cuboid. [206] One mechanism of injury, sudden forceful dorsiflexion of the foot and a violent reflex contraction of the peroneus muscles, is the same mechanism that produces a dislocation of the peroneal tendons. [807] [810] The other mechanism proposed (repetitive inversion injuries) is more subtle, [806] [809] [810] and thus recognition of this injury requires knowledge of its existence, careful examination of the lateral aspect of the foot, and standard radiographs that include anteroposterior, lateral, and calcaneal views.

The differential diagnosis for this combined injury includes lateral ankle ligament sprains, avulsion fractures at the insertion of the PBT, peroneal tendon subluxation, and tenosynovitis of the peroneal tendons. With a fracture of the os peroneum and disruption of the tendon, there is point-specific tenderness over the area of the sesamoid, and either wide separation of the fracture fragments or proximal migration of the sesamoid is evident on the standard radiographs. [805] [808] [809] [810] [811]

PERONEAL TENDINITIS

Far more common than complete disruption of one or both of the peroneal tendons are interstitial tears and chronic tendinitis. The evaluation and management of chronic tendinitis and tenosynovitis of the peroneal tendons have been addressed in only a limited number of studies. [667] [717] [795] [796] [815] [822] [831] Scheller and colleagues postulated that peroneal tendinitis is due to the pulley action and abrupt change in direction of these tendons at the lateral malleolus. [62] They suggested that the oblique stress placed on the tendon at the lateral malleolus produces an area of decreased vascularity that predisposes the tendon to injury. Burman concluded that tenosynovitis of the peroneal tendons occurs in three specific locations: posterior to the lateral malleolus (peroneal sulcus), at the peroneal trochlea (a tunnel created by the attachment of the calcaneal fibular ligament to the peroneal tubercle of the os calcis), and at the plantar surface of the cuboid. [210] Burman also concluded that stenosing tenosynovitis of the peroneus longus and brevis occurs mainly in individuals with well-developed peroneal tubercles, and noted that anatomic studies have documented that 37% to 44%

of humans have prominent peroneal tubercles. He operated on 6 of 25 patients who had chronic peroneal tenosynovitis at the level of the peroneal tubercle. In all six cases, the tendons were found to be intact, but the fibrous sheath and fibrous septum of the retinaculum were thickened. After resection of the stenotic sheath, the patients experienced relief from their peroneal pain. [210]

Trevino and coworkers reported on four cases of peroneal tenosynovitis that were treated surgically. [112] They noted that the site of involvement was either the peroneal sulcus (two cases) or the peroneal trochlea. Roggatz and Urban described three cases of peroneal tendinitis that were located distal to the peroneal trochlea at the lateral border or plantar surface of the cuboid bone. [226] Peroneal tendinitis also can be caused by congenital anomalies of the peroneal tendons. [782] [832] [833] [834] Of the four cases reported to date, three had an anomalous bifurcation or trifurcation of the PBT, [782] [832] [834] and one had an accessory peroneal muscle and tendon. [228] In all cases, resection of the anomalous tendon and muscle produced good results.

The evaluation and management of interstitial tears (splits) of the peroneus longus and brevis tendons have been addressed in an increasing number of studies. [800] [812] [813] [814] [815] [816] [817] [818] Interstitial tears of the PLT can be classified as acute or chronic. Acute tears occur after a single event, after which symptoms develop. With chronic tears, there is no acute traumatic episode, and symptoms are insidious in onset and develop over a long period. [228]

Acute tears may occur as primary injuries or may occur in conjunction with other ankle injuries. Bassett and Speer reported on eight athletes who sustained longitudinal ruptures of PLT (five patients) or PLB (three patients) as a result of a plantar flexion-inversion ankle injury. [208] Sammarco and Brainard chronicled the evaluation and management of 14 cases of PLT tears, of which 8 had acute onset of symptoms. [228] In these two series of patients, delay in diagnosis was common, ranging from 7 days to 6 months, and from 7 to 48 months, respectively. [813] [833] In Bassett and Speer's study, all eight athletes noted persistent lateral ankle swelling, popping, and retrofibular pain after their initial sprain. [208] They all complained of a subjective feeling of ankle instability but were able to compete in their sport by taping the ankle. On physical examination, all ankles were stable, and all had retrofibular tenderness, synovial sheath fullness, and palpable retrofibular popping with active foot rotation. None had evidence of clinical subluxation of the peroneal tendons. They and others [814] [835] have concluded that splits in the PLT and PBT that go unrecognized may be the cause of residual pain after ligamentous repairs of lateral ankle injuries.

In the past decade, longitudinal tears (split lesions) of the PBT have been reported with greater frequency; however, despite the recent focus on this problem, fewer than 50 cases have been reported in the literature. [813] [814] [816] [817] [818] These studies stress that chronic PBT tears are frequently overlooked or misdiagnosed and are often found in cases of chronic ankle laxity. A great majority (90%) of patients with PBT tears have swelling and tenderness over the peroneal tendons. Tenderness with resisted eversion of the foot also is a common finding, [816] [818] and re-creation of the retromalleolar pain during an anterior drawer test usually indicates a tear. [209] Sobel and colleagues coined the term *peroneal tunnel compression test* to describe the maneuver by which the superior peroneal retinaculum is compressed against the posterior ridge of the fibula while the patient forcefully dorsiflexes and everts the foot against resistance. [213] The test is positive if this maneuver re-creates the patient's pain.

Imaging studies for evaluation of peroneal tendon tears have limited value. Standard radiographs of the ankle (including anteroposterior, lateral, and oblique views) usually are normal. [208] However, MRI has been found useful in differentiating longitudinal split tears in the peroneal tendons from other lateral ankle disorders. [230] Several studies have described the features and characteristics of peroneal tendon tears on MRI, [836] [837] [838] and some have attempted to define the accuracy of MRI. [816] [838] [839] Rosenberg and associates did a retrospective review of ankle MRI done on 27 patients who had clinical "evidence" of longitudinal PBT splits. [233] Seven (26%) of the 27 patients had surgery, and the tears were confirmed in all 7 cases. Yao and associates reported on 4 patients who had MRI performed before surgical exploration of the "peroneal tunnels." [234] Surgery and MRI revealed two PBT and three PLT splits. They concluded that axial MRI through the ankle and hindfoot can help distinguish tendinitis from longitudinal tendon splits. [234] Other studies, however, have noted a 27% false-negative rate for detection of peroneal tendon splits. [211] Thus, it appears that appropriate treatment of clinically diagnosed peroneal tendon splits should not be predicated on the findings on MRI until prospective studies have established its accuracy and better diagnostic criteria.

Ultrasound also has been recommended for the diagnosis of peroneal tendon tears. Grant and associates evaluated 60 peroneal tendons that had preoperative ultrasound examinations and found that the sensitivity, specificity, and accuracy of ultrasonography for detecting the 25 tears found at surgery were 100%, 85%, and 90%, respectively. [235]

Treatment Options

Successful treatment of peroneal tendon disruptions with associated fractures of the os peroneum has been achieved with 6 weeks of cast immobilization, [203] internal (suture) fixation of the sesamoid fracture, [205] and excision of the sesamoid fragments and repair of the tendon. [805] [806] [809] [811] Peroneal tendon disruptions *without* an associated fracture of the os peroneum have been variably treated by immobilization, [831] [833] tenodesis of the proximal and distal tendon ends to the peroneus brevis, [796] [798] [809] [841] suture to the cuboid, [200] and primary repair of the tendon. [795] [811] Although operative treatment with primary repair [795] [811] and tenodesis [796] [798] [809] [841] have produced uniformly good long-term results, the outcome of nonoperative treatment, reported to be successful in one case, [203] has not been well documented. [640] [791]

Initial treatment of peroneal tendinitis should include decreased activity, immobilization, anti-inflammatory medications, and protected (crutch) ambulation. Some authors state that they have never found it necessary to operate on a patient with peroneal tendinitis for relief of symptoms. [667] [831] Most published reports, however, have found that operative treatment that includes release of the peroneal tendon sheath, along with débridement and repair of the tendon, often is necessary to alleviate the patient's symptoms. [717] [813] [814] [815] [816] [817] [842] All the methods of surgical treatment reported include débridement of the tendon and either primary repair of the tendon, tenodesis of the tendon to the PLT, or a tendon graft. [646] [717] [732] [779] [814] [843] [844] Krause and Brodsky based the method of surgical treatment on the cross-sectional area of tendon involvement. [211] If 50% or more of the tendon remained after débridement of the damaged portion (grade I tear), they repaired the tendon. If less than 50% remained (grade II), proximal and distal tenodesis of the PBT to the longus tendon was performed. [211] All of the aforementioned studies emphasize that these procedures must be augmented by a peroneal tendon stabilization procedure when there is concomitant, underlying subluxation of the peroneal tendons. One study concluded that the reduction of pressure on the tendon that occurs with deepening of the peroneal groove may be advantageous for treatment of recalcitrant peroneal tendinitis. [240] The results of surgery from either single or combined procedures have been uniformly good or excellent. [717] [806] [814] [815] [816] [817] [842]

Recently, tendoscopy for treatment of peroneal tendon disorders has been advocated. [237] Van Dijk and Kort reported the results of peroneal tendoscopy on nine consecutive patients. [237] The indications for the arthroscopy were diagnostic (five), snapping sensation (two), removal of exostosis (one), and partial tendon rupture (one). After surgery performed through a two-portal technique, three of four patients with adhesions resected and one patient with symptomatic prominent peroneal tubercle removed were symptom free, and one patient had a longitudinal rupture of the PBT successfully repaired. [237] Scholten and Van Dijk reported similar results in 23 patients who underwent peroneal tendoscopy. [241]

Authors' Preferred Method

Our preferred method of treatment is based on the duration, site, and cause of the peroneal tendinitis. It has been our experience that most cases of tendinitis are secondary to either subluxation of the peroneal tendon or chronic lateral ankle instability. We have treated operatively four patients with longitudinal tears in their peroneal tendons. They were managed as has been outlined for the treatment of posterior tibial tendinitis. The central degenerated fibers were excised, and the peripheral intact fibers were repaired. It was not necessary to perform a tenodesis in any of these cases. However, we agree with those who note that pure peroneal tendinitis can usually be treated successfully with a nonoperative regimen of decreased activity, immobilization (when necessary), anti-inflammatory medications, protected ambulation, and a stretching and strengthening program.

SUBLUXATION OF PERONEAL TENDONS

Traumatic dislocation of the peroneal tendons, either acute or chronic, is an uncommon injury. When the condition is recurrent, however, it causes disabling pain and usually prevents participation in sports. Although Monteggia originally described this injury in a ballet dancer in 1803, only sporadic reports were published on this topic in North America before 1960. [847] [848] [849] [850] Subsequently, there has been an abundance of publications on this topic that have elucidated the incidence, mechanism of injury, and difficulties of establishing the diagnosis. [667] [790] [812] [825] [843] [845] [851] [852] [853] [854] [855] [856] [857] [858] [859] [860] [861] [862] [863] [864] [865] [866] [867] [868] [869] [870] [871] [872] [873] [874] The injury most commonly occurs in skiing, [851] [852] [863] [875] [876] and it is estimated that complete peroneal dislocations occur in 0.5% [35] and sprains of the peroneal retinaculum in 2.5% of all skiing injuries. [246] Moritz reported that four to five complete peroneal dislocations occur each year at Sun Valley. [271] Although skiing accidents are the primary cause of this injury, soccer, basketball, football, ice skating, and a variety of other activities have precipitated this injury as well. [667] [812] [851] [856] [858] [860] [864] [865] [877]

Peroneal tendon dislocations can be classified as acute or chronic. Several studies have noted that acute injuries are often misdiagnosed as lateral ankle sprains and that owing to the lack of early diagnosis and proper treatment, recurrent dislocations invariably occur. [645] [812] [853] [858] [859] [868] [877] The aforementioned reports indicate that this injury occurs in all age groups, the youngest and oldest patients being 13 and 54 years, respectively.

Pertinent Anatomy

Several authorities have stated that patients sustaining peroneal dislocations usually have predisposing anatomic abnormalities, which include a shallow peroneal groove and an incompetent or absent peroneal retinaculum. [825] [858] [867] [868] Anatomic texts state that the posterior surface of the lateral malleolus usually has a definitive sulcus to accommodate the peroneal tendons. Several studies have shown, however, that the contour of the posterior surface of the distal fibular is quite variable. Edwards found that 11% of cadaveric fibulas had a flat posterior surface with no lateral edge, and 7% had a convex posterior surface. [242] In the remaining 82%, a groove was present but was never more than 2 to 3 mm deep. She concluded that the lateral ridge of the fibula, when present, was not of sufficient proportion to maintain the peroneal tendons in their groove. She also noted that the length of the sulcus was not measurable because of its gradual inception and that the width of the sulcus ranged from 5 to 10 mm. [242] Szczukowski and associates performed CT on eight normal ankles and on two patients with peroneal dislocations. [262] They found that the eight normal ankles had well-defined, concave fibular grooves and that the two patients with dislocations had convex peroneal grooves. Other authors have found convex or shallow grooves in 40% to 100% of patients operated on for recurrent dislocations. [825] [858] [864] [868]

Eckert and Davis examined the distal fibulas of 25 amputated legs and found that the posterior surface was essentially flat; when a groove was present, it was so variable in depth, length, and orientation that it was unrealistic to attempt a description of its “usual” anatomy. [220] They also documented that there is a discrete ridge of dense collagen and elastin fibers that extends along the posterior lip of the lateral malleolus. [220] This ridge, which is most pronounced near the tip of the fibula, tapers proximally and is usually 3 to 4 cm in length. The superior retinaculum lacked strong connections to this dense strip of collagen and attached mainly to periosteum of the lateral aspect of the malleolus. Based on 73 cases of acute dislocations that were surgically explored, they concluded that rupture of the retinaculum occurs rarely, but instead the retinaculum strips the periosteum from the lateral malleolus or avulses a thin cortical shell. These authors based their conclusions on the aforementioned series of patients in whom they found no tears of the retinaculum, and they described three patterns of injury (grades I, II, and III). In grade I injuries, the retinaculum and periosteum were elevated from the lateral malleolus, and the tendons lay between the periosteum and the bone. This lesion, which occurred in 51% of their patients, also has been described by Das De and Balasubramaniam. [246] In grade II injuries (33% of their patients), the distal 1 to 2 cm of the fibrous ridge was elevated along with the retinaculum, and in grade III injuries (16% of their patients), a thin cortical rim of bone was avulsed along with the retinaculum, the fibrous lip, and the periosteum. [220] The peroneal tendons lay between the two exposed cancellous surfaces of the bone. In grade I injuries, the peroneal tendons, once reduced, were unstable only when placed under tension; in grade II and III injuries, the tendons were very unstable after they were reduced. [220] Oden described a similar classification scheme based on 100 cases, but defined a type II injury as a tear rather than an elevation of the retinaculum from the fibula, and added a type IV injury, an avulsion of bone from the posterior rather than anterior fibular insertion site. [258]

Anatomic variations in the superior peroneal retinaculum also have been cited as a factor contributing to dislocation of the peroneal tendons. Congenital absence of this structure has been reported, and acquired laxity may occur in individuals subject to habitual pronation of the foot such as jockeys. [848] [850] [876] [878] Stover and Bryan concluded that primary sprains of the retinaculum may result in permanent laxity, predisposing the tendons to dislocation. [245] They also concluded, however, that although the absence or convexity of the posterior fibular groove or the laxity of the retinaculum contributes to dislocation of the tendons, the presence of these factors is not a prerequisite for the occurrence of a dislocation.

Relevant Biomechanics

The most commonly described mechanism of injury is sudden, forceful, passive dorsiflexion of the ankle with the foot in slight eversion. [667] [790] [825] [843] [850] [858] [865] [866] This results in a sudden, strong reflex contraction of the peronei, which are the dynamic lateral stabilizers of the ankle. This sequence of events produces the various grades of injuries summarized previously. Eversion of the ankle tends to tense the tendons against the retinaculum, making dislocation easier if there is preexisting retinacular laxity. Eversion of the ankle also tenses the calcaneofibular ligament, which decreases the width of the fibro-osseous tunnel and forces the tendons against the retinaculum. [850] [868] The position of dorsiflexion causes a maximal change of direction of the tendons at the lateral malleolus and, in combination with eversion, also thrusts the tendons against the superior retinaculum. The key component of the injury, however, is the forceful reflex contraction of the peroneal muscles.

Clinical Evaluation

In acute injuries, the athlete usually notes a popping or snapping sound or sensation at the posterolateral aspect of the ankle, accompanied by intense localized pain behind the fibula and above the joint line. [220] There is usually diffuse lateral swelling and ecchymosis. The pain usually subsides rapidly and becomes relatively mild. If the individual is examined within hours of injury, one usually finds swelling localized over and just posterior to the lateral malleolus. If the swelling is not extensive, the peroneal tendons often can be palpated over the lateral malleolus, or their displacement can be appreciated when the ankle is dorsiflexed (Fig. 25D-5). When there is an associated fracture of the lateral malleolus, the bone fragment may be palpable in the deep tissues. In most cases, there are no signs of injury to the anterior talofibular, fibular calcaneal, or anterior tibiofibular ligaments as is commonly seen with lateral ligament injuries.

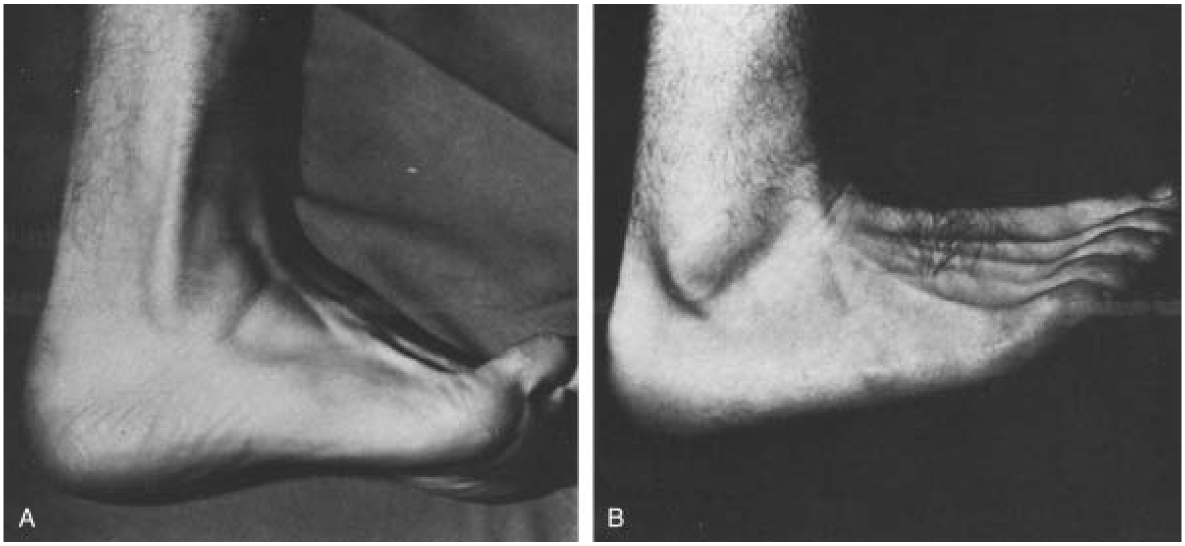

Figure 25D-5 Subluxation of the peroneal tendons. With dorsiflexion and eversion of the right foot (**A**), the peroneal tendons dislocate anteriorly over the lateral malleolus. The dislocated tendons reduce when the foot is inverted (**B**).

If the injury is evaluated 4 or more hours after the injury, the lateral aspect of the ankle often is markedly swollen, and palpation reveals only tenderness at the posterior lip of the lateral malleolus. At this juncture, the swelling may obscure frankly dislocated tendons and makes the diagnosis difficult. One must therefore attempt to stress the retinaculum by eliciting active eversion of the foot with the ankle held in dorsiflexion. In patients with an acute subluxation, this test may produce apprehension or severe pain, or may show obvious dislocation, all of which are diagnostic of the problem. Safran and associates advocate that this provocative testing be done with the patient prone and the knee flexed to 90 degrees. [260] They concluded that this position is a more comfortable position for the examiner and that dynamic instability is better visualized.

Because of the rapid resolution of symptoms and an initial diagnosis of an ankle sprain, most patients seek medical evaluation when chronic subluxation is established. Patients with recurrent subluxation complain of lateral ankle instability and pain. They also note a popping or snapping sensation in the ankle just before the ankle giving way. [667] [790] [843] [855] [856] [859] [861] [865] [866] [868] The snapping or popping sensation is noted particularly during such activities as jogging or walking on uneven ground.

Chronic subluxation may tend to occur with increasing frequency owing to progressive stretching of the residual lateral soft tissue structures. [207] Many authors state that they can reproduce the tendon subluxation in all their patients by having the patient contract the peroneal muscles with the foot in eversion. [667] [853] [858] [864] [865] [866] [868] Marti, however, stressed that one must examine the other ankle to identify those individuals with bilateral congenital subluxation of the peroneal tendon. [253] In addition, DeHaven reported that one can reproduce the dislocation in only about 50% of patients with chronic dislocation. [274]

The differential diagnosis of this injury includes longitudinal tears of the peroneal tendons, sprains of the lateral ligaments of the ankle, and in children, Salter I fractures of the lateral malleolus. Often a longitudinal tear of one of the tendons will result in internal snapping of one tendon over another, mimicking a subluxation. [855] [860] Patients with chronic dislocation of the peroneal tendons do not have instability of the ankle or subtalar joints. Specifically, they usually have a negative anterior drawer test and no increased lateral laxity in comparison with the opposite ankle. Salter I fractures can be documented by obtaining radiographs of the opposite ankle and noting the location of the pain. In patients with acute and chronic peroneal dislocations, the pain is located along the posterior lip of the lateral malleolus and not over the anterior tibial-talar area.

Radiographic evaluation of this injury should include anteroposterior, lateral, and mortise views of the ankle. Stress views, to assess increased talar tilt and lateral ankle instability, may be performed as indicated. Radiographs will be diagnostic only in patients with grade III injuries, those with an associated rim avulsion fracture of the lateral malleolus. [850] [875] [876] [877] The fragment, which is 1 to 1.5 cm in length, is best seen on the mortise view. This fracture occurs in 15% to 50% of individuals sustaining peroneal tendon dislocations, [272] and when this finding is present, it is pathognomonic of a dislocation of the peroneal tendons. [790] [850] [853] [858] [864] [865] [868] [875] [876] [877] [880] MRI will give further information regarding interstitial tendinopathy, [837] [838] [839] and dynamic ultrasound imaging will demonstrate peroneal tendon subluxation. [276] However, these imaging modalities are not routinely necessary in cases of isolated peroneal instability.

Treatment Options

Nonoperative Treatment

Although most authorities agree that there is no place for nonoperative treatment of chronic dislocations, the treatment of acute injuries remains controversial. [667] [825] [850] [853] [870] [882] Stover and Bryan [245] stated that all acute cases should be treated conservatively with a well-molded cast and non–weight-bearing ambulation for 5 to 6 weeks. They reported on 5 patients treated in this manner, and all five had excellent results; however, 2 other patients in their series were immobilized for a shorter interval, and 6 of 10 patients treated by taping of the ankle in a neutral position for 4 to 6 weeks had recurrent dislocations. Thus, of a total of 17 acute peroneal dislocations treated nonoperatively, 7 ultimately needed surgical repair. [245] In contrast, Scheller and colleagues treated seven patients with taping and a lateral felt pad over the fibula, and all seven had recurrent dislocations. [62] Similarly, Escalas and associates [248] reported that 28 of their 38 patients with acute dislocations, who were treated by immobilization and a compressive bandage for several weeks, had recurrent dislocations. Eckert and Davis [220] noted that only 1 of their 7 patients who were treated with either adhesive strapping or casts for 4 weeks had a stable pain-free ankle, whereas four had recurrent dislocations.

McLennan reported on 16 athletes with subluxations or dislocations of the peroneal tendons. [277] Of the 6 patients with acute injuries (diagnosis within 1 week), 1 had immediate surgery, and 5 had 3 weeks of taping of the ankle with a crescent-shaped, laterally placed pad. Three (60%) of these 5 athletes later had operative correction because of recurrent dislocations. Ten athletes with subacute and chronic injuries (diagnosis at 2 weeks to 2 years) also were treated with the aforementioned regimen of taping. Four (40%) of these athletes ultimately had surgery. [277] McLennan concluded that although nonoperative treatment gives satisfactory results in more than 50% of the cases, operative treatment of acute injuries is indicated in athletes seriously involved in sports, particularly those with rim fractures. [277] All the aforementioned authors who have experienced failures with nonoperative treatment still recommend treating acute cases initially with a short leg, non–weight-bearing cast for a minimum of 4 weeks. [667] [853] [882] They reserve surgical treatment for patients who have subsequent dislocations.

Operative Treatment

The results of operative treatment of acute dislocations have been reported in only a limited number of studies. [825] [858] [877] In Eckert and Davis's series of 73 patients, grade I (retinaculum and periosteum elevated from lateral malleolus) and grade II (fibrous ridge and periosteum elevated) lesions were repaired by suturing the retinaculum to the fibrous ridge or the ridge and retinaculum to the malleolus, respectively. [220] Grade III injuries (rim fractures) were treated with open reduction and K-wire fixation. At follow-up 6 or more months after surgery, the tendons had redislocated in 3 patients, all with grade II injuries. Twelve additional patients experienced mild pain after vigorous activity, but none thought their symptoms warranted further treatment. [220]

Results of acute surgical repairs also have been reported by Marti [253] and Murr. [272] All three of Murr's patients had rim fractures of the lateral malleolus and were treated by open reduction and internal (suture) fixation. Within 1 year of surgery, all three patients had returned to skiing and were symptom free. [272] Marti reported on five patients with acute injuries, one of whom had a rim fracture, and concluded that to achieve the best results, acute injuries should be treated operatively with primary repair of the torn peroneal retinaculum. [253] At an average follow-up of 3½ years, all five patients were symptom free and had normal ankle motion. In all the aforementioned series of acute injuries, the patients were immobilized in a short leg cast for 6 weeks after surgery.

Although more than 18 surgical procedures have been proposed for treatment of recurrent dislocations, there are only five basic types of procedures. These are bone block procedures, [849] [858] [859] [880] [882] rerouting procedures, [790] [855] [864] reattachment of the retinaculum and reinforcement with local tissue, [677] [851] [856] [865] [871] [873] [880] [884] reconstruction of the superior peroneal retinaculum with tendon slings, [848] [853] [854] [860] [861] [866] and groove-deepening procedures. [843] [867] [868] [870] [872] [874]

The procedures that alter the osseous structure of the fibula are designed to contain the peroneal tendons either by deepening the peroneal groove or by augmenting the lateral osseous ridge of the fibula. Augmentation of the lateral border of the fibula usually is achieved with a bone block procedure. In the original procedure described by Kelly in

1920,[244] a “veneer-like graft, almost wholly composed of compact bone,” was created by two saw cuts (one sagittal and one horizontal) in the distal 2 to 3 cm of the fibula. The graft was then rotated posteriorly so that it overlapped the posterior cortex of the fibula by 5 to 6 mm and was fixed in its new position with two countersunk screws (Fig. 25D-6 **A**). Kelly subsequently described a modification that eliminated screw fixation by dovetailing the graft so that it was wider anteriorly and was held tightly in its bed when it was displaced posteriorly (see Fig. 25D-6 **B**).

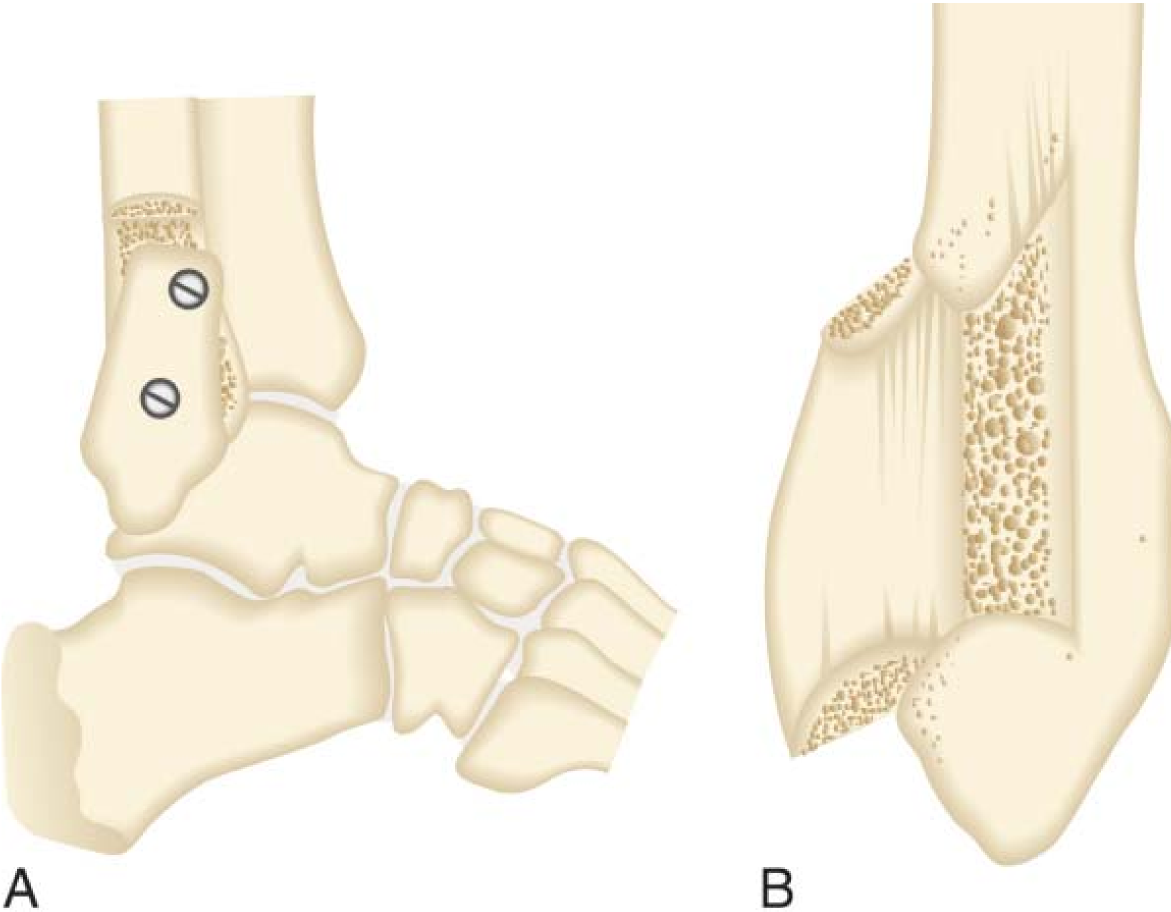

Figure 25D-6 Kelly’s bone block procedures. The original technique (**A**) is shown on the left, and the modified procedure (**B**) is shown on the right.

DuVries modified Kelly’s technique by using a wedge-shaped section of distal fibula that was 2 cm wide and half the depth of the lateral malleolus.[275] The wedge was displaced posteriorly 0.5 cm and held in place with a small screw or autogenous bone peg (Fig. 25D-7). Patients treated in this manner were immobilized in a short leg cast for 5 to 8 weeks.

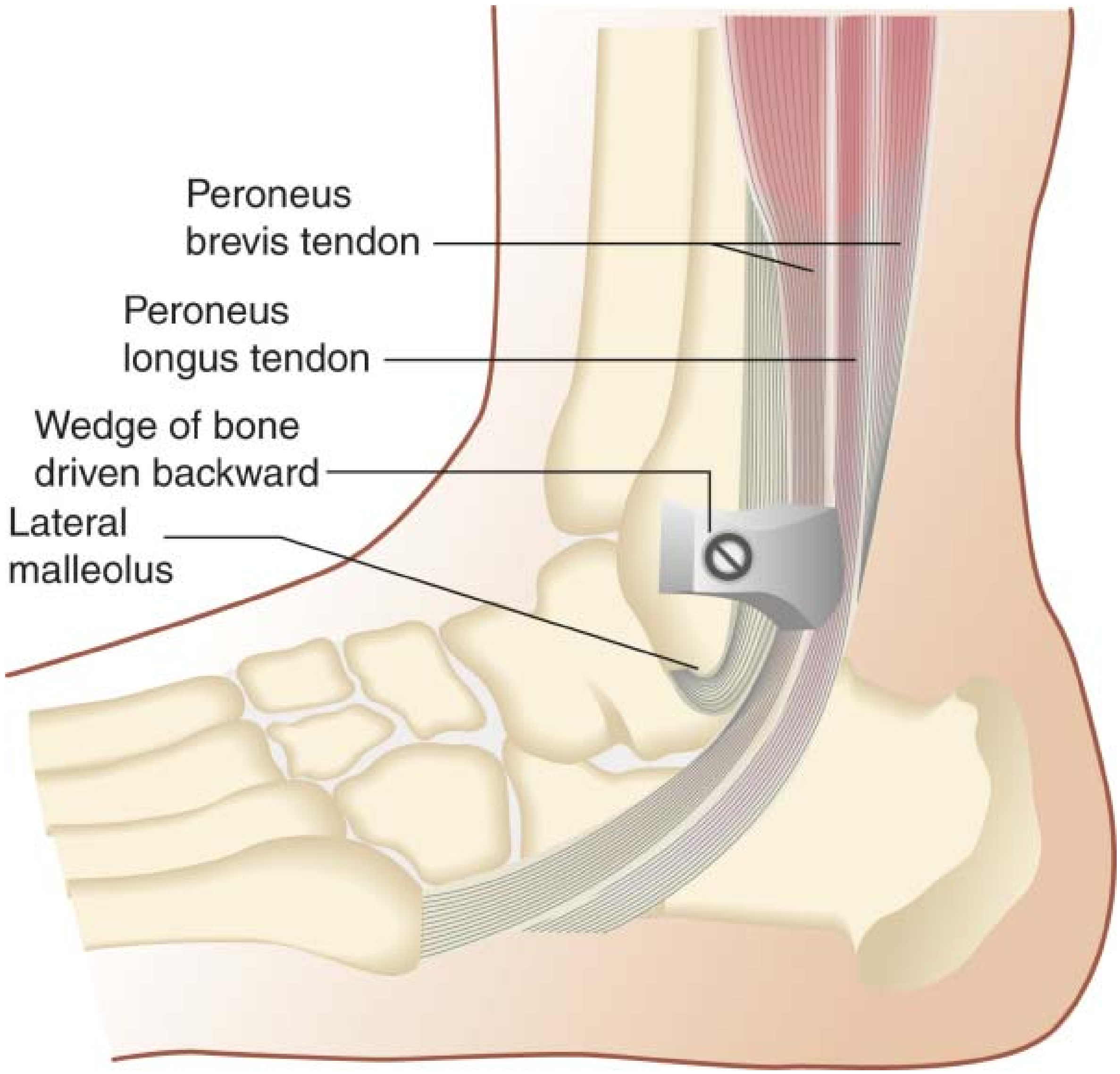

Figure 25D-7 The Du Vries modification of Kelly's bone block procedure.

The largest series of patients (11 to 12 cases) treated by the modified Kelly procedure were reported by Marti in 1977, [253] Micheli and colleagues in 1989, [278] and Mason and Henderson in 1996. [254] None of Marti's had redislocations 2 or more years after surgery, but two patients had crepitation of the tendons. He concluded that the crepitation was caused by insufficient posterior displacement of the bone block, which resulted in a shallow groove with a sharp inferior edge. In the more recent series of sliding, [278] or rotational, [254] bone block procedures, similar results have been reported.

In patients with a shallow, flat, or convex peroneal groove, the groove-deepening procedure described by Zoellner and Clancy appears to be an effective method of correcting the basic deformity. [263] With this technique, a 1 × 3-cm osteoperiosteal flap is elevated from the posterior aspect of the distal part of the fibula and lateral malleolus. The posterior medial border of the cortical flap is left intact to act as a hinge. The flap is elevated and swung posteriorly, and the cancellous bone from the posterior aspect of the fibula is removed to a depth of 6 to 9 mm. The osteoperiosteal flap is then tapped back into place, and the peroneal groove is deepened over a length of 3 to 4 cm (Fig. 25D-8). Zoellner and Clancy used this technique in 10 patients and stated that the results were excellent at an average follow-up of 2 years (range, 7 months to 3 years and 10 months). [263] In 6 of the 10 patients, however, the peroneal retinaculum was so tenuous that an additional (1 × 1 cm) periosteal flap was fashioned from the lateral surface of the malleolus and sutured to the medial part of the peroneal retinaculum. After surgery, the patients were immobilized in a short leg cast for 3 weeks and then in a short leg cast with an ankle hinge for an additional 3 weeks. Zoellner and Clancy recommended this technique because it (1) is technically easy to perform, (2) does not require metallic fixation, (3) corrects the basic deformity of a shallow peroneal groove, and (4) allows early motion because prolonged immobilization for union of bone or tendon is not needed. [263] Three subsequent studies have also reported excellent results with this technique in 17 additional patients, [812] [843] [867] and one study reported excellent results with a similar but indirect groove-deepening procedure. [265]

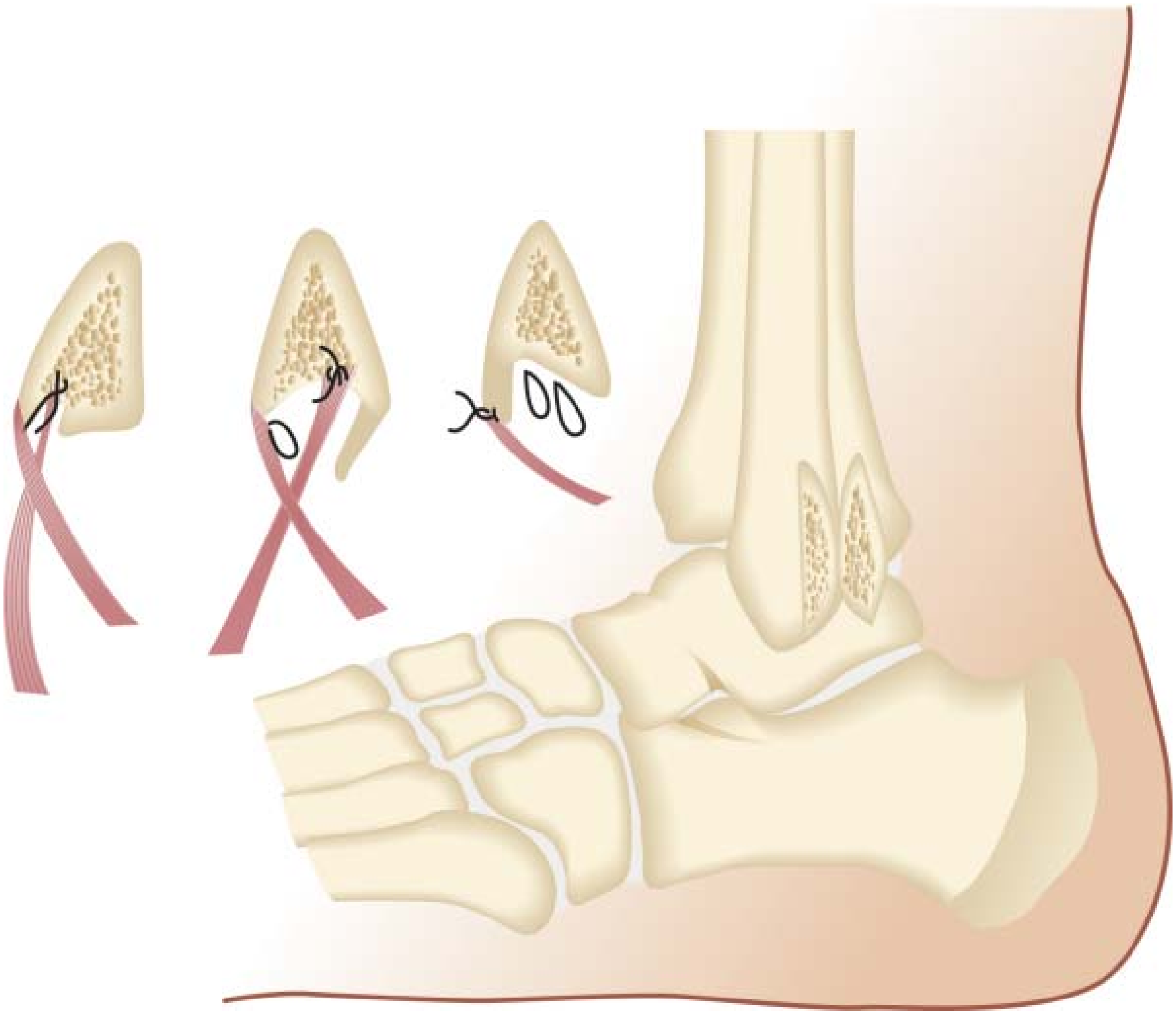

Figure 25D-8 Deepening of the peroneal groove through an osteoperiosteal flap.

Results of the various soft tissue procedures (e.g., rerouting, periosteal flaps, and tendon slings) generally are very good. To date, however, there has not been a controlled study that compares the results of the different procedures to determine whether one is better than another.

Rerouting of the peroneal tendons has been advocated by Sarmiento and Wolf [185] and by Poll and Duijfjes. [259] Sarmiento and Wolf reported one case in which the tendons were divided, repositioned under the calcaneofibular ligament, and then repaired with a Bunnell stitch. At 3-year follow-up, the patient participated in athletic activities and had had no further dislocations. [185]

In 1984, Poll and Duijfjes reported on 10 patients in whom the insertion of the calcaneofibular ligament was mobilized and lifted with a cancellous bone block from the calcaneus. The peroneal tendons were then brought under the ligament, and the bone block was replaced and fixed with a small cancellous screw. After surgery, the ankle was immobilized for 6 weeks in a short leg plaster cast, but weight-bearing was allowed after 2 weeks. Poll and Duijfjes reported excellent results in all 10 patients and recommended the procedure because it precluded scarring and adhesions of the peroneal tendons to the surrounding structures. [259] They also described and critiqued similar procedures proposed by Platzgummer and by Leitz. Platzgummer divided the calcaneofibular ligament near the fibula, and Leitz osteotomized the lateral malleolus and refixed it with Kirschner wires. [259] Poll and Duijfjes stated that the disadvantage of the first technique was that the integrity of the ligament was disturbed, and a disadvantage of the second technique was that the osteotomy was near the articular surface of the fibula. Thus, these procedures increased the risk for adhesions forming between the tendons and the ligament or bone. Recently, Harper reported a good result in one patient who had transfer of both tendons under the calcaneofibular ligament after detachment of the ligament from the fibula. [250]

The use of a tendon sling to reconstruct the peroneal retinaculum was first described by Jones in 1932 (Fig. 25D-9). [243] He reconstructed the retinaculum in a 22-year-old football player with a strip of tendon that was fashioned from the Achilles tendon at its calcaneal insertion. The tendon slip, which was 21⁄2 inches in length and about 1⁄4 inch in width, was passed through a transverse drill hole in the fibula, 1 inch above the tip of the lateral malleolus, looped posteriorly, and sutured to the periosteum of the fibula and to the tendon slip itself. Jones stressed that the tendon slip should be anchored with the foot held in full dorsiflexion and supination. After surgery, the athlete was placed in a short leg cast for 6 weeks. At 6 weeks, he was permitted to return to full activity and subsequently returned to football without symptoms 3 months later.

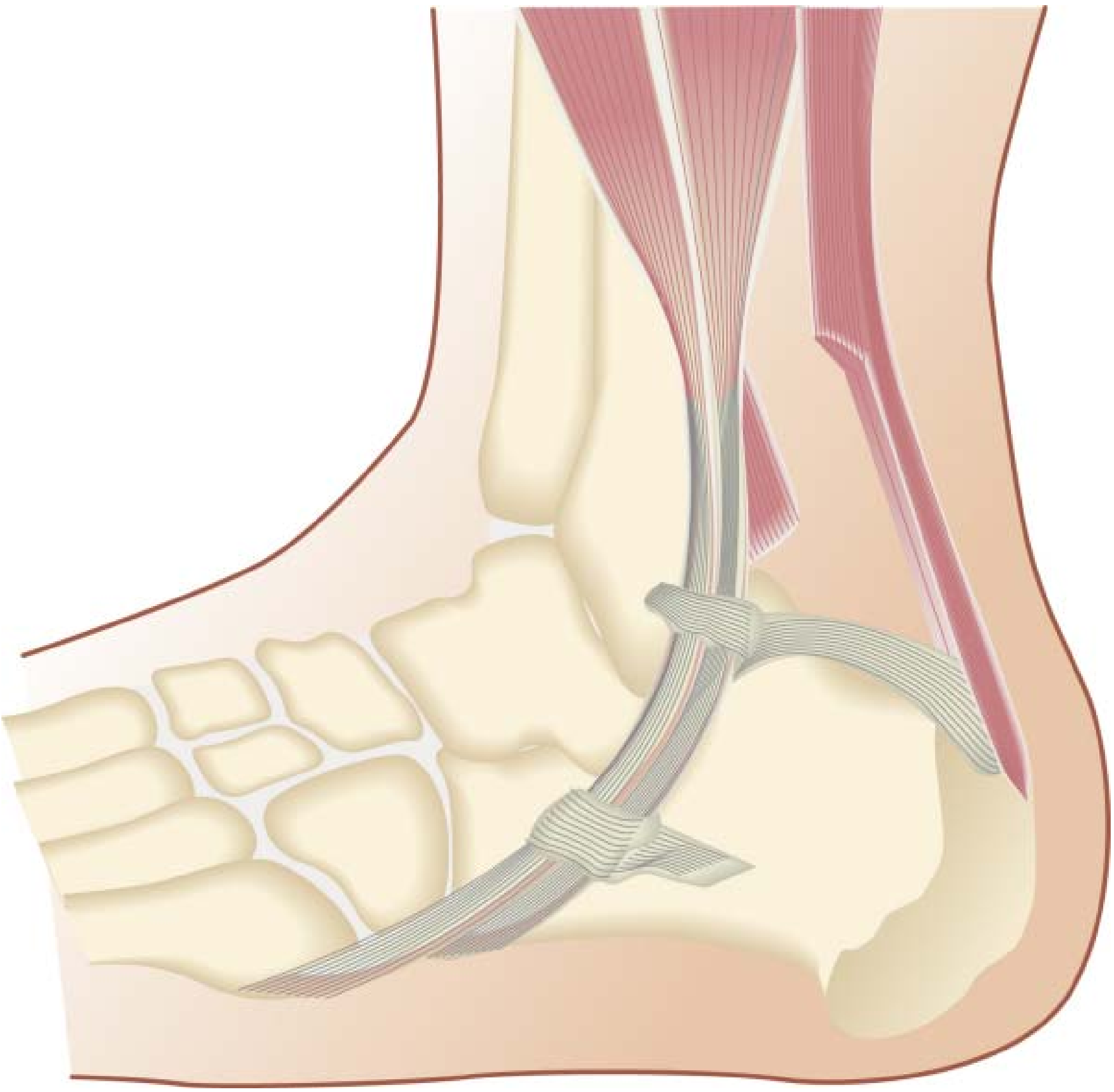

Figure 25D-9 The Jones (tendon-sling) procedure.

The largest series of patients treated with the Jones procedure was reported by Escalas and colleagues in 1980. [248] They performed the procedure in 28 patients, 15 of whom were followed for an average of 6.8 years (range, 3 to 11 years). Fourteen of the 15 patients had excellent results and returned to sports activities after an average of 4.2 months. One patient reported instability of the ankle, but no instability could be demonstrated on physical examination. Three of the 15 patients had a moderate decrease in inversion of the hindfoot, and four lost up to 7 degrees of dorsiflexion. The authors noted that minor loss of motion occurred despite the fact that the tendon slip was sutured in all patients with the foot held in maximal dorsiflexion.[248]

Tendon slings have also been created from a portion of the PBT[261] and by transposition of the peroneus quartus tendon.[256] Stein used a free graft consisting of 50% of the diameter of the tendon of the peroneus brevis and anchored it through two drill holes to the fibula at a point 1.5 cm proximal to the inferior tip of the lateral malleolus.[261] The graft was passed through the distal drill hole in an anteroposterior direction, looped over the peroneal tendons, and passed through the proximal medially placed hole in a posterior-to-anterior direction. The free tendon was then sutured to itself with sufficient tension to restrain anterolateral subluxation of the tendons. Stein described the use of this technique in a 17-year-old quarterback who was placed in a short leg cast for 6 weeks after the operation. The athlete was able to resume full sports-related activities 3 months after the operation and was asymptomatic when examined 3 years later. Stein believed that his procedure was superior to that of Jones[243] because the latter procedure had two disadvantages: (1) it weakened the Achilles tendon and (2) restoring full ankle motion was a problem because the slip of tendon was left attached to the calcaneus.

Mick and Lynch[256] reported a case in which the peroneus quartus was used to reconstruct the superior and inferior peroneal retinaculum. The peroneus quartus, which is estimated to occur in 13% of the population, originates on the posterior surface of the fibula and inserts on the calcaneus. They rerouted this tendon anteriorly over the peroneus longus and brevis tendons and placed it in a 5-mm deep oblique groove created in the anterior aspect of the lateral malleolus. The periosteum and soft tissue on either side of the groove were sutured over the transferred peroneus quartus tendon. At follow-up 1 year later, their patient was performing all required duties as a firefighter and was asymptomatic.[256]

Reconstruction of the peroneal retinaculum with an osteoperiosteal flap was conceived and described by Watson-Jones in 1955.[279] With this technique, a flap of periosteum (1.0 × 1.35 cm) is elevated from the distal fibula. The posterior periosteum and soft tissue structures are left intact. The flap is reflected posteriorly and sutured to the remaining portion of the superior retinaculum and the fascia of the flexor muscles of the great toe. The tendon sheath of the peroneal muscles is not opened, and the groove is not deepened. After surgery, the patient is placed in a short leg cast for 4 to 6 weeks and then rehabilitated until he or she has gained full range of motion and full strength.

The results of this procedure have been reported in only a limited number of studies. [677] [851] [873] Scheller and colleagues performed this procedure in seven patients and subsequently described their follow-up at a minimum of 15 years.[62] All seven patients had excellent results and returned to their preinjury sporting activities. In contrast, Das De and Balasubramaniam noted that two of three patients treated by them in this manner had recurrent dislocations and poor results.[246] They concluded that the failures occurred because the procedure did not address the primary disease involved. They operated on seven subsequent patients with recurrent dislocations and noted that in all cases the periosteum was stripped from the lateral malleolus but remained in continuity with the superior retinaculum. The detached periosteum extended forward to the anterior margin of the lateral malleolus and down to its tip, creating a false pouch into which the peroneal tendons easily dislocated. They suggested that this injury was similar to that found in Bankart's lesion in recurrent dislocations of the shoulder. They repaired the avulsed periosteum by scarifying the outer surface of the lateral malleolus and then suturing the periosteal flap to the lateral malleolus through the drill holes in the lateral edge of the peroneal groove. Their results in all seven cases were excellent at a minimum follow-up of 2 years.[246] In 1998, they published the results of 21 patients treated as outlined previously. They reported that 18 had good results and had returned to their preinjury level of activity. The three fair results were due to painful scars or neuromas.[251] Maffulli and colleagues performed anatomic repairs of the superior peroneal retinaculum on 14 patients with chronic, recurrent peroneal tendon subluxations. At follow-up of 38 months, none had experienced recurrent subluxations, and all had returned to their normal activities.[266]

Subluxation of the peroneal tendons within the peroneal sheath has been described by McConkey and Favero,[255] Harper,[250] and Raikin and collegues.[264] McConkey and Favero reported on a 28-year-old runner who, 5 years after a plantar flexion inversion injury, complained of intermittent pain and clicking in the area of the peroneal tendons during jumping and pivoting activities. There was no evidence of anterior subluxation of the peroneal tendons, but the patient was able to reproduce the painful clicking of the tendons with dorsiflexion and eversion of the foot. Direct compression over the tendons eliminated the clicking. When nonoperative treatment was unsuccessful, the peroneal tendons were surgically explored under a local anesthetic. At surgery, it was evident that dorsiflexion and eversion of the foot caused the peroneus brevis to rise out from under the peroneus longus and to displace posterolaterally. This maneuver produced the click that the patient had been experiencing. They corrected this problem by taking strips of the retinaculum and passing them under the peroneus longus but over the peroneus brevis. At follow-up 4 years later, the patient had returned to athletic participation, including basketball, without recurrence of the symptoms.[255]

Harper described two cases of subluxation of the tendons within the peroneal groove. One occurred insidiously in a 13-year-old girl, and the other developed in a 26-year-old man after several inversion injuries to the ankle.[250] Both were treated successfully, one with tenodesis of the brevis tendon to the longus after failure of a tendon-sling procedure, and the other with rerouting of the tendons. [250]

Raikin and colleagues described 14 patients in whom preoperative ultrasound demonstrated intrasheath peroneal tendon subluxation during dorsiflexion and eversion of the ankle. [264] At surgery, two types of intrasheath subluxation were found. In 10 patients, the subluxation occurred when the intact tendons switched their normal anatomic positions, and the peroneus longus came to lie deep to the peroneus brevis tendon. In the other 4 patients, there was a split in the peroneus brevis tendon through which the longus tendon subluxated. All 14 patients were treated with a peroneal groove–deepening procedure with a retinacular reefing and had excellent results. [264]

Authors' Preferred Method

The authors' experience parallels that of Eckert and Davis and of Murr, who concluded that the results of closed methods of treatment are disappointing and doomed to failure. [825] [877] Thus, our preferred method of treatment for acute dislocations is to reduce the tendons and tape the ankle with a felt pad over the perineal groove to allow the athlete to attempt to complete the current season. At the end of the season, a reconstruction of the retinaculum and deepening of the groove (as detailed later) are performed.

One author has treated one athlete who presented with dislocated peroneal tendons that could not be reduced by closed methods. The injury had occurred 1 month before my evaluation of the athlete. During that interval, the individual had been examined by two physicians who, despite the mother's insistence that the tendon was dislocated, failed to recognize and diagnose the injury. This individual sought another opinion because he was unable to jog or participate in sports owing to pain, weakness, and lateral ankle instability. This athlete was surgically treated with the method outlined later for chronic dislocations.

The authors' surgical treatment of chronic dislocations includes deepening the retromalleolar groove and reconstructing the superior peroneal retinaculum. Deepening of the peroneal groove is achieved using the method described by Zoellner and Clancy. [263] In this technique, a 5- to 7-mm J-shaped incision is made over the posterior aspect of the lateral malleolus along the course of the peroneal tendons. The superior and inferior retinaculi are incised, and the tendons are freed from their sheath and retracted anteriorly over the lateral malleolus. A small drill bit is then used to define the borders of an osteoperiosteal flap (4 cm in length and 1 cm in width) in the peroneal sulcus of the distal fibula and lateral malleolus. A small osteotome or an oscillating saw with a 3⁄8-inch blade is used to connect the drill holes along the superior, lateral, and inferior margins of the flap. The periosteum of the posterior medial border of the flap is left intact to act as a hinge. The flap is then raised and swung posteriorly to allow access to the cancellous bone beneath it. The cancellous bone from the posterior aspect of the fibula is removed to a depth of 8 to 10 mm. One should also remove the cancellous bone that remains on the posterior aspect of the flap and the cancellous bone under the intact medial edge of the sulcus to prevent the flap from springing open when it is tapped back into place. The flap will usually hold firmly in place when it is tapped back into position. If the flap does not stay in place, it can be held in its new bed with sutures that are passed through drill holes in the flap and the anterolateral cortex of the fibula.

The peroneal tendons are replaced in the new groove, and the ankle is put through a full range of motion. If the tendons do not remain well seated and show a tendency to subluxate, the groove should be deepened further by removing more cancellous bone. We also routinely reconstruct the superior retinaculum with a periosteal flap (1 cm [2]) fashioned from the lateral surface of the malleolus, hinged on its posterolateral side, and sutured to the medial part of the peroneal retinaculum.

Although we have not found it necessary to perform additional procedures, we would not hesitate to augment the lateral edge of the fibula and further deepen the groove by using the modified bone block procedure described by Kelly. [244] The subcutaneous tissues are closed with a running 2-0 absorbable suture, and the skin is closed with a running subcuticular 3-0 nonabsorbable suture.

After surgery, the patient is placed in a short leg boot for 2 weeks. At that juncture, the running subcuticular suture is removed, and the patient is allowed to bear full weight as wound healing and the patient's pain dictate. Three weeks after surgery, the patient is instructed to take off the boot and begin dorsiflexion and plantar flexion exercises 3 to 4 times per day. Immobilization is discontinued 6 weeks after surgery, and the patient begins further range of motion and strengthening exercises. The criteria for return to sports participation are the achievement of full range of motion and full strength, and completion of a running program.

The running program is initiated when the patient has a full range of motion and strength equal to 80% of that of the contralateral extremity. The athlete begins by jogging one quarter of a mile and then, if there is no pain or limp, progressing to 1 mile. At that time the athlete is instructed to perform six 40-yard "sprints" sequentially at half, three-quarters, and full speed. Athletes are allowed to increase their speed only when they have had no pain or limp at slower speeds. They subsequently repeat the same routine, alternately "cutting off" the injured and uninjured ankle every 10 yards. Most athletes are able to obtain a full range of motion and normal strength and to complete the running program within 12 to 16 weeks of surgery.

ACHILLES TENDON INJURIES

Injuries of the Achilles tendon include peritendinitis, tendinosis, and partial or complete ruptures. Several authors report that peritendinitis of the Achilles is the most common overuse syndrome seen in sports medicine clinics. [630] [885] [886] [887] [888] [889] Although the true incidence of peritendinitis and tendinosis in athletes is not known, Achilles tendon pain accounted for 6.5% to 11% of the complaints of runners who were examined for lower extremity problems, [885] [886] and it has been reported in a variety of other sports such as soccer, football, tennis, volleyball, basketball, badminton, and handball. [628] [631] [888] Similarly, although there have been several studies of partial ruptures of the Achilles tendon, the true incidence of this phenomenon also has not been documented. [890] [891] [892] [893] [894] Reported estimates of the prevalence of Achilles tendinitis, however, are 11% in runners, [242] 9% in dancers, [290] 5% in gymnasts, [291] 2% in tennis players, [293] and less than 1% in football players. [292] In the various published reports to date, the mean age of patients with peritendinitis and tendinosis ranged from 24 to 30 years, and the youngest and oldest were 16 and 52 years, respectively. [885] [888] [890] [895] [896] [897] [898]

Basically, Achilles peritendinitis and tendinosis are overuse phenomena and are the result of accumulative impact loading and repetitive microtrauma to the tendon. [627] [628] [631] [885] [886] [889] In Clement and colleagues' series of 109 runners with Achilles tendinitis, the average distance run per week for males was 34 miles (range 12 to 70 miles) and for females was 24 miles (range, 10 to 50 miles). [280] There are, however, both intrinsic and extrinsic factors that predispose an athlete to these injuries. Intrinsic factors include areas of decreased vascularity, [626] [899] aging and degeneration of the tendon, [627] [629] [632] and anatomic deviations such as heel-leg or heel-forefoot malalignment, [626] [886] [900] and poor gastrocnemius-soleus flexibility. [885] [887] [889] Clement and colleagues proposed that a varus position of the heel or supination of the forefoot produced functional overpronation of the foot during running. He and others have concluded that this type of mechanical deformation may result in a whipping action in the tendon and increased friction between the tendon and the peritendinous tissues. [627] [885]

Extrinsic factors that predispose an athlete to tendinitis include a sudden increase in training intensity, interval training, change of surface (soft to hard), fluoroquinolone antibiotics (e.g., levofloxacin), and inappropriate or worn-out footwear. [885] [888] [900] [900] [901] [902] McCrory and coworkers performed a discriminant analysis of significant variables to determine causal factors associated with Achilles tendinitis. [299] Their study revealed that plantar flexion peak torque, touch-down angle, and years running were the strongest discriminators between runners afflicted with Achilles tendinitis and runners who had no history of overuse injury. [300] Although complete ruptures most often occur in middle-aged persons after a specific precipitating event, partial ruptures occur in younger individuals (20 to 30 years of age) who have reached their highest level of performance. [631] [890]

Anatomic Considerations

The tendocalcaneus (Achilles tendon), which is the thickest and strongest tendon in the body, is formed about 15 cm above the heel at the confluence of the soleus and the gastrocnemius muscles. Although the tendon begins where the muscle belly of the gastrocnemius ends, it continues to receive muscle fibers on its anterior surface from the soleus almost to the malleolar level. The soleus and gastrocnemius components of the tendon can be separated and identified almost to the tendon's insertion at the calcaneus, which is about 1.5 cm distal to the tip of the superior tuberosity. Between its origin and insertion, the tendon twists laterally (about 90 degrees) so that the tendinous fibers from the gastrocnemius insert into the posterolateral and those from the soleus insert into the posteromedial aspect of the calcaneus. Proximal to the site of insertion of the tendon, the retrocalcaneal bursa is interposed between the tendon and the upper part of the bony surface of the calcaneus. The narrowest part of the tendon is 4 cm proximal to its insertion, and throughout its length, the tendon is separated from the deep muscles by areolar and adipose tissue. The small saphenous vein and sural nerve are located along its lateral side. Lagergren demonstrated that the Achilles tendon has an avascular zone 2 to 6 cm above its insertion into the calcaneus. [21] Stein and colleagues confirmed these findings using a new method with injection of radioisotopes. [301] They found that the intravascular volume of the middle part of the tendon (3 to 6 cm above the calcaneal insertion) was significantly lower than the proximal or distal parts of the tendon. This area of avascularity is the most common site of peritendinitis, tendinosis, and rupture of the tendon. [631] [714] [885] [888] [889] [906] [907] [908] [909] [910] [911] [912] Hastad and colleagues demonstrated through isotope clearance studies that there is deterioration in the nutrition of the tendon with advancing age. [294]

The pathologic changes in Achilles tendon injuries span a continuum of abnormalities but generally can be defined by three stages. In stage I, the tendon is normal, but there are inflammatory changes in the peritendinous tissue that include thickening and adherence of the sheath to the tendon and on occasion an exudative fluid around the tendon. This stage is most aptly described as peritendinitis. [628] [629] In two series of patients with Achilles tendinitis (a total of 188 tendons) in which the tendon was surgically explored, isolated peritendinitis was observed in 50% to 59% of the cases. [888] [889] The terms *tendinosis* and *peritendinitis with tendinosis* best describe the second stage, which is characterized either by degenerative and inflammatory changes within the tendon or by degenerative changes within the tendon and associated inflammation of the peritendinous tissue, respectively. Macroscopic examination of the tendon may reveal nodular thickening, areas of metaplastic calcification, or loss of the normal luster of the tendon at several locations. [629] [631] [632] [888] [891] [912] Microscopic examination reveals areas of mucoid degeneration, fibrinoid necrosis, and tearing of the tendon fibers. [629] [632] [888] [889] In general, tendinosis does not affect the whole tendon but may affect nonadjacent areas of the tendon. In the series of Nelen and associates [283] and Schepsis and Leach, [284] tendinosis (intrinsic degeneration) without macroscopic ruptures was observed in 20% and 33% of the operated tendons, respectively.

In the final stage of injury, macroscopic, visible disruption of the tendon occurs. These macroscopic tears occur in both the peripheral and central areas of the tendon. In the studies cited earlier, macroscopic tears and partial disruption of the tendon were found in 21% of the cases. [888] [889]

Clinical Evaluation

The predominant symptom of Achilles tendinitis is pain, which is localized to the area of the tendon 2 to 6 cm above its insertion. [627] [621] [885] [888] [889] [890] [913] [914] [915] In Nelen and associates' series of 91 patients who were operated on for chronic Achilles tendinitis, the extrinsic factors that contributed to the onset of symptoms were sudden changes of training in 31% of the patients, inappropriate training surface in 15%, inappropriate shoes in 7%, and direct trauma in 10%. [283] Clement and colleagues studied 109 runners and concluded that the three most prevalent causal factors were overtraining (75%), functional overpronation (56%), and poor gastrocnemius-soleus flexibility (38%). [280]

In the early stages of tendinitis, the athlete experiences pain only with prolonged running. The pain usually subsides rapidly with rest but may be exacerbated by climbing stairs. In the subacute stage, the pain is present at the start of a run and becomes worse with sprinting, sometimes forcing the athlete to stop or to cut down on sports activity. In the advanced stages, when there is tendinosis or a partial rupture, the athlete is unable to run and experiences pain at rest. The athlete may also complain of weakness and intermittent swelling. [289] Several authors think that the presence and severity of morning stiffness is a good standard by which to evaluate the seriousness of the condition. [887] [888] Nelen and associates found the following incidence of symptoms in the 91 patients whom they explored surgically for chronic Achilles tendinitis. [283] Thirty-five percent had pain only during sports activities, 65% had pain with daily activities, 86% had morning stiffness, 10% had pain at rest, and 35% had acute sharp pain, felt during a sprint or acceleration while running. [283] The aforementioned constellation of symptoms caused 54% of these athletes to decrease their sports activity and 46% to discontinue it. [283]

The physical findings in patients with Achilles peritendinitis and tendinosis include soft tissue swelling, local tenderness, and crepitus. [629] [887] [888] [900] [916] [917] [918] In the early stages, there is focal swelling and tenderness limited to a small area, usually no larger than the breadth of the palpating fingertips. The area of tenderness can be defined by squeezing the diseased tendon segment between the thumb and index finger. Crepitus, which is the result of an exudation around the tendon, is more commonly found in the acute stage and is accentuated by active dorsiflexion and plantar flexion of the foot. In all stages of tendinitis and tendinosis, including partial ruptures, Thompson's test is negative.

In chronic tendinitis, the area of tenderness is somewhat larger, and often the area is thickened and nodular. When one detects nodularity in the tendon, one should be suspicious of tendinosis or a partial rupture of the tendon. [283] In Denstad and Roass's series of 58 partial ruptures that were treated surgically, there was localized tenderness in 56 (97%), focal swelling in 53 (91%), but a palpable defect in the tendon in only 2 (3%). [285] In contrast, Skeoch examined 11 patients (16 tendons) with partial ruptures and concluded that palpation of the tendon generally reveals a partial defect. [289]

Differential diagnosis of patients with posterior heel pain includes retrocalcaneal bursitis and superficial tendo-Achilles bursitis or "pump bumps." [627] [887] The superficial bursa of the Achilles tendon lies between the tendon and the skin and becomes inflamed as a result of friction from the heel counter of a shoe. The retrocalcaneal bursa lies anterior to the tendon, and inflammation of this structure usually is associated with prominence of the posterosuperior tuberosity of the os calcis.

Standard anteroposterior and lateral radiographs of the ankle rarely show calcification of the soft tissues around the tendon or in the tendon itself; however, a prominent superior tuberosity of the os calcis will be evident on the lateral radiograph. [282] Lateral radiographs taken with a soft tissue technique may show localized swelling, particularly in the area of Kager's triangle, [25] which includes the retrocalcaneal bursa. [630] [890] The incidence of calcification within the Achilles tendon is not known because bone formation itself does not cause symptoms. [286] As noted in subsequent sections of this chapter, however, rupture of the Achilles tendon may occur through the ossified area [308] or in the tendon adjacent to the site of ossification. [286]

Studies on sonographic evaluation of athletes with chronic Achilles tendinosis have found that this modality accurately demonstrates both tendinitis and tendinosis (and associated microtears) of the tendon. [919] [920] [921] [922] Ultrasound also has shown that the increase in neovascularization seen after an eccentric training program correlates highly with the amount of improvement in outcome/VISA scores. [316]

Treatment Options

Nonoperative Treatment

Most cases of Achilles peritendinitis and tendinosis are successfully managed nonoperatively. The basic modalities of nonoperative treatment are rest or a decrease in the runner's weekly mileage, use of a 1⁄4- to 3⁄8-inch heel lift, oral nonsteroidal anti-inflammatory medications, use of an orthotic to correct excessive pronation, ultrasound, and stretching exercises. [885] [887] [888] [889] Total rest usually is not required, but the athlete should be instructed to avoid hill work and interval training. [887] [889] Clement and colleagues suggest a form of modified rest in which the athlete is allowed to participate in swimming and cycling activities but is not allowed to resume running for 7 to 10 days after the symptoms have subsided. [280]

Although several authorities advise the use of a 1⁄4- to 3⁄8-inch heel lift to decrease tension on the Achilles tendon, studies have differed on the effectiveness of these viscoelastic pads. [878] [923] In a blind-observer, random, prospective study of 33 subjects with Achilles tendinitis, Lowdon and associates found that subjects treated with ultrasound and exercises showed more profound improvement at both the 10-day and 2-month assessments than the two patient groups that received heel pads and exercises. [273] In a recent randomized, controlled clinical trial, Petersen and associates found that an AirHeel brace was as effective as eccentric training for treatment of chronic Achilles tendinopathy. [318]

The positive effects of ultrasound therapy on the repair of Achilles tendon injuries have been documented (in rats) by Jackson and coworkers. [287] They found that ultrasound increased the rate of collagen synthesis and the breaking strength of the Achilles tendon. The breaking strengths of treated tendons were significantly greater than those of untreated tendons 5, 9, 15, and 21 days after injury.

Stretching exercises have been proposed by several authorities as a key to nonoperative treatment of Achilles tendinitis. [885] [887] [888] [889] Leach and associates stressed that the common finding in patients with posterior heel pain due to Achilles tendinitis is loss of passive dorsiflexion. [282] There are two recommended methods of stretching, but with either method, the stretching should be slow and should be sustained for 20 to 30 seconds. With one method, the individual leans against a wall with the knees straight and the heel flat on the floor. With the other method, the involved foot is placed forward and both the knee and the ankle are flexed while the heel is held flat on the floor. It has been our experience that the former method of stretching affects the more proximal tendon and musculotendinous junction, whereas the latter method has a more profound effect on the insertion and distal aspect of the Achilles tendon.

Immobilization of the ankle for a period of 7 to 10 days may be indicated in individuals with severe acute symptoms. [282] However, a recent study found that continued sports activity during rehabilitation for the tendinopathy using a pain-monitoring model produced the same results as active rest. [319] Steroid injections, however, should be limited to the area of the retrocalcaneal bursa and should be employed only in patients with recalcitrant retrocalcaneal bursitis. [887] [889] There is a growing amount of evidence that steroid injections in and around the Achilles tendon may increase the risk for tendon rupture. [637] [887] [925] [926] Kleinman and Gross reported on three cases of Achilles tendon ruptures that occurred 2 to 6 weeks after the injection of steroids into the tendon. [321] In all cases, the rupture was the result of minor trauma, and the tears were transverse. They concluded that the tears were directly attributable to the steroid injection because rupture of the Achilles tendon usually is a sudden traumatic event, and in such patients, surgical exploration reveals that the tendon ends are shredded and interdigitated. In the three patients of Kleinman and Gross, however, the tears were transverse, and the tendon ends were rounded, with obvious preexisting degenerative changes. Astrom and Rausing analyzed 342 cases of partial ruptures in 298 patients who were operated on for chronic painful Achilles tendinopathy. [312] A logistic regression analysis of age, gender, physical activity, and preoperative steroid injections found that only preoperative steroid injections and male gender predicted a partial rupture. [312]

A study by Balasubramaniam and Prathap [320] supports the conclusions reached by Kleinman and Gross [321] and Astrom and Rausing. [312] They created central tears in the Achilles tendon of rabbits and then injected hydrocortisone acetate into one side and compared the effects of the injection with the rabbit's contralateral tendon. They subsequently sacrificed the animals at various intervals and observed the following changes on the injected side. Within 45 minutes there was separation of collagen bundles, and by 24 to 72 hours, areas of necrosis within the collagen were evident. At both 1 and 8 weeks after injection, they found that there was no evidence of repair of the Achilles tendon lesions, and in some animals, there was evidence of dystrophic calcification. [320]

Although most authors recommend nonoperative treatment of Achilles tendinitis, only Clement and colleagues have reported the results of a large series of patients (109 runners) treated in this manner. [280] Of these 109 athletes, 86 had complete follow-up, and 85 had good or excellent results (e.g., they had achieved preinjury training levels and either were symptom free or had minor symptoms on occasion). Clement and colleagues and other authorities, however, stress that athletes will not remain symptom free unless they understand the extrinsic factors that caused the injury. [885] [887] [889] [914] They should also be instructed in preventive measures, which include warming the Achilles tendon before running, applying ice for 10 to 15 minutes after running, and monitoring their shoewear, particularly the posterolateral aspect of the shoe. The most important preventive measure is stretching the posterior structures to prevent contractures and loss of passive dorsiflexion. [885] [887] [889]

Over the past decade, many new nonoperative methods of treatment have been advocated. These include extracorporeal shock-wave therapy (ESWT) [927] [928] [929] [930] heavy-load eccentric training, [921] [923] [931] topical medications, [932] [933] and intratendinous injections of proteinase inhibitors, [934] [935] sclerosing agents, [331] and dextrose. [331a]

Alfredson and colleagues prospectively studied the effect of heavy-load eccentric training in 15 athletes who had chronic Achilles tendinosis. [309] All 15 experienced fast recovery in eccentric and concentric calf muscle and returned to previous running activity symptom free. They concluded that there was little place for surgery in the treatment of chronic Achilles tendinosis located at the 2- to 6-cm level in the tendon. [309] However, Woodley and associates reviewed all of the controlled trials using eccentric exercise (EE) for the treatment of Achilles and other tendinopathies and concluded that there was a dearth of high-quality research in support of the clinical effectiveness of EE over other methods of treatment. [333]

The effectiveness of ESWT has been evaluated in several recent randomized, double-blind, placebo-controlled studies. [927] [930] Rassmussen and colleagues reported on the 4, 8, and 12-week results of 48 patients with chronic Achilles tendinopathy that were treated with either active ESWT or sham ESWT. [322] Significantly better results were seen in the intervention group at both 8 and 12 weeks. Rompe and colleagues reported on 50 patients with chronic, insertional Achilles tendinopathy. Twenty-five patients received eccentric training, and 25 patients received repetitive low-energy ESWT. At the 4-month primary follow-up, all of the outcome measures (e.g., VISA-A scores, pain ratings) were significantly better in the group that received ESWT. They concluded that eccentric training as applied in their study gave inferior results to ESWT for the treatment of chronic, insertional Achilles tendinopathy. [325]

Operative Treatment

Operative treatment should be considered when a comprehensive nonoperative treatment program of several months' duration has failed and the athlete is not willing to alter or abandon the precipitating sports activity. Surgical treatment is required in about 25% of athletes with Achilles tendon overuse injuries. [334] Although the surgical procedure performed should depend entirely on the disease found at the time of the operation, there are basically four distinct surgical procedures. These include release of the Achilles tendon sheath; excision of degenerated segments of tendon and side-to-side repair; excision of degenerated tissue and reconstruction of the tendon with fascial flaps from the gastrocnemius; and excision of the retrocalcaneal bursa and ostectomy of the superior tuberosity of the os calcis. [628] [887] [888] [889] [890] [891] [915] [916] [917] [918] [919]

In cases of pure peritendinitis, it is recommended that the sheath of the Achilles tendon be released from the musculotendinous junction to the insertion of the tendon. [628] [887] [888] [889] [900] Adhesions between the tendon and the sheath should be released but only on the dorsal, medial, and lateral sides of the tendon. Circumferential dissection of the tendon will damage the anterior vascular supply of the tendon and cause excessive scarring. [885] [887] [888] [889] Several authors also make a number of vertical slits in the tendon to "vent" the tendon and encourage ingrowth of new blood vessels. [627] [628] [629]

Endoscopic and ultrasound-guided percutaneous tenotomies also have been advocated for the treatment of chronic Achilles tendinopathy. [922] [940] Testa and associates reviewed the 3-year results of 63 patients who had ultrasound-guided percutaneous longitudinal tenotomy of the Achilles tendon. [317] At that juncture, there were 35 excellent, 12 good, 9 fair, and 7 poor results. Nine of the 16 patients with fair or poor results underwent an open exploration of the Achilles tendon 7 to 12 months after the percutaneous procedure. They concluded that percutaneous ultrasound-guided tenotomy should be considered in patients with recalcitrant Achilles tendinopathy. However, they noted that in patients with diffuse or multinodular tendinopathy, an open exploration with stripping of the paratenon and multiple longitudinal tenotomies may be preferable. [317]

Maquirriain and coworkers reported results of seven patients with chronic Achilles tendinopathy who had endoscopic peritenon release, débridement, and longitudinal tenotomies performed. [335] According to their scoring system, all seven patients improved after surgery from an average score of 39 points preoperatively to 88 points postoperatively. The only complications were a minor hematoma and edema that resolved spontaneously. Endoscopic Achilles tenodesis also has been described for the treatment of chronic insertional Achilles tendinopathy. [336]

In cases with focal tendinosis and partial rupture of the tendon, the diseased area of the tendon should be excised and the tendon repaired by side-to-side suture of the remaining normal tendinous fibers. [888] [889] [890] One must carefully inspect and palpate the tendon throughout its length because nonadjacent areas of tendinosis are common. The tendon should be opened in zones that have lost their normal luster and in areas of nodularity. However, a recent study has documented that normal-appearing areas adjacent to Achilles tendon lesions also will have histologic and biochemical degenerative changes. [337] In addition, a recent study has shown that patients with focal intratendinous lesions have poorer surgical results than those with only peritendinous adhesions. [338]

In cases in which excision of the degenerated tissue disrupts the continuity of the tendon, one should reconstruct and reinforce the tendon with a turn-down flap of fascia from the gastrocnemius. In these cases, a modified Lindholm [339] repair, using only one flap of gastrocnemius fascia, will cover the defect. An FHL transfer also can be used for repair of large defects caused by the excision of degenerated segments of the tendon. [340]

When it is evident preoperatively that the patient has a retrocalcaneal bursitis, the bursa should be excised, and, if prominent, the posterosuperior tuberosity of the os calcis should be removed. [888] [889] Ostectomy of the superior angle of the os calcis should begin just superior to the insertion of the Achilles fibers at an angle of 45 degrees to the long axis of the tendon. After excision of the fragment, any rough edges should be removed with a rasp or rongeur. [887] [889] The foot should then be put through a full range of motion to make sure that all areas of bony impingement have been excised. Leitze and associates have reported that endoscopic decompression of the retrocalcaneal space, including bursectomy and excision of a Haglund spur, produces results equal to or better than open techniques. [341]

Overall results of the aforementioned operative procedures were rated as good or excellent in 84% to 100% of the patients in the various series published to date. [887] [888] [889] [890] [894] [900] [916] [917] [918] In several studies, the results of specific procedures were reported. [888] [889] [916] [918] In Nelen and associates' series of 143 tendons that were surgically explored, release of the tendon sheath alone produced good or excellent results in 89% of the 93 tendons with peritendinitis. Excision of the degenerated tissues and side-to-side repair produced good or excellent results in 73% of the 26 tendons with tendinosis, and turned-down gastrocnemius flaps produced good or excellent results in 87% of the 24 tendons with more extensive tendinosis. [283] Complications from the aforementioned procedures included six cases of skin edge necrosis, two superficial wound infections, and one case of phlebitis.

Schepsis and Leach reported the results of 45 patients who underwent surgical exploration. [284] In the 28 patients who had release of the sheath (15 cases), excision of areas of calcification (4 cases), or débridement of partial tears and repair of the tendon (9 cases), the results were good or excellent in 89%. In the 11 patients who had excision of the retrocalcaneal bursa and a partial ostectomy of the os calcis, the results were good or excellent in 71% of the cases. In the 6 remaining cases, a release of the tendon sheath, a retrocalcaneal bursectomy, and partial ostectomy of the os calcis were performed. All 6 patients had good or excellent results. They concluded that the higher percentage of unsatisfactory results in those treated for retrocalcaneal bursitis alone was due to technical errors. Specifically, either an inadequate amount of bone had been removed or concomitant adjacent Achilles tendinitis had not been appreciated at the time of the initial operation. In a subsequent long-term follow-up study that included an additional 21 patients (66 total), Schepsis and coworkers reported satisfactory results in 75% and 86% of the patients with retrocalcaneal bursitis and insertional tendinitis, respectively. [313] They also reported that the highest percentage of satisfactory results (89%) were obtained in the paratendinitis group, and the lowest (67%) in the tendinosis group. [313]

In a recent study and the largest to date, Paavola and colleagues reported the results and complications of surgical treatment of Achilles tendon overuse injuries in 432 consecutive patients. [311] There was a total of 46 (11%) complications. Sixteen complications (35%) were classified as major (14 skin edge necroses, 1 new partial rupture, 1 deep vein thrombosis), and 30 (65%) were considered minor (11 superficial wound infections, 5 seromas, 5 hematomas, 5 extensive scar or fibrotic reactions, and 4 sural nerve irritations). Fourteen (30%) of the 46 patients with complications had reoperations. They noted that every 10th patient treated surgically suffered a postoperative complication that clearly delayed recovery. [311] They suggested that these complications could be prevented through the meticulous adaptations in surgical techniques they describe.

Postoperative Management and Rehabilitation

In cases of peritendinitis in which only the sheath is divided, the postoperative protocols in the aforementioned series range from no immobilization [627] [900] [916] to cast immobilization for 2 to 6 weeks. [887] [888] [889] A recent study found no advantages in recovery of muscle strength after surgery with a short immobilization time (2 weeks) compared with a longer (6 weeks) period. [310] In individuals with tendinosis and side-to-side repairs of the tendon, a non–weight-bearing splint is worn for 2 weeks, and a short leg walking cast is worn for an additional 2 weeks. [888] [916] Some authors, however, recommend no immobilization even after side-to-side repairs of the tendon. [285] When a flap of gastrocnemius has been used to reconstruct the Achilles tendon, most authorities recommend immobilization for a period of 5 to 7 weeks. [887] [888] [889] [918]

Stretching and strengthening of the Achilles tendon are initiated after immobilization has been discontinued. Patients then progressively increase their activity from walking and swimming to bicycling, jogging, and running. After a tendon reconstruction, they usually are able to jog within 8 to 12 weeks of surgery, but full recovery takes 5 to 6 months. [887] [888] [889] [916] [917]

AUTHORS' PREFERRED METHOD

Initial treatment of Achilles peritendinitis and tendinosis always is conservative and focuses on control of pain and inflammation, correction of functional malalignment, and rehabilitation of the gastrocnemius-soleus muscle-tendon complex. In athletes who present within 1 to 2 weeks of the onset of symptoms, a short course (7 to 10 days) of oral, nonsteroidal anti-inflammatory medications and 2 weeks of rest usually will allow them to return to running, symptom free. In addition, they are counseled about the extrinsic factors (e.g., errors in training) that may have caused their problem and about prophylactic measures (proper shoes, stretching) that can prevent Achilles tendinitis.

In most cases, however, the athlete seeks treatment only after he or she has attempted to "run through" the pain and has had to curtail or stop sports activities. In these cases, successful nonoperative treatment requires a more comprehensive program. First, the athlete must stop the precipitating activity (e.g., interval training, sprints, and so on) but is allowed to maintain aerobic fitness with alternative activities (such as swimming or cycling) if such activities can be performed without symptoms. This program of modified rest should be continued for 7 to 10 days after the Achilles tendon pain has subsided. Second, the athlete is started on a daily program of gastrocnemius-soleus stretching and strengthening exercises. Stretching is done in the manner outlined previously, and strengthening, initiated when pain has subsided, is achieved by performing toe raises with the heel hanging over the edge of a stair and moving the ankle through a full range of motion. Eccentric strengthening is accomplished with the toe raises by progressively increasing the speed of the heel drop. Third, functional components of malalignment are corrected with orthotics. Off-the-shelf flexible leather longitudinal arch supports (with or without a medial heel wedge) often are all that are needed to correct overpronation of the foot. Custom-made, rigid orthoses are obtained for patients with more severe or complex foot deformities.

Ten days to 2 weeks after symptoms subside, the athlete begins a gradual return to the preinjury level of activity with a running program staged to include a progressive increase in intensity and duration. The rate at which an athlete is able to return to preinjury training levels varies according to the severity and duration of each individual's symptoms. I use steroid injections only in athletes who have retrocalcaneal bursitis. In those individuals, the bursa (not the tendon) is injected with 1 mL of lidocaine (Xylocaine), 0.25% bupivacaine (Marcaine), and 40 mg of dexamethasone. After the injection, the athlete is instructed to refrain from all physical activity for a minimum of 3 days. At that juncture, he or she begins the rehabilitation program outlined earlier for nonoperative treatment of Achilles tendinitis.

Operative treatment is offered to athletes who have Achilles pain after completing a 2- to 3-month program that includes "modified rest," oral anti-inflammatory medications, use of orthoses (arch supports and heel lifts), and physical therapy (stretching and ultrasound). The operation is performed with the patient prone on the operating table, and the extent of the surgical procedure is dictated by the operative findings. In all patients, the tendon sheath is released throughout the length of the tendon, and the tendon is examined from its musculotendinous junction to its insertion on the calcaneus. In patients with peritendinitis and no macroscopic evidence of tendinosis, adhesions between the tendon and the tendon sheath are released, and vertical slits are made in the tendon in the area or areas that correspond with the patient's preoperative pain. If these vertical incisions reveal intrinsic degeneration of the tendon, the degenerated areas are excised, and a side-to-side repair of the tendon is performed.

In patients with partial ruptures or macroscopic evidence of tendinosis (nodularity, calcification, or loss of normal luster of the tendon), each area is opened through a longitudinal incision, and the abnormal segments of the tendon are excised. The method used to repair the tendon is predicated on the amount of diseased tissue removed. A side-to-side repair is performed when 20% or less of the width of the tendon is excised. When wider segments are removed, an end-to-end repair and a turned-down flap of gastrocnemius fascia are used to reconstitute the integrity of the tendon. Details of the surgical technique and postoperative protocol for this procedure are provided in the following section on repair of Achilles tendon ruptures.

Patients who undergo a release of the tendon sheath or a side-to-side repair of the tendon are put in a short leg, removable plastic boot for 2 weeks to facilitate wound healing. During that interval, they are allowed to bear weight on the extremity as tolerated. When the wound has healed, they begin the rehabilitation program outlined under the section on nonoperative treatment of Achilles

ACHILLES TENDON RUPTURES

Spontaneous ruptures of the Achilles tendon generally occur in healthy, vigorous, "young" adults with no previous history of calf or heel pain. [721] [908] [938] [948] [949] Although the exact incidence of this injury is not known, two studies determined the incidence in two larger populations of people. [949] [950] Nistor reported that over a 4-year period, 107 people (0.02%) from a population of 500,000 were treated at his hospital (the only one in the area) for this injury. [344] Cretnik and Frank found that during a 5-year period, 116 (0.04%) of 273,609 inhabitants of their region sustained this injury. [345] In published series on this injury, the average age of the patients ranged from 37 to 43.5 years, [631] [844] [908] [910] [911] [937] [948] [949] [950] [951] and 75% were between the ages of 20 and 49 years. [631] [844] [909] [911] In most series, most individuals with this injury were men in their third to fifth decades of life who were participating in recreational sports activities. [844] [862] [908] [909] [912] [937] [948] [949] [951] The left Achilles is ruptured more frequently than the right, [866] [952] possibly because of the higher prevalence of individuals who are right-hand dominant and thus push off with their left foot. The reported risk for subsequently rupturing the contralateral tendon ranges from 6% to 26%. [848] [950] In many series, basketball and racket sports accounted for more than half of the injuries, [909] [912] [937] [949] and in one large study, 58 (52%) of 111 patients were playing badminton at the time of their Achilles tendon rupture. [348] One report noted a high (14.3%) incidence of gout in their patients compared with the normal prevalence in the general population of 0.2% to 0.3%. [332]

Although ruptures of the gastrocnemius musculotendinous junction (so-called tennis leg) occur, they are extremely rare injuries, [64] and the Achilles tendon most commonly ruptures 3 to 4 cm proximal to its insertion on the calcaneus, within the area of decreased vascularity. [626] [906] The pathogenesis of Achilles tendon rupture has been attributed to both degeneration of the tendon and excessive mechanical forces. Arner and Lindholm reported that all 92 ruptured tendons that they examined histologically had degenerative changes. [349] Kannus and Józsa found that 864 (97%) of the 891 spontaneously ruptured tendons they examined histologically had degenerative changes. [116] If tendon ischemia and secondary degeneration, however, were major factors, patients older than 30 years of age would be expected to have a higher rate of rupture, which is not the case. [631] [844] [909] [910] [911] [937] In addition, pathologic specimens removed from acute ruptures often reveal the presence of hemorrhage and inflammation and no associated peritendinitis or tendinosis. [629] [909] [937] Thus, several authors have concluded that ruptures are due to a sudden overloading of the musculotendinous unit in a poorly conditioned individual rather than to underlying pathologic processes in the tendon. [908] [909] In contrast, Tallon and associates have shown that ruptured Achilles tendons histologically have a significantly greater degree of degeneration than tendons with tendinopathy, and with both conditions, the tendons are significantly more degenerated than normal tendons. [350]

The common precipitating event in 90% to 100% of individuals that sustain this injury is active forceful, sometimes unexpected, plantar flexion of the foot. [909] [937] Arner and Lindholm classified the trauma that resulted in rupture in 92 patients into three catagories. [349] The mechanism in the first category was pushing off with the weight-bearing foot while extending the knee. This type of movement, seen with sprint starts and in jumping sports, accounted for 53% of the ruptures in their series. The mechanism in the second category was sudden, unexpected dorsiflexion of the ankle. This mechanism, seen when an individual steps in a hole, accounted for 17% of the ruptures. The third category was violent dorsiflexion of a plantar flexed foot as occurs after a fall from a height. This mechanism was reported by 10% of the patients. [349]

The major current controversy regarding this injury is whether surgical repair or cast immobilization is the most appropriate method of treatment.

Clinical Evaluation

Most patients who sustain spontaneous ruptures of the Achilles tendon note a sudden snap in the heel region at the time of injury and subsequent pain with flexion of the foot. [631] [721] [909] [912] [937] [948] [956] Kannus and Józsa reported that only 297 (33%) of the 891 patients in their study had symptoms before rupture of their Achilles tendon. [116] Many patients do not seek immediate treatment because they still are able to plantar flex their ankle, and about 70% of patients complain of pain in the ankle or heel only at the time of their initial medical evaluation. This presenting complaint, along with a moderate limp and weakness in the ankle, may suggest a mild sprain rather than a heel cord rupture. Thus, the correct diagnosis is missed at the time of initial evaluation in 25% of cases. [862] [888] [908] [909]

In acute cases, physical examination will reveal a palpable depression over the area of the tendon rupture, weakness of plantar flexion, and positive results on the calf squeeze or Thompson's test. [631] [733] [908] [909] [937] Thompson's test is performed with the patient prone on a table with the feet extending over the end of the table. The calf muscles are then squeezed between the examiner's thumb and forefingers in the middle third (the musculotendinous junction) below the place of widest girth. A “normal reaction” is shown by passive plantar movement of the foot. A “positive reaction” occurs when there is no plantar movement of the foot, indicating a rupture of the heel cord. [127] The test also can be performed with the patient kneeling on a chair.

The accuracy of Thompson’s test for the diagnosis of fresh ruptures has been reported to be between 96% and 100%. [733] [909] [957] O’Brien, however, concluded that Thompson’s test can be falsely positive. [352] He noted that with Thompson’s test, tension in the Achilles tendon is produced by lifting and functionally shortening the gastrocnemius-soleus complex. When the gastrocnemius aponeurosis is torn and is no longer connected to that of the soleus, Thompson’s test will indicate a complete rupture when in fact the Achilles tendon is intact. [352] Thus, O’Brien described and recommended a needle test for diagnosis of complete ruptures of the tendon.

The needle test is performed by inserting a 25-gauge needle into the calf just medial to the midline at a point 10 cm proximal to the superior border of the calcaneus. The foot is then passively dorsiflexed and plantar flexed, and the movement of the hub of the needle is noted. Movement of the needle in the direction opposite the direction of the foot indicates that the tendon is intact throughout its distal 10 cm.

O’Brien performed this test on 10 patients with suspected rupture of the Achilles tendon and found that two patients had a positive Thompson’s test but a negative needle test. At surgery, both of these patients were found to have a partial rupture of the musculotendinous junction of the gastrocnemius, but the Achilles tendon was intact. [352] He further commented that false-positive results on Thompson’s test may cause unwitting errors when the results of operative and nonoperative treatment of “complete” ruptures are compared.

A recent prospective study of 174 patients found that palpation of the gap was the least sensitive clinical test for an Achilles tendon rupture. [351] Although both the calf squeeze and O’Brien tests had a high positive predictive value, the calf squeeze test was significantly more sensitive (0.96 versus 0.8) for diagnosis of an Achilles tendon tear. [351]

In chronic cases, defects in the tendon may not be evident or palpable owing to hematoma formation at the site of rupture and generalized swelling about the heel and ankle. Most patients do not have ecchymosis, swelling, or point tenderness as are usually found with acute ruptures, [109] and in one series, Thompson’s test was positive in only 80% of the chronic cases. [332]

Standard radiographs usually are not diagnostic of an Achilles tendon rupture. Although Arner and colleagues reported that deformation of the contour of the distal stump was pathognomonic of an Achilles tendon rupture, [353] most authorities have concluded that standard radiographs are helpful only in confirming the diagnosis of rupture in patients with calcification of the tendon (Fig. 25D-10). [714] [937]

Figure 25D-10 Lateral radiographs of a 56-year-old patient with calcification of the Achilles tendon. The radiograph on the left (**A**) was obtained when the patient was evaluated for plantar fasciitis. Three years later (**B**), the Achilles tendon was avulsed from the calcaneus.

With the advent of MRI, studies have been published that assess the diagnostic capabilities of this technique. One study found that MRI was extremely accurate for assessing the condition (e.g., shredded, uniform) and orientation (e.g., antegrade, retrograde) of the torn fibers and the width of the diastasis (with and without ankle flexion) between the ends of the tendon. [354] A second study identified four types of Achilles tendon lesions: type I, inflammatory reaction; type II, degenerative change; type III, incomplete rupture; and type IV, complete rupture. [355]

Ultrasound also has been promoted as an excellent imaging method for confirming the clinical diagnosis of an Achilles tendon rupture. [356] Margetic and associates reported that ultrasound imaging correctly identified complete Achilles tendon ruptures in 86 (98%) of 88 patients treated operatively. [356] The other patients had partial ruptures. However, the authors do not believe that MRI or ultrasound imaging is indicated for evaluation of Achilles tendon injuries because clinical tests are extremely reliable for diagnosis of complete ruptures, and treatment of lesser injuries should be predicated on the results of nonoperative treatment.

Treatment Options

Controversy about Treatment Method

The best method of treatment for complete ruptures of the Achilles tendon remains controversial. A review of the literature reveals that there are comparable numbers of studies that advocate operative [239,302-305,307,332,342,345,348,357-363] and nonoperative [904] [907] [911] [949] [952] [969] [970] [971] methods of treatment. The advantages of nonoperative treatment are absence of wound complications, decreased patient cost, and lack of a scar. The disadvantages of nonoperative treatment are a re-rupture rate as high as 39% (Table 25D-1), a higher percentage of dissatisfied patients, and a significant loss of power, strength, and endurance compared with surgically treated patients. [908] [909] [910] [937] In addition, the results of surgical repair of re-rupture after nonsurgical treatment are not as good as those of primary repair. [342]

TABLE 25D-1 -- Comparison of the Rate of Re-rupture in Reported Series of Nonoperatively Treated Achilles Tendon Ruptures

Study	No. of Cases	No. of Re-ruptures	Percentage of Cases
Nistor, 1981 [344]	60	5	8
Lea & Smith, 1972 [306]	66	7	11
Lildholdt & Munch-Jorgensen, 1976 [364]	14	2	12
Stein & Luekens, 1976 [347]	8	1	13
Gillies & Chalmers, 1970 [302]	7	1	14
Jacobs et al, 1978 [305]	32	7	22
Edna, 1980 [357]	10	3	30
Persson & Wredmark, 1979 [360]	20	7	35
Saleh et al, 1992 [368]	40	2	5
Inglis et al, 1976 [303]	23	9	39
Cetti et al, 1993 [348]	55	7	13
Ingvar et al, 2005 [365]	196	14	7
Van der Linden-van der Zwaag et al, 2004 [366]	80	4	5
Moller et al, 2002 [363]	53	11	21
TOTALS	664	80	12.1

The advantages of surgical repair are a much lower rate of re-rupture, which ranges from 0% to 5% (Table 25D-2), a higher percentage of patients returning to sports, and a greater recovery of strength, power, and endurance. [909] [910] [937] [962] [964] [965] [968] [971] The disadvantages of surgical repair are the cost of hospitalization and the major and minor complications of surgery (Table 25D-3). Complications such as deep venous thrombosis and pulmonary embolism have been reported with both methods of treatment. [952] [964] [965] [970]

TABLE 25D-2 -- Comparison of the Rate of Re-rupture in Reported Series of Surgical Repairs of Achilles Tendon Ruptures

Study	No. of Cases	No. of Re-ruptures	Percentage of Cases
Jacobs et al, 1978 [305]	26	0	0

Study	No. of Cases	No. of Re-ruptures	Percentage of Cases
Inglis & Sculco, 1981 [304]	159	0	0
Lennox et al, 1980 [307]	20	0	0
Inglis et al, 1976 [303]	44	0	0
Percy & Conochie, 1978 [359]	74	0	0
Shields et al, 1978 [391]	32	0	0
Gillies & Chalmers, 1970 [302]	6	0	0
Ma & Griffith, 1977 [239]	18	0	0
Beskin et al, 1987 [332]	42	0	0
Jessing & Hansen, 1975 [376]	108	2	2
Arner & Lindholm, 1959 [349]	92	4	4
Nistor, 1981 [344]	44	2	5
Cetti et al, 1993 [348]	56	3	5
Soldatis et al, 1997 [361]	23	0	0
Mortensen et al, 1999 [371]	71	0	0
Speck & Klaue, 1998 [373]	20	0	0
Aoki et al, 1998 [370]	22	0	0
Sölveborn & Moberg, 1994 [372]	17	0	0
TOTALS	874	11	1.1%

TABLE 25D-3 -- Comparison of the Rate of Postoperative Complications in Reported Series of Surgical Repairs of Achilles Tendon Ruptures

Study	No. of Cases	No.of Major [*] Complications (%)	No. of Minor [†] Complications (%)
Inglis & Sculco, 1981 [304]	159	20 (13)	—
Jessing & Hansen, 1975 [376]	108	7 (7)	18 (17)
Arner & Lindholm, 1959 [349]	92	22 (24)	49 (53)
Percy & Conochie, 1978 [359]	74	16 (22)	7 (10)
Inglis et al, 1976 [303]	44	2 (5)	—
Nistor, 1981 [344]	44	4 (9)	29 (64)
Beskin et al, 1987 [332]	42	3 (7)	—
Shields et al, 1978 [391]	32	1 (3)	—
Jacobs et al, 1978 [305]	26	5 (19)	—
Lennox et al, 1980 [307]	20	4 (40)	1 (5)
Ma & Griffith, 1977 [239]	18	0 (0)	2 (11)
Gillies & Chalmers, 1970 [302]	6	1 (17)	—
Soldatis et al, 1997 [361]	23	0 (0)	2 (9)
Cetti et al, 1993 [348]	56	2 (4)	15 (27)
TOTALS	744	91 (12.2)	123 (16.5)

* Major complications include wound infection, delayed wound healing, skin slough, sinus tract formation, and re-rupture.

† Minor complications include adhesions of the tendon to the skin and sural nerve injury. (In several series, the number of minor complications was not reported.)

The limited number of studies (Table 25D-4) comparing operative and nonoperative methods of treatment have reached contradictory conclusions. [907] [908] [910] [949] [971] [972] Nistor performed a prospective randomized study and found only minor differences in the results of the two methods of treatment. [344] He concluded that the treatment of choice is nonsurgical because in patients treated conservatively, there were fewer complications, fewer complaints, and no hospitalization. Gillies and Chalmers also concluded that owing to the complications of surgery, the results of operative repair of fresh ruptures were not significantly superior to those achieved with nonoperative management. [302] Moller and associates also concluded that if re-rupture is avoided, both surgical and nonsurgical treatment produce good functional outcomes. [363] In all three of these studies, the authors found no significant difference in the functional strength achieved by surgically and nonsurgically treated patients (see Table 25D-4).

TABLE 25D-4 -- Summary of Studies Comparing the Re-rupture Rate and Strength Achieved after Operative and Nonoperative Treatment of Achilles Tendon Ruptures

	Operative				Nonoperative			
Study	No. of Patients	No. of Re-ruptures	Percentage of Cases	Strength [*] Achieved (%)	No. of Patients	No. of Re-ruptures	Percentage of Cases	Strength [*] Achieved (%)
Nistor, 1981 [344]	44	2	5	83	60	5	8	79
Gillies & Chalmers, 1970 [302]	6	0	0	84	7	1	14	80
Jacobs et al, 1978 [305]	26	0	0	75	32	7	22	65
Inglis et al, 1976 [303]	44	0	0	88	23	9	39	62
Van der Linden-van der Zwaag et al, 2004 [366]	212	10	4.7	N/A	80	4	5	N/A
Moller et al, 2002 [363]	59	1	1.7	80	53	11	20.8	80
TOTALS	391	13	3.3		255	37	14.5	

* Strength of the normal leg was used to calculate the percentage of strength achieved after surgery.

In contrast, studies by Inglis and colleagues, [303] Jacobs and associates, [305] and Cetti and coworkers [348] not only documented a much higher rate of re-rupture with nonoperative treatment but also found that surgical repair resulted in significantly better restoration of strength, power, and endurance (see Table 25D-4). Both studies concluded that surgical treatment is the treatment of choice, particularly for active individuals. Two other relevant studies that examined the postoperative strength of patients found that surgical repair of re-rupture after failed conservative treatment and early (<1 month of injury) surgical repair of acute ruptures produced better functional (strength) results than a second (8-week) period of casting and late surgical repair, respectively. [909] [965]

Nonoperative Treatment

The largest series of patients treated nonoperatively were reported by Nistor, [344] Lea and Smith, [911] [970] and Ingvar. 365 Nonoperative treatment was popularized by Lea and Smith, who reported on 66 patients in 1972. [306] They immobilized their patients in a short leg walking cast with the foot in a gravity equinus position for 8 weeks and allowed weight-bearing as soon as the cast was dry. The length (8 weeks) of immobilization was important because they documented a higher incidence of re-rupture with shorter periods of casting. After the cast was removed, the patient began active gastrocnemius strengthening exercises and used a 2.5-cm heel lift for 4 weeks.

In their series of patients, the average time to immobilization was 2.6 days (range, 1 to 42 days), and the majority (56%) of patients were placed in a cast within 24 hours of injury. At an average follow-up of 26 months (range, 5 months to 6.5 years), re-ruptures had occurred in 7 (11%) of these 66 patients. All re-ruptures occurred within 1 to 4 weeks after the

8-week period of immobilization was completed. When a re-rupture occurred, the authors recommended a second full 8-week period of immobilization to ensure a good result.[306]

Nistor[344] modified this treatment protocol by using a below-knee gravity equinus cast for 4 weeks, a similar cast with less equinus for an additional 4 weeks, and a 2.5-cm heel lift for 4 subsequent weeks. In his series of 60 patients, there were five (8%) re-ruptures. All re-ruptures, which occurred between 9 and 16 weeks after the initial rupture, were treated with plaster casts for 7 to 9 weeks.

In 2005, Ingvar and associates reported the results of nonoperative treatment in 196 consecutive patients.[365] The re-rupture rate was 7%, 7 patients suffered other complications (7 had deep vein thrombosis, 1 had pulmonary embolism), and 62% of the patients reported a full recovery. The authors concluded that the low re-rupture rate challenged the claim of other studies that acute Achilles tendon ruptures should be treated operatively.[365]

More recent studies have found that patients treated nonoperatively with splinting that allows controlled early mobilization are able to return to normal activities sooner. [905] [973] [974] In 1997, McComis and colleagues reported on 15 patients who had nonoperative treatment of their Achilles tendon rupture with a functional bracing protocol.[300] The brace allowed immediate weight-bearing and active plantar flexion of the ankle, but limited dorsiflexion of the ankle. They graded their results (100-point scoring system) as excellent in 3 patients, good in 9, fair in 2, and poor in 1. Five patients had a positive result on Thompson's squeeze test at their 2-year follow-up.[300] In 2007, Twaddle and Poon found that the common denominator between operative and nonoperative treatment was early motion.[369] They found no differences in the outcomes of operative and nonoperative treatment in patients who were treated with early motion controlled in a removable orthosis.

Operative Treatment

Surgical treatment of Achilles tendon ruptures initially recommended by Abrahamson in 1923 and by Queru and Stainovitch in 1929 has become increasingly popular since Arner and Lindholm published their results in 1959.[349] Open surgical procedures proposed for acute disruptions (<4 weeks after injury) include end-to-end repair with a Bunnell, modified Kessler, or pullout wire suture [909] [910] [912] [937] [964] [975] [976] [977] [978] [979] [980]; end-to-end repair with the three-tissue bundle technique[332]; and direct repair and augmentation with tendon grafts, [937] [964] fascial flaps and grafts, [909] [912] [937] [981] or synthetic (e.g., Dacron, Gore-Tex, and the like) grafts.[358] Closed surgical methods of repair include percutaneous suture [844] [967] [982] [983] [984] [985] [986] [987] and external fixation.[258]

In general, repairs are performed with the aid of a tourniquet and with the patient in the prone position. Although local anesthesia has been used successfully for repair of Achilles tendon ruptures, [975] [976] [988] [989] a general or regional anesthetic usually is administered to the patient for open repairs. [909] [937] [964] [978] [981] Many studies stress the importance of delaying surgery if there is substantial soft tissue swelling. They note that there is no difference in the functional result if the tendon is repaired within 30 days of rupture, and therefore optimizing local skin conditions is preferred over hasty surgical intervention. [909] [912] [937] [951]

For open repairs, an incision medial to the medial border of the Achilles tendon is recommended to minimize sural nerve injury. Inglis and colleagues recommend a darted incision, with darts created 1 inch apart at a 30-degree angle, to distribute skin pressure more evenly along the line of the incision and to increase the operating area of the incision, [909] [980] while Lansdaal advocated a minimally invasive procedure.[375] The subcutaneous tissues are then opened to expose the deep (crural) fascia of the leg. The Achilles tendon is not a subcutaneous structure but lies beneath the deep crural fascia. The fascial layer is sharply dissected so that it can be closed carefully at the completion of the tendon repair. Closure of the deep fascia not only reduces tension on the skin closure but also acts as an interface between the subcutaneous tissue and the tendon repair and helps to prevent adherence of the tendon to the subcutaneous tissues and skin.

Although numerous surgical procedures have been proposed for repairing ruptures of the Achilles tendon, there is no single, uniformly superior technique. Analysis of the efficacy of one type of repair has been poorly documented because in most published series, several techniques were employed (Table 25D-5). It is evident from Table 25D-2 , however, that all the techniques used in the different studies have been uniformly successful. In one study, Jessing and Hansen compared the results of end-to-end suture (54 patients) with those of direct repair augmented with a turn-down graft of gastrocnemius fascia (48 patients).[376] There was one re-rupture in each group, and they concluded that there were no significant differences in the functional results of these two operative procedures. They did note, however, that although the rate of re-rupture with either technique was very low, the risk for sustaining a rupture of the contralateral tendon was quite high. Eight (26%) of the 31 patients in their series who resumed full sporting activities ruptured the other Achilles tendon within 2 to 14 years of their ipsilateral rupture. Aroen and colleagues reported that 6% of their 168 patients with Achilles tendon repairs subsequently ruptured their contralateral tendon.[385] The basic concepts and principles of specific types of repairs are summarized in the following paragraphs.

TABLE 25D-5 -- Comparison of the Frequency of Various Procedures Performed for Open Repair of Acute Ruptures of the Achilles Tendon

Study	No. of Ruptures	Primary [*] Suture (%)	Local [†] Graft (%)	Three-Tissue Bundle (%)	Other [‡] (%)
Inglis & Sculco, 1981 [304]	162	79 (49)	81 (50)	—	2 (1)
Beskin et al, 1987 [332]	42	19 (45)	18 (43)	5 (12)	—
Lennox et al, 1980 [307]	20	16 (80)	4 (20)	—	—
Jacobs et al, 1978 [305]	25	16 (64)	9 (36)	—	—
Shields et al, 1978 [391]	32	—	26 (81)	—	6 (19)
Jessing & Hansen, 1975 [376]	102	54 (53)	48 (47)	—	—
TOTALS	383	184 (48)	186 (49)	4 (1)	8 (2)

* In the primary suture group, direct repair with either a Bunnell or an amplified Kessler suture often was secured with a pullout wire.

† Local grafts included flaps of gastrocnemius fascia or augmentation with the plantaris or peroneus brevis tendon.

‡ Includes reinsertion of the Achilles tendon into the os calcis via drill holes and os calcis bolt fixation.

Although some authors recommend excision of the frayed tendon ends, [349] most remove only the intervening hematoma and repair the tendon in its frayed state. [909] [937] [981] End-to-end repairs are accomplished with either a Bunnell suture, a Krackow locking loop stitch, or a modified Kessler suture. [910] [912] [964] [975] [976] [977] [978] [991] A recent study tested the Krackow locking loop technique and found that increasing the number of locking loops had minimal effect on the strength of the repair. [386] However, using a second interlocking suture placed at 90 degrees to the first significantly increased the load to failure of the repair. Two other studies found that the overall strength of the repair can be further enhanced by the edition of an epitenon suture. [992] [993] However, load to failure was significantly improved only when the epitenon was sutured with a cross-stitch rather than a running stitch technique. [992] [993]

The three-tissue bundle technique, used exclusively for repair of acute ruptures, was first described in the American literature in 1987. [332] With this technique, the disorganized tendon fibers are gathered into three bundles by means of three heavy nonabsorbable sutures (Fig. 25D-11 **A** and **B**). The three bundles are sutured together in a functional position (see Fig. 25D-11 **C**), and then the tendon sheath is closed over the repair. A recent cadaveric study using eight pairs of fresh-frozen Achilles tendons compared the tensile strength of the triple-bundle technique with the Krackow locking loop technique. [389] Biomechanical testing revealed that the triple-bundle technique was almost 3 times stronger (average load to failure, 387 N versus 161 N).

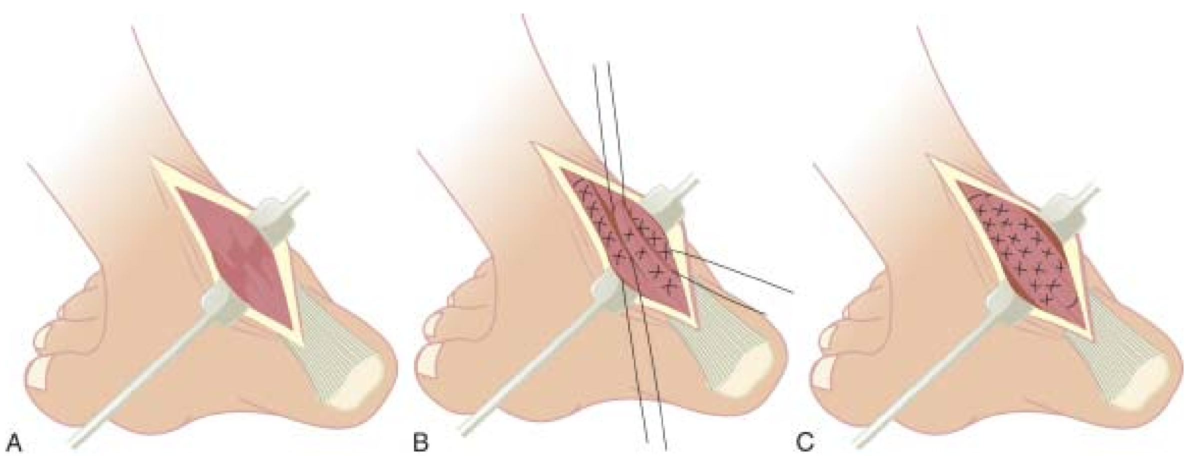

Figure 25D-11 The three-tissue bundle technique. The torn tendon fibers (**A**) are gathered into three bundles with three nonabsorbable (Bunnell) sutures (**B**). The three bundles are then interdigitated and sewn to each other with the suture ends and additional nonabsorbable sutures (**C**). *(Redrawn from Beskin JL, Sanders RA, Hunter SC, Hughston JC: Surgical repair of Achilles tendon ruptures. Am J Sports Med 15:1-8, 1987.)*

After surgery, the foot and ankle are immobilized in below-the-knee plaster splints with the foot in a relaxed equinus position for 5 to 7 days. The patient is instructed to keep the leg elevated with the knee flexed 45 degrees but is allowed to move around on crutches without bearing weight to perform necessary daily functions. The splints are removed 1 week after surgery, and active dorsiflexion is initiated. When 0 or more degrees of dorsiflexion are achieved (usually in 1 to 3 days), a short leg walking cast is applied with the ankle in a neutral position and is worn for 6 to 8 weeks. After the cast is removed, the patient is placed on crutches and returns gradually to full weight-bearing over 4 to 6 weeks. Active range of motion is encouraged as soon as the cast is removed, and no further immobilization or heel lifts are employed. The authors found that range of motion at the time of cast removal was significantly better in patients treated with the three-bundle technique than with other surgical procedures. Although this difference was not present at long-term follow-up, the greater range of motion after cast removal did imply an earlier return to normal activities. Their long-term follow-up (average, 4.7 years) documented that most patients returned to their preinjury level of activity and that most patients did not seek further follow-up more than 6 to 12 months after their surgery. 332

Augmentation of end-to-end repairs has been recommended by several authorities. [937] [944] [964] [981] [995] Lindholm described a method of augmentation that reinforces the repair and prevents adhesion of the repaired tendon to the overlying skin. [339] Through a standard medial incision, direct repair of the tendon is performed as described previously. Subsequently, two fascial flaps, 1 cm wide and 7 to 8 cm long, are fashioned from the proximal tendon and aponeurosis of the gastrocnemius muscle. The flaps, which are left attached at a point 3 cm proximal to the site of the repair, are rotated 180 degrees and twisted so that the smooth external surface faces the subcutaneous tissues. The two flaps are then sutured to the distal stump of the Achilles tendon and to each other so that they completely cover the site of the repair. Lindholm stresses that during wound closure, one must be careful to approximate the sheath over the site of the repair. [339]

Jessing and Hansen described a modification of the Lindholm technique using only one flap of gastrocnemius fascia. [376] The strip of fascia, about 4 cm in width, is rotated on its distal pedicle and turned distally to cover the rupture site (Fig. 25D-12). The smooth outer surface of the gastrocnemius fascia is thus interposed between the site of repair and the crural fascia.

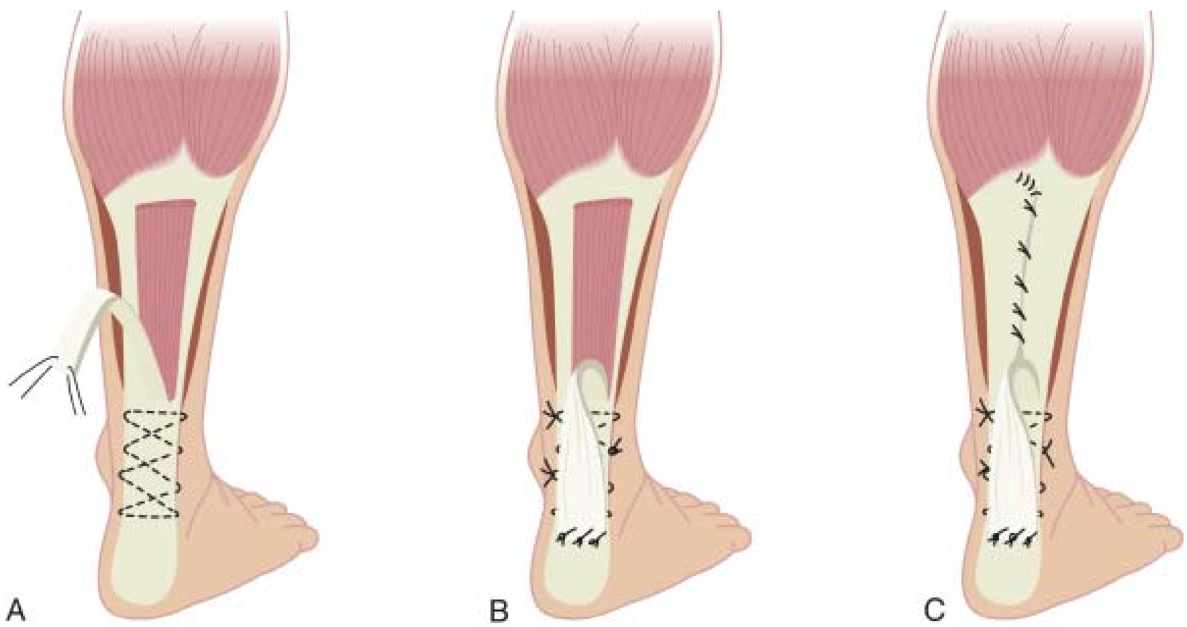

Figure 25D-12 Augmentation of a direct repair of the Achilles tendon with one strip of gastrocnemius fascia. After repair of the tendon (**A**), a strip of gastrocnemius fascia about 4 cm wide and 12 cm long is twisted 180 degrees upon itself and is then turned down over the repair so that its smooth surface underlies the tendon sheath and subcutaneous tissues (**B**). The defect in the gastrocnemius fascia is closed, and the flap is secured to the Achilles tendon (**C**).

Lynn described a method of augmenting end-to-end repairs in which the plantaris tendon is fanned out to make a membrane an inch or more wide for reinforcing the tendon. [390] This method can only be applied to acute injuries, however, because after 2 weeks, the plantaris often becomes incorporated in the scar and cannot be identified. With this technique, a standard medial incision 12 to 18 cm long is made parallel to the medial border of the tendon. The ends of the tendon are sewn together, and the plantaris tendon is divided at its insertion on the calcaneus. The tendon is then fanned out to form a membrane, and the membrane is placed over the repair and sutured in place with interrupted sutures. When possible, the Achilles tendon is covered for 1 inch both proximal and distal to the repair. Closure of the wound and postoperative treatment are the same as those described for other primary repairs.

After the aforementioned surgical procedures, the foot is placed in 20 to 30 degrees of equinus, and a long leg cast is applied. [909] [912] [964] [996] Excessive equinus is not desirable because it produces local subcutaneous tissue and skin ischemia. [304] Three weeks after surgery, a short leg cast is applied and is worn for 3 to 4 additional weeks. At that juncture, immobilization is discontinued, and the patient is given heel lifts, 2- to 2.5-cm in height, for both heels. In general, weight-bearing is not permitted until immobilization is discontinued. [909] [912] [964] [996] The elevated heels are worn for 3 to 4 weeks, and during that time the patient is allowed to swim and exercise on a stationary bicycle with instructions to increase force gradually as power returns to the gastrocnemius-soleus complex. Light jogging and athletics are not initiated until about 3 months after surgery.

In recent years, several studies have advocated immediate motion and early full weight-bearing after surgical repair of acute Achilles tendon ruptures. [974] [975] [976] [977] [978] [997] [998] Sölveborn and Moberg studied 17 patients who were allowed free ankle motion in a patellar tendon–bearing plaster cast with an outrigger frame under the foot that made weight-bearing possible immediately after surgery. [372] They reported that no re-ruptures or other complications occurred, and range of motion, strength, and recovery time were better than results of traditional ankle immobilization.

Several subsequent studies have reported similar results. [975] [976] [978] In 1998, Speck and Klaue prospectively evaluated the clinical outcomes of 20 patients who had 6 weeks of early full weight-bearing in a removable ankle-foot orthosis after an open repair of a torn Achilles tendon. [373] All 20 patients reached their preoperative level of sports activity and had no significant side-to-side difference in ankle mobility and isokinetic strength. There were no re-ruptures. In a larger prospective study, Mortensen and colleagues randomly assigned 71 patients who had repairs of acute Achilles tendon ruptures to either conventional postoperative management (a cast for 8 weeks) or early restricted motion of the ankle in a below-the-knee brace for 6 weeks. [371] They found that early motion patients had a smaller initial loss in range of motion, and returned to work and sports activities sooner than those managed with a cast. There were no re-ruptures in either group. As noted previously, Twaddle and Poon found that outcomes of operative and nonoperative treatment were the same when controlled early motion was used. [369] Kangas and colleagues found that tendon elongation after operative repair was less in patients treated with early motion and that patients who had less elongation had better isokinetic calf muscle strength and significantly better clinical outcomes. [392]

Closed techniques for repair of acute ruptures have been described in several reports. [844] [862] [967] [982] [983] [984] [985] [986] [987] [999] In 1977, Ma and Griffith reported on 18 tendons that were repaired using a percutaneous suturing technique. [239] Through stab wounds, sutures were passed through the proximal and distal stumps of the tendon with straight and curved needles. Through tension on the two ends of the suture, the ends of the tendon were approximated while the ankle was positioned in maximal equinus. The suture was then cut short, tied with a surgeon's knot, and pushed subcutaneously. The six small puncture wounds (three medial and three lateral) were left open and were covered with dry sterile dressings. Aftercare included a short leg, non–weight-bearing cast for 4 weeks, and then a low-heeled, weight-bearing, short leg cast. When the cast was removed, the patient started toe-heel raising for 4 weeks and then performed heel cord stretching exercises for an additional 4 weeks. Ma and Griffith stated that the procedure could be performed under a local, regional, or general anesthetic.

Results of the percutaneous suture were no ruptures in the 18 patients and only two minor complications. One was a skin retraction dimple at the operative site, and the other was a tender nodule at the site of the surgical knot. Small, nontender nodules generally were present in most of the 18 patients at the site of the surgical knot, but the sutures were removed only in the two patients who were symptomatic. [239]

In the past decade, there have been additional studies to assess long-term results, [377] present new percutaneous techniques, [967] [983] [1000] and compare the results of percutaneous and open surgical repair. [394] In 1990, Bradley and Tibone compared the results of 15 patients treated by open repair augmented with a gastroc-soleus fascial graft, and 12 patients treated by percutaneous (Ma and Griffith type) repair. [394] They found that percutaneous repair resulted in strength levels similar to open repair but had a re-rupture rate of 12%, significantly higher than the results of open repairs. They concluded that open repair should be performed in all high-caliber athletes who cannot afford any chance of re-rupture. [394]

Recently, new percutaneous surgical techniques have been developed to increase the strength of repairs [395] and decrease complications such as sural nerve injury. [967] [984] [985] [986] [987] Webb and Bannister reported results of 27 patients who had repairs through three midline stab incisions rather than Ma and Griffith's six (three lateral, three medial) skin incisions.[362] They had no sural nerve injuries, compared with the 13% injury rate reported with the Ma and Griffith technique. Amlang and associates described percutaneous repairs using the Dresden instrument in 61 patients and reported that very good and good results were achieved in 78% and 20% of the patients, respectively.[379] Cretnik and coworkers compared the results of 105 patients who had open repairs with those of 132 patients who had percutaneous repairs.[380] There were slightly more re-ruptures (3.7% versus 2.8%) and sural nerve injuries (4.5% versus 2.8%) in the group of percutaneous repairs, but there were significantly more major complications (12.4% versus 4.5%, $P = .03$) in the group of open repairs. More recent reports have advocated endoscopically assisted percutaneous Achilles tendon repairs.[986] [987] In 2003, Halasi and colleagues compared the results of 89 patients with percutaneous repairs with those of 57 patients who had endoscopically assisted percutaneous repairs.[381] They reported excellent to good results in 88% and 89% of the percutaneous and endoscopic-percutaneous groups, respectively. They noted that the re-rupture rate was lower (2 total, 3 partial versus 1 partial) in the endoscopic group.

In 1985, Nada described a method of external fixation for treatment of acute ruptures that he used in 33 consecutive patients.[257] With this technique, Kirschner wires (0.08 and 12 cm long) are inserted through stab wounds into the calcaneus and the proximal stump of the tendon. The ankle is put into an equinus position, and the proximal wire is pulled distally (1 to 1.5 cm) to approximate the ends of the tendon. The two K-wires are then maintained in their approximated position with an external fixator. The operation was performed under local anesthesia in 26 of the 33 patients.

Four weeks after the operation, the clamps and wires were removed, and the patients were placed in an equinus short leg walking cast for an additional 4 to 5 weeks. After 8 weeks, immobilization was discontinued, and the patients were given shoes with the heels built up 2.5 cm. Results at a mean follow-up of 2.4 years (range, 9 months to 4 years) were good or excellent in 30 of the 33 patients. Two patients had a fair result owing to sural nerve injuries, and one had a poor result owing to a Sudeck's atrophy. All except the patient with the poor result returned to their original level of activity, and there were no re-ruptures.[257]

In patients with neglected Achilles tendon ruptures, end-to-end repair often is not possible after excision of the intervening scar tissue. In these cases, grafts of fascia lata, local tendons (plantaris and peroneus brevis), and strips of Achilles tendon or free autologous gracilis tendon grafts have been used to bridge the gap.[950] [1001] [1002] [1003] [1004] Mann and colleagues opposed the use of free fascia and turn-down fascial grafts because they are avascular structures that must be revascularized to be incorporated into the repair.[17] They also noted that transfer of the peroneus brevis carries the risk for changing the balance between the everters and inverters of the foot. Thus, they recommended transfer of the flexor digitorum longus and reported good and excellent results in six of the seven patients in which this procedure was performed.[17] In contrast, Sebastian and associates recommended the use of either peroneus brevis or flexor hallucis longus for reconstruction of chronic Achilles tendon ruptures.[398]

Abraham and Pankovich also found that results of repair of neglected rupture with strips of Achilles tendon and fascia lata were unsatisfactory.[400] They concluded that only an end-to-end repair of the chronic lesion would promote optimal functional recovery and restore muscle strength. Thus, they described a technique by which an end-to-end anastomosis was made possible by a proximal V-Y tendinous flap. With this technique, the tendon ends are appropriately trimmed, and the length of the defect in the tendon is measured with the knee in 30 degrees of flexion and the ankle in 20 degrees of plantar flexion. An inverted V incision, with arms that are 1½ times the length of the defect, is made over the central aponeurosis of the gastrocnemius. The incisions extend through the fascia and underlying muscle tissue of the gastrocnemius. The flap is then pulled distally and sutured to the distal stump of the ruptured tendon. Maffuli and Leadbetter reported the use of free autologous gracilis tendon grafts in 21 patients and concluded that the procedure is safe but technically demanding and affords good results even in neglected ruptures of 9 months duration.[399]

Postoperatively, the patient is placed in a long leg cast for 6 to 8 weeks and then a short leg, weight-bearing cast for an additional 4 to 6 weeks. The patient is subsequently placed in shoes with 3- to 5-cm heel lifts, which are worn for 1 month. Progressive resistive exercises are initiated immediately after the second cast is removed. In their series of four patients, there was only one surgical complication (a neuroma of the sural nerve), and three of the four patients regained full strength of the triceps surae muscle. The other patient had some residual weakness but was happy with the result.[400] [400]

AUTHORS' PREFERRED METHOD

Operative repair of Achilles tendon ruptures is our treatment of choice because most of our patients are intercollegiate or recreational athletes. Nonoperative treatment is always offered and discussed but usually is not recommended owing to the reported rate of re-rupture of 7% to 39% (see Table 25D-1) and the loss of strength after this method of treatment (see Table 25D-4). We have, however, treated acute ruptures with cast immobilization or a removable orthosis in patients who declined to have surgery, in elderly and chronically ill patients, and in middle-aged, sedentary, executive-type individuals who feel they cannot afford the time away from work required for operative treatment.

Our preferred method of repair is shown in Figure 25D-13 . The procedure is performed on an outpatient basis, under a general or regional anesthetic. The patient is placed prone on the operating table, and the ruptured tendon is approached through a medial incision that parallels the medial border of the Achilles tendon. The skin and subcutaneous tissue are gently retracted with skin hooks and narrow retractors to expose the crural fascia. The fascia is incised along the medial border of the tendon so that its closure will be offset from that of the skin incision.

The ends of the tendon (see Fig. 25D-13 **A**), which are not routinely débrided, are reapproximated with a Krackow locking loop stitch of 5-0 nonabsorbable suture. The peripheral fibers of each stump subsequently are sewn together with a 2-0 absorbable suture using a cross-stitch technique (see Fig. 25D-13 **B**). The repair is then reinforced with a strip of gastrocnemius fascia that is twisted 180 degrees on its distal pedicle and rotated distally to cover and extend 2 to 3 cm beyond the site of repair (see Fig. 25D-13 **C**). A strip that is 7 to 8 cm long and 1.2 to 1.4 cm wide usually is sufficient because the fascia can easily be stretched to a width of 2 to 3 cm with tissue forceps to encompass the width and length of the sutures used for the primary repair. The fascial strip is secured to the distal stump and to the medial and lateral edges of the tendon with a running 2-0 absorbable suture (see Fig. 25D-13 **C**). The ankle is then placed in a neutral position to assess the degree of tension on the repair. The defect in the gastrocnemius fascia is closed with a running 0-0 absorbable suture, and the sheath of the Achilles tendon is closed with interrupted and running 2-0 absorbable suture (see Fig. 25D-13 **D**). The subcutaneous tissues are closed with buried 2-0 absorbable sutures, and the skin is closed with a running subcuticular, 3-0 nonabsorbable suture and 3 to 4 escape stitches. The leg is placed in a short leg plastic boot with the ankle in 20 degrees of plantar flexion.

Two weeks after surgery, the subcuticular suture is removed, the angle of flexion of the ankle hinge on the boot is decreased to 10 degrees, and the patient is allowed full weight-bearing with the ankle locked in 10 degrees of plantar flexion. Three weeks after surgery, the ankle is brought up to a neutral position, and the patient is allowed to continue full weight-bearing on the extremity. Immobilization is discontinued 4 weeks after the repair, and the patient begins active and active-assisted range of motion exercises, swimming, and stationary bicycle activities and is allowed to ambulate in a shoe that has a 1-cm heel lift. When the patient has achieved a full range of motion (usually in 2 to 4 weeks), strengthening (e.g., one-leg heel raises) of the extremity is initiated. Patients begin the previously described running program when isokinetic strength testing reveals that their strength is at least 70% of that of the nonoperated extremity. They are allowed to return to athletic activities when they have full strength and full endurance and have completed the aforementioned running program. We also encourage athletes to complete an ankle joint proprioception training program. Bressel and colleagues have shown that patients with a previous history of an Achilles tendon rupture have proprioception deficits in both limbs, which adversely affect their function.[401] In most cases, the athletes have achieved full strength and completed the running and proprioceptive programs within 6 months of

Critical Points

- Early recognition and management of tendon injuries of the foot and ankle can optimize outcome.
- A thorough history of the injury and physical examination to evaluate potential tendon injuries are usually adequate for diagnosis; however, MRI or ultrasound examination are useful for diagnosis of partial injuries, evaluation of associated structures, and determination of the location of the injury and tendon ends.
- Most closed disruptions of tendons of the foot and ankle are preceded by chronic degeneration of the tendon.
- Early surgical intervention in young active patients with tendon disruptions to the foot and ankle may lead to improved outcomes compared to nonoperative management.
- After a rupture of the Achilles tendon, an MRI of the Achilles tendon is needed only if nonoperative treatment is selected to determine if the ends of the tendon are apposed when the ankle is in plantar flexion.
- After Achilles tendon repairs, early motion and weight bearing produce the best results.

Suggested Readings

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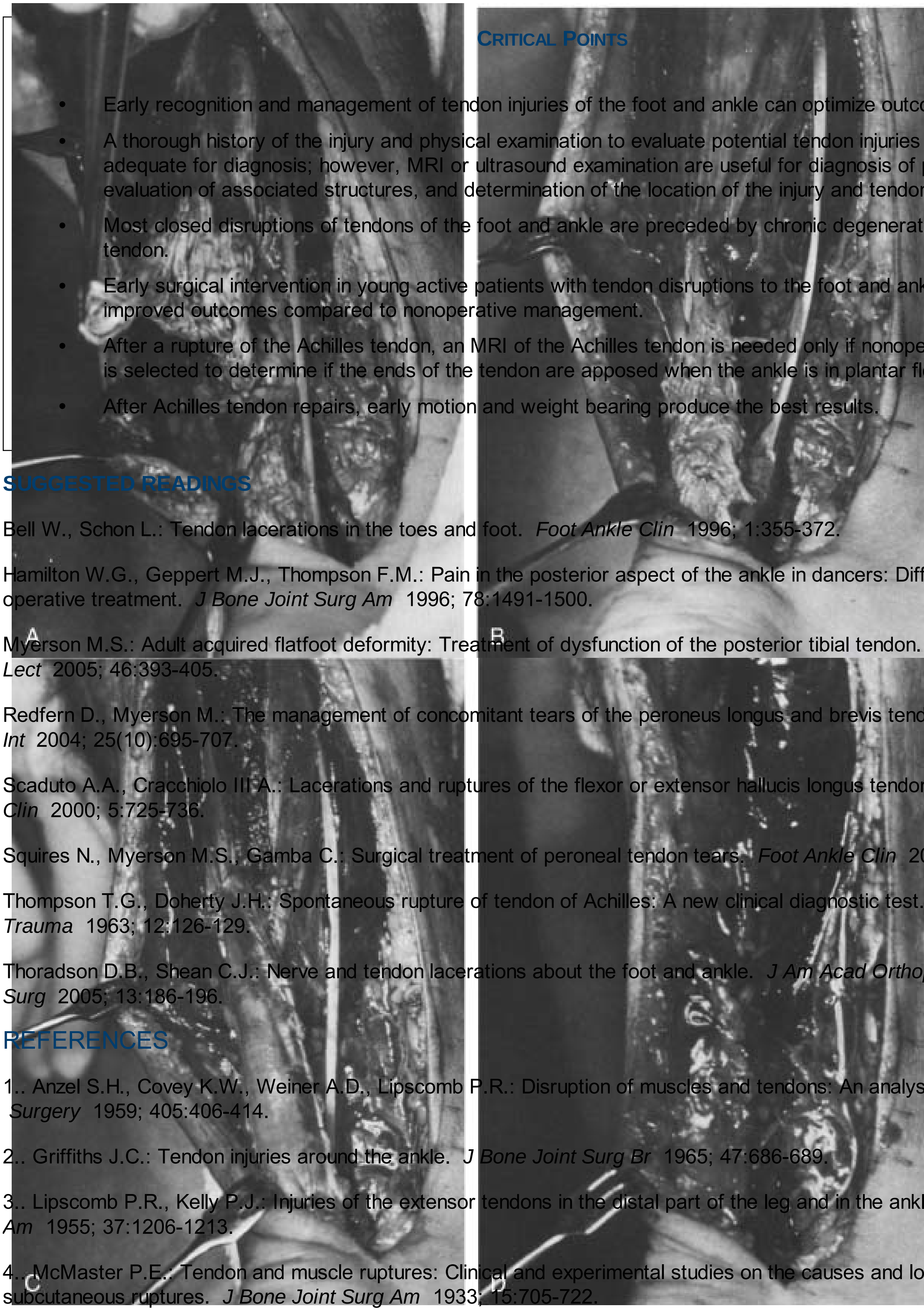

Figure 25D-13 The authors' method for repair of Achilles tendon ruptures. The frayed ends of the tendon (**A**) are not routinely débrided. The ends of the tendon are reapproximated with a No. 5 nonabsorbable suture (**B**), and the peripheral fibers are aligned and repaired with a running 2-0 absorbable suture. **C**, The repair is reinforced with a turned-down flap of gastrocnemius fascia. The defect in the gastrocnemius fascia and the Achilles tendon sheath are repaired (**D**) before closure of the wound.

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SECTION E Stress Fractures of the Foot and Ankle

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Repetitive stress or "overuse" injuries of the bones of the foot and ankle cause numerous problems and disturbances in training and recreation. Beginning even before President John F. Kennedy's public awareness program in the early 1960s, fitness and recreation have gained increasing attention and importance in modern American society. As sports and exercise have become the focus of personal health programs, as well as preventive medicine projects that promote healthier lifestyles, stress-related injuries have become more and more commonplace. They are now estimated to account for up to 10% of a sports medicine practice. [1]

The first description of a stress fracture was recorded in 1885 by the Prussian military physician, Breithaupt, who described soldiers with swollen, painful feet. After the discovery of roentgenographs in the late 19th century, this condition was attributed to a metatarsal shaft fracture, and the term *march fracture* was coined. Through the ensuing 100 years, numerous reports have described fatigue-type fractures; most of the early literature identified these injuries in a military setting. [1016] [1017] [1018] [1019] [1020] [1021] [1022] [1023] [1024] The literature continues to abound with reports of many different kinds of stress fractures that affect all manner of athletes, sportspersons, and individuals engaged in health-conscious fitness programs. A wide range of the population is affected as well, including both sexes and all ages from adolescence to senescence, as well as athletes participating in virtually every kind of sporting endeavor. [1025] [1026] [1027] [1028] [1029] [1030] [1031] [1032] [1032] [1034] [1035] [1036] [1037] [1038] [1039] [1040] [1041] [1042] [1043] [1044] [1045] [1046] [1047] [1048] [1049] [1050] Recent literature has placed a new emphasis on the nature and occurrence of stress injuries in elite athletes as well as on nutritional and hormonal contributions to disease. [1015] [1026] [1027] [1031] [1033] [1042] [1044] [1051]

A stress fracture can be defined as a partial or complete fracture of bone that results from repeated application of a stress lower than that required to fracture the bone in a single loading situation. [15] Stress fractures can be divided into two types—fatigue fractures and insufficiency fractures. Fatigue fractures result when histologically normal bone is subjected to repetitive stress below the monotonic fracture threshold, leading to incomplete repair and eventual mechanical failure. These tend to occur in specific anatomic sites based on particular loading patterns associated with specific activities (Table 25E-1). Insufficiency fractures, in contrast, occur when mechanical failure of the bone results from relatively normal stresses applied to a histologically abnormal bone, which is deficient in mineral or abnormally inelastic, or both. [38] Insufficiency fractures typically occur in postmenopausal osteoporotic women. [25] They are also seen in patients with underlying conditions such as Paget's disease, diabetes mellitus, hyperparathyroidism, [39] rheumatoid arthritis, [1052] [1054] and postirradiation osteopathy. [1]

TABLE 25E-1 -- Characteristics of Stress Fractures

Bone	Location	Activity
Hallucal sesamoids	Medial > lateral	Running, football, golf, gymnastics
Metatarsal shaft, neck	2nd, 3rd, 4th	Military recruits, running, athletes
Metatarsal base	2nd	Ballet dancers
Metatarsal shaft base (Jones' fracture)	5th	Basketball, football, soccer
Navicular	Dorsal, middle	Track and field, basketball, football, soccer
Medial malleolus		Running, jumping, basketball
Tibia	Shaft or distal metaphysis	Distance runners

ANATOMY

Depending on what one includes, the foot and ankle is composed of about 30 bones (i.e., tibia, fibula, talus, calcaneus, navicular, cuboid, three cuneiforms, five metatarsals, fourteen phalanges, and two hallux sesamoids) and associated accessory ossicles and variable sesamoids joined by myriad ligamentous structures. Extrinsic and intrinsic musculotendinous units power the articulations that allow for both motion and dissipation of force. The normal walking cycle consists of a stance phase and a swing phase, with the stance phase consuming about 62% of the walking cycle. The stance phase is divided into three intervals: heel strike, foot flat, and push-off. During the first interval, the foot is being loaded with the weight of the body while the ankle undergoes rapid plantar flexion under control of the anterior musculature. The foot is in pronation that originates at the subtalar joint, allowing absorption and dissipation of the forces generated by ground contact. The second interval begins with foot flat and forward transfer of the center of gravity over the foot. The ankle initially dorsiflexes, and the subtalar joint demonstrates progressive inversion brought on by multiple factors. The progressive inversion helps transform the flexible hindfoot and midfoot into a rigid lever. In the third interval, rapid plantar flexion of the ankle by concentric contraction of the posterior musculature occurs with further inversion of the subtalar joint, reaching a maximum at toe-off. When the heel is elevated at the time of lift-off, the weight of the body is normally shared by all of the metatarsal heads. Because of the oblique nature of the metatarsophalangeal break, the foot must supinate to distribute the body weight evenly among the metatarsal heads.

Alterations and variations in anatomy and range of motion may affect the loading of the foot during the stance phase of gait, which can lead to localized overloading and subsequent stress fracture. Patients with rigid foot deformities such as cavovarus foot or tarsal coalition are poorly able to absorb the forces during the first interval of the stance phase. [1055] [1056] [1057] Stress is subsequently transferred to the midfoot (navicular, base of metatarsals), laterally (fifth metatarsal), or distally to the forefoot. Inability to invert (supinate) the hindfoot during the second and third intervals leads to failure to distribute the body weight evenly among the metatarsals. Kaufman and colleagues showed that pes planus increases the risk for stress fracture [42] and does not protect the foot, as other studies have suggested. [41] Clinically, overloading of the medial column of the foot occurs with sesamoid symptoms. Many authors accept that an abnormally short first metatarsal (congenital, traumatic, iatrogenic) or an abnormally long second or third metatarsal (Morton's foot) contributes to a stress fracture because of loss of the normal metatarsophalangeal break, or abnormal pattern of pressure distribution within the forefoot, which results in isolated overloading of specific metatarsals. [44] Other authors showed that the lengths of the first and second metatarsals and first and second toe lengths had no effect on metatarsal stress fracture incidence. [45] Conversely, some authors demonstrated that the relative length of the second metatarsal is a risk factor regarding stress fracture at the base of the second metatarsal. [46] No correlation was found between first ray hypermobility and second and third metatarsal stress fractures. [47] Kaufman was unable to demonstrate a relationship between loss of ankle or hindfoot motion and stress fracture. [42] There is one report of a stress fracture of the anterior process of the calcaneus, in essence a portion of the coalition, in the presence of a calcaneonavicular coalition that caused restricted subtalar motion. [48] In an inverse scenario, there is also a report of talar stress fracture developing after resection of a talocalcaneal coalition. [49]

BIOMECHANICS

Bone is a dynamic organ that is capable of repair and reconfiguration. Wolff's law states that stresses applied to a bone stimulate remodeling of the bony architecture for optimal withstanding of the forces being applied. Rapid disuse osteopenia results when normal forces are withdrawn from a healthy bone. This phenomenon is clearly demonstrated in experimental models as well as in humans experiencing weightlessness. [50] During steady state, daily stresses applied to bone stimulate development of a bony architecture that is optimally fit in terms of strength and density, according to Wolff's law. When activities and therefore stresses are increased, remodeling of the bone takes place. The exact mechanism that initiates this process is unknown. Several authors state that it is likely due to the creation of microfractures. [1] Microfractures stimulate osteoclastic resorption in initiation of the repair process. As osteoclasts resorb bone, osteoblasts fill in the voids with new osteons. If high levels of activity continue, microfractures and osteoclastic resorption exceed osteoblastic new bone formation, resulting in metabolic imbalance. If stresses continue to accumulate at a rate beyond that occurring in the repair process, fatigue fracture may result. Although microfractures of the bone are a physiologic process that responds to Wolff's law, disease is apparent when reparative processes cannot keep pace with the fracture process.

Stresses to bone result from both tension and compression. Tensile forces occur on the convex sides of long bones; compressive forces occur on the concave side. Muscular activity and muscle fatigue can attenuate and increase, respectively, these forces applied to bones. In normal, nonmaximal activity, muscular contractions tend to dampen the forces applied with weight-bearing. As activity level and duration increase, muscles fatigue, and the dampening effect of muscle diminishes and the stresses at the bones increase. [1065] [1066] Microfractures begin to accumulate, thus shifting the balance in favor of bone damage. Continued impact forces from weight-bearing lead to damage that exceeds repair capabilities and eventually to fatigue fracture. Fatigue fracture is a function of the number of loading cycles and the amount of force applied. Because the metabolic balance in normal healthy bones relies on the ability of bone to heal, osseous vascularity plays an important role in this balance. As such, "watershed areas" of bone with relative hypovascularity (proximal metaphyseal-diaphyseal junction of the fifth metatarsal; central third of tarsal navicular; metatarsal necks; anterior tibia) are known to be predisposed to fatigue fractures.

RISK FACTORS

Various risk factors for development of a lower extremity stress fracture have been identified. Clearly, running is responsible for the greatest number of stress fractures in the nonmilitary population. [1] Running is the athletic activity that produces the greatest number of repetitions of the same (high) impact-loading activity. The force of each stride is a magnification of body weight, as has been shown in the gait studies of Mann and associates. [53] Thus, a 70-kg individual dissipates 120 tons of force per foot per mile walked, and this increases to 210 tons of force per foot per mile in running. [53] It is not hard to see why runners and joggers develop stress injury after running multiple miles, several times a week, on concrete streets. In this context, it is hard to understand why the incidence of stress fractures is not higher still. Other predisposing factors may include poor training techniques, such as abruptly increasing the intensity, duration, or type of training. Alterations in warm-ups, running surface or grade, shoewear, and orthoses may be predisposing factors, although Milgrom and colleagues showed no difference in stress fracture rate in military recruits when using insoles as a prophylactic measure. [54] Other impact activities such as jumping, sprinting, and hurdles can independently lead to stress fractures; they also increase the risk for stress injury in runners.

Anatomic factors play a role in stress-related injuries. The height of the arch has been shown by Simkin and colleagues [43] to correlate with an increased risk for stress fracture. A cavus foot deformity tends to be rigid and unable to absorb forces imparted during physical activities. Leg-length discrepancies overload the longer limb, resulting in the application of higher stresses. Hallux valgus deformities and long second metatarsals (Morton's foot) lead to second ray overload, with development of stress fractures as well as to the rare stress fracture of the first proximal phalanx. [55] Excessive external rotation at the hip has been shown to increase the risk for stress fracture. [4]

Female gender as a risk factor has received increased attention in the recent literature on stress fractures. In their study of stress fractures in dancers, Kadel and coworkers [24] showed that excessive training and prolonged duration of amenorrhea contribute independently to the risk for fracture. Bone mineral density has been shown to be significantly decreased in amenorrheic athletes compared with eumenorrheic athletes and controls. [1042] [1070] Girls with anorexia nervosa are at higher risk for stress fractures, and the bone appearance on magnetic resonance imaging (MRI) is altered and thus may interfere with stress fracture diagnosis. [57] Although estrogen supplementation has been shown to be clearly beneficial in preventing postmenopausal bone loss, as well as to increase bone mineral density in amenorrheic woman, [58] the literature is contradictory as to whether estrogen supplementation decreases the incidence of stress fracture among amenorrheic athletes. [1070] [1072] [1073] Prophylactic treatment with risedronate did not appear to decrease the risk for stress fractures in military recruits. [60]

Previous surgical procedures to the lower extremities may expose the bones to new or varied forces. Metatarsal osteotomies, either for hallux valgus or for lesser ray problems, may lead to overloading of adjacent rays and increased risk for subsequent development of stress fractures. [61] Ankle and hindfoot procedures to fuse or realign the joint can lead to increased loading, especially if there is a malunion. Sammarco and Idusuyi [62] reported a case of a third metatarsal stress fracture following endoscopic plantar fascia release. They postulated that excess force was created because of the loss of the stress-relieving function of the plantar fascia. Stress fractures following total joint replacement have been reported, presumably owing to newly increased stresses in a poorly conditioned, previously underused extremity. [63]

Age has been shown to be a risk factor for stress fracture, likely related to osteoporosis. Myburgh and associates [28] showed that athletes with stress fractures had lower bone mineral density in both the appendicular and the axial skeleton. They also showed that these same athletes had lower dietary calcium intake than did control subjects. White race has also been shown to be a risk factor.

DIAGNOSIS

History

Although history and physical examination have classically been the standard for diagnosis of stress injuries, imaging studies have evolved to aid significantly in the diagnosis. Patients sustaining stress fractures of the foot and ankle generally present with a history of insidious onset of a vague, aching pain. The patient usually does not remember a specific traumatic event but may note a recent change in activity level or a change in type or duration of training. For the casual athlete, a new recreational sport or a diet-related exercise program may be the only change. For more highly trained athletes, the change may be as simple as new athletic shoes, a different surface for running, or minor increases in speed or distance. Routine daily activities without a history of strenuous activity may produce insufficiency stress fractures in patients with osteoporosis or underlying diseased bone. [25] Dull, aching pain is characteristically present midway through or near the end of the activity; it usually diminishes with cessation of the activity. The patient can usually localize the pain to a specific area. This pain progresses and eventually may develop into a sharp, severe pain that results from all weight-bearing activities as the stress fracture becomes complete. Although a history of menstrual irregularities certainly aids in the diagnosis of stress fracture, a high index of suspicion is necessary early in the course, especially if a history of increased activity level is not forthcoming.

Physical Findings

The most reliable physical finding is a discrete, localized area of tenderness and swelling over the affected bone. Sixty-six percent of the athletes with stress fractures in Matheson's study had localized tenderness, and 25% had soft tissue swelling.[64] Erythema and warmth may be present but are much less likely. Patients may present with a limp but rarely with muscular atrophy. Joint range of motion is usually not affected. Percussion and vibratory and ultrasonic stimulation have been reported to aid in the diagnosis but are usually unnecessary.[65]

Physical examination should also include assessment of ankle and hindfoot position. Equinus contracture or hindfoot varus can predispose an athlete to increased levels of forefoot or lateral foot stress, respectively. Alignment and range of motion of the knee and hip should be assessed as should leg lengths.

Imaging Studies

A patient's history of temporally related altered activity level, along with findings of localized tenderness, is often sufficient for the diagnosis of stress fracture to be made and treatment initiated. Plain radiographs, however, should still be obtained. Standard three-view studies of the ankle and of the foot are initially performed (anteroposterior, lateral, and oblique views of the foot; anteroposterior, lateral, and mortise views of the ankle). Special oblique studies can be obtained as required by the particular case. Not uncommonly, in fact, more often than not, the initial radiographs are negative, with radiographic changes lagging behind clinical symptoms by at least 2 to 3 weeks. [1022] [1039] Cortical hypertrophy may be present, suggesting a condition of longstanding overload. Linear radiolucencies from osteoclastic resorption may eventually appear. More commonly, increased radiodensity as the body attempts a healing response can be present. Prather and colleagues[8] found radiographic assessment alone to be diagnostic only 64% of the time.

In many patients with nonarticular involvement, diagnosis of stress fracture by history and physical findings, despite negative plain radiographs, is sufficient (and reasonable) for treatment to be initiated. With higher risk lesions or in high-caliber or elite athletes, further diagnostic studies are appropriate. Bone scintigraphy with radionuclide technetium-99m diphosphonate has an advantage because of its ability to demonstrate early physiologic abnormality, thus increasing sensitivity.[66] Bone scans are routinely positive as early as 2 days after injury and may be so as early as 24 hours.[67] The isotope is incorporated into the bone by the activated osteoblasts. Even in subradiographic stress fractures, scintigraphic uptake is usually intense (Fig. 25E-1). The possibility of a false-positive study should be considered because it has been shown that in cases of os trigonum, no correlation was found between technetium-99m diphosphonate uptake and the clinical picture.[68] Some fractures have a characteristic appearance on bone scan, and certain fractures (e.g., navicular, sesamoid, talar processes) often require further anatomic definition with computed tomography (CT).

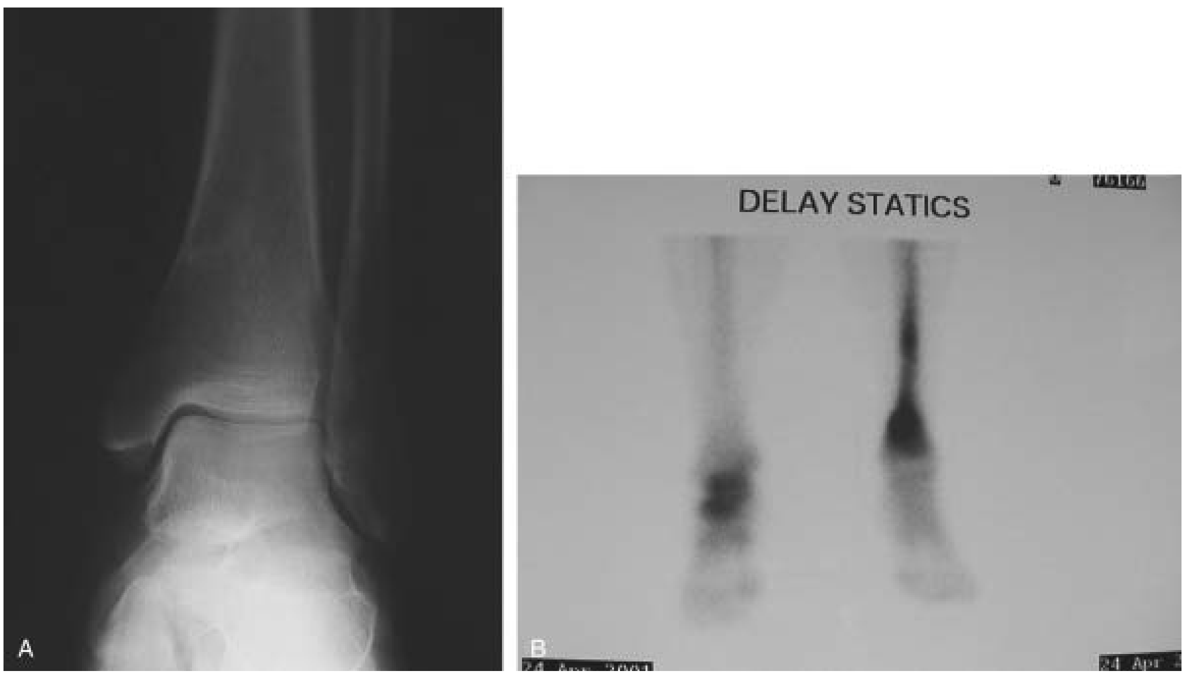

Figure 25E-1 **A,** Minimal radiographic findings with no cortical involvement. **B,** Diagnosis of tibial stress fracture confirmed by technetium-99m bone scan. *(Courtesy of James W. Brodsky, MD.)*

MRI is the most commonly used adjunct in the diagnosis of stress fractures (Fig. 25E-2). Stress fractures have a characteristic appearance on MRI, that is, low signal intensity on T1-weighted images and high signal intensity on T2-weighted images. [69] T1 and short T1 (tau) inversion recovery (STIR) sequence images demonstrate the lesion best when it is out of the acute phase. [70] MRI has the advantage of better accuracy in diagnosis than bone scan in terms of definition of the anatomy of the fracture site. [69] The benefits may be offset by the higher cost, and often the MRI diagnosis does not change the treatment decisions. Thus, this approach should not be used indiscriminately in all patients with suspected stress fracture.

Figure 25E-2 **A** and **B,** Tibial stress fracture: incomplete medial to lateral, but cortical portion facilitates diagnosis. **C,** Magnetic resonance imaging also demonstrates the lesion with high sensitivity. *(Courtesy of James W. Brodsky, MD.)*

With the more frequent use of MRI and considering its high sensitivity, more subtle findings may be addressed as stress fractures. A more rigorous definition of the term *stress fracture* would be reserved for cases in which a fracture line can be identified either on T1, T2, or STIR sequences. Other findings, such as bone edema without a definite fracture line, could be addressed as *stress reaction,* although there is obvious ambiguity between the two terms, presumably because the physiologic process is on a continuum.

TREATMENT PRINCIPLES

The treatment of stress fractures of the foot and ankle may vary somewhat in duration, timing, and the election of aggressive intervention in comparison with the treatment of those fractures caused by acute, discrete traumatic incidents (Table 25E-2). These variations are discussed later according to the specific fractures. The basic principles of fracture care must still be followed, however, to achieve predictable healing. The two most basic of these principles are rest and immobilization of the injured limb. Rest takes on an additional (and negative) nuance of meaning for many athletes in sports-induced stress fractures because it is most often the highly repetitive motions and impacts of the sport itself that induce the injury. Rest, then, does not simply suggest the sensible alteration of everyday activity that comes to mind in the nonathlete; rather, it means that the athlete must severely modify or even suspend the previously intense pursuit of the sport. This can, and often is, met with howls of protest and anger, given the passion or payment associated with participation in amateur or professional sports, respectively. The physician or surgeon is, in turn, subjected to pressure from patient, parent, coach, trainer, or agent (sometimes subtle and often overt) to violate these basic principles so that the athlete may return to the sport as quickly as possible. Sometimes the doctor feels that the demand is not just for a prognosis, nor even treatment, but for something just short of magic, as if the fracture will be healed if the doctor would only acquiesce to a quick return to the sport. In addition to the science discussed elsewhere in this chapter, successful treatment usually requires the skillful application of the art of medicine, encompassing that unique blend of rational decision making, sympathetic coercion, and thoughtful compromise.

TABLE 25E-2 -- Authors' Preferred Method of Treatment and Time to Return to Sport

Stress Fracture	Treatment	Return to Sport (mo) [*]
Tibia	Conservative—NWB	4-5
Medial malleolus	Open bone grafting and plate fixation	3-4
Distal fibula	Conservative—WBAT in a CAM walker	2-3
Calcaneus	Conservative—NWB	2-3
Talus	Conservative—NWB	3-6
Navicular	Open bone grafting and fixation with two lag screws	4-6
Metatarsals 2-4	Conservative—WBAT in a CAM walker	1.5-2
Proximal metaphyseal-diaphyseal junction of the 5th metatarsal	Early (no sclerosis)—closed fixation with an intramedullary screw Late (sclerosis)—open bone graft and intramedullary screw fixation	3-4
Medial sesamoid	Conservative—off-loading orthosis; if displaced fracture, sesamoidectomy and FHB reconstruction or bone grafting	2-4

FHB, flexor hallucis brevis; NWB, non–weight-bearing; WBAT, weight-bearing as tolerated.

* Approximate time from treatment.

Rest, then, signifies decrease in, or cessation of, the sport, together with the judicious use of cross-training, so generalized deconditioning is minimized while the specific injury to the limb is protected from further microtrauma and repetitive stress.

Traditionally, immobilization has signified cast application. The advent of prefabricated braces, orthoses, and walking boots, as well as rigid-soled shoes, has made it easier to comply with immobilization. Although the use of casts has been supplanted in many cases, there are still many instances in which the superior immobilization provided by a cast is required in treatment of the fracture. The advantage of the removable devices is that even in fractures that require strict non–weight-bearing, the benefit of being able to remove the device for bathing is exceeded by the benefit of the patient being able to perform active range of motion exercises to maintain some muscle tone and bulk and to reduce joint stiffness and the period of rehabilitation once the fracture is healed. Most stress fractures of the foot and ankle heal by the application of these two principal treatments alone. Patient compliance with instructed use should never be forgotten as possible cause of treatment failure when using removable orthotic devices and other forms of treatment with a discretionary component.

Dietary change and metabolic treatment are important adjuncts to treatment of stress fracture in the patient with insufficiency fracture, less for the healing of the specific stress fracture than for the prevention of future stress fractures because stabilization or reversal of the disease process is slow. Fractures in osteoporotic bone are not slower to heal than those in normal bone; however, they heal with the same porotic bone. Dietary and metabolic interventions are frequently supervised by an internal medicine or endocrine specialist. These include adequate intake of calcium with vitamin D in a diet that has sufficient calories and protein to ensure that the patient is not in a catabolic state. These interventions also frequently include hormone replacement therapy in postmenopausal women (unless there is a medical contraindication such as a history of estrogen-sensitive breast cancer) and often the use of bisphosphonates such as alendronate (Fosamax), risedronate (Actonel), and ibandronate (Boniva), or the parathyroid hormone–like drug teriparatide (Fortéo). The use of osteoblast-inhibiting medications as a preventive measure has not shown to reduce the risk for stress fracture. [60]

Although surgery is required in the minority of stress fractures of the foot and ankle, the need for surgery follows the same basic indications as for other fractures: nonunion, malunion, or displacement that would lead to either of the two. In practical terms, there are several stress fractures of the foot and ankle for which surgery is more the rule than the exception, although the opposite is true for the rest. Those fractures that frequently require surgery are vertical stress fractures, distal medial tibia fractures, stress fractures of the tarsal navicular, and Jones' fifth metatarsal fracture at the junction of the proximal metaphysis and diaphysis of the fifth metatarsal. Fractures that not infrequently require surgery are stress fractures of the base of the second metatarsal, disruption of the synchondrosis between the accessory and primary navicular bones, and stress fractures of the sesamoids of the hallux. Specific recommendations are discussed in the following sections.

It should be emphasized to all patients with fractures, injuries, or surgeries of the foot or ankle that it is typical to have a great deal of swelling, and for the swelling to be persistent, often lasting 6 months or longer.

SPECIFIC FRACTURES AND THEIR TREATMENT

Ankle Fractures

Stress fractures of the supramalleolar area are relatively uncommon. Fractures of the distal tibial metaphysis are most often associated with overuse, usually in the absence of deformity. Factors that increase the risk for a distal tibial stress fracture include previous prolonged immobilization, change in the modulus of elasticity of the bone caused by adjacent internal fixation, osteoporosis, and sudden increase in physical activity. Radiographic appearance often does not include a cortical component of the fracture. The initially negative radiographs later demonstrate condensation of new bone within the diaphysis (see Fig. 25E-2 A and B), which seldom progresses to a complete fracture or to any discernible displacement (Fig. 25E-3). Treatment consists of immobilization and patience because these tend to be slow to heal. An initial period of non–weight-bearing of 6 to 8 weeks is followed by weight-bearing immobilization until symptoms subside entirely. Weaning out of the removable walking brace is followed by participation in a rehabilitation program.

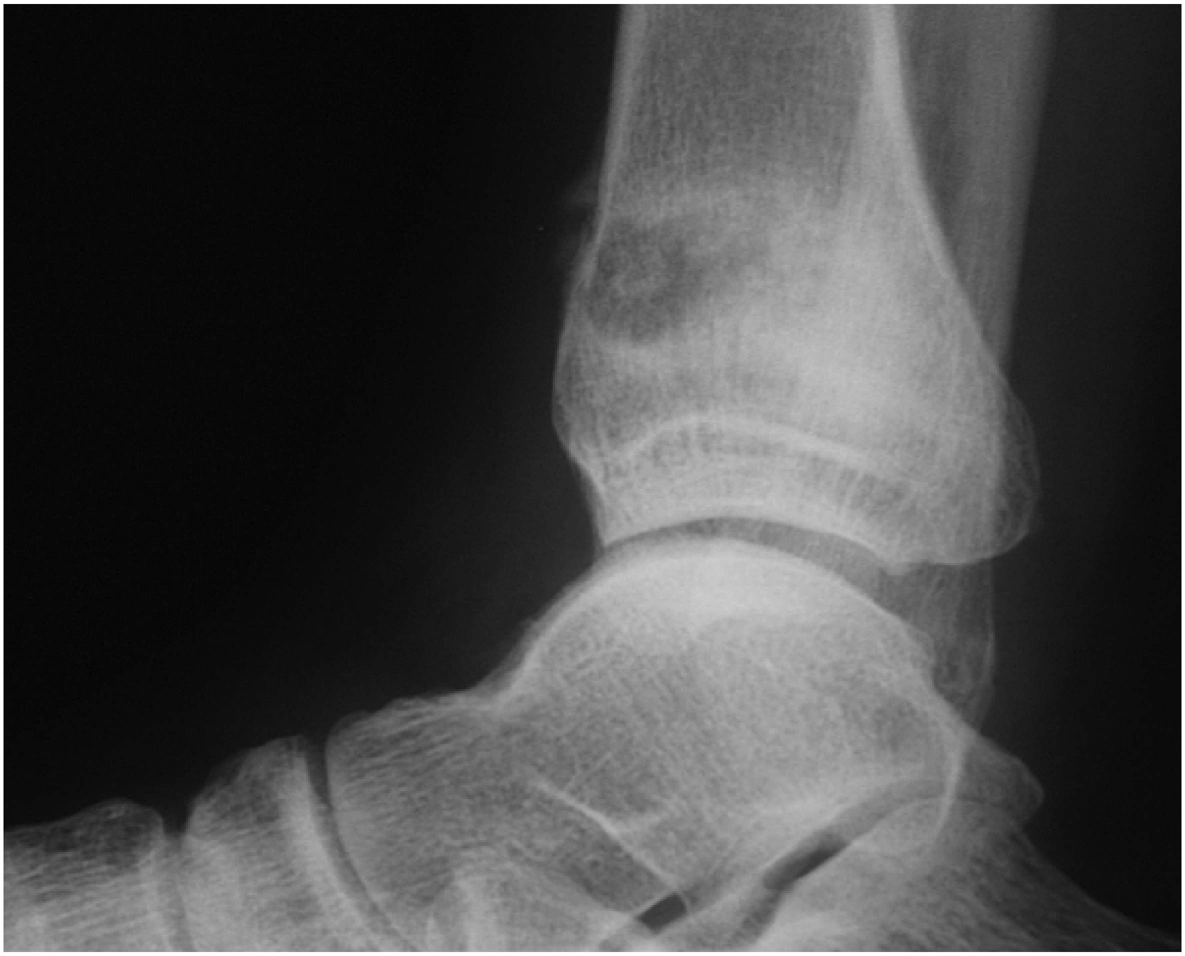

Figure 25E-3 Less typical finding of cystic resorption at tibial stress fracture site. *(Courtesy of James W. Brodsky, MD.)*

Vertical fractures of the distal medial tibia, including and extending superiorly from the medial malleolus, require special consideration of mechanical factors. Vertical forces involved are apparent relative to orientation of the fracture line. Predisposing factors include varus at the ankle or of the hindfoot that causes wedge-like force from the medial corner of the talus against the medial ankle mortise, although this is not always present. Open reduction with internal fixation requires an antiglide buttress plate to counteract the vertical forces. Two screws in the medial malleolus are usually inadequate and likely to fail (see Fig. 25E-6). Figure 25E-4 shows a medial malleolus fracture before displacement in high school quarterback. The plate was placed percutaneously as an outpatient procedure, and the patient returned to play after 3 months. The symptoms subsided completely.

Figure 25E-4 A, An intra-articular, vertical stress fracture of the medial malleolus in a high school quarterback. B, Healed fracture, one year after percutaneous planting of the fracture.

In contrast to distal tibial fractures, stress fractures of the distal fibular diaphysis and metaphysis are not infrequently associated with deformity. Typically, this would be a valgus hindfoot that increases biomechanical stress on the distal fibula (Fig. 25E-5). Otherwise, the same predisposing factors apply as those enumerated in the discussion of stress fractures of the distal tibia. As early as 1940, Burrows subclassified stress fractures of the distal fibula into two groups—young male athletes with fractures 5 to 6 cm proximal to the distal tip of the lateral malleolus, and middle-aged women with fractures 3 to 4 cm proximal to the malleolar tip (Fig. 25E-6). [1039] [1040] [1085] Treatment in the two groups is substantially the same, that is, immobilization with or without activity modification. In the second subgroup, consideration should be given to metabolic status and appropriate diagnostic tests, or referral should be considered. As with all stress fractures, clinical healing may precede completion of radiographic fracture healing. Surgery is seldom required.

Figure 25E-5 This patient with valgus hindfoot (**A**) developed a fibular stress fracture (**B**) after initiation of a walking program. *(Courtesy of James W. Brodsky, MD.)*

Figure 25E-6 Finding in a 50-year-old physician with ankle pain after inception of a new jogging regimen. **A** and **B,** Initial radiographs. **C,** After 7 weeks of immobilization, note cystic resorption and new peripheral bone formation. *(Courtesy of James W. Brodsky, MD.)*

Fractures of the medial malleolus, which are even less common than supramalleolar fractures of either the tibia or fibula, tend to be fatigue-type fractures due to overexertion in athletes or patients initiating a new exercise program. Although general treatment principles should be followed, treatment is more likely to be surgical because of a greater proclivity for nonunion. Nonsurgical treatment with immobilization can be used initially in most patients. Exceptions include fractures that are fully intra-articular, displaced fractures, and fractures in selected professional or other serious athletes who require the fastest possible return to sport. Depending on the facture pattern, percutaneous screw fixation may not be sufficient mechanical stabilization, especially if the fracture has displaced or has developed a delayed union or nonunion. Medial plating and bone grafting is usually required in cases of nonunion or delayed union.

Illustrated in Figure 25E-7 is a professional basketball player with recurrent stress fractures of both medial malleoli. Neither pedobarographic analysis nor gait laboratory evaluation revealed an underlying biomechanical abnormality to explain the occurrence of the fractures. Treatment in this individual was variably successful, with failures and recurrences both after periods of immobilization and after internal fixation with and without bone grafting.

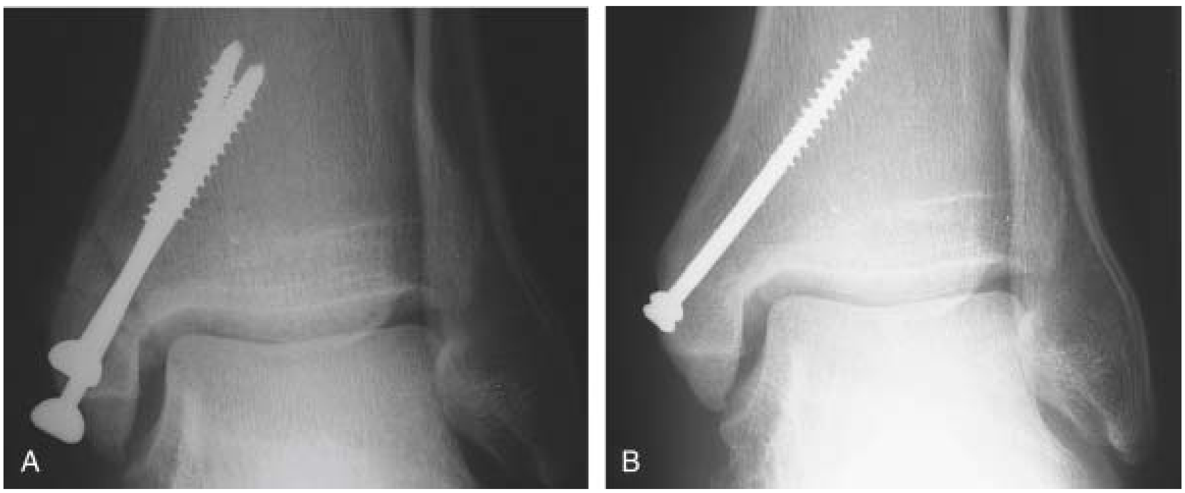

Figure 25E-7 Professional basketball player with bilateral recurrent stress fractures of the medial malleolus. **A,** Nonunion after open reduction with internal fixation. **B,** Healing after revision of fixation and bone grafting. This degenerated again to a recurrent stress fracture after resumption of play but rehealed with conservative therapy. *(Courtesy of James W. Brodsky, MD.)*

Fractures of the Hindfoot and Midfoot

Stress fractures of the talus are relatively uncommon and are seen most often in runners and military recruits but can occur in dancers and jumping athletes such as gymnasts. Fractures classically occur in the talar neck, but several reports describe talar process (i.e., lateral, posteromedial, posterolateral) stress fractures as well as talar body stress fractures in gymnasts. [72] Treatment is usually nonoperative, but the challenge is to make the diagnosis promptly. Plain radiographs are typically unrevealing, and an imaging study such as MRI or technetium scanning is required in most cases. Misdiagnosing talar osteochondral lesion as a stress fracture should be avoided. Making the diagnosis can be difficult owing to the misleading, deep, but vague localization of the patient's pain. Healing time varies, averaging between 3 and 4 months, but occasionally longer.

Fractures of the posterolateral process of the talus and disruption of the synchondrosis between an os trigonum and the posterior body of the talus are also uncommon injuries. Scintigraphy has been shown to be nondiagnostic in these instances because 63% of recruits who had technetium-99m diphosphonate uptake at the os trigonum had no pain over that area. [68] They are most typically associated with classical ballet because of the hyperplantar flexion of the tibiotalar joint when the female dancer is dancing on toe (*en pointe*) (Fig. 25E-8). [1087] [1088] An important variation of stress fracture is the posterior impingement of the os trigonum against the posterior tibia, which causes direct mechanical pain (

Fig. 25E-8). This often requires excision of the os trigonum through a posterior or posterolateral approach, usually with excellent results. [1087] [1088] [1089]

Figure 25E-8 Professional ballerina with posterior ankle pain when dancing *en pointe*. Comparison of lateral radiographs in standing (**A**) and "on-toe" (**B**) positions reveals not only compression of the os trigonum but also displacement of the os trigonum away from the talus as well as anterior subluxation of the tibiotalar joint. *(Courtesy of James W. Brodsky, MD.)*

Stress injuries of the lateral process of the talus are unusual. Cases of acute fracture, although uncommon, are much more common than is stress injury. An example of an MRI of a stress injury of the lateral process is demonstrated in Figure 25E-9 . The treatment of these fractures should be tailored according to the duration of symptoms, the displacement, and whether there are the radiographic characteristics of nonunion. Cases in which a sclerotic line is found on CT require surgical intervention with excision of the fragment unless it is a large intra-articular fragment, for which bone grafting and internal fixation is the treatment of choice.

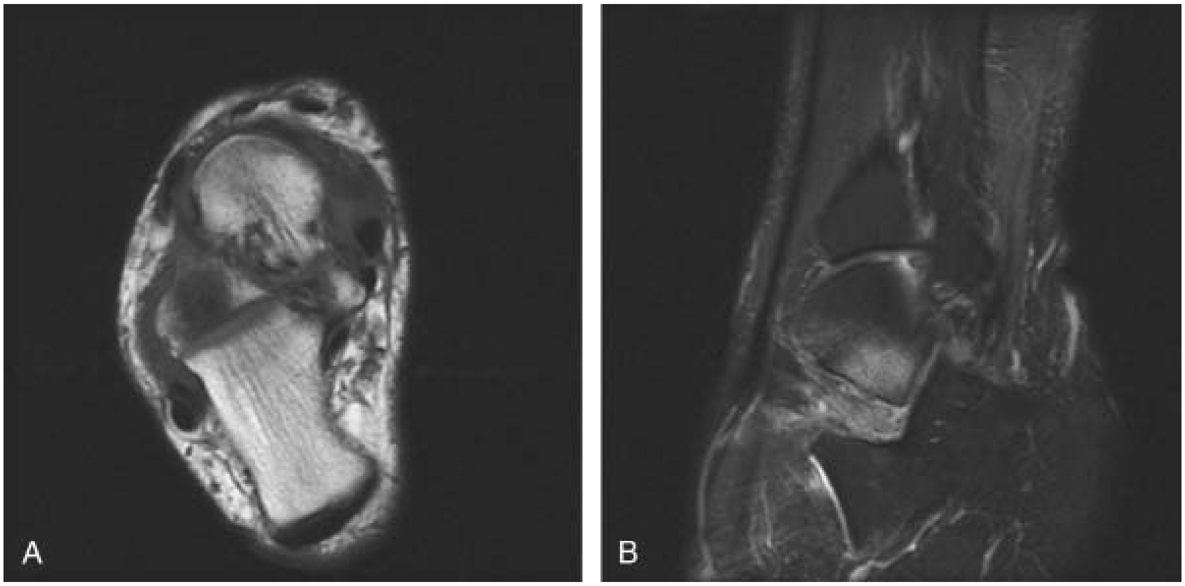

Figure 25E-9 This athlete underwent magnetic resonance imaging for persistent lateral hindfoot pain. T1-weighted (**A**) and T2-weighted (**B**) sequences demonstrate the marrow edema indicative of a nondisplaced stress fracture of the lateral process. *(Courtesy of James W. Brodsky, MD.)*

Although calcaneal pain is one of the most common if not the single most common affliction of the foot in both athletes and nonathletes, few of these cases are true stress fractures. Most heel pain in both groups is attributable to soft tissue conditions that are frequently manifested at the interface between tendon or ligament and bone, but are not stress fractures, nor even true osseous lesions. These diagnoses typically include plantar fasciitis, insertional Achilles tendinitis, plantar heel pain syndrome, and nerve entrapments. The symptoms may correspond to the anatomic location of these structures, most commonly pain on the plantar surface of the heel or on the posterior aspect of the heel. Owing to their extreme rarity in relation to other common heel pain diagnoses, stress fractures of the calcaneus are frequently overlooked because the initial radiographs are nearly always negative, and the pain is frequently quite diffuse and nonspecific. Although imaging studies such as MRI or technetium bone scan may be required to make the diagnosis, repeat radiographs after an interval of several weeks eventually show the typical, albeit often subtle, radiographic appearance. Calcaneal stress fractures are typically incomplete and manifest as a vertical condensation or radiodensity within the cancellous bone of the calcaneal tubercle. This usually occurs in the dorsal two thirds, as can be seen on the lateral standing radiograph of the foot, and does not extend down to the plantar cortex (Fig. 25E-10). Rarely, the fracture might be posterior in the tubercle (Fig. 25E-11). Recently, a review of 44 cases of calcaneal stress fractures using MRI found a certain percentage of stress fractures to appear in the middle and anterior part of the calcaneus [62] as well as a case report of a stress fracture of a calcaneonavicular coalition in a runner. [48] Changes on the imaging studies correspond to the same anatomic distribution as that described for radiographs. Technetium bone scans that show increased uptake on the plantar surface of the calcaneus do not signify stress fracture, but rather are typical of plantar fasciitis. The inflammatory process at the insertion of the plantar fascia involves the periosteum and Sharpey's fibers, which produce the positive uptake on the scan.

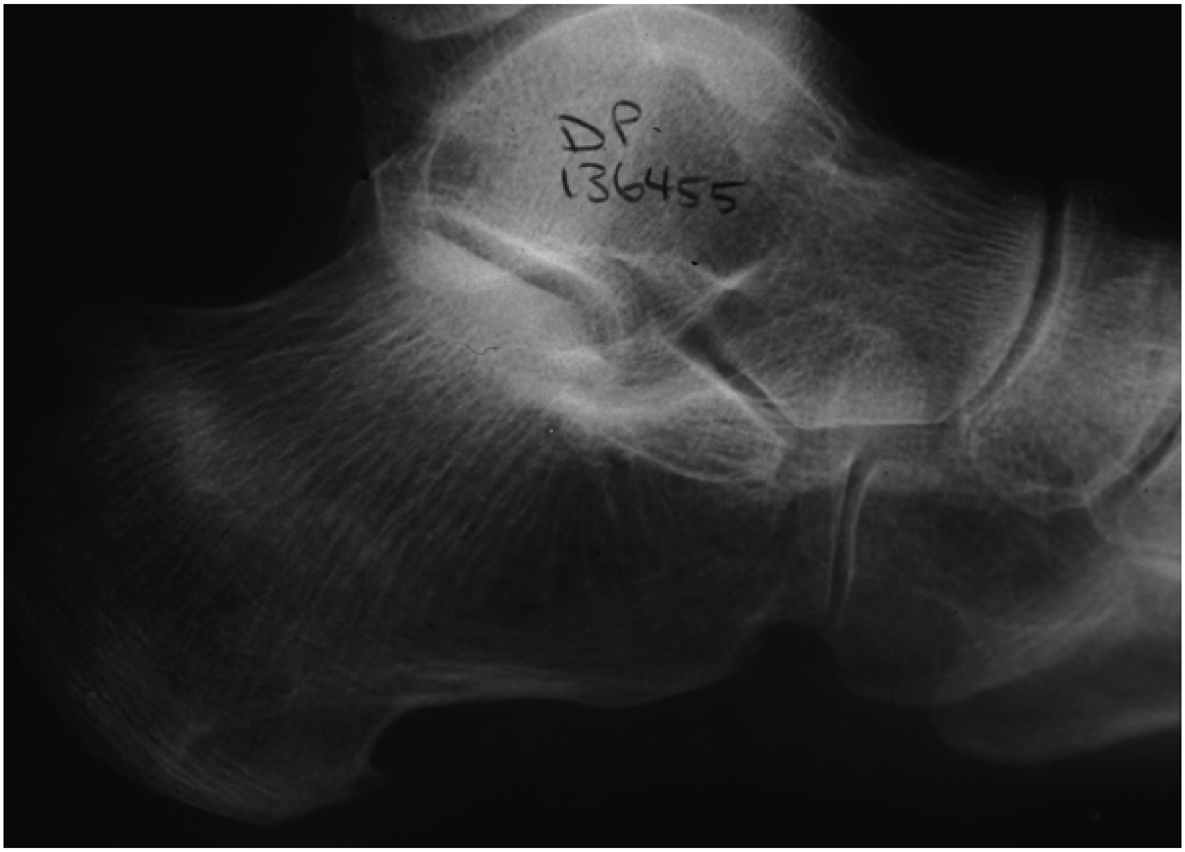

Figure 25E-10 Calcaneal stress fracture in a runner. The propagation of the fracture from the superior cortex of the tubercle is the most common pattern. *(Courtesy of James W. Brodsky, MD.)*

Figure 25E-11 Persistent heel pain in this 29-year-old woman was initially attributed to plantar fasciitis at another institution, where her radiographs were negative. After an interval of 4 weeks, the stress fracture of the calcaneal tubercle became apparent. *(Courtesy of James W. Brodsky, MD.)*

On close physical examination, the distinguishing characteristic of calcaneal stress fracture is that the pain is dorsal and anterior on the calcaneal tubercle, and the patient will point to the medial and lateral sides of the heel anterior to the Achilles tendon as the center of the pain. Treatment consists of diminished activity with variable immobilization until symptoms subside, usually between 2 and 3 months.

Stress fractures of the tarsal navicular are the most difficult stress fractures of the foot and ankle to treat because of the typically slow and recalcitrant healing. Presentation is one of vague, poorly localized pain that is gradual in onset. On careful questioning, the patient will point to the dorsum of the hindfoot, but the examiner can easily be misled into thinking that the pain emanates from the adjacent tibiotalar joint. These relatively uncommon fractures occur primarily in running athletes. The largest series to date is that undertaken by Torg and associates.[34] They showed that all 21 of the fractures in their study were in the sagittal plane, in the central one third of the navicular. Strict enforcement of non–weight-bearing has a markedly positive effect on healing of the fracture.

The routine, three-view foot radiographic evaluation is frequently unrevealing because the navicular is underpenetrated and the fracture is in the sagittal plane. Cone-down views assist in diagnosis. Telltale radiographic signs include sclerosis in the subchondral bone adjacent to the talonavicular joint. Although bone scan or MRI adequately screens for the presence of a navicular stress fracture, the study of choice is CT. Sections in two planes are recommended. Images in the coronal plane should be parallel to the talonavicular joint and roughly parallel to the plantar surface of the foot. The typical appearance of the fracture on CT is a vertical fracture line (in the sagittal plane) that begins on the dorsum of the navicular, proceeds plantarward, and is almost always incomplete (Fig. 25E-12 A to C). Although Torg and associates [34] described these as occurring in the central third, the location is usually more lateral than medial, and often at the junction of the medial two thirds and the lateral third. Sclerosis often is noted on both sides of the fracture, depending on the age of fracture, which in the horizontal plane extends to and through the proximal subchondral bone of the navicular at the talonavicular joint. The location of the fracture line has been demonstrated to correspond to a zone of avascularity in the tarsal navicular. Microangiographic studies showed the central one third of the navicular to be relatively avascular. [34] The intraosseous blood supply has been shown to enter from medial and lateral poles of the bone, and to diminish in the zone where the fracture occurs.

Figure 25E-12 A, Defensive lineman with vague, persistent hindfoot pain. Subtle linear lucency in the navicular is seen on the radiograph. **B** and **C,** Computed tomography confirmed the diagnosis. **D,** The patient was treated with internal fixation and bone grafting. *(Courtesy of James W. Brodsky, MD.)*

Tarsal navicular stress fractures frequently require surgical intervention because diagnosis is commonly delayed. If treatment is initiated early, an attempt at non–weight-bearing immobilization may be tried. Surgical treatment consists of bone grafting and internal fixation; the surgical procedure is technically challenging. The incision should be on the dorsolateral aspect of the hindfoot, corresponding to the central one third of the navicular bone. The fracture site is exposed and carefully débrided. The major pitfall is to lose the three-dimensional orientation of the navicular bone, so one must remember that the medial and lateral parts of the articular surface (and cartilage) are more proximal, and the articular surface is farther distal in the midportion of the bone. Thus, it is easy to penetrate the central portion of the proximal articular surface, damaging the talonavicular joint. Further risks to the joint are posed by the placement of the internal fixation screws, again attributable to the sharply curved proximal articular surface of the navicular. The screws are best and most easily placed from lateral to medial because the medial fragment is larger, thus affording a greater area for placement of the screw threads. For damage to the talonavicular joint to be avoided, thoughtful screw placement requires a more distal starting point on the lateral aspect of the navicular (see Fig. 25E-11 D). It is advisable to place the screws under fluoroscopic control; the process may be facilitated by the use of 3.5- or 4.0-mm cannulated screws, placed over guidewires. It is necessary to warn the patient about the typically prolonged healing time of 3 to 5 months for this fracture.

Postoperatively, non–weight-bearing immobilization in a cast is recommended for at least 8 weeks, followed by a walking cast for at least 1 month, and a removable walking boot thereafter, with modification based on the rate of fracture healing, as judged by radiographs, and if necessary, repeat computed tomographic scan.

Disruption of the synchondrosis between the accessory navicular and the medial tubercle of the navicular is uncommon but must be mentioned among the stress injuries of the hindfoot. Although this may occur as a result of chronic loading in a running athlete, the examiner's index of suspicion should be raised, especially in jumping athletes such as hurdlers or long-jumpers. Because this is the site of attachment of the tibialis posterior tendon, this injury also occurs as a result of acute forced eversion. The injury also may be subacute or superimposed on chronic trauma. Physical examination reveals tenderness at the medial hindfoot, overlying the most distal portion of the tibialis posterior tendon, right at its insertion. This tenderness is exacerbated by passive eversion and active inversion. There may be increased medial prominence and swelling. Radiographic diagnosis may be delayed because of difficulty in visualizing the accessory navicular on routine foot radiographs. A reverse oblique (medial oblique) plain radiograph is the view of choice. Even excellent radiographs can be inadequate in assessing whether the attachment of the accessory navicular has been disrupted. The plane between the accessory navicular and the tuberosity of the navicular is oblique in all three planes, making it difficult to assess if it has retracted proximally. Technetium-99m bone scan is the screening test of choice, if making this determination is not possible on radiograph and physical examination. Highly collimated views with appropriately adjusted gain are necessary for localization of the increased scintigraphic uptake to the synchondrosis (Fig. 25E-13).

Figure 25E-13 Images of a 15-year-old female basketball and softball player with persistent medial pain over the hindfoot. Technetium-99m bone scans (**A** and **B**) and comparison view of the contralateral foot (**C**) confirmed disruption of the synchondrosis between the accessory navicular and the navicular. *(Courtesy of James W. Brodsky, MD.)*

Surgery consists of advancement of the tibialis posterior tendon to prevent incompetence and an acquired pes planus. This may be accomplished by excising the accessory navicular, then reattaching the tendon directly to the medial navicular tubercle, or by arthrodesis of the accessory navicular to the medial tubercle. The latter is possible only if the accessory bone is large (type II). The result is enhanced if the medial tubercle of the navicular is reduced in size, which serves to tighten the tendon by advancing the insertion distally and laterally, while reducing the external prominence on the medial border of the hindfoot. Either way it is essential to advance the tibialis posterior tendon to ensure it is reconstructed under sufficient tension, that it will be subsequently functional. It is wise to err on the side of greater than lesser tension in the reconstructed tendon.

Stress fractures of the cuneiforms are reported but rare, and they require conservative management as outlined previously. Stress fractures of the middle and lateral cuneiforms have been reported more frequently than those of the medial cuneiform, presumably because of the propagation of forces from the bases of the second and third metatarsals. [1023] [1046] Stress fractures of the cuboid are somewhat less rare but nonetheless very uncommon. One should look for predisposing mechanical factors that cause overload of the lateral column of the foot, such as subtalar ankylosis or a varus position of the hindfoot or ankle, or as a complication after plantar fascia disruption. [76] As with cuneiform stress fractures, the cancellous nature of the cuboid makes healing very likely. Once healing has resulted from conservative measures, consideration should be given to the use of a soft-soled but supportive shoe, in addition to a custom-molded, cushioning, accommodative insole to compensate for the stiffness or varus.

A rare stress fracture is a fracture of the os peroneum that is embedded in the peroneus longus tendon as it passes under the cuboid. Only a few cases of stress fracture of the os peroneum have been reported in the literature. [1091] [1092] Some have been associated with a peroneus longus tear, whereas others were shown to be associated with an intact tendon. [78]

Metatarsal Fractures

Stress fractures of the metatarsals are the most common stress fractures of the lower extremity. [15] These occur commonly in military recruits, dancers, and other athletes. The most common metatarsal stress fractures occur in the shaft (diaphysis) or neck of the bone. Although insufficiency fractures are known to occur in the metatarsals, most metatarsal stress fractures seen in clinical practice are of the fatigue-type. The most common history involves a precipitous increase in the duration and intensity of weight-bearing sports or exercise activity, such as a sudden doubling of running distance, or the institution of a new and vigorous exercise program in previously sedentary individuals. As with other stress fractures, the initial radiographs are negative more often than not. The diagnosis can usually be made on the basis of the history described earlier in the paragraph, a high index of suspicion, tenderness to palpation directly over the metatarsal (more so than the intermetatarsal web space), and one specific physical finding, that is, the presence of a well-circumscribed swelling only over the dorsum of the forefoot that does not extend to the medial or lateral border of the foot (Fig. 25E-14). Pain is almost always described as dorsal in location.

Figure 25E-14 Classic metatarsal stress fracture. **A,** The initial film shows a minor crack in the diaphyseal cortex. **B,** Subsequent radiograph shows the exuberant callus formation. **C,** Note the foot swelling, especially on the dorsum. *(Courtesy of James W. Brodsky, MD.)*

Treatment, besides attention to predisposing conditions, is symptomatic and usually consists of a rigid-bottom surgical shoe or a stiff-soled shoe and cessation of inciting activities (e.g., dancing, running, marching). Healing time is variable, but on average, resumption of the use of normal shoewear begins between 6 and 8 weeks after onset. Advancement to unrestricted shoewear may require several months, depending on the severity of swelling and the rate at which it recedes.

The primary complication of metatarsal stress fractures is displacement with subsequent malunion, and its primary result is transfer metatarsalgia. In occasional cases, surgery might be warranted to reconstruct such a forefoot situation, although shoe modification with a cushioning orthosis would usually be tried first. The reconstruction could include plantar flexion osteotomy and bone grafting of the malunion, with or without elevating osteotomy of the symptomatic adjacent metatarsal.

Fractures of the proximal fifth metatarsal are of two types, and improper use of the eponyms by some authors has led to unnecessary confusion of these distinct entities. The most common fracture of the foot and ankle is fracture of the base of the fifth metatarsal through the cancellous bone of the proximal metaphysis (properly known as a *dancer's fracture*) (Fig. 25E-15). These are acute fractures (not stress fractures) that are sustained by a sudden inversion mechanism, which is similar to the twisting injury that produces damage to the lateral collateral ligaments of the ankle. Most of these injuries usually do not extend into the metatarsal-cuboid joint. Most are mildly or minimally displaced and need only conservative treatment, which consists of immobilization. Treatment of choice is usually a removable below-knee walking boot for 6 to 8 weeks. It is important to point out to the patient that there is always a lag between clinical and radiographic healing. Persistent lucencies at the fracture site are common, presumably owing to the displacement of the fracture and the time needed to fill in. Nonunion cannot be diagnosed before 6 months after the initial fracture. Complete evidence of radiographic healing is usually not present until after 3 to 4 months.

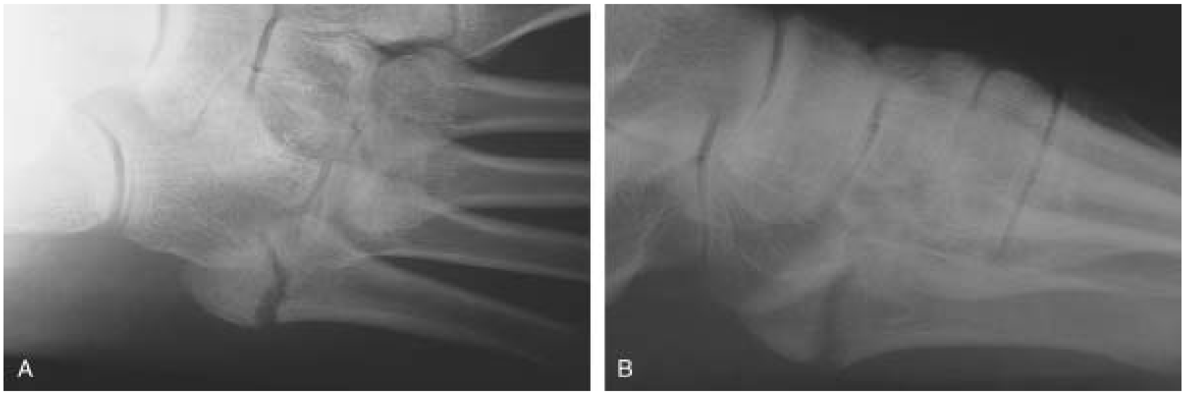

Figure 25E-15 A “dancer’s fracture.” This is the most common foot fracture, that is, avulsion fracture of the base of the fifth metatarsal associated with acute inversion injury. *(Courtesy of James W. Brodsky, MD.)*

Surgical intervention is required if there is severe displacement, especially if the proximal fragment is highly rotated, but this is uncommon. Most cases are treated nonoperatively.

The second type of fracture is properly referred to by the eponym of *Jones’ fracture*, in reference to its original report by Sir Robert Jones, [79] in which he described the injury as it occurred in his own foot. It is a fracture of the metaphyseal-diaphyseal junction. Although many of these fractures have an acute component as in the original fracture, many, if not most, are superimposed on chronic stress injury to the bone. This injury is an incomplete stress fracture, which explains the intense bony sclerosis, hypertrophic bone formation, and incomplete nature of the fracture that is seen on the radiograph even immediately after the acute twisting injury. The fracture is based plantar and lateral, with the dorsomedial cortex often intact (Fig. 25E-16).

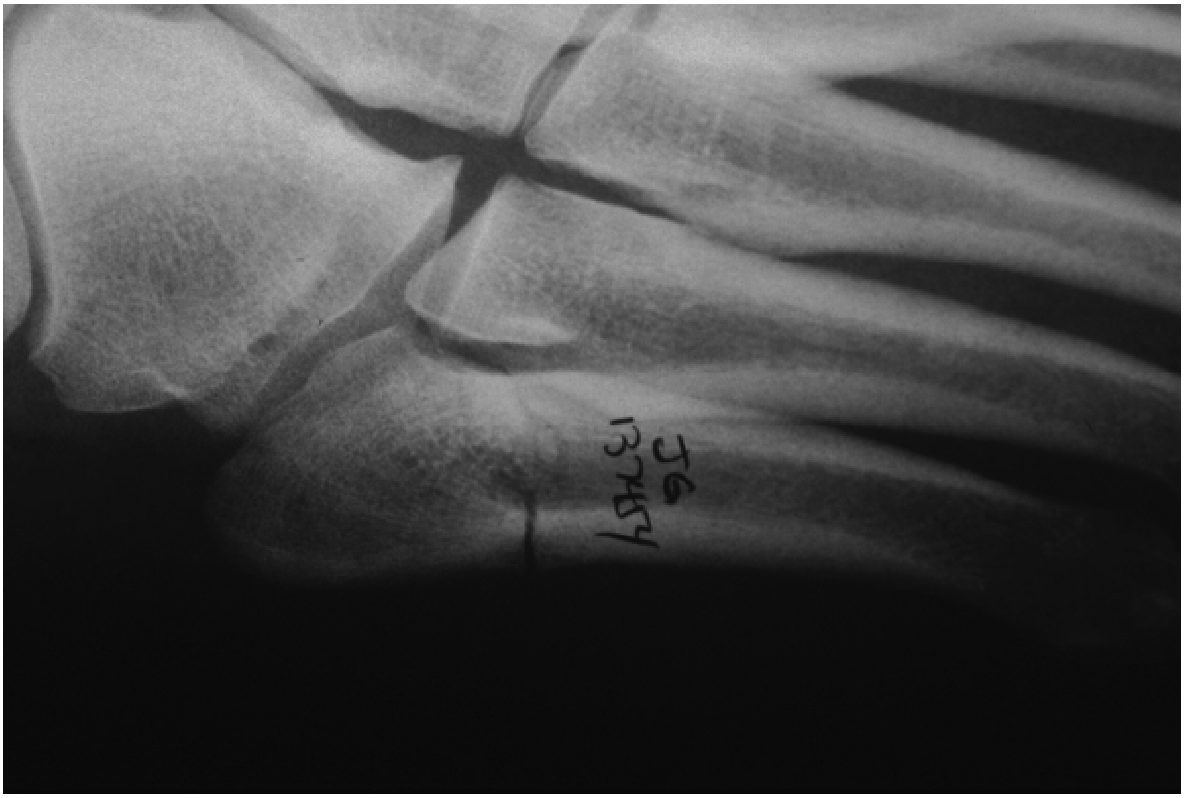

Figure 25E-16 Acute Jones' fracture at the diaphyseal-metaphyseal junction of the fifth metatarsal. *(Courtesy of James W. Brodsky, MD.)*

Nonoperative treatment is appropriate for injuries with an acute component in patients who do not have an established pseudarthrosis or chronic bony sclerosis at the margins of the fracture on radiographic evaluation. Nonoperative treatment consists of immobilization in a cast or below-knee boot with strict non–weight-bearing for at least 6 to 8 weeks. A significant number of these fractures heal without surgery, but radiographic consolidation takes at least 3 months on average. Fractures with less chronic stress change (as judged by less sclerosis and less hypertrophic spurring) are more likely to heal nonoperatively; surgical treatment is more often necessary in fractures with evidence of chronic stress injury. Surgical treatment is indicated in cases in which nonoperative treatment has failed, as determined by persistent, unresolving pain or established pseudarthrosis, or in which there is a need to return a high-performance athlete to his or her sport as quickly as possible. In many athletes, it is almost routine to treat these fractures with early surgery because it provides speedier and more reliable healing, especially in the middle of an athletic season. The surgery typically consists of internal fixation with an intramedullary screw (usually between 4.5 and 6.5 mm diameter) placed from the proximal tubercle in a distal direction past the fracture site (Fig. 25E-17). Although several authors have recommended the use of cannulated 4.5-mm screws [1094] [1095] in some cases, cannulated screws will bend and break either in cases of nonunion or a recurrent stress fracture. This may be augmented by débridement and bone grafting of the fracture site, especially in cases with evidence of a hypertrophic nonunion, or chronic stress injury. Six weeks of cast immobilization and non–weight-bearing is recommended after surgery, followed by appropriate progression, based on radiographic findings, to weight-bearing in a cast followed by a below-the-knee boot. In high-performance athletes, it is recommended that the screw be retained after healing, as long as the player continues to compete, because of the risk for recurrent fracture. Once the fracture is healed, strong consideration should be given to the use of a supportive athletic shoe and a custom-molded cushioning orthosis. A good example is a dual-density device made of heat-molded polyethylene foam (e.g., medium-grade Plastazote or Pelite) combined with non-deforming open-cell foam (e.g., Poron). To the plantar surface of the orthosis should be added a lateral wedge to counter the varus thrust of the hindfoot and to facilitate the transfer of weight to the medial side of the midfoot.

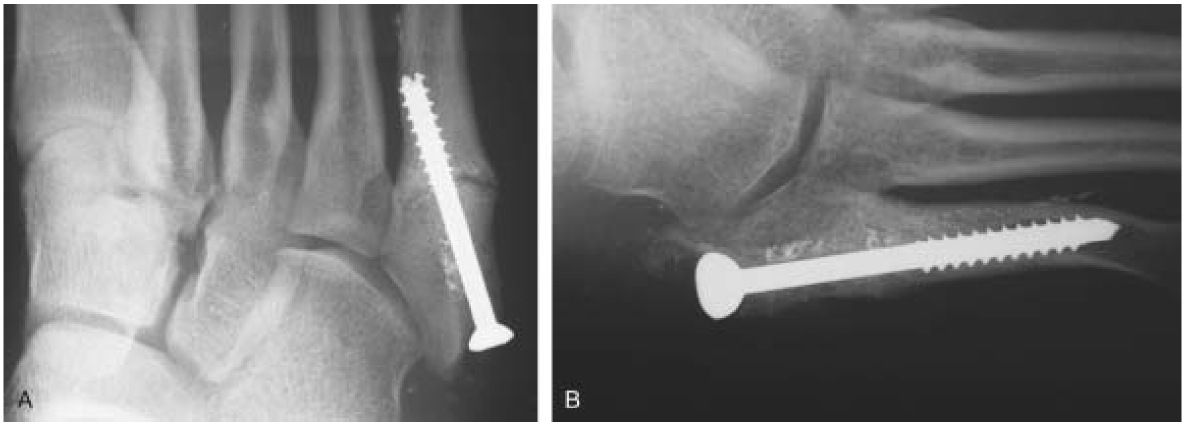

Figure 25E-17 Images of an 18-year-old collegiate soccer player with recurrent pain and nonunion of Jones' fracture after previous surgery. **A,** Note the hypertrophic beaking and sclerosis at the fracture site. **B,** Healing was achieved 10 weeks after revision intramedullary fixation and bone grafting. *(Courtesy of James W. Brodsky, MD.)*

Risk factors for Jones' fracture include a high level of activity in a running or jumping athlete. This fracture is typically seen in soccer and basketball players, although its occurrence is not exclusive of other sports and activities. Athletes with hindfoot varus of any cause have a predilection for this fracture because of lateral column overloading.

Basilar metatarsal stress fractures are less common among the general population but are the most common stress fracture seen in ballet dancers. [1033] [1044] The typical location is at the proximal metaphyseal-diaphyseal junction, with possible extension into the tarsometatarsal joint. Multiple theories exist as to the mechanism for a basilar metatarsal fracture; all these theories center around the *en pointe* position.

Fractures of the bases of the metatarsals are less common than are those of the diaphysis and neck, but they are not rare. [30] Most typically, this fracture occurs at the base of the second metatarsal (Fig. 25E-18). By the time it is diagnosed, radiographs demonstrate sclerosis without displacement. The patient gives a history of chronic aching in the midfoot region, which must be distinguished from tarsometatarsal arthritis. These fractures are chronically symptomatic to varying degrees, although they seldom heal with immobilization alone. Most require surgical intervention with bone grafting, with or without internal fixation. If the fracture is very close to the second tarsometatarsal joint, it may be technically impossible to achieve fixation to the narrow proximal fragment. In these cases, it may be necessary to do an arthrodesis of the adjacent joint in order to gain stabilization and fixation (Fig. 25E-19). An unusual case of a stress fracture of the base of the first metatarsal in a skeletally immature boy that involved the physis and the first tarsometatarsal joint (Salter-Harris III) was reported. This fracture healed with conservative measures, [82] but stress fractures of the first metatarsus are rare.

Figure 25E-18 A 66-year-old woman with large joint osteoarthritis developed this stress fracture of the proximal second metatarsal while participating in a walking program for knee rehabilitation. Note the chronic hypertrophy of the metatarsal. *(Courtesy of James W. Brodsky, MD.)*

Figure 25E-19 A, Image of a 27-year-old woman with persistent dull midfoot pain. The correct diagnosis of stress fracture of the base of the second metatarsal was made after 1 year of multiple consultations. B, Operative treatment required bone grafting as well as arthrodesis of the metatarsal-cuneiform joint. *(Courtesy of James W. Brodsky, MD.)*

Stress injury to the hallux sesamoids is relatively common in active populations. The hallux sesamoids are the points of weight transfer along the medial column of the forefoot, and they are covered with little soft tissue padding. Injuries to the sesamoids vary from acute fracture and stress fracture to sesamoiditis. Sesamoid stress fractures can occur in either the medial or the lateral sesamoid. The medial sesamoid is usually slightly larger than the lateral, and fracture of the medial sesamoid is somewhat more common. [35] Radiographic details of the sesamoids are often difficult to visualize on standard radiographs because of the obscuring shadow of the first metatarsal head (Fig. 25E-20).

Figure 25E-20 Stress fracture of the medial sesamoid. Note the irregular edges, the elongation compared with the lateral sesamoid, and the difference in density of the proximal and distal portions. *(Courtesy of James W. Brodsky, MD.)*

Patients with sesamoid stress fractures are generally active, although most do not necessarily give a history of a recent change in activity level. They present with insidious onset of medial forefoot pain, primarily with weight-bearing activities; this pain is exacerbated by running, jumping, and participating in toe-off activities. Physical examination reveals tenderness localized to the plantar first metatarsophalangeal joint (tenderness may localize to a specific sesamoid), which is worsened with passive dorsiflexion and plantar flexion of the hallux. Ecchymosis and swelling are unlikely. Radiographs may demonstrate a transverse fracture of the sesamoid, but differentiation from a bipartite sesamoid becomes problematic (Fig. 25E-21). If physical examination does not absolutely localize the pain to a specific sesamoid, anteroposterior and sesamoid views with a metallic marker are helpful. In most patients, treatment can be initiated without additional studies. If an absolute diagnosis is necessary, bone scan (from the plantar surface of the foot) may be the modality of choice [83] because even MRI sometimes fails to diagnose these stress fractures. [84]

Figure 25E-21 Sesamoid fracture equivalent. There is disruption of the synchondrosis of this bipartite sesamoid. Note the separation between the two poles. *(Courtesy of James W. Brodsky, MD.)*

Treatment is largely conservative, with activity modification and the use of off-loading orthoses being the mainstay. Protected weight-bearing in a cast or boot is used in recalcitrant cases and in those with a definite acute fracture line on plain films. Surgical treatment is reserved for cases that are recalcitrant to nonoperative treatment and symptomatic cases with radiographically documented distraction between the fragments. Sesamoid excision and reconstruction of the flexor hallucis brevis tendon and intersesamoid ligament is the appropriate surgical treatment. This approach has led to good results in the authors' hands, with little evidence of late hallux deformity. However, the soft tissue reconstruction is critical to obtaining a good result because the position of the remaining sesamoids must be maintained and late deviation of the great toe must be prevented. (The risk is that hallux valgus deformity may develop after medial sesamoid excision, and hallux varus after excision of the lateral sesamoid.) Other authors have suggested bone grafting of nondisplaced sesamoid nonunions, with satisfactory results. This approach has been reported in a small series of young, primarily collegiate, athletes.[85] In Figure 25E-22 , a medial sesamoid stress fracture in a teenage athlete is illustrated, which was treated with this method of bone grafting.

Figure 25E-22 A, Teenage athlete with a medial sesamoid stress fracture treated with local bone grafting. **B,** The result of a completely healed sesamoid. *(Courtesy of James W. Brodsky, MD.)*

Although partial excision of one of the poles of the fractured sesamoid has been suggested, this seldom is feasible unless one piece is much smaller than the other. Leaving half-sesamoids usually results in a painful condition because of the poor tracking of the fragment under the metatarsal condyles, but a recent study reported six cases of proximal partial sesamoidectomy, with five out of the six cases achieving complete pain relief. [84] The authors recommend partial excision only when the fragment is small, composing 25% to 30% of bone at most.

Stress fracture of the first proximal phalanx is unusual. Only about 25 cases have been reported in the literature. About 90% were associated with hallux valgus. [1069] [1100] These fractures occurred at the medial base of the proximal phalanx. Most healed with conservative treatment, whereas a few of them were treated with open reduction and bone grafting. [55]

CRITICAL POINTS

- Repetitive microtrauma in a muscle-fatigued athlete is the cause of stress fracture.
- Clinical presentation with radiographic imaging is still the mainstay of diagnosis.
- MRI is the most common adjunct to diagnosis.
- Most stress fractures heal with cast or boot immobilization.
- Fractures that benefit from early surgical intervention, especially in a high-performance athlete, are fractures of the medial malleolus, navicular, and base of fifth metatarsal (Jones' fracture).
- Return to sport should occur only after the fracture has healed and patient is pain free, depending on the specific case.

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SECTION F Heel Pain

Keith L. Wapner,
Selene G. Parekh

RETROCALCANEAL BURSITIS (HAGLUND'S DISEASE, ENLARGEMENT OF THE SUPERIOR TUBEROSITY OF THE OS CALCIS)

Pain in the posterior-superior portion of the calcaneus may be multifactorial, caused by retrocalcaneal bursitis, enlargement of the superior bursal prominence of the calcaneus, insertional Achilles tendinosis, or inflammation of an adventitious bursa between the Achilles tendon and the skin (Fig. 25F-1). [1101] [1102] [1103] [1104] [1105] [1106] [1107] [1108] [1109] [1110] [1111] [1112] Each of these entities may exist as an isolated condition or may be part of a symptom complex. Careful analysis of the patient's subjective complaints and objective findings are required to arrive at the correct diagnosis.

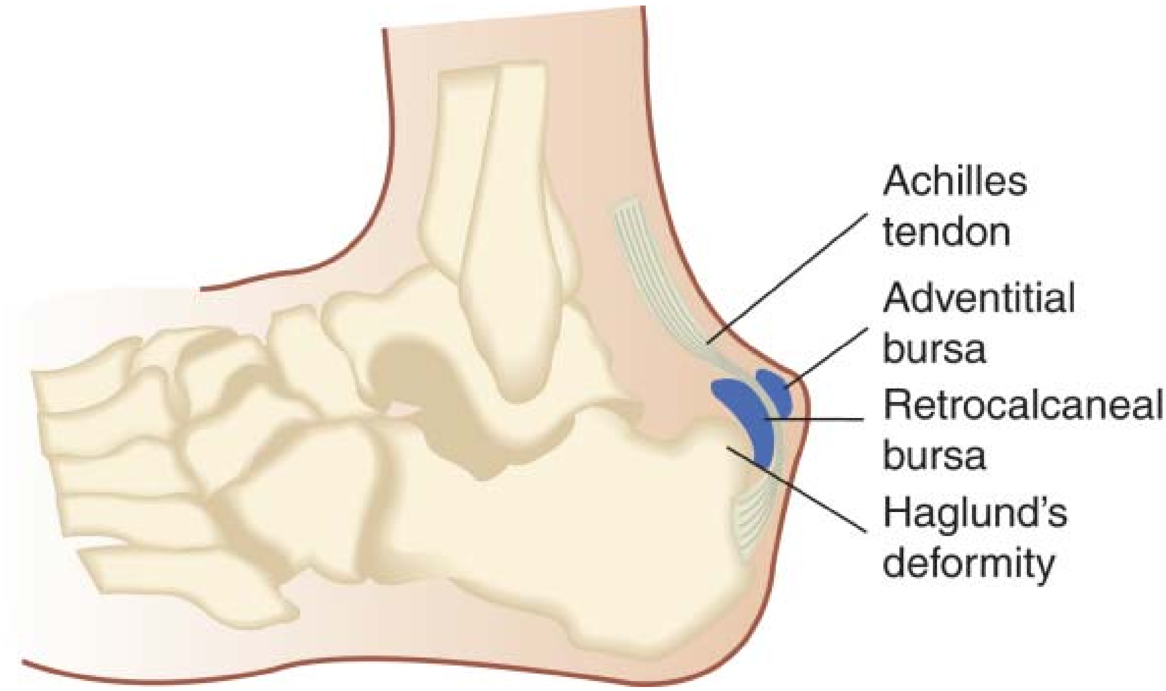


Figure 25F-1 Illustration of Haglund's deformity with a retrocalcaneal bursa between the Achilles tendon and the superior bursal prominence and an adventitious bursa between the Achilles tendon and the skin.

Retrocalcaneal bursitis may occur as an isolated entity but is more commonly associated with the prominent posterior-superior bursal portion of the calcaneus, or Haglund's deformity. When Achilles tendinosis occurs concomitantly with this condition, it is generally located in the area of the Achilles tendon just at or above the insertion of the Achilles at the posterior portion of the os calcis and not more proximally as occurs with deterioration in the area of decreased vascularity 2 to 6 cm above the posterior tuberosity of the os calcis. Calcification may occur within the Achilles tendon at this area and probably represents calcification in a degenerative area of the tendon. [3]

The adventitious bursa, which occurs between the Achilles tendon and the overlying skin, is usually caused by pressure of the counter of the shoe against the prominent area. It is more common in women and less common in athletes.

Mann noted that Haglund called attention to the possible relationship between the shape of the calcaneus and the appearance of "pump bumps," Achilles tendinitis and retrocalcaneal bursitis, and small spurs that are attached to the Achilles tendon. [8] Variations in the shape of the superior tuberosity of the os calcis may also play a role in the development of this retrocalcaneal symptom complex (Fig. 25F-2). [8]

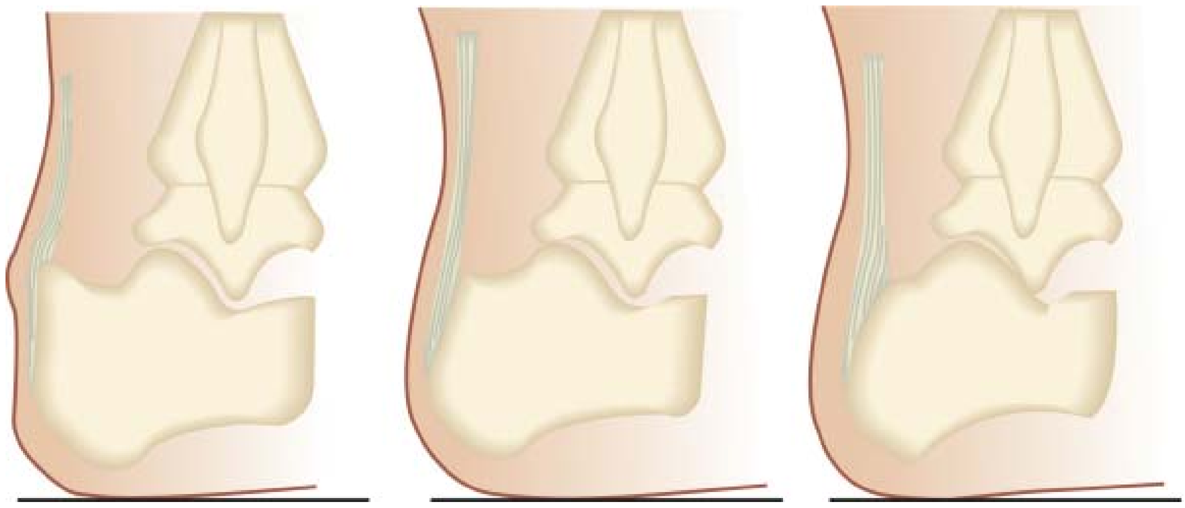


Figure 25F-2 Variations in the shape of the superior tuberosity of os calcis. *Left,* hyperconvexity; *middle,* normal; *right,* hypoconvexity. *(Redrawn from DuVries HL: Miscellaneous afflictions of the foot. In Mann RA [ed]: Surgery of the Foot, 5th ed. St. Louis, CV Mosby, 1986, p 248.)*

Pertinent Anatomy

The Achilles tendon, covered by a thin paratenon, inserts into the middle of the posterior part of the posterior surface of the calcaneus, [13] inserting on average between 10 and 13 mm from the superior aspect of the calcaneal tuberosity, extending 19 mm inferiorly, and extending further medially than laterally. [1114] [1115] A retrocalcaneal bursa located between the Achilles tendon and the superior tuberosity of the calcaneus is a constant finding. [1113] [1116] [1117] Dorsiflexion of the foot and ankle produces increased pressure in the retrocalcaneal bursa, whereas plantar flexion decreases the pressure in the retrocalcaneal bursa. [10]

Anatomically, the retrocalcaneal bursa has an anterior bursal wall composed of fibrocartilage laid over the calcaneus, whereas the posterior wall is indistinguishable from the thin epitenon of the Achilles tendon. [16] It is a disk-shaped structure lying over the posterior-superior aspect of the calcaneus, fitting like a cap over the calcaneus and having a concave aspect anteriorly (Fig. 25F-3). [16]

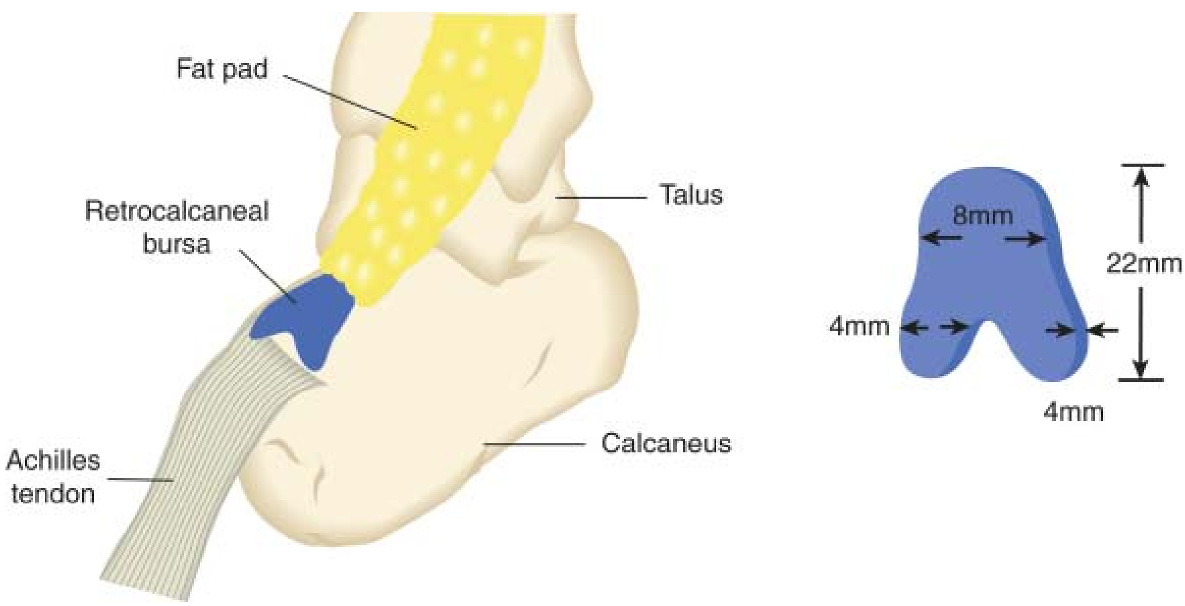


Figure 25F-3 Demonstration of the disk-shaped retrocalcaneal bursa. *(From Frey C, Rosenberg Z, Shereff MJ: The retrocalcaneal bursa: Anatomy and bursography. Presented at the American Orthopaedic Foot and Ankle Society Specialty Day Meeting, Las Vegas, February, 1989.)*

The superior tuberosity of the os calcis may be hyperconvex, normal, or hypoconvex. [8] The radiographic anatomy of the os calcis has been described by Heneghan and Pavlov [1104] [1109] in terms of the following anatomic landmarks on the lateral projection (Fig. 25F-4). The superior aspect of the talar articulation marks the most proximal portion of the posterior facet. The bursal projection is the area of the superior tuberosity of the os calcis. The tuberosity of the posterior surface marks the site of the Achilles insertion. The medial tubercle is the site of insertion of the central portion of the plantar aponeurosis.

Figure 25F-4 Important radiographic landmarks of the os calcis: T, superior aspect of talar articulation; BP, bursal projection; P, posterior tuberosity indicating attachment of Achilles tendon; M, medial tuberosity attachment of plantar aponeurosis; A, anterior tubercle. *(From Pavlov H, Heneghan MA, Hersh A, et al: The Haglund syndrome: Initial and differential diagnosis. Radiology 144[1]:83-88, 1982.)*

Relevant Biomechanics

The retrocalcaneal bursa is a constant, horseshoe-shaped bursa found between the Achilles tendon and the rounded superior bursal prominence of the os calcis. It maintains the relatively constant distance between the axis of the ankle joint and the insertion of the Achilles tendon. [10] If the posterior prominence were not present, during dorsiflexion there would be shortening of the distance of the ankle joint axis and the insertion of the Achilles tendon. As this lever arm shortens, the ability of the gastrocnemius-soleus muscle to function is affected. This mechanism allows the tension of the gastrocnemius-soleus muscle group through the Achilles tendon to remain constant with dorsiflexion and plantar flexion. [10]

Canoso and colleagues showed by magnetic resonance imaging (MRI) the dynamic aspects of the retromalleolar fat pad. [18] They said that the tongue-like extension of the fat pad may be viewed as a freely moving spacer with a sliding motion between cartilage and tendons and is facilitated by hyaluronic acid, presumably secreted by its own lining. Because the Achilles tendon inserts in the middle third rather than in the more proximal upper portion of the posterior calcaneal surface, the plantar flexion lever is increased. The retrocalcaneal bursa, by accepting the fat pad, allows the necessary separation of tendon and bone that occurs in plantar flexion without creating excessive tissue tension.

Retrocalcaneal pain syndrome is commonly associated with the high-arched cavus foot and the varus heel. [1102] [1110]

The combination of these factors tends to produce a foot that does not dorsiflex as readily as a normal foot. There is prominence of the heel, which is more susceptible to increased pressure from the tendons and the counter of the shoe. Ruch stated that retrocalcaneal bursitis generally occurs in the circumstances of compensated rearfoot varus, compensated forefoot valgus, and a plantar flexed first ray because of the abnormal motion of the subtalar joint and the frontal and sagittal plane relationships. [10]

Clinical Evaluation

The history is generally that of slow onset of dull, aching pain in the retrocalcaneal area aggravated by activity and certain shoewear. A common complaint is start-up pain after sitting or when arising out of bed in the morning. At times there may a history of acute onset of pain, sometimes associated with a traumatic incident. When this occurs, one must think of a tear or calcification of the Achilles tendon as perhaps the initiating factor.

Physical examination reveals swelling in the area of the retrocalcaneal bursa between the Achilles tendon and the calcaneus. [1] There is generally a prominence in the area of the superior portion of the heel. The swelling in the retrocalcaneal bursa will be found just anterior to the Achilles tendon. By palpating medially and laterally at the same time and with the aid of ballottement, one can sometimes feel fluid within the bursa (Fig. 25F-5). With careful and discrete palpation, one can generally differentiate between swelling in the Achilles tendon and swelling in the retrocalcaneal bursa. The swelling of the Achilles tendon associated with retrocalcaneal bursitis is usually at the level of the tendon at or just proximal to the insertion. Dorsiflexion of the foot usually increases the pain in the area. A great deal of swelling and inflammation on examination may indicate involvement of the retrocalcaneal bursa and involvement of the Achilles tendon. There may be redness and swelling between the Achilles tendon and the skin, usually due to an adventitious bursitis produced by pressure of the shoe counter against the Achilles tendon. There may be an area of periostitis, which is a discrete localized area of tenderness of the os calcis, usually on the lateral side of the posterior portion of the os calcis and produced by pressure of the shoe counter.

Figure 25F-5 Illustration demonstrating area of the swelling; retrocalcaneal bursitis with swelling is anterior to the Achilles tendon.

Diagnostic Studies

Radiographic Studies

A lateral view of the foot is taken with the patient standing. This allows biomechanical evaluation of the foot as well as evaluation of the specific points of the os calcis. The points of the os calcis are identified as the posterior margin of the posterior facet, the superior bursal projection, the tuberosity indicating the site of the Achilles tendon insertion, the medial tubercle, and the anterior tubercle. [9] The shape and appearance of the superior bursal prominence are noted.

Evaluation of the lateral radiograph may be performed using the method of Fowler and Philip, which measures the posterior calcaneal angle (Fig. 25F-6). [19] Fowler and Philip consider the bursal projection prominent if the angle is greater than 75 degrees. Some authors have concluded that a combination of the Fowler angle and the angle of calcaneal inclination is more effective in correlating the radiographic appearance with symptoms than the Fowler and Philip angle alone, [1110] [1120] the combined angle being greater than 90 degrees in patients with symptomatic Haglund's disease.

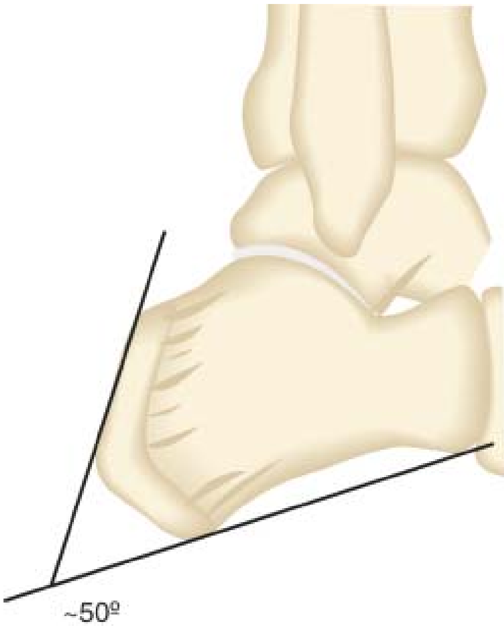


Figure 25F-6 Measurement of the Fowler and Philip angle. The normal angle is shown on the *left* and abnormal on the *right*. Upper level of normal is considered to be 69 degrees. Drawing at *right* indicates an abnormal angle of 75 degrees.

Parallel pitch lines have been used by Heneghan and Pavlov [4] to determine the prominence of the bursal projection (Fig. 25F-7). The base line is constructed by placing a line along the medial tuberosity and the anterior tubercle and a parallel line from the posterior lip of the talar articular facet. The bursal prominence is considered abnormal if it extends above this line.

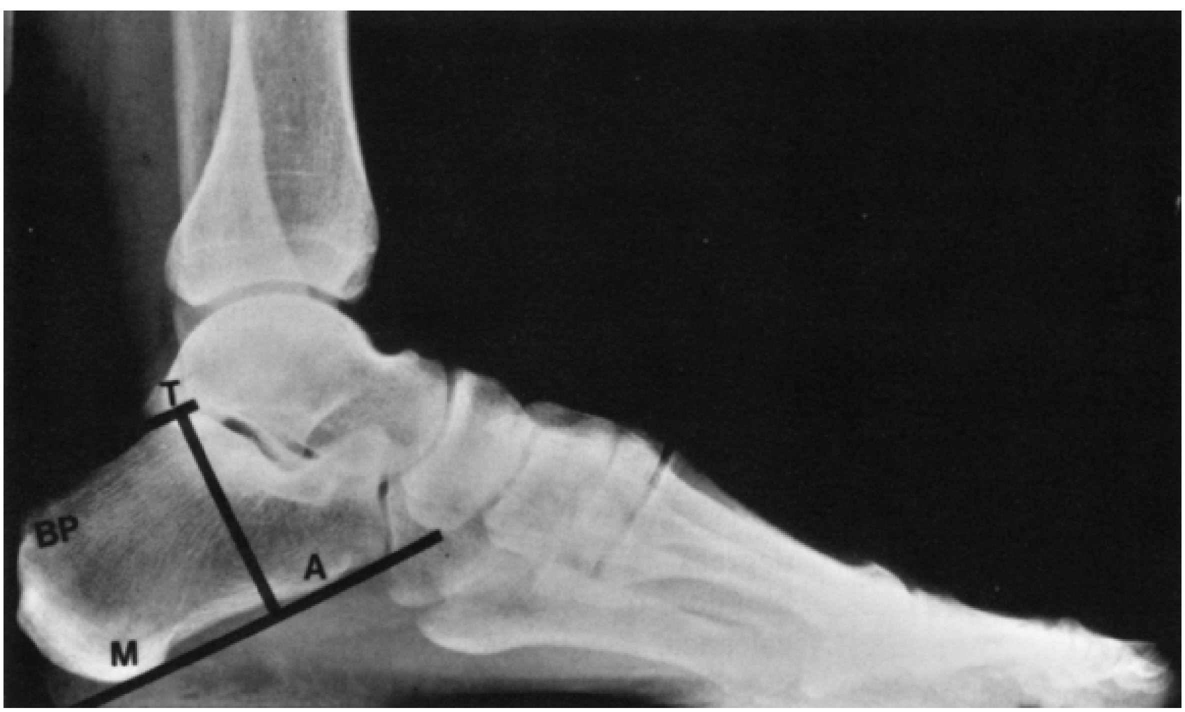


Figure 25F-7 Parallel pitch lines used to determine the prominence of the bursal projection. A line is drawn along the medial tuberosity and the anterior tuberosity. A parallel is constructed from the superior prominence of the posterior facet. If the bursal projection is above the superior line, the projection is considered abnormally large. *(From Pavlov H, Heneghan MA, Hersh A, et al: The Haglund syndrome: Initial and differential diagnosis. Radiology 144[1]:83-88, 1982.)*

Pavlov and associates noted that the recessions seen on the lateral radiograph extend 2 mm inferior to the bursal projection, and with retrocalcaneal bursitis, there is loss of the sharp interspace with the tendoachilles. [9] They also pointed out that the pre-Achilles fat pad outlines the anterior surface of the Achilles tendon, which normally measures 9 mm from anterior to posterior, 2 mm above the bursal projection.

Frey and colleagues described the retrocalcaneal bursa in a study of 12 fresh cadavers, 15 patients with signs and symptoms of retrocalcaneal bursitis, and 8 normal patients. [21] They found that in symptomatic patients, the amount of contrast material accepted averaged 0.92 mL, and the outline of the bursa was irregular in 100% of the patients in this group. The asymptomatic group accepted an average of 1.22 mL of contrast material, and 71% were noted to have a smooth bursal outline. The average area of the bursa on the lateral radiograph was 0.77 cm [2] in the normal patient and 1.18 cm [2] in the abnormal patient. Eighty-three percent of the patients noted significant (greater than 80%) improvement of their symptoms when 1% lidocaine was injected into the retrocalcaneal bursa.

Burhenne and Connell used xeroradiography to assess soft tissue and calcaneal detail in patients suffering from painful swelling localized in the heel. [22] They noted that neither a posterior calcaneal angle (of Philip and Fowler) of more than 75 degrees (see Fig. 25F-6) nor the parallel pitch line (see Fig. 25F-7) proved to be a reliable index. They evaluated 4 patients with heel pain and swelling out of 100 control patients and found that the radiographic triad of retrocalcaneal bursitis, superficial tendoachilles bursitis, and Achilles tendon thickening, in the presence of an intact posterior-superior calcaneal margin, were readily evaluated with xeroradiography.

Canoso and coworkers reported finding bursal fluid in cadavers without rheumatic disease and aspirating the retrocalcaneal bursa in four patients, three with Reiter’s syndrome and one with pseudogout. [23] They reported that the findings in bursal and synovial joint fluids were similar. In patients with retrocalcaneal bursitis, one should always be aware of the possibility that the bursitis may be a manifestation of systemic arthritis or gout. This should be addressed by the history, general physical examination, and laboratory studies to rule out these disorders.

Gerster and Piccinin reported on the painful heel syndrome with plantar fasciitis or Achilles tendinitis in 33 of 150 patients suffering from a seronegative spondyloarthritis. [1124] [1125] By contrast, Achilles tendinitis was not encountered in 220 patients with rheumatoid arthritis. Gerster and colleagues also reviewed 30 patients with severe heel pain, of whom 24 were diagnosed as having seronegative spondyloarthritides in which talalgia is frequently the first symptom of the disease. [26] Talalgia is defined as heel pain located either posteriorly (along the Achilles tendon or its insertion on the calcaneus) or at the attachment of the superficial aponeurosis on the plantar surface of the calcaneus. Of these 24 patients, 5 had involvement of the Achilles tendon with peritendinitis, and 6 had plantar fasciitis plus Achilles peritendinitis. Gerster’s group, unlike others, thought that rheumatoid arthritis was not a common cause of severe talalgia.

MRI has provided clearer insight into the anatomic abnormalities associated with posterior heel pain. [1127] [1128] [1129] [1130] The imaging allows visualization of the Achilles tendon and the bursa as well as demonstrating any bony abnormalities in the posterior-superior calcaneus. In patients refractory to nonoperative treatment, preoperative MRI defines which anatomic structures need to be addressed (Fig. 25F-8) The degree of tendinosis present in the Achilles tendon is easily visualized and distinguished from isolated bursitis.

Figure 25F-8 **A,** Axial magnetic resonance imaging (MRI) of normal Achilles tendon showing normal shape of the Achilles. **B,** Sagittal MRI of normal Achilles tendon showing normal shape of the Achilles. **C,** Sagittal MRI showing increased signal in the insertion of the Achilles tendon consistent with tendinosis. There is increased fluid demonstrating an inflamed bursa surrounding Haglund's deformity. **D** and **E,** Axial and sagittal MRI demonstrating chronic tendinosis of the Achilles with marked fusiform swelling of the tendon.

Treatment Options

Puddu and associates [31] proposed three stages of inflammation occurring at the insertion of the Achilles. Stage 1, peritendinitis, involves inflammation of the paratenon only. Peritendinitis with tendinosis, stage 2, is characterized by macroscopic thickening, nodularity, and microscopic focal degeneration of the Achilles tendon in addition to inflammation of the paratenon. Stage 3 is characterized by degenerative lesions of the substance of the tendon itself without associated peritendinitis.

Clain and Baxter, [32] divided Achilles tendon pathology into insertional and noninsertional dysfunction. Insertional tendinosis occurs within and around the Achilles tendon at its insertion and may be associated with Haglund's deformity or spur formation within the tendon itself. In the presence of associated tendinosis, they advocated transfer of either the flexor digitorum longus, as advocated by Mann and colleagues, [33] or the flexor hallucis longus (FHL), as advocated by Wapner and coworkers. [1134] [1135] Schepsis and colleagues [36] concurred that tendon transfer should be considered because it may enhance the blood supply and reinforce the Achilles tendon.

Nonoperative Therapy

Initial goals in the treatment of retrocalcaneal bursitis are to control pain and attempt to allow the patient to return to normal function and activity. Rest, particularly soon after the onset of symptoms, can be helpful. The duration of rest may be prolonged, depending on increasing symptom durations. [1137] [1138] Cross-training with low-impact exercises, such as an elliptical machine, swimming, [1116] [1137] or biking, [39] may prevent deconditioning in the athlete. In the athlete who is unwilling to cross-train, modified regimens may be suggested. In patients with exquisite tenderness, immobilization in a CAM boot or short leg walking cast may be helpful. [1116] [1140] In the athlete, immobilization should be used cautiously because patients can have resultant tendon and muscle atrophy, degeneration, and decreased blood supply. [1139] [1141]

Nonsteroidal anti-inflammatory drugs (NSAIDs), orally or locally delivered as a patch, may decrease local inflammation. [1141] [1142] [1143] Cryotherapy and ice can decrease pain, swelling, and inflammation as well. [1141] [1142] [1143] Corticosteroids, orally, injected locally, or applied topically, have been used. [44] Injections must be used cautiously because they can lead to a higher incidence of tendon ruptures and tendinopathy. [1145] [1146] Animal model studies with intratendinous injections have been shown to result in localized tendon necrosis and decreased mechanical strength. If used, the steroid injection must be placed anterior to the tendon, in the area of the retrocalcaneal bursa. [1141] [1142]

Physical therapy is a useful modality in the treatment of Achilles tendinitis. Eccentric loading of the tendon has been shown to have beneficial effects on the microcirculation of the tendon. [1147] [1148] Stretching of the contractures of the gastrocnemius and soleus muscles may limit Achilles tendon strain. Traditionally, night splints or dynamic joint stretching devices have been thought to decrease the Achilles contracture. [40] However, a recent study by de Vos and associates demonstrated that a night splint, in addition to eccentric exercises, is not beneficial in the treatment of chronic midportion Achilles tendinopathy. [49]

Orthoses may help these patients by providing a heel lift function, correcting hyperpronation, or minimizing leg-length discrepancies. [1141] [1142] Care must be exercised when correcting hindfoot pronation deformities because overcorrection can result in inflexibility of the hindfoot with decreased shock absorption. [42] Heel cups can decrease the strain on an Achilles tendon and elevate a prominent superior calcaneal tuberosity away from the tendon. High-heels, clogs, open-backed shoes, gel braces, [50] and horseshoe pads [40] can also provide symptomatic relief for the patient.

Brisement therapy is a technique whereby saline is forcibly injected between the paratenon and the tendon. The principle of this technique is to lyse adhesions between these two structures. [1140] [1142] Successful reports have been published in the literature using glycosaminoglycan polysulfate [41] and deproteinized hemodialysate injections. [51] In addition, sclerosing therapy in insertional tendinopathy has shown promising results in a pilot study. [52]

Finally, mixed results have been published on shock-wave therapy for the treatment of chronic Achilles tendinopathy. Rompe and coworkers, in a randomized, controlled trial of low-energy shock-wave therapy and eccentric loading, demonstrated inferior results in the low-energy shock-wave treated cohort. [53] No difference was found by Costa and colleagues in a double-blind randomized placebo controlled trial comparing shock wave to placebo. [54] Furia published successful treatments with high-energy shock-wave therapy. [55] These studies suggest that high-energy, rather than low-energy, shock-wave therapy may be beneficial for chronic Achilles tendinopathy.

Operative Therapy

Fiamengo and colleagues reviewed the charts of patients in whom a diagnosis of Haglund's disease, retrocalcaneal bursitis, or pump bumps had been made. [3] They found 19 patients who met the criteria, and radiographs of 12 of these patients were available for review. They also reviewed 104 control cases in which calcaneal spurs and Achilles tendon calcification with a posterior calcaneal step were present. This step is a horizontal ledge in the middle of the posterior portion of the os calcis corresponding to the level at which the Achilles tendon inserts into the calcaneus. The Fowler and Philip angle was measured, and no difference was found between the two groups. However, the symptomatic heels had a significantly longer horizontal calcaneal length, measured by a horizontal line between the most anterior and most posterior portions of the os calcis. The incidence of Achilles tendon calcification and a posterior calcaneal step was higher in patients who had chronic posterior heel pain compared with the control population. Fiamengo and colleagues recommended that in cases of chronic posterior heel pain, resection of the posterior-superior aspect of the calcaneus, as well as excision of the degenerative and calcific soft tissue in and about the distal Achilles, should be performed. [3]

Fox and colleagues reported on 32 patients with Achilles tendon disease who were operated on between 1968 and 1973. [56] They divided the patients into two groups, those with an acute rupture without antecedent complaints and those with a history of chronic pain, weakness, and functional loss. They did not describe distal retrocalcaneal bursitis in these patients. They stated that degenerative disease of the Achilles tendon should be recognized and treated not as a simple injury but as a pathologic lesion.

Heneghan and Pavlov noted that in their experimental model, the osseous projection on the plantar surface of the calcaneus produced a more prominent bursal projection, and heel elevation decreased the pitch angle, allowing the prominent bursal projection and the foot to slip forward, thus displacing the posterior calcaneus away from the shoe counter (Fig. 25F-9). [4]

Figure 25F-9 Demonstration of calcaneal angle or pitch angle, which is an angle drawn between the horizontal baseline and the inferior portion of the os calcis.

Ippolito and Ricciardi-Pollini described three patients with invasive retrocalcaneal bursitis in whom there were a large bursa and invasion of the os calcis. [57] Pathologic examination revealed lymphoplasmacellular infiltrates containing proportionately more plasma cells than lymphocytes. Removal of the bursa provided clinical relief, and no systemic rheumatic disease developed in later years.

Keck and Kelly reported on 13 patients with 20 symptomatic heels that were treated surgically. [6] In 17 heels, the superior bursa was excised, and in 3 heels, a dorsally based closing wedge osteotomy was performed. Good results were reported in 15 of the heels treated. The initial results were good in all but 2 patients whose pain recurred as a manifestation of generalized rheumatoid arthritis. Osteotomy was used to reduce the posterior prominence, and results were rated good in 2, fair in 1, and poor in 2 heels. The authors thought that too few osteotomies were performed in this series to evaluate this method. However, the osteotomy had the disadvantage of requiring a longer convalescence. [6] Zadek reported in 1939 on a closing wedge osteotomy of the superior part of the os calcis in three patients for treatment of adventitious bursitis. [58] These patients were relieved of their symptoms.

Kennedy and Willis reported on the effects of local steroid injections in tendons. [59] They found that the most significant effect was actual collagen necrosis and that a return to normal failing strength in the injected tendons occurred by 14 days after the injection. The conclusion was that local steroid placed directly in a normal tendon weakened it significantly for up to 14 days. Therefore, in a patient with posterior heel pain, any injection should be made into the retrocalcaneal area, not into the tendon. However, even then there is some contact with the Achilles tendon. [59]

Lagergren and Lindholm reported on the vascular supply of the Achilles tendon. [60] They found that ruptures of the Achilles tendon are usually limited to the segment of the tendon that lies between 2 and 6 cm proximal to its insertion in the os calcis and that this was an area of decreased vascularity and nutrition. This is an important finding relative to the retrocalcaneal bursal syndrome because this classic type of Achilles tendinosis is proximal to the area usually associated with the retrocalcaneal bursal syndrome. This may suggest that insertional tendinosis is brought on by impingement on the tendon rather than decreased circulation.

A soleus muscle anomaly associated with symptoms simulating retrocalcaneal bursitis has been reported. [61] In this entity, a soft bulge due to a large mass of anomalous soleus muscle in Kager's triangle was noted. The condition responded satisfactorily to excision.

Pavlov and colleagues reported the use of the parallel pitch line measurement in 10 symptomatic feet and 78 control feet. [9] They thought that the symptoms correlated statistically with a positive posterior pitch line but not with an abnormal posterior calcaneal angle. They concluded that radiographically, the syndrome is characterized by (1) retrocalcaneal bursitis (loss of the lucent retrocalcaneal recess between the Achilles tendon and the bursal projection), (2) Achilles tendinitis (an Achilles tendon measuring more than 9 mm located 2 cm above the bursal projection), (3) superficial tendoAchilles bursitis (a convexity of the soft tissues posterior to the Achilles tendon insertion), and (4) a cortically intact but prominent bursal projection with a positive parallel pitch line.

In a series of 65 patients with Haglund’s disease reported by Ruch, [10] 17 patients were operated on by resection of the posterior-superior portion of the os calcis, resecting the posterior-superior aspect both medially and laterally and removing sufficient bone to render the previous palpable prominence entirely absent. They were evaluated 6 months to 5 years postoperatively. Fifteen demonstrated good to excellent results with elimination of symptoms. Three of the patients required a second procedure to obtain the desired result.

Vega and colleagues reported 20 cases of Haglund’s deformity. [12] They noted that the combination of the Fowler and Philip angle and the calcaneal angle, when greater than 90 degrees, correlated with the manifestation of symptoms. This was similar to the findings of Ruch. [10] Vega and associates reported conservative treatment with pads, shoes, braces, and injections and resorted to surgical treatment only if conservative treatment was not successful. The surgical incision recommended was the lateral para-Achilles tendon approach.

Clancy stated that an articular-like surface lines the calcaneus at its superior surface where it comes into contact with the Achilles tendon. [2] He found that a bursa may form from constant overuse, enlargement of the bony prominence, or external pressure. He recommended steroid injection behind but not through or into the tendon. In patients who did not obtain relief with conservative measures, ostectomy proved successful. Clancy noted that most of those who required surgery had significant cavus deformities.

Schepsis and Leach [11] reported that most athletes, particularly runners, who presented with acute or chronic posterior heel pain were successfully managed nonoperatively using a combination of (1) a decrease in or cessation of the usual weekly mileage, (2) temporary termination of interval training and workouts on hills, (3) change from a harder bank surface to a softer surface, (4) a 1⁄4- to 1⁄2-inch lift inside the shoe or added to the shoe, and (5) a program designed to stretch and strengthen the gastrocnemius-soleus complex. These measures were combined with oral anti-inflammatory medications and an occasional injection of corticosteroid into the retrocalcaneal bursa. Postural abnormalities were treated with orthotics. Schepsis and Leach studied retrospectively 45 cases of chronic posterior heel pain treated surgically in 37 patients. [11] All but 2 of these patients were competitive long-distance runners who ran an average of 40 to 120 miles per week before the onset of symptoms. Their ages ranged from 19 to 56 years.

The surgical approach used by Schepsis and Leach was a longitudinal incision 1 cm medial to the Achilles tendon that was continued transversely to form a J-shaped incision if necessary (Fig. 25F-10). The patient was placed in a cast for 2 to 3 weeks with weight-bearing permitted after 1 week. When there was pathology within the tendon requiring excision and repair, immobilization was continued for 1 to 2 weeks longer. Range of motion exercises were emphasized. A graduated program of swimming and stationary bicycling, combined with isometric, isotonic, and isokinetic strengthening of the calf muscles, was prescribed. Jogging was permitted after 8 to 12 weeks, rarely sooner. Full return to a competitive level of sports activity usually required 5 to 6 months.

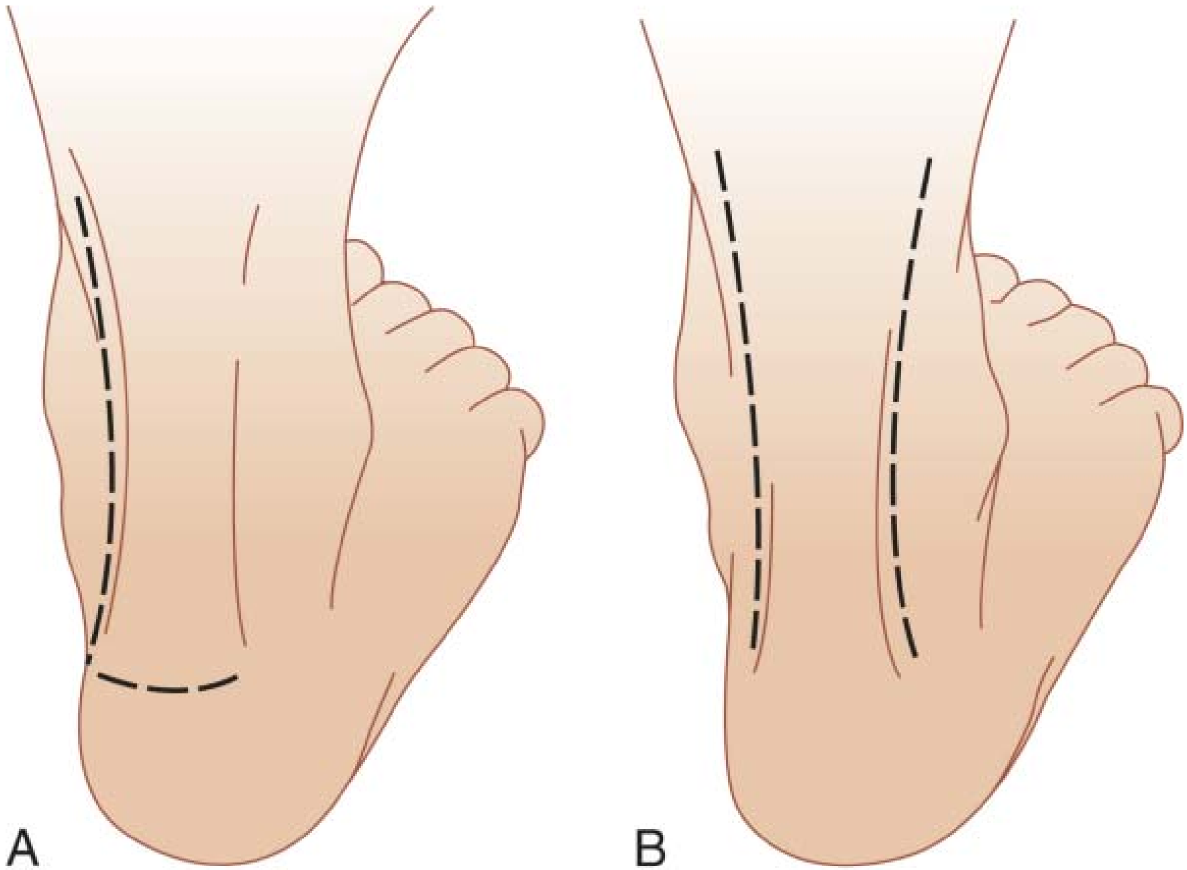


Figure 25F-10 Illustration of surgical incisions for retrocalcaneal bursitis. **A,** Medial approach with J extension as described by Schepsis and Leach [11] ; **B**, medial and lateral approach incisions as described by Jones and James. [5]

The patients were divided into three groups: those with Achilles tenosynovitis-tendinitis, those with retrocalcaneal bursitis, and those with a combination of both. In a group of 24 patients with Achilles tenosynovitis-tendinitis, there were 15 (63%) excellent results, 7 (29%) good results, 1 (4%) fair result, and 1 (4%) poor result. In the 14 patients with retrocalcaneal bursitis, there were 7 (50%) excellent, 3 (21%) good, and 4 (29%) fair results. In the group with a combination of both, there were 5 (71%) excellent and 2 (29%) good results. It was noted that 4 of the 6 unsatisfactory results occurred in the group with retrocalcaneal bursitis.

Jones and James [5] reported on 10 patients who underwent partial calcaneal exostosectomies for retrocalcaneal bursitis. They suggested that conservative measures should be attempted before considering surgical intervention. These included a decrease in the usual weekly mileage, elevation of the heel, instruction in Achilles tendon strengthening, removal of external pressure from the heel, use of oral anti-inflammatory medications, and evaluation and treatment of postural foot deformities. They also suggested immobilization of the leg in a short leg walking cast for a brief period, allowing the athlete to continue cardiovascular maintenance on an exercise bicycle. Steroid injection was used as a last resort before surgery.

During an 8-year period, Jones and James operated on 10 patients with retrocalcaneal bursitis. [5] Six patients were competitive long-distance runners, and 4 were avid recreational runners. Their symptoms consisted of pain and tenderness in the retrocalcaneal area that developed either immediately or after running several miles. The patients ranged in age from 21 to 42 years. Surgery was performed through a longitudinal incision on both sides of the Achilles tendon (see Fig. 25F-10) and included exostosectomy and excision of the bursa. Jones and James emphasized that the ridge of bone at the insertion site must be carefully removed with a small curet and rongeur so that no prominence of bone is left beneath the Achilles tendon posteriorly. A short leg walking cast was used for 8 weeks, with partial weight-bearing allowed for the first 2 weeks and then full weight-bearing. After casting, a 1-inch heel elevation was used until the foot assumed the neutral position easily. General muscle conditioning was carried out until Cybex testing revealed symmetrical muscle strength. All the patients went back to their desired level of activity within 6 months.

Endoscopic techniques for the débridement of the retrocalcaneal space and the posterior-superior tuberosity of the calcaneus have been reported in the literature. Ortmann and McBryde reported on 28 patients and 30 heels treated with endoscopic surgery; 86.67% of patients reported excellent results, with one major complication of an Achilles tendon rupture.[62] Although the medial column of the Achilles tendon, the sural nerve, and the plantaris tendons are at risk during this technique,[63] the purported benefits of lower morbidity and recovery time make this a desirable option for some surgeons.

Sullivan,[20] addressing the problem of recurrent pain in the pediatric athlete, stated that heel pain may be due to osteochondrosis of the apophysis of the calcaneus (Sever's disease) or to Achilles tendinitis, which is characterized by pain on palpation of the tendon just above its insertion. In severe cases, there may crepitation of the tendon. He recommended treatment consisting of rest and aspirin or other mild anti-inflammatory agents. In differentiating these two entities, it may be helpful to note that the pain of osteochondrosis occurs on the inferior portion of the os calcis and the pain associated with Achilles tendinitis is felt proximal to the insertion of the Achilles tendon on the os calcis.

In summary, retrocalcaneal bursitis is a condition characterized by inflammation of the retrocalcaneal bursa, the Achilles tendon just above its insertion, and at times the tissue between the Achilles tendon and the skin. It is generally managed by conservative measures consisting of anti-inflammatory medication, decreased activity, padding to prevent pressure on the affected area, orthoses or heel lifts, and strengthening and stretching exercises. If it does not respond to these modalities, surgical intervention may be considered. Surgery generally consists of excision of the exostosis and the retrocalcaneal bursa and at times the adventitious bursa, if it is present, and correction of the Achilles tendon pathology with tendon transfer if necessary. Although most series do report good results after surgery, in the athlete, this condition may present a serious threat to continued full activity even after surgical intervention.

AUTHORS' PREFERRED METHOD OF TREATMENT

The patient is first evaluated to ascertain the exact reason for the pathology. Adventitious bursitis is usually seen in women and does not appear to be a prominent problem in athletes. It is generally treated conservatively by softening the heel counter, using a small U-shaped pad (Fig. 25F-11) to relieve the pressure of the shoe or counter against the inflamed area, and anti-inflammatory medications. If the pain is refractory to these modalities, an injection of steroid directly into the inflamed area of the bursa, with care to avoid the tendon, may be tried once. Because of the risk for Achilles rupture after injections, the foot is immobilized in a removable cast walker for 2 weeks. Surgical intervention performed solely for adventitious bursitis is unusual.

Nonoperative management of insertional Achilles tendinitis is determined by the degree of inflammation and tendinosis present. When mild, use of a U-shaped heel pad, home stretching of the gastrocnemius-soleus muscle, avoidance of activities such as running, cross-training with bike riding, and use of a night splint to keep the foot in neutral is generally successful. The addition of NSAIDs can be considered.

In moderate to severe cases of tendinosis, a period of immobilization with a molded ankle-foot orthosis can be used. This allows decreased load across the tendon but does not completely immobilize the tendon. The brace should have a relief molded into it to avoid direct contact on the posterior-superior aspect of the calcaneus. The patient is allowed to continue to ambulate with full weight-bearing and may do activities such as stationary bike riding with the brace. The brace is continued until the swelling and tenderness are resolved. Aggressive stretching of the gastrocnemius-soleus complex and strengthening are then started, and the patient is weaned out of the brace. Shoe modifications with a U-shaped heel pad and soft heel counter are used.

If nonoperative management fails, an MRI is obtained before surgery to assess the degree of tendinosis within the insertion of the Achilles tendon. This allows for preoperative discussion with the patient regarding the need for possible tendon transfer to reinforce the Achilles tendon.

Surgical procedures are usually performed for retrocalcaneal bursitis associated with the superior bony prominence. The retrocalcaneal bursa and the superior bursal prominence are excised. The adventitious bursa is excised if it is prominent. If the adventitious bursa is excised, the surgeon must take care to excise it carefully and meticulously to prevent damage to or an adverse effect on the blood supply or the skin overlying this area because a skin slough would be a serious complication.

A medial, lateral, or combination of incisions 1.5 cm anterior to the Achilles tendon is made as determined by the location and width of the bony prominence (see Fig. 25F-10). The incision is carried directly to the level of the paratenon, and dissection is performed at this level to maintain full-thickness skin flaps to avoid skin loss. Attention should be paid to the calcaneal branch of the sural nerve on the lateral side and the medial calcaneal nerve on the medial side. The Achilles tendon is inspected to confirm the degree of tendinosis. The retrocalcaneal bursa is excised. An exostosectomy is performed, removing the bone from the area of insertion of the Achilles tendon to the superior portion of the posterior facet of the os calcis (Fig. 25F-12). Adequate bone is removed, and the edges are smoothed with a rasp.

If significant tendinosis is present, the patient is positioned supine. A medial incision is used to expose the Achilles tendon. Dissection is carried down to the level of the paratenon, and the paratenon is opened and the tendon débrided. If significant calcification is present within the tendon, it should be removed. At times, this necessitates partial release of the insertional fibers of the Achilles tendon. Once the tendon is adequately débrided, the decision must be made about whether to add a tendon transfer. If more than 30% of the tendon is involved, I prefer to add a transfer of the FHL (Fig. 25F-13).

Attention is first directed to the medial border of the foot, where the FHL tendon is harvested. A longitudinal incision is made along the medial border of the midfoot, just above the level of the abductor muscle from the navicular to the head of the first metatarsal. The skin and subcutaneous tissues are sharply divided down to the level of the abductor hallucis fascia. The abductor is then reflected plantarward, and a small Weitlaner retractor is placed in the wound. The flexor hallucis brevis is then reflected plantarward, exposing the deep midfoot anatomy. In some instances it is necessary to release the origin of the short flexors to assist visualization.

The FHL and flexor digitorum longus tendons are identified within the midfoot. They are generally covered by a layer of fatty tissue. Identification of the tendons is assisted by placing a finger over the lateral wall of the short flexor and manually plantar flexing and dorsiflexing the first toe proximal interphalangel (PIP) joint. The motion of the tendon can be felt, and dissection can be carried down to identify the tendons of the FHL medially and the flexor digitorum longus laterally. The FHL is divided as far distally as possible, but one must allow an adequate distal stump to be transferred to the flexor digitorum longus. The proximal portion is tagged with a suture. The distal limb of the FHL is then sewn into the flexor digitorum longus with all five toes in a neutral posture, providing flexion to all five toes through the flexor digitorum longus.

Attention is again turned to the posterior medial incision. The fascia overlying the posterior compartment of the leg is then incised longitudinally directly over the muscle belly of the FHL. The tendon is then retracted from the midfoot into the posterior incision.

A transverse drill hole is placed just distal to the insertion of the Achilles tendon halfway through the bone from medial to lateral. A second, vertical, drill hole is made just anterior to the level of resection of the posterior-superior prominence to meet the first hole. A large towel clip is used to augment the tunnel created. A suture passer is placed through the tunnel from distal to proximal. The suture is then pulled through the tunnel, thus drawing the FHL tendon through the drill hole.

If the Achilles insertion has been detached, suture anchors can be used to reattach the tendon before securing the FHL transfer. The FHL is then woven from distal to proximal through the Achilles tendon using a tendon weaver. The tendon weaver is passed through the Achilles, creating a “tunnel” in the tendon. The tag suture on the flexor hallucis is then grasped and pulled back through the tunnel, bringing the flexor tendon through the Achilles. This process is repeated to use the full length of tendon harvested. The tendon is secured with multiple sutures of No. 1 Cottony Dacron. After completion of the reconstruction, the paratenon is repaired. The subcutaneous tissue and skin are closed. Compressive dressings and plaster splints are applied to maintain 15 degrees of ankle plantar flexion.

The patient is placed in a short leg non–weight-bearing cast at 15 degrees of equinus for 4 weeks. When the patient returns at 4 weeks, the dressing is removed, and the forefoot is placed on a foot rest with the patient seated on an examining table and with the hip flexed and allowed to stay in this position until the foot reaches neutral. The foot is then placed into a short leg walking cast or removable cast walker with the ankle at neutral for an additional 4 weeks, and weight-bearing is begun. A rehabilitation program for strengthening and range of motion is begun 8 weeks before surgery. The patient is maintained in a removable cast walker for community ambulation until 10 degrees of dorsiflexion is obtained and grade 4/5 strength is demonstrated. In-home ambulation is allowed with a 7⁄16-inch heel lift during this time. The patient is then advanced to regular shoewear and continued on a home strengthening program with Thera-Band. Athletic activity is restricted for 6 months after surgery.

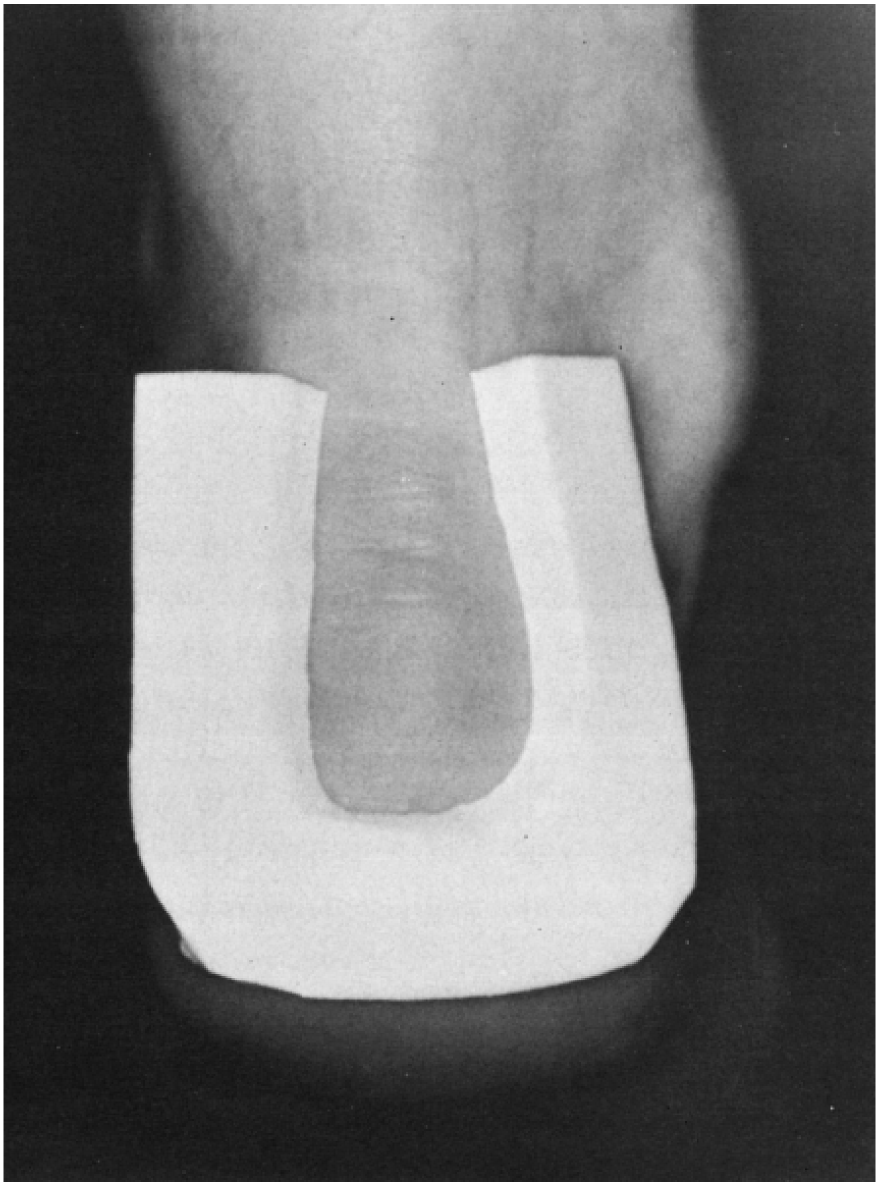


Figure 25F-11 Demonstration of use of U-shaped pad to remove pressure on the bony prominence of the heel.

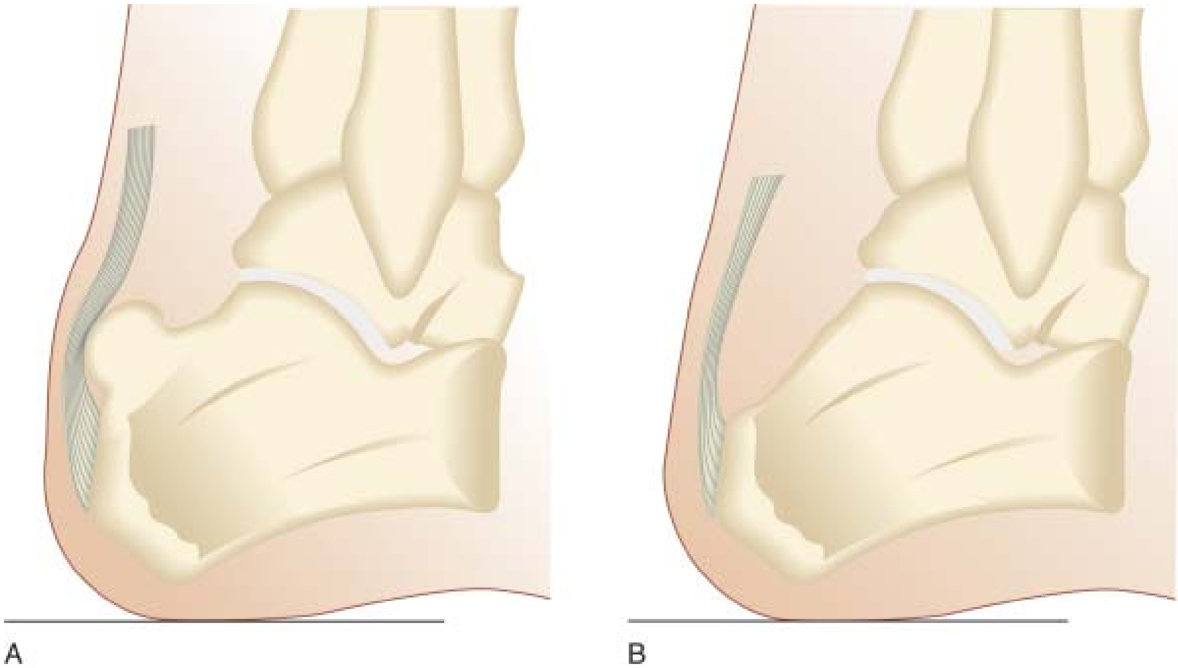
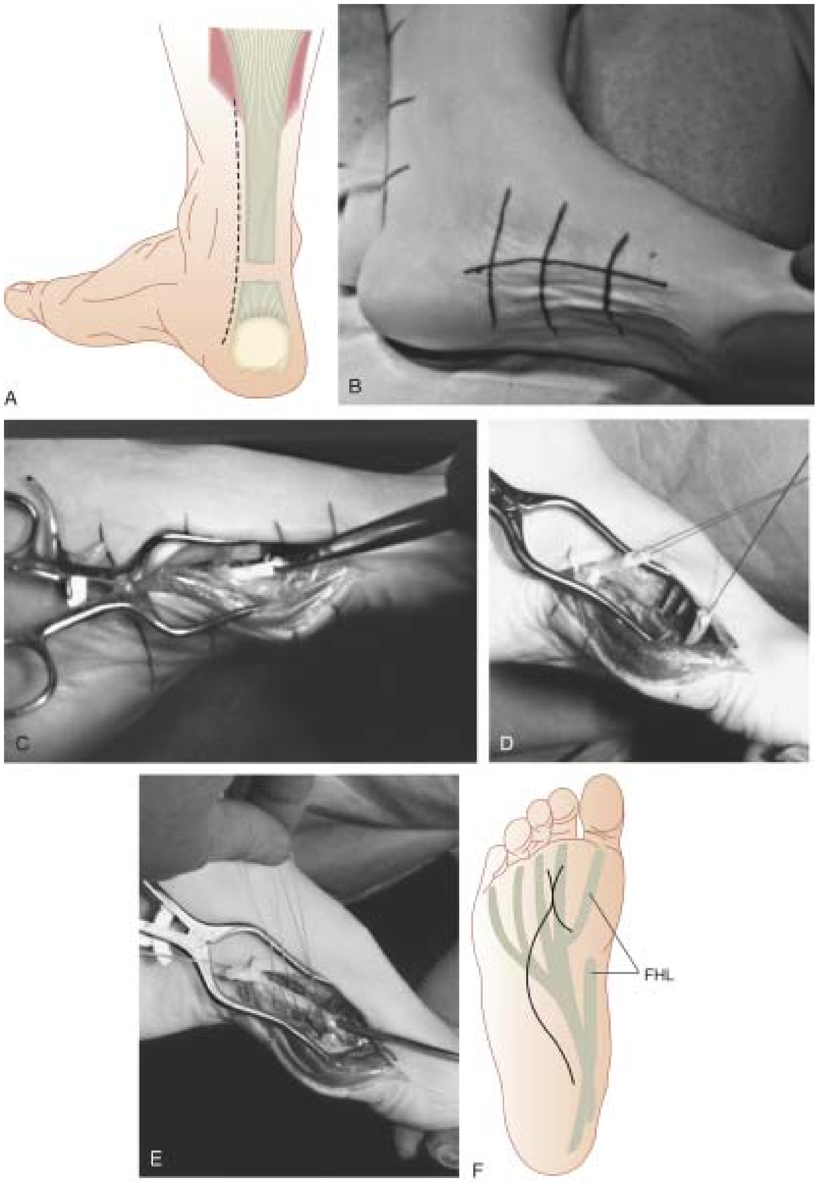


Figure 25F-12 **A,** Haglund's deformity with prominence of posterior superior portion of os calcis. **B,** Appearance of os calcis after surgical resection of posterior superior prominence for symptomatic Haglund's deformity.


A
B
C
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FHL

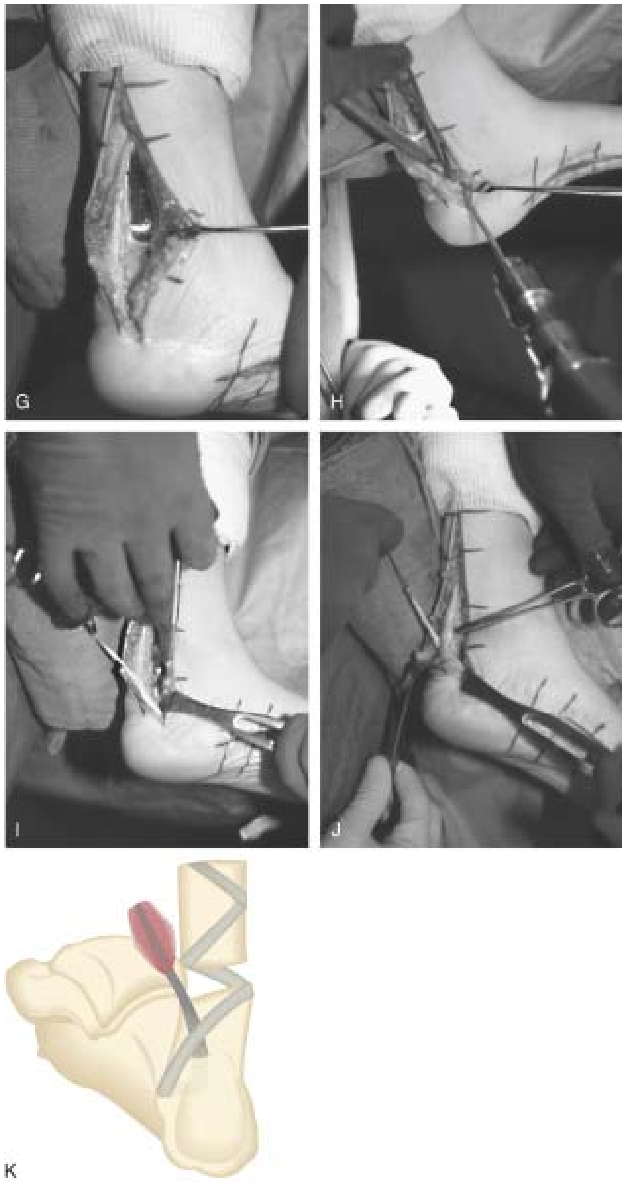

Figure 25F-13 **A,** Diagram of incisions used for flexor hallucis longus (FHL) reconstruction of Achilles tendon. **B,** Clinical demonstration of incisions. **C,** Abductor and flexor hallucis brevis are reflected plantar and the FHL tendon identified. **D,** The FHL has been tagged and divided at the level of the proximal third of the first metatarsal. **E,** The flexor digitorum longus (FDL) is identified and tagged. **F,** Diagram of FHL and FDL anastomosis. **G,** Posterior incision is made, and the posterior fascia of the leg is opened to allow transfer of the FHL into the wound. **H,** Two drill holes are made, one superior and the other medial, to intersect in the posterior body of the calcaneus to create a tunnel for tendon transfer. **I,** The FHL tendon is passed through the superior hole and out the medial side of the tunnel. **J,** The FHL is woven through the Achilles tendon using a tendon weaver. **K,** Diagram of completed weaving of FHL through the Achilles tendon demonstrating the orientation of the tunnel through the posterior calcaneus.

Criteria for Return to Sports Participation

The time needed for return to sports participation will depend on the severity and perniciousness of the condition, the relationship of the pain component to the patient's sporting activity, and the extent of involvement of the Achilles tendon. Should the situation be severe enough to force the patient to stop the desired athletic activity, the following plan is used.

If retrocalcaneal bursitis is present with a normal Achilles tendon, conservative therapy is tried until the patient has been asymptomatic for 4 to 6 weeks. The patient may then return to sports participation starting with limited activity and working up to full activity within 4 to 12 weeks, assuming that the patient has recovered full strength and mobility without pain. If retrocalcaneal bursitis is associated with degeneration of the Achilles tendon, nonoperative treatment should be used until the patient is asymptomatic. A gradual increase in activity is then allowed over a 6- to 12-week period. If surgery has been performed on the tendon in combination with excision of the retrocalcaneal bursa and exostosectomy, immobilization is continued for 8 weeks, but active range of motion is begun at 3 weeks. Strengthening and stretching exercises are started at 6 to 9 weeks. Increased activity according to tolerance can be started at 12 weeks, and return to strenuous activity is allowed at 4 to 6 months if local symptoms have resolved. When tendon transfer is used, the protocol is as described in the previous section.

PLANTAR FASCIITIS ASSOCIATED WITH PAIN IN MEDIAL TUBEROSITY (HEEL SPUR SUBCALCANEAL PAIN SYNDROME)

Pain in the region of the medial tuberosity of the heel associated with increased pain with activity and sometimes related to a spur of the os calcis has been described for many years. Initially, this condition was thought to be associated with gonococcus and was described as "gonorrheal spurs." [1164] [1165] Later it was thought that this entity was due to pull of the plantar fascia and musculature. [1166] [1167] [1168] [1169] [1170] [1171] More recently, Przylucki and Jones [72] and Baxter and Thigpen [64] have attributed this pain to entrapment of the nerve to the abductor digiti quinti arising from the lateral branch of the plantar nerve. Freeman [73] and others [1166] [1174] have attributed this pain to irritation of the medial calcaneal nerve. Other authors have attributed the pain to herniation or to compression of fat nodules. [1175] [1176] Bordelon [1166] [1167] described a clinical syndrome characterized by pain beneath the heel that is aggravated by ambulation and is not associated with any trauma. He proposed that this condition be considered in light of the structures that are present in the area. Specific treatment should be directed toward the structures that are inflamed, the theory being that inflammation in one structure may produce inflammation in other structures. [1166] [1167] In reviewing the literature, it is apparent that many different theories exist about the etiology of subcalcaneal pain, and hence many different methods of treatment have been suggested for it. [1164] [1165] [1166] [1167] [1168] [1169] [1171] [1172] [1173] [1174] [1175] [1176] [1177] [1178] [1179] [1180] [1181] [1182] [1183] [1184] [1185] [1186] [1187] [1188] [1189] [1190] [1191] [1192] [1193] [1194] [1195] [1196] [1197] [1198] [1199] [1200] [1201] [1202] It has been said that although this condition is familiar to all orthopaedic surgeons, it is probably fully understood by none. [67]

Snook and Chrisman [100] noted that there is conflicting literature on this subject on two salient points. The first is that there is no accepted explanation of the etiology of the condition. Second, there is no generally approved method of treatment. They thought that perhaps the basic cause lay in the subcalcaneal pad, which in some unknown manner lost its compressibility, either by local loss of fat with thinning or by rupture of the fibrous tissue septa. Ali [103] stated that "the painful heel is due to a fibrotic response, similar to plantar fibromatosis and not to the spur of bone which is the end result of recurrent strain on the plantar fascia." Tanz [101] thought that "inferior heel pain is often due to irritation of a branch of the medial calcaneal nerve." Similarly, Baxter and Thigpen [64] and Przylucki and Jones [72] advanced the thought that the heel pain is due to an entrapment neuropathy that involves the branch of the lateral plantar nerve to the abductor digiti quinti. It has been noted that this branch passes more proximally than is shown in most anatomic studies and is in the area of the heel spur.

Mann [71] wrote that "in the early stages, fibrositis of low chronicity, with or without pain, anterior to the calcaneal tuberosity represents the pathological change. Continuation of the process leads to osteophytic changes and bone deposits on the sulcus just anterior to the tuberosity."

Kopell and Thompson [104] stated that calcaneodynia, or painful heel, "is usually ascribed to an inflammatory reaction based on mechanical stress at the common muscular and fascial origins on the anterior inferior surfaces of the calcaneus and in many cases, the inflamed structures are the calcaneal nerves which innervate the region of the common origin." Leach and co-workers [1169] [1170] also thought that the cause of plantar fasciitis appears to be repetitive trauma, which produces microtrauma in the plantar fascia near its attachments and leads to attempted repair and chronic inflammation.

Katoh and colleagues [105] reported on objective analysis of foot function during gait using vertical impulse distribution along the sole of the foot during the load-bearing period of gait. This was demonstrated to be reliable in distinguishing between patients with painful heel pads and those with plantar fasciitis.

Although there has been much discussion about the relationship of the calcaneal heel spur to subcalcaneal pain (Fig. 25F-14), the relationship has not been definitely established. Tanz [101] stated that a heel spur is located in the origin of the short toe flexors and not in the plantar fascia. He noted that 15% of normal asymptomatic adult feet have subcalcaneal plantar spurs, whereas about 50% of adult feet with plantar heel pain have spurs. He thought that heel spurs contributed to the plantar heel pain, although many patients with plantar heel pain did not have spurs. Snook and Chrisman [100] agreed with this. Their report on 27 patients with subcalcaneal pain noted that 13 had a calcaneal spur and 11 did not. Mann [71] stated that "over a long period of time, proliferative bony changes at the origin of the fascia may lead to the formation of a spur."

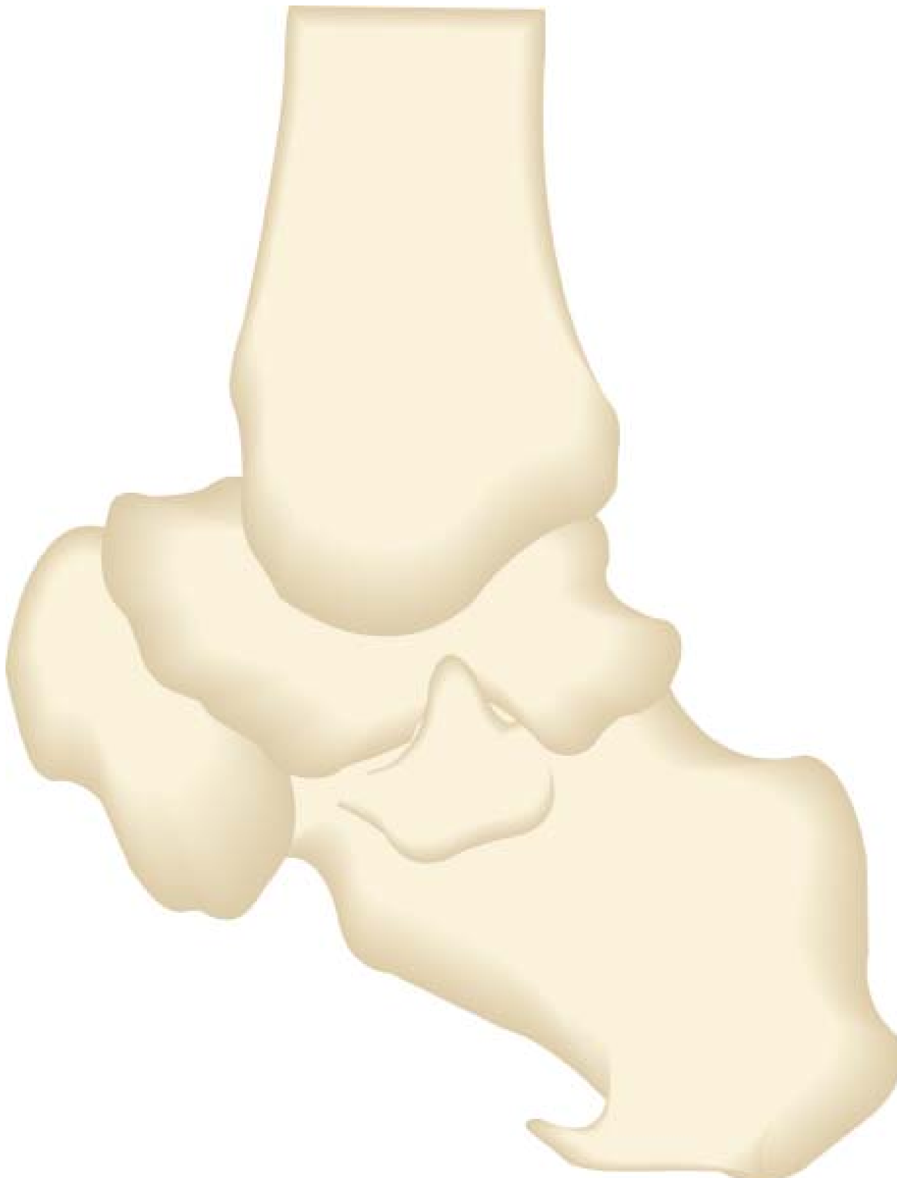

Figure 25F-14 Lateral view of a foot demonstrating a spur in the region of the medial tuberosity. The spur is in the region of the origin of the short flexors. Half of symptomatic patients have heel spurs.

Shmokler and colleagues [106] reviewed 1000 patients at random with radiographs of the foot. There was a 13.2% incidence of heel spurs. Only 39% of those with heel spurs (5.2% of the total sample) reported any history of subcalcaneal heel pain. Shmokler and coworkers believed that these statistics tended to support the premise that the presence of a heel spur did not mandate pain.

Leach and colleagues [1169] [1170] stated that the spur is located in the short toe flexor origins as opposed to the plantar aponeurosis, casting doubt on the concept that the heel spur contributes to the pain in the plantar fascia. In evaluating 45 patients with 52 painful heels, Williams and associates [107] found that 75% of those with painful heels had a heel spur compared with 63% of the opposite nonpainful heels. Comparing 63 heels in 59 age- and sex-matched controls, the incidence of heel spur was 7.9%. Warren and Jones [1208] [1209] attempted to predict which factors would be associated with plantar fasciitis but found that a set of predictable variables was not present. Heel spurs may be present or absent and may or may not be the primary pathologic entity in heel pain. However, they have to be considered in the context of the entire syndrome.

Pertinent Anatomy

The plantar aponeurosis arises from the os calcis and is composed of three segments (Fig. 25F-15). [1210] [1211] [1212]

[1213] The central segment is the largest and arises from the plantar aspect of the posteromedial calcaneal tuberosity. It inserts into the toes. The lateral portion arises from the lateral process of the tuberosity of the os calcis and inserts into the base of the fifth metatarsal. The medial portion is thin and covers the undersurface of the abductor hallucis.

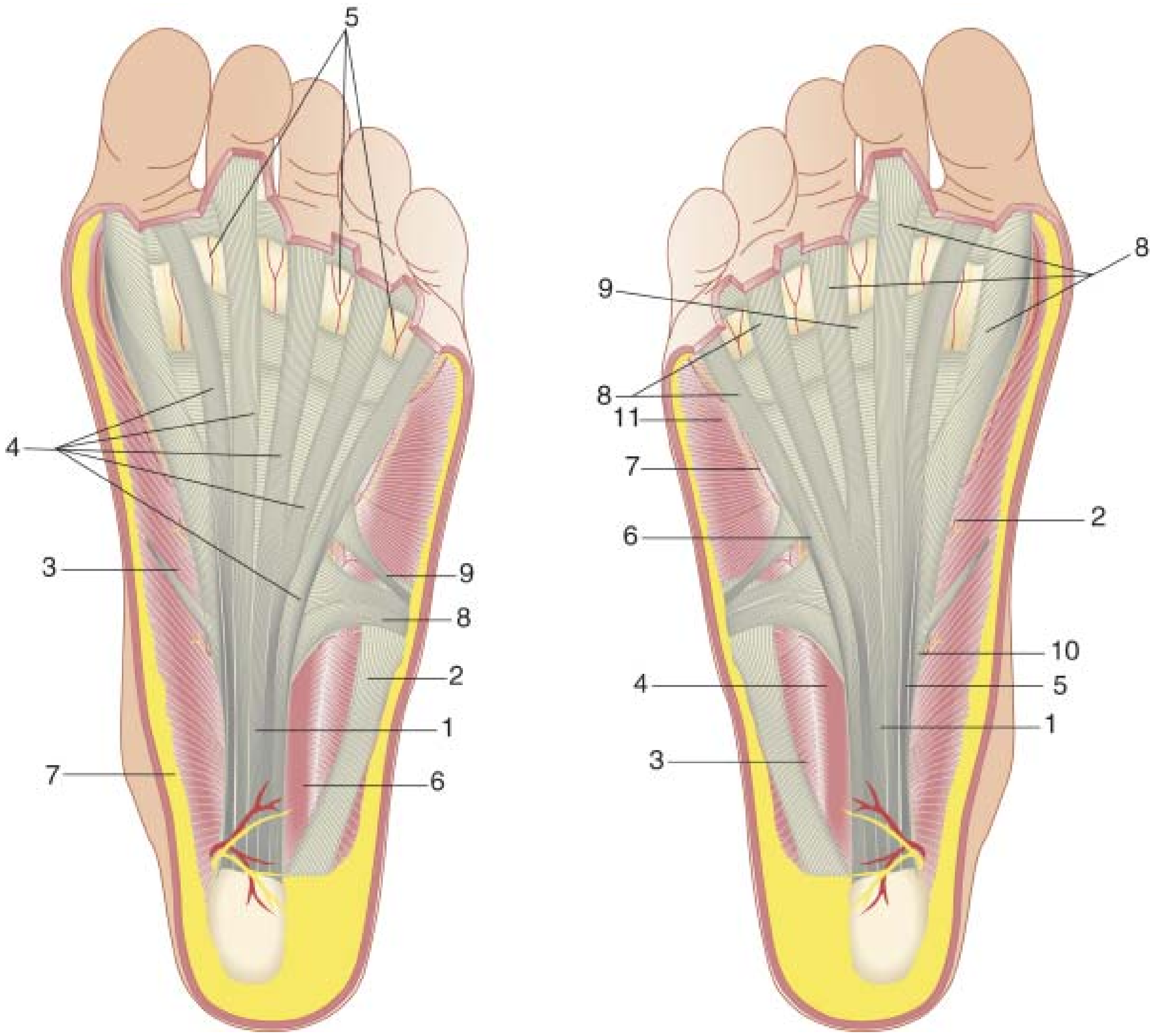

Figure 25F-15 Plantar aponeurosis composed of three parts: (1) central component of plantar aponeurosis, (2) medial component of plantar aponeurosis, (3) lateral component of plantar aponeurosis. From the clinical standpoint, the central portion is considered to be in the plantar aponeurosis. *(Redrawn from Rondhuis JJ, Hudson A: The first branch of the lateral plantar nerve and heel pain. Acta Morphol Neerland-Scand 24[4]:269-279, 1986.)*

Clinically, when considering the plantar aponeurosis, one is generally referring to the central portion, which extends from the medial tuberosity of the os calcis to the toes. It originates from the os calcis and passes to the proximal phalanges of the lesser toes through the longitudinal septa, to the big toe through the sesamoids, and into the skin of the ball of the foot through the vertical fibers. [114] Hyperextension of the toes and the metatarsophalangeal joints tenses the plantar aponeurosis, raises the longitudinal arch of the foot, inverts the hindfoot, and externally rotates the leg. This mechanism is passive and depends entirely on bony and ligamentous instability. This mechanism, whereby the arch is raised and supported with dorsiflexion of the toe, providing more stability to the foot, has been termed the *windlass mechanism* by Hicks. [115]

The posterior tibial nerve is located on the medial side of the foot behind the medial malleolus and beneath the flexor retinaculum (Fig. 25F-16). The medial calcaneal nerve arises at the level of the medial malleolus or below and passes superficially to innervate the skin of the heel. It may consist of one or two branches. The important anatomic point is that this nerve passes in the subcutaneous tissue between the plantar fascia and the skin. The next nerve, which is the nerve to the abductor digiti quinti and which branches off the lateral plantar nerve, passes deeper, beneath the plantar ligament and underneath the spur, if present, to innervate the abductor digiti quinti (Fig. 25F-17). It is important to differentiate the medial calcaneal nerve from the nerve to the abductor digiti quinti. [1166] [1167] [1168] They can be readily differentiated because the medial calcaneal nerve passes in the subcutaneous tissue and is superficial to the plantar aponeurosis, whereas the nerve to the abductor digiti quinti is deeper and passes beneath the plantar fascia.

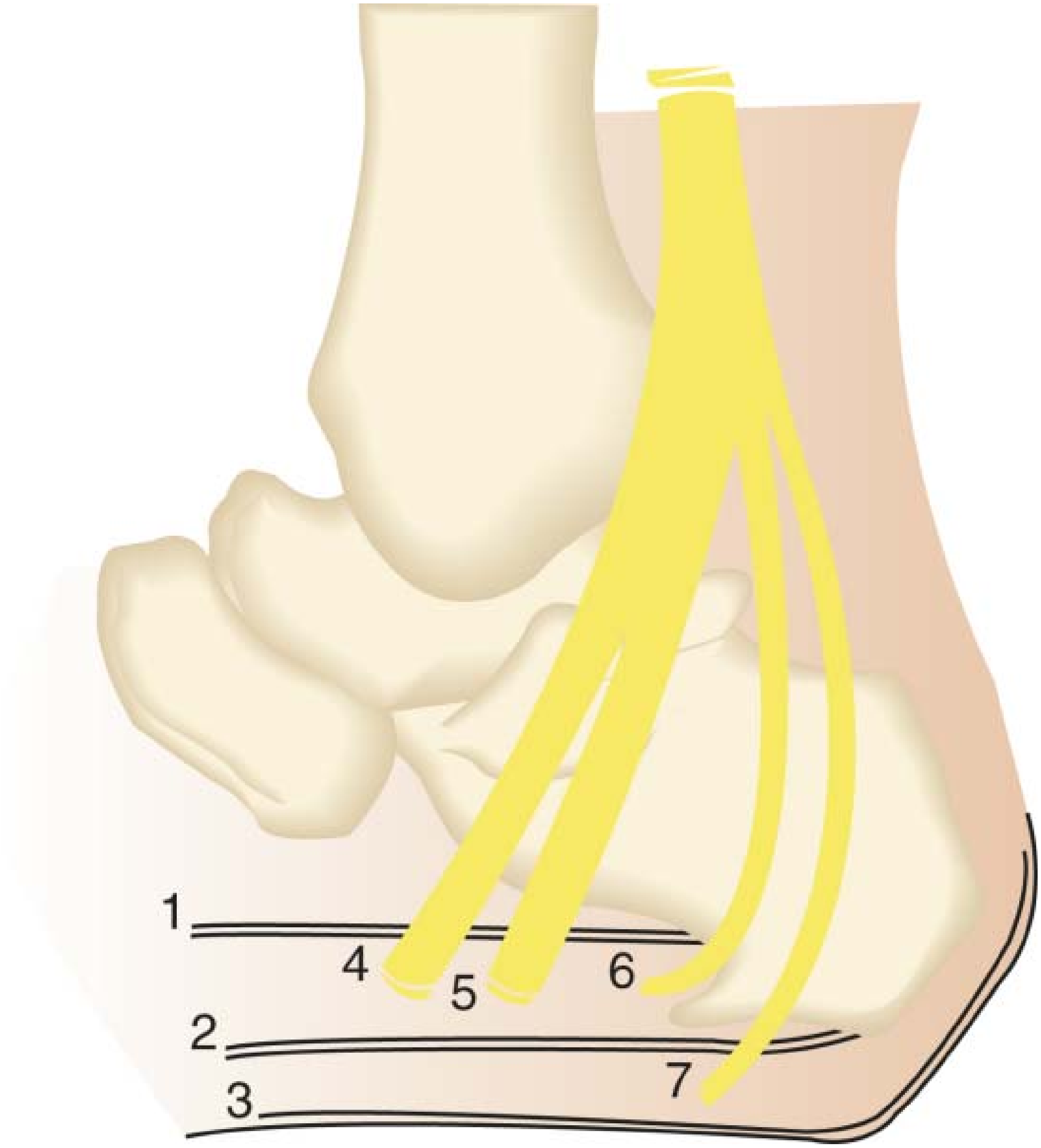

Figure 25F-16 Relationship of structures commonly associated with heel pain: (1) long plantar ligament, (2) plantar fascia, (3) skin, (4) medial plantar nerve, (5) lateral plantar nerve, (6) nerve to abductor digiti quinti, and (7) medial calcaneal nerve. Note that the medial calcaneal nerve supplying sensation to the heel passes superficial to the plantar fascia. The nerve to the abductor digiti quinti passes deep to the plantar fascia and beneath the spur.

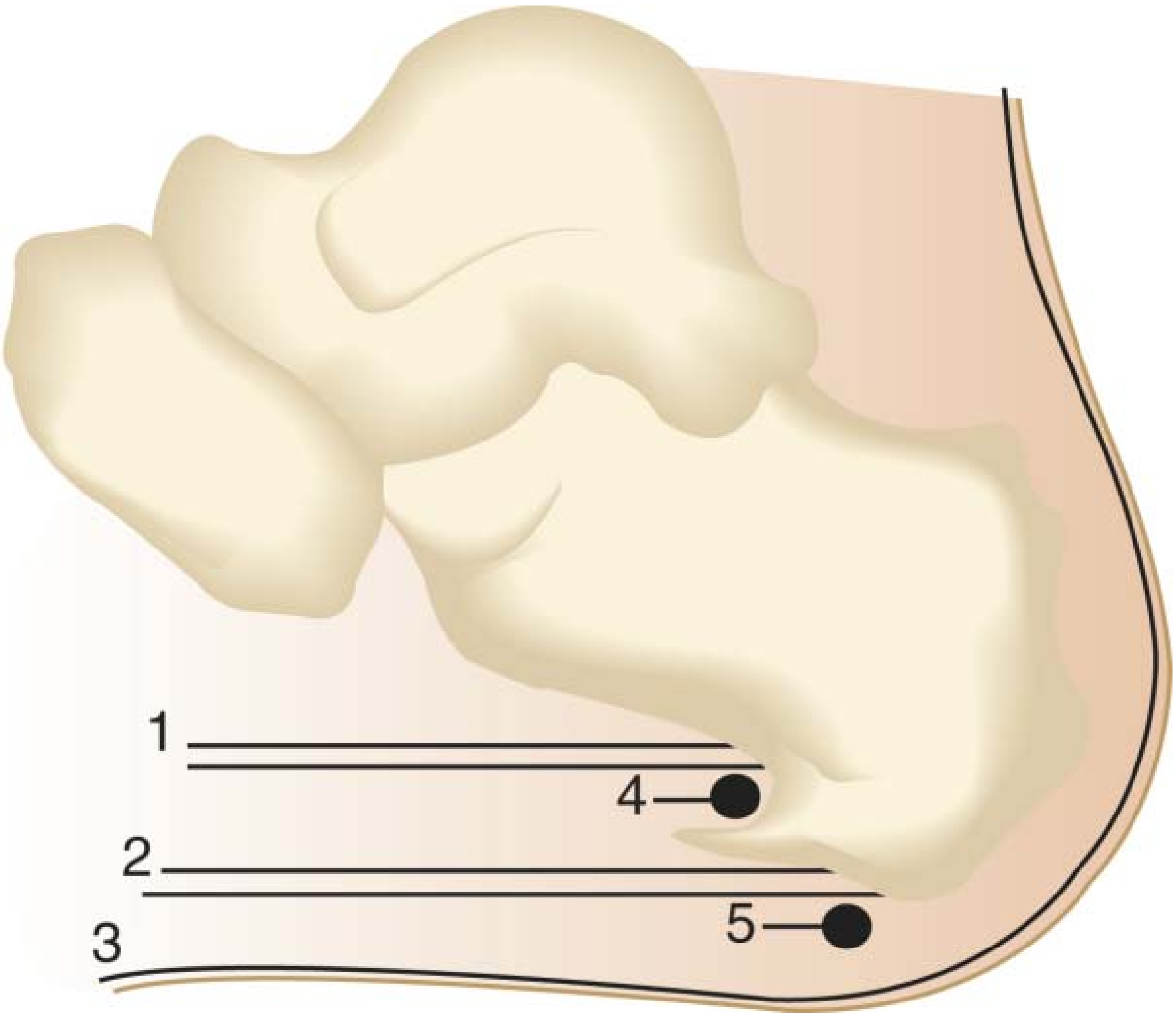

Figure 25F-17 Sagittal section of os calcis showing relationship of the nerves to the abductor digiti quinti and the medial calcaneal nerve as described by Baxter and Thigpen [64] and Przylucki and Jones. [72] (1) Long plantar ligament, (2) plantar fascia, (3) skin, (4) nerve to abductor digiti quinti, (5) medial calcaneal nerve.

Rondhuis and Hudson [116] noted that entrapment of the nerve to the abductor digiti quinti occurs between the abductor hallucis and the medial margin of the medial head of the quadratus plantae muscle. They did not find a perforation of the fascia as described by Baxter and Thigpen, [64] nor did they find any bursa in the origin of the plantar aponeurosis. They concluded that there are fibers that innervate the perichondrium and that sensory fibers are present and form free endings, producing pain sensation. They found motor branches to the flexor digitorum brevis and abductor digiti quinti muscles (Fig. 25F-18).

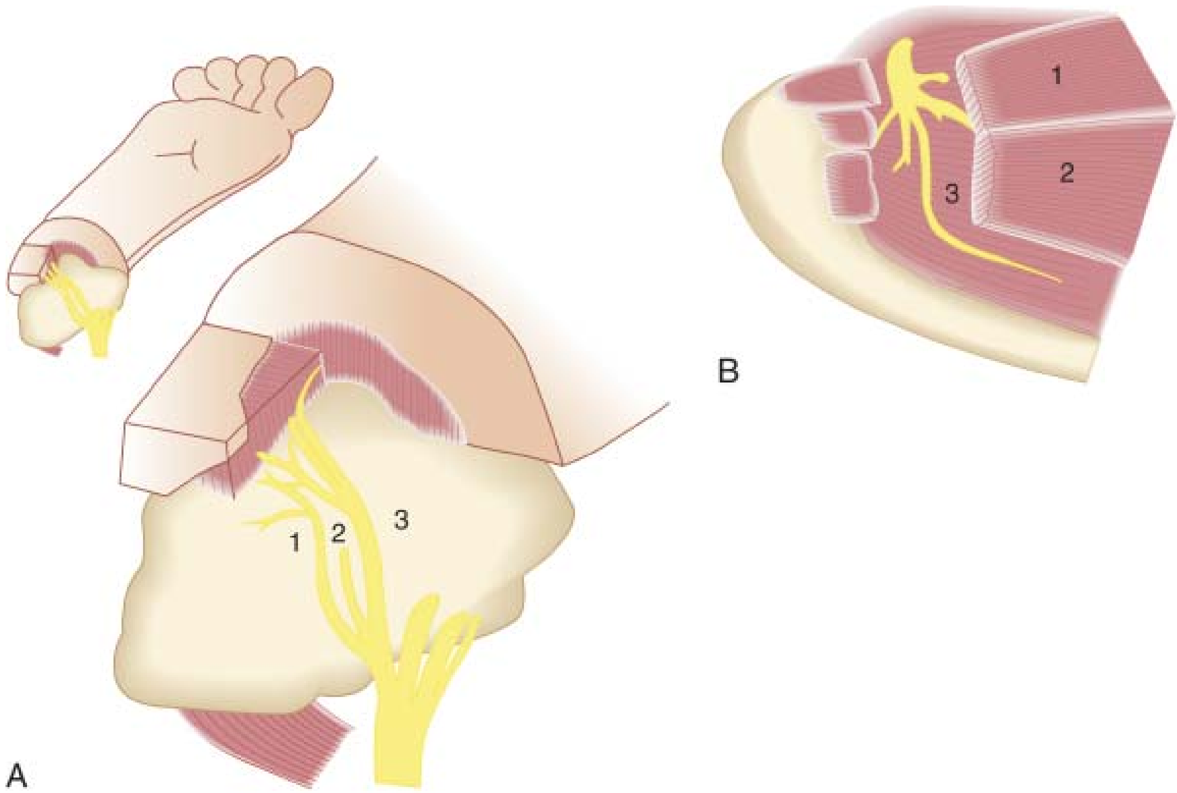

Figure 25F-18 A, Illustration of the nerve to the abductor digiti quinti. (1) Branch running to the medial process of the calcaneal tuberosity, bifurcating into a branch covering the perichondrium of the medial process and into another one running to a more lateral part of the calcaneal perichondrium. (2) Branch to the flexor digitorum muscle. (3) Branches to the abductor digiti minimi muscle. **B,** Drawing of the nerve to the abductor digiti quinti as dissected in an adult foot showing parts of the abductor hallucis (1) and flexor digitorum (2) muscles, which have been removed; the nerve runs across the quadratus plantae muscle (3) in a plantar direction, then turns into a horizontal plane and proceeds laterally. *(Redrawn from Rondhuis JJ, Hudson A: The first branch of the lateral plantar nerve and heel pain. Acta Morphol Neerland-Scand 24[4]:269-279, 1986.)*

The medial and lateral plantar nerves continue to the foot and pass through prospective foramina of the abductor muscles. When considering entrapment of the posterior tibial nerve, it is important to note that this nerve may be entrapped either beneath the flexor retinaculum at the level of the medial malleolus or at the point where the medial and lateral plantar nerves exit through separate foramina in the abductor muscles (Fig. 25F-19).

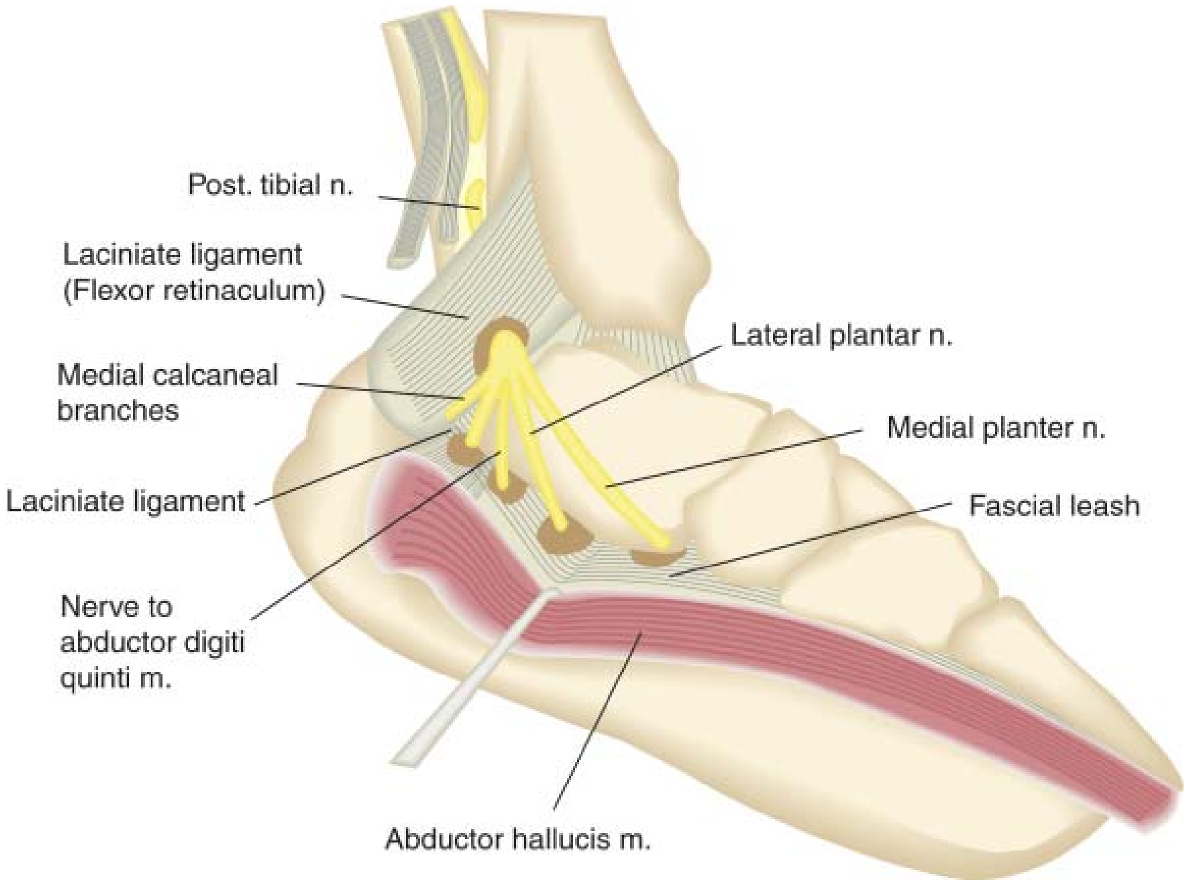

Figure 25F-19 Site of entrapment of the posterior tibial nerve and its branches, demonstrating possible entrapment beneath the laciniate ligament and at the point at which the nerve passes through the fascia of the abductor hallucis muscle. *(Redrawn from Baxter DE, Thigpen CM: Heel pain—operative results. Foot Ankle 5[1]:16-25, 1984. ©American Orthopaedic Foot and Ankle Society, 1984.)*

Relevant Biomechanics

The foot must be evaluated to ascertain which specific type of foot and which biomechanical components are involved.[68] A supple foot with a tendency toward a flatfoot deformity will place increased strain on the origin of the plantar fascia on the calcaneus because the windlass mechanism will be under increased strain in maintaining a stable arch during the propulsive phase of gait. Biomechanically, in an effort to avoid this strain, one could consider an orthotic device to correct the biomechanical deformity and to increase the support of the foot during the stance phase. Strapping with tape may also be used to hold the forefoot in adduction and the heel in varus to relieve the pressure on the origin of the plantar aponeurosis during propulsion. When a cavus foot is present, there may be excessive strain on the heel area because the foot lacks the ability to evert to absorb the shock and adapt itself to the ground. With a cavus foot, a soft cushioning material may be used to decrease the shock component and increase the area of contact. The goals of these orthotic devices are to reduce the stress on the medial tuberosity and the plantar fascia (Fig. 25F-20).

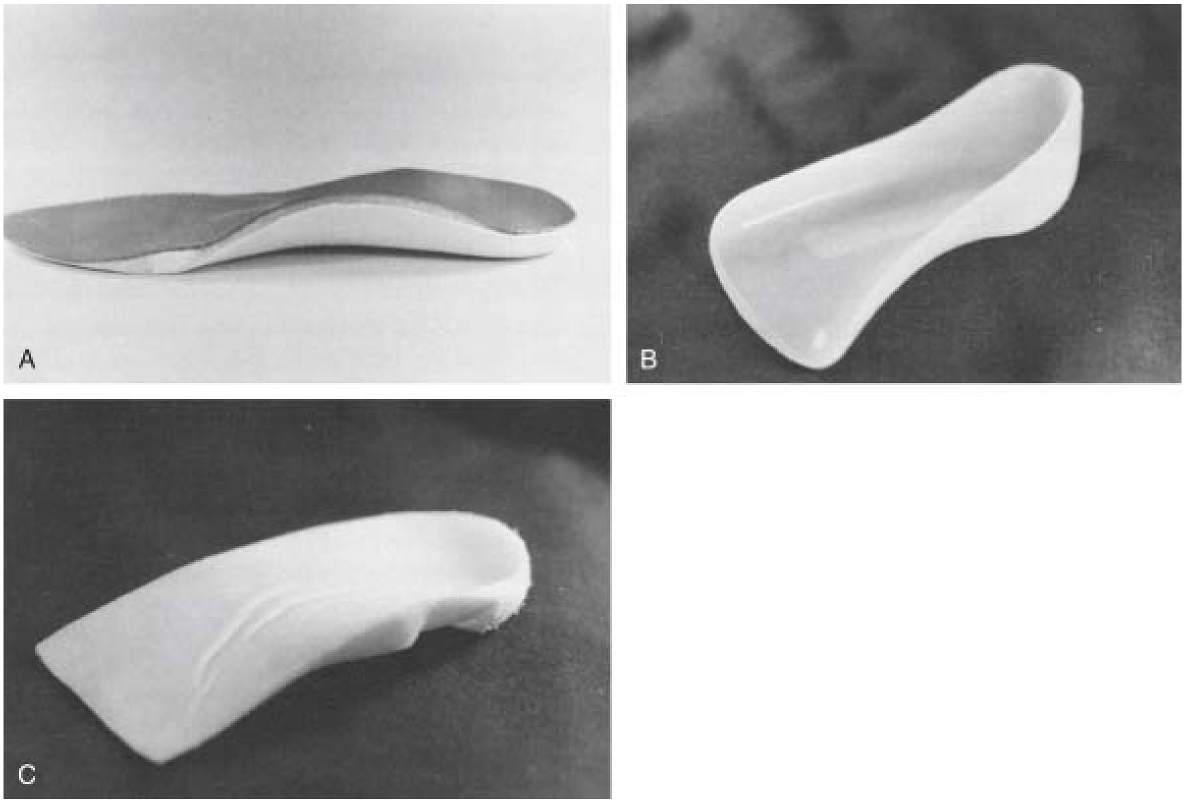

Figure 25F-20 A and **B,** Orthotic devices for flatfoot deformity due to increased strain on plantar fascia. **A,** Orthotic with forefoot post to prevent collapse of foot and thus strain on plantar fascia. **B,** Orthotic device constructed of polypropylene designed to prevent eversion and abduction of the foot to prevent strain on plantar fascia. **C,** Soft orthotic device constructed from polyethylene closed-cell foam thermoplastic material to provide cushioning and increased contact of arch.

Clinical Evaluation

History

The history usually reveals a slow but gradual onset of pain along the inside of the heel. [1166] [1167] [1168] Occasionally, the pain may be associated with a twisting injury of the foot, producing an abrupt onset of pain. [112] However, the clinical course is generally similar regardless of the onset. The location of the pain is generally described as along the medial side of the foot at the bottom of the heel. The pain is worse upon first arising in the morning and then decreases with increased activity. However, it may increase after prolonged activity. Periods of inactivity are generally followed by an increase in pain as activity is started again. Numbness of the foot is not present. When severe pain is present, the patient is unable to bear weight on the heel and will bear weight on the forepart of the foot.

Physical Examination

Physical examination consists of evaluating the foot to determine what type of foot is present. [68] As with any examination of the foot, examination must include the entire lower extremity as well. Specific examination of the foot reveals acute tenderness along the medial tuberosity of the os calcis. This tenderness may be at the origin of the central slip of the plantar fascia, or it may be deep, in which case it probably represents a deep inflammation, perhaps with involvement of the nerve to the abductor digiti quinti. The medial calcaneal nerve is palpated and tapped to search for paresthesias of the medial calcaneal nerve in the subcutaneous tissue; if found, this condition indicates inflammation and entrapment of the medial calcaneal nerve in this area. The plantar fascia is palpated to determine whether the plantar fascia is tender just at its origin or throughout its course. The plantar fascia is also palpated for nodules, the presence of which suggests plantar fibromatosis. The plantar fascia is palpated both with the toes flexed so that it is supple and with the toes extended, which places tension on the plantar fascia. The tarsal tunnel is palpated and percussed to elicit any tenderness, inflammation, or Tinel's sign of the posterior tibial, medial or lateral plantar, or medial calcaneal nerves. Sensation of the foot is evaluated by light touch and pinprick to ascertain the status of the sensory nerves. The subtalar joint and ankle are examined for motion and mobility, both actively and passively to rule out referred pain from these areas. Active motor power of the muscles that cross or affect this area, such as the posterior tibial, anterior tibial, peroneus longus and brevis, and toe flexors and extensors, are checked to determine their motor power and also to see whether any pain is produced with active motor function. Neurologic examination of the remainder of the lower extremities and back is performed as indicated.

Diagnostic and Radiographic Studies

Standing, full-weight bearing radiographs of the heel including the foot in the anteroposterior and lateral standing projections are taken. Radiographs taken in this manner provide information about the osseous structures of the foot as well as specific details of the os calcis. Standing radiographs also provide information about the biomechanical status of the foot because they are taken in the base and angle of gait and with the foot loaded. They thus represent the position and relationships of the bones of the foot during the stance phase of gait. Such radiographs also help to classify the foot as to type: normal, flat, or cavus. The presence of a spur or calcification along the medial tuberosity are also shown. Axial non–weight-bearing views of the os calcis may be taken to provide information about the os calcis in a second plane. Graham [86] reported on radiographs of the heel taken at a 45-degree angle that showed bony condensation on the medial side (Fig. 25F-21). This was thought to represent a fatigue fracture. Ninety-eight percent of his cases were associated with a positive bone scan on the side of the heel that had pain. A technetium-99m bone scan may be performed to provide objective evidence of an inflammatory abnormality in this area (Fig. 25F-22). Sewell and associates [117] interpreted these bone scans in five patients with heel pain to show that the uptake is increased at the site of insertion of the plantar fascia into the calcaneus in patients with clinical signs of plantar fasciitis and that such signs may occur in the absence of any radiologic change. Changes in intensity of tracer uptake reflected symptomatic improvement. They suggested that radiologic imaging allowed quantitative assessment of inflammation of the “enthesis” and enabled therapy to be evaluated. Vasavada and colleagues [118] obtained early blood pool images to detect soft tissue inflammation (plantar fasciitis) when delayed images were normal.

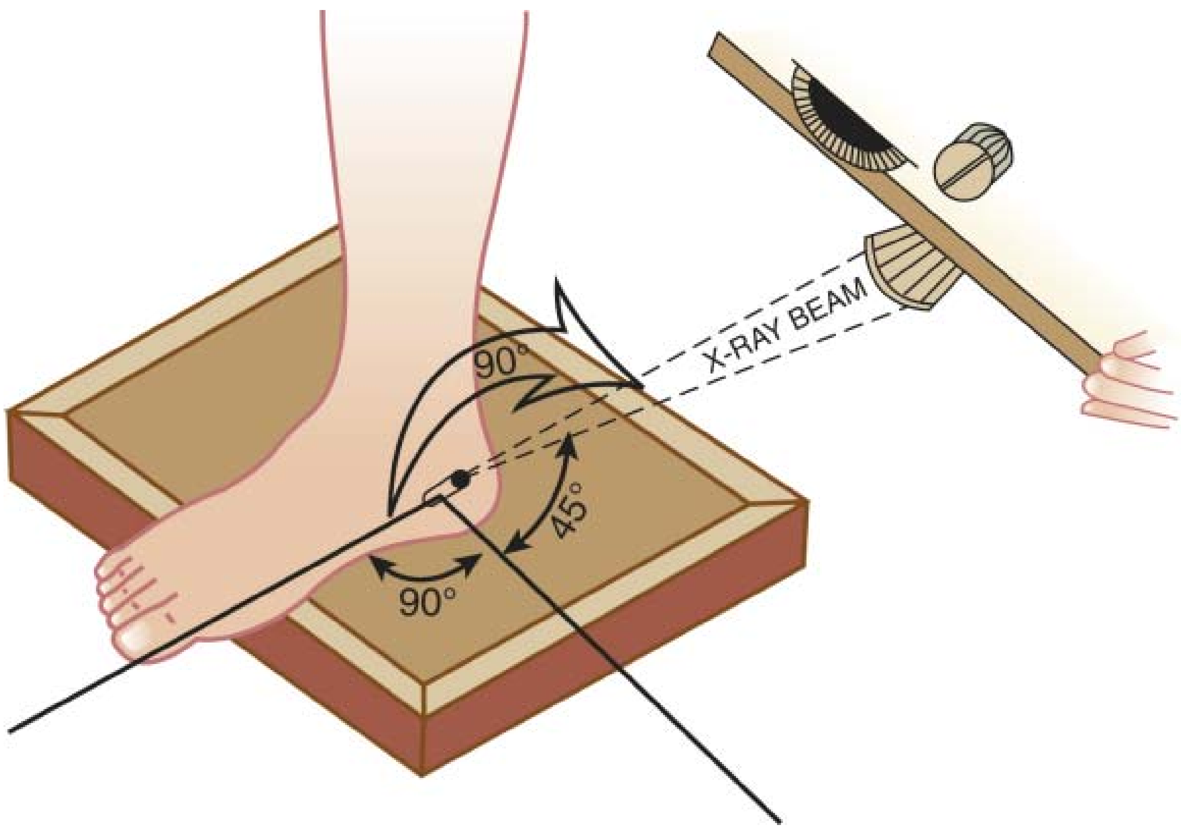

Figure 25F-21 Position for roentgenogram of the heel to demonstrate fatigue fracture on the medial side of the os calcis as described by Graham. The x-ray beam is angled downward from the heel, making a 90-degree angle to the toe-heel plane of the foot and a 45-degree angle to the horizontal plane of the cassette. *(Redrawn from Graham CE: Painful heel syndrome: Rationale of diagnosis and treatment. Foot Ankle 3[5]:261-267, 1983. ©American Orthopaedic Foot and Ankle Society, 1983.)*

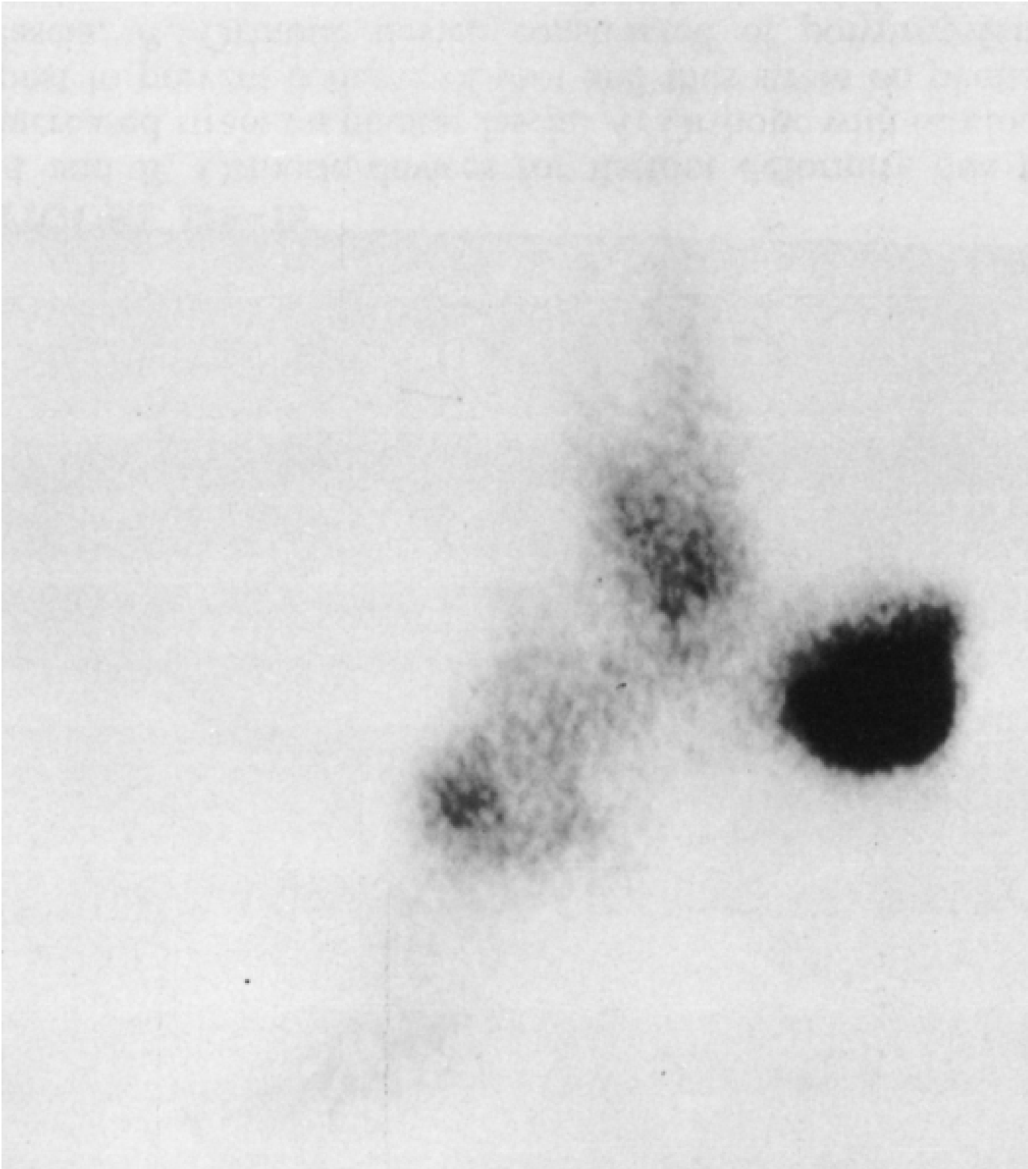

Figure 25F-22 Bone scan showing increase in uptake in the calcaneal area associated with subcalcaneal pain syndrome.

Williams and colleagues [107] evaluated a total of 45 patients with painful heel syndrome without evidence of associated inflammatory arthritis. These patients were studied using technetium-99m isotope bone scans and lateral and 45-degree medial oblique radiographs of both feet. Of the 52 painful heels in these 45 patients, 59.6% showed increased uptake at the calcaneus. These authors found that patients with scans showing increased uptake tended to have more severe heel pain and responded more frequently to a local hydrocortisone injection. Their findings suggested that patients with normal scans had some noninflammatory lesion such as simple trauma or entrapment of the medial calcaneal nerve. Their findings showed no evidence of stress fractures in contrast to Graham's findings. [86]

Intenzo and Wapner [119] studied 15 patients complaining of chronic heel pain that underwent three-phase technetium-99m MDP bone scintigraphy. Ten patients demonstrated abnormal scan findings consistent with plantar fasciitis with uptake only in the early soft tissue phase and had responded to conventional therapy. Two patients were found to have calcaneal stress fractures, and one patient demonstrated a calcaneal spur that required no treatment. The remaining two patients had normal scans and did not appear clinically to have plantar fasciitis. They found the three-phase bone scan useful in diagnosing plantar fasciitis and in distinguishing it from other causes of the painful heel syndrome.

Laboratory studies in most patients with subcalcaneal pain syndrome are normal. When the subcalcaneal pain syndrome is present, and especially if it is persistent and severe, consideration must be given to the diagnosis of a systemic disorder such as seronegative arthropathy. It has been reported that in patients with the subcalcaneal pain syndrome, there may be an incidence as high as 16% of subsequent development of a systemic arthritic disorder. [72]

Eastmond and associates [120] described 26 patients with seronegative pauciarticular arthritis and positive HLA-B27. They noted that low back and buttock pain, Achilles tendinitis, and dactylitis of the toes were more frequent in HLA-B27–positive patients. They suggested that arthritis and an increased frequency of HLA-B27 may occur in adults who do not have clinical or radiologic signs of any other seronegative arthritis. The authors believe that this test should be considered part of the systemic work-up for a patient with chronic, recurrent, and incapacitating heel pain. Gerster [121] found the painful heel syndrome with plantar fasciitis or Achilles tendinitis in 33 of 150 patients suffering from a seronegative spondyloarthritis. An HLA-B27 antigen was found in 91% of the patients. He contrasted this study with a study of 220 cases of rheumatoid arthritis. In these patients, he found that Achilles tendinitis was not encountered and that plantar fasciitis was exceptional.

Gerster and Piccinin [114] also described severe heel pain in 4 of 18 cases of juvenile-onset seronegative spondyloarthropathy. The plantar fascia was affected in each case. Four other patients among these 18 had mild heel pain. Gerster and colleagues [122] also reported on 30 cases of severe heel pain in patients with seronegative spondyloarthropathy. Four patients underwent surgical intervention with either disinsertion of the plantar aponeurosis or rasping of the calcaneal spur. This surgery failed, and Gerster and colleagues stated that surgery is contraindicated in patients with severe heel pain associated with seronegative spondyloarthritis.

Proximal neurologic causes of heel pain should also be considered. Tarsal tunnel syndrome may be present with referred pain to the heel and sole of the foot. A positive Tinel's sign may suggest this diagnosis. Electromyographic and nerve conduction studies should be used to rule out this condition. [68] Heel pain may also be referred from the lumbar spine. If a spinal or proximal cause appears to be a possibility, appropriate laboratory and radiographic studies should be performed as indicated.

MRI has shed light on the anatomic structures involved in subcalcaneal heel pain. [123] Heel pain can be caused by disorders of the plantar fascia, calcaneus, tendons, or adjacent nerves. Because these conditions can lead to pain located in a small area of the heel, a precise clinical diagnosis may be difficult. This article describes some of these various causes of heel pain and how MRI helps to characterize them.

Grasel and colleagues [124] evaluated various MRI signs of plantar fasciitis and to determine whether a difference in these findings exists between clinically typical and atypical patients with chronic symptoms resistant to conservative treatment. They found signs on MRI that included occult marrow edema and fascial tears. Patients with these manifestations appeared to respond to treatment in a manner similar to that of patients in whom MRI revealed more benign findings.

Treatment Options

Nonoperative Therapy

Management of subcalcaneal heel pain should initially begin with nonoperative treatment. [125] Plantar fasciitis is a common cause of heel pain, which frustrates patients and practitioners alike because of its resistance to treatment. Although normally managed with conservative treatment, plantar fasciitis is frequently resistant to the wide variety of treatments commonly used, such as NSAIDs, rest, pads, cups, splints, orthotics, corticosteroid injections, casts, physical therapy, ice, and heat. Although there is no consensus on the efficacy of any particular conservative treatment regimen, there is agreement that nonsurgical treatment is ultimately effective in about 90% of patients. Because the natural history of plantar fasciitis has not been established, it is unclear how much of symptom resolution is in fact due to the wide variety of commonly used treatments.

Gill and Kiebzak [126] studied 411 patients with a clinical diagnosis of plantar fasciitis who were assessed for predisposing factors. Each patient completed an outcomes assessment survey instrument that ranked effectiveness of various nonsurgical treatment modalities. Listed in descending order of effectiveness, the treatment modalities assessed were short leg walking cast, steroid injection, rest, ice, runner's shoe, crepe-soled shoe, aspirin or NSAID, heel cushion, low-profile plastic heel cup, heat, and Tuli heel cup. Treatment with a cast ranked the best. The Tuli heel cup ranked the poorest. Most of the treatments were found to be unpredictable or minimally effective. However, in their study, stretching and night splints were not included.

A randomized trial by Landorf and associates in 2006 found that foot orthoses produce only small short-term benefits in function and pain in people with plantar fasciitis. [127] When compared with a sham device, these orthoses do not have any long-term benefits. However, another study performed by Roos and colleagues demonstrated that foot orthoses and anterior night splints are equally effective in both short- and long-term times. [128] Further studies will be necessary to elucidate the role of foot orthoses in the treatment of plantar fasciitis.

O'Brien and Martin [94] studied 58 painful heels in 41 patients who received conservative treatment. Seventy percent were classified as having excellent results (no remaining symptoms), 26% were classified as having good results (50% or less of the symptoms remained), and only 3.5% were symptomatic and classified as having poor results. Injection therapy was most successful when the preceding symptoms had been present for an average of 2.6 months. Orthotic therapy was most successful when the duration of the preceding symptoms was 21⁄2 years.

Shikoff and coworkers [98] reported a retrospective study of 195 patients with heel pain. The typical patient was middle-aged and overweight; 91% of the respondents were classified as having above normal weight for their sex and height. About half of the patients continued to wear heel padding or to take oral medication, or both, for months after the initial visit. Thirty percent experienced only marginal relief from pain or had an unsatisfactory result.

The efficacy of oral NSAIDs was evaluated by Donley and colleagues. [129] In this randomized, prospective, placebo-controlled study, 29 patients were treated with a conservative regimen. In addition, patients were randomly assigned to placebo or NSAIDs. The study concluded that NSAIDs, particularly when taken for 2 months, increased pain relief and decreased disability when used in conjunction with a conservative treatment regimen.

Callison [80] reviewed 400 consecutive patients with heel pain who were seen in his office during a 40-month period from October 1985 to February 1989. Radiographs of all were obtained. Heel spurs were present in 45% and absent in 53%. The series consisted of 65% women and 35% men; 30% of the women were obese, but only 10% of the men were obese. Seven percent were involved in active sports. Patients were treated with steroid injections, orthoses, calf-stretching exercises, and NSAIDs. Plaster immobilization was occasionally used. Results showed that 73% improved significantly within 6 months, 20% failed to improve, and 7% did not return and were lost to follow-up.

Davis and coworkers [130] studied 105 patients (70% female and 30% male; average age, 48 years) with 132 symptomatic heels who were treated according to a standard nonoperative protocol and then reviewed at an average follow-up of 29 months. The treatment protocol consisted of NSAIDs, relative rest, viscoelastic polymer heel cushions, Achilles tendon stretching exercises, and, occasionally, injections. Obesity, lifestyle (athletic versus sedentary), sex, and presence or size of heel spur did not influence the treatment outcome. Ninety-four patients (89.5%) had resolution of heel pain within 10.9 months. Six patients (5.7%) continued to have significant pain but did not elect to have operative treatment, and 5 patients (4.8%) elected to have surgical intervention. They concluded that despite attention to the outcome of surgical treatment for heel pain in the current literature, initial treatment for heel pain is nonoperative. The treatment protocol used in this study was successful for 89.5% of the patients.

Wolgin and associates [131] evaluated the long-term results of patients treated conservatively for plantar heel pain. After eliminating those patients with worker's compensation-related complaints and those with documented inflammatory arthritides, data on 100 patients (58 females and 42 males) were available for review. The average patient age was 48 years (range, 20 to 85 years). The average follow-up was 47 months (24 to 132 months). Clinical results were classified as good (resolution of symptoms) for 82 patients, fair (continued symptoms but no limitation of activity or work) for 15 patients, and poor (continued symptoms limiting activity or changing work status) in 3 patients. The average duration of symptoms before medical attention was sought was 6.1, 18.9, and 10 months for the three groups, respectively. They concluded that although the treatment of heel pain can be frustrating because of its indolent course, a given patient with plantar fasciitis has a good chance of complete resolution of symptoms.

The use of steroid injection in the treatment of painful heel syndrome has been advocated but is controversial. [1231] [1232] [1233] Few reports on the long-term efficacy of injection are available. Miller and colleagues [134] evaluated the results of a single injection of corticosteroids in patients with painful heel syndrome. Twenty-seven heels in 24 patients were injected with a combination of 1 mL of lidocaine and 1 mL of betamethasone (6 mg). These patients had never previously received an injection to their heels and had continued symptoms of pain after a trial of other nonoperative treatment modalities. After the injection, patients were seen and surveyed periodically for a period of 5 to 8 months. The amount of pain relief that they obtained, the length of time this lasted, and the amount of heel pain present at the final follow-up were recorded. At final follow-up, the pain had returned to its preinjection level in 13 feet. Based on the results of our study, they believed that a steroid injection is a reasonable adjunct in the treatment of painful heel syndrome, but that it is unlikely to provide permanent pain relief.

Complications from steroid injection have been reported and can be severe. Rupture of the plantar fascia and irreversible fat pad atrophy [1235] [1236] [1237] [1238] have been reported. Loss of the plantar fad pad decreases the physiologic protection in the subcalcaneal region and increases the risk for the return of intractable symptoms. Injection should be used with caution in patients with a competent fat pad and not repeated more than once in any 3-month interval. If fat pad atrophy occurs, repeat injection should be avoided.

In one study published in 2007, Lee and Ahmad evaluated the results of patients injected with intralesional autologous blood injection compared with corticosteroid injections. [139] Sixty-four patients were randomized in this prospective, controlled trial. Patients were followed for 6 months. As compared with corticosteroid treatment, intralesional autologous blood injection is not as expeditious or effective in treating chronic plantar fasciitis.

Iontophoresis has been used as an adjunct to conservative care by physical therapists. Acetic acid or dexamethasone can be used. A recent study by Osborne and Allison demonstrated that six treatments of acetic acid iontophoresis combined with taping provided greater relief from stiffness and equivalent pain relief when compared with dexamethasone iontophoresis with taping. [140]

Pfeffer and associates [141] reviewed a 15-center prospective randomized trial to compare several nonoperative treatments for proximal plantar fasciitis (heel pain syndrome). Included were 236 patients with duration of symptoms of 6 months or less. Patients with systemic disease, significant musculoskeletal complaints, sciatica, or local nerve entrapment were excluded. Patients were randomized prospectively into five different treatment groups. All groups performed Achilles tendon and plantar fascia stretching in a similar manner. One group was treated with stretching only. The other four groups stretched and used one of four different shoe inserts, including a silicone heel pad, a felt pad, a rubber heel cup, or a custom-made polypropylene orthotic device. Patients were re-evaluated after 8 weeks of treatment. Combining all the patients who used a prefabricated insert, they found that their improvement rates were higher than those assigned to stretching only (P=.022) and those who stretched and used a custom orthosis (P = .0074). We conclude that when used in conjunction with a stretching program, a prefabricated shoe insert is more likely to produce improvement in symptoms as part of the initial treatment of proximal plantar fasciitis than a custom polypropylene orthotic device.

Wynne and associates reported on the effect of counterstrain treatment in patients with plantar fasciitis. [142] This single-blind, randomized controlled study of crossover design evaluated 20 patients treated with either counterstrain or placebo. This group found a significant relief of symptoms that were most pronounced in the first 48 hours after treatment. However, a 2-week stretching program has been shown to not statistically benefit first-step pain, foot pain, foot function, or general foot health when compared with patients not stretching. [143] Other studies have demonstrated a tissue-specific plantar fascia-stretching protocol as a key component in the treatment of chronic plantar fasciitis. [1244] [1245] Long-term use of this therapy produces a marked decrease in pain and functional limitations and a high rate of satisfaction.

The use of night splints to prevent plantar flexion of the ankle, as an adjunct to the treatment of subcalcaneal pain, has received considerable interest in the past few years. Wapner and Sharkey [146] originally reported the results of the use of molded ankle-foot orthosis night splints for the treatment of recalcitrant plantar fasciitis in 14 patients with a total of 18 symptomatic feet. All patients had symptoms for greater than 1 year and had previously undergone treatment with NSAIDs, cortisone injections, shoe modifications, and physical therapy without resolution. All patients were provided with custom-molded polypropylene ankle-foot orthoses in 5 degrees of dorsiflexion because no commercially manufactured splints were yet available. With continued use of NSAID medication, Tuli heel cups, Spenco liners, and general stretching exercises, successful resolution occurred in 11 patients in less than 4 months. There were three failures. It is thought that night splints provide a useful, cost-effective adjunct to current therapeutic regimens of plantar fasciitis by assisting in maintaining the flexibility gained by stretching exercises and relieving morning start-up pain, and thus reducing the time to resolution of symptoms. Multiple subsequent studies have demonstrated the usefulness of these devices, and they are now readily commercially available. [1179] [1247] [1248] [1249] [1250]

Martin and colleagues [151] reported on an outcome study of 400 patients with chronic plantar fasciitis treated nonoperatively and concluded that patients could expect a good outcome. Compliance with their protocols did not have a correlation with outcome, with one exception. Patients with chronic conditions who were compliant with the use of the night splint had a better outcome. Patients' subjective perceptions were that stretching, night splints, and heel pads were of equal importance in their treatment. This study suggests that within the first 12 months of onset, early, aggressive, nonsurgical treatment offers the best chance of a good outcome.

Hyland and associates performed a randomized controlled trial comparing calcaneal taping, sham taping, and plantar fascia stretching for the short-term management of plantar heel pain. [152] They found calcaneal taping to be more effective for the relief of plantar heel pain than stretching, sham, taping, or no treatment.

Alvarez [1177] [1253] was one of the first to report on the OssaTron as another alternative for management of heel pain syndrome after failure of nonoperative management and before surgical management. His study evaluated primarily the safety and early preliminary efficacy of the OssaTron in the treatment of patients with plantar fasciitis unresponsive to nonoperative management. Twenty heels of 20 patients were treated with 1000 extracorporeal shock waves from the OssaTron to the affected heel after administration of a heel block. Each patient was evaluated by radiography, Kin Com, range of motion, and physical examination, including evaluation of point tenderness by means of a palpometer and according to a 10-point visual analog scale. The control was the contralateral heel. Patients also performed self-evaluation by means of patient activities of daily living questionnaire and pain reported by a 10-point visual analog scale. There were no complications or adverse effects attributed to the procedure of orthotripsy. Of the 20 patients treated, 17 were improved or pain free. Eighteen of the 20 subjects treated stated that they would undergo the procedure again instead of surgery. Based on these results, Alvarez concluded that orthotripsy is a safe and effective method of treating heel pain syndrome that has been unresponsive to nonoperative management.

Zingas and associates [154] studied the safety and efficacy of musculoskeletal shock-wave therapy in the treatment of chronic plantar fasciitis in 29 patients with chronic plantar fasciitis who were enrolled in a randomized, 1:1 allocated, placebo-controlled, prospective, double-blind clinical study with two groups: one receiving extracorporeal shock-wave therapy with the Dornier Epos Ultra and the other receiving sham treatment. The authors hypothesized that shock-wave therapy will be useful in the treatment of chronic plantar fasciitis that has failed conventional conservative methods. Preliminary results indicate that shock-wave therapy is a safe and efficacious treatment for chronic plantar fasciitis with only minor, transient adverse effects. The U.S. Food and Drug Administration has approved this treatment for chronic plantar fasciitis.

Since that time, numerous studies have been published evaluating the efficacy of shock-wave therapy for the treatment of chronic proximal plantar fasciitis. One of the most compelling studies is a double-blind, multicenter, randomized controlled study performed by Malay and associates. [155] The cohort in this study was followed for 1 year after one treatment of shock-wave therapy. The authors found that patients treated with shock-wave therapy had objectively and subjectively less pain than those treated with sham intervention. Similar results have been published by other groups in the literature. [1256] [1257] [1258] [1259] [1260] Although some of these studies use one treatment of high-energy shock-wave therapy, whereas others use three weekly treatments of low-energy shock-wave therapy, it is unclear what constituted "high-energy" treatments. One study evaluating moderate-dose shock-wave therapy has demonstrated no effect on outcome measures, 6 months after treatment when compared with sham therapy. [161] There is no correlation between the presence or absence of a heel spur and the eventual treatment outcome. [162] Location of the treatment probe for shock-wave therapy has been found to be most effective when placed, with patient aid, at the most tender site. [163] As extracorporeal shock-wave therapy continues to be studied, its effectiveness and efficacy in the treatment of chronic plantar fasciitis will continue to be elucidated.

Operative Therapy

Surgical management of chronic plantar fasciitis remains controversial. The American Orthopaedic Foot and Ankle Society (AOFAS) issued a position statement on the timing of surgical intervention (Box 25F-1).

BOX 25F-1

AMERICAN ORTHOPAEDIC FOOT AND ANKLE SOCIETY POSITION STATEMENT: ENDOSCOPIC AND OPEN HEEL SURGERY

1. Nonsurgical treatment is recommended for a minimum of 6 months and preferably 12 months.
2. More than 90% of patients respond to nonsurgical treatment within 6 to10 months.
3. When surgery is considered in the remaining patients, a medical evaluation should be considered before surgery.
4. Patients should be advised of complications and risks if an endoscopic or open procedure is not indicated.
5. If nerve compression is coexistent with fascial or bone pain, an endoscopic or open procedure should not be attempted.
6. The AOFAS does not recommend surgical procedures before nonoperative methods have been used.
7. The AOFAS does support responsible, carefully planned surgical intervention when nonsurgical treatment fails and work-up is complete.
8. The AOFAS supports cost constraints in the treatment of heel pain when the outcome is not altered.
9. The AOFAS recommends heel padding, medications, and stretching before prescribing custom orthoses, and extended physical therapy.
10. This position statement is intended as a guide to the orthopaedist and is not intended to dictate a treatment plan.

Review of the literature provides no clear consensus on the role of surgery. Snook and Chrisman,[100] reporting on 22 patients with 25 painful heels, found that 16 patients with 18 painful heels obtained relief from pain with a variety of conservative measures, including a plastic heel cup. Seven of the patients (8 painful heels) required surgical therapy consisting of excision of the medial and inferior tuberosities of the os calcis. All obtained pain relief. The follow-up period in these patients ranged from 2 to 7 years. Goulet[85] reported on the use of soft orthoses for treating plantar fasciitis.

Ali[103] reported that steroid injections provided relief in 13% of the patients treated in his series. Plantar fasciotomy provided permanent relief in 75%, and plantar fasciotomy with excision of the calcaneal spur produced a cure in 85%. However, Mann[71] stated that surgical treatment of calcaneal spurs gives only 50% to 60% satisfactory results.

Ward and Clippinger[102] used a curved oblique plantar incision on the proximal aspect of the medial longitudinal arch to release the plantar fascia in eight feet with recalcitrant plantar fasciitis (Fig. 25F-23). Seven feet became pain free, and the eighth was 75% improved. Normal sensation was preserved in all cases. There were no painful scars or neuromas.

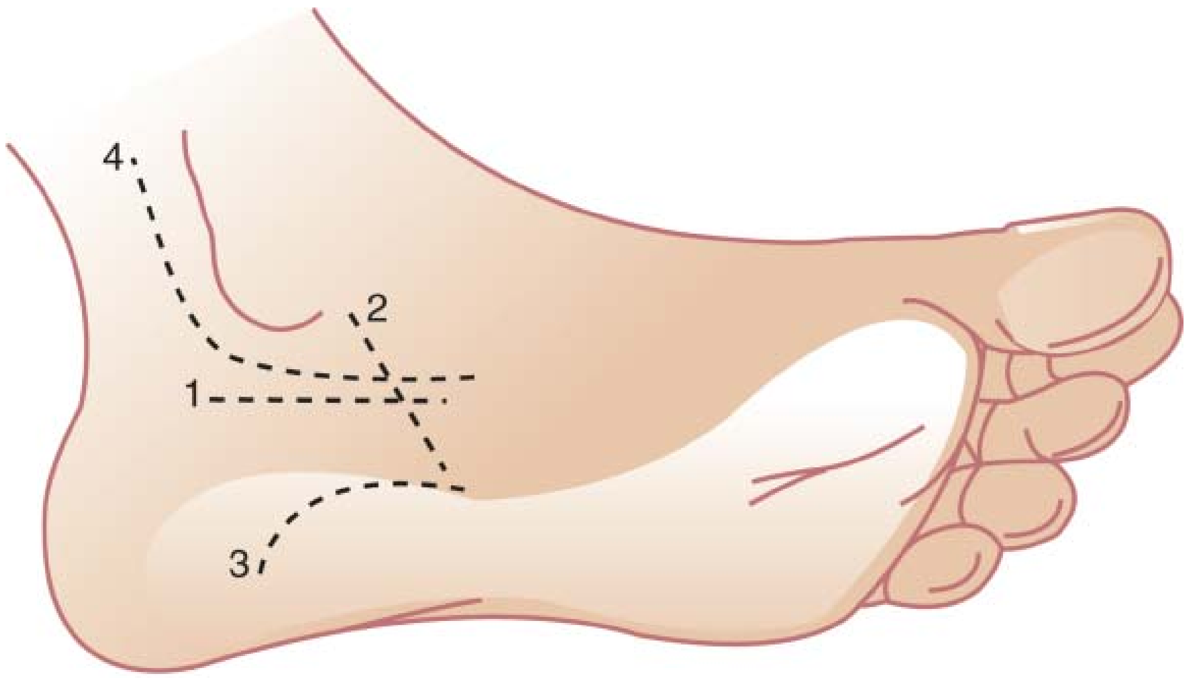

Figure 25F-23 Incisions used in subcalcaneal pain surgery: (1) medial incision, (2) oblique incision, (3) incision of Ward and Clippinger, (4) incision for complete release and exploration.

Shmokler and colleagues[106] used fluoroscopy to aid in the removal of heel spurs. They used this modality in 130 patients between January 1, 1984 and September 5, 1987 but did not provide a statistical analysis.

Contompasis[81] operated on 129 patients through a medial horizontal incision, performing exostosectomy and fasciotomy, fasciotomy only, or exostosectomy only. Most heels underwent more exostosectomies (115 cases) than fasciotomies. Overall, there was complete improvement in 43%, some improvement in 38%, no improvement in 17%, but no worsening of symptoms in any patient. DuVries[84] in 1957 treated 37 patients with medial incision and a fasciotomy and rasping of the spur; he reported good results without recurrence of symptoms.

Hassab and El-Sherif[87] drilled the os calcis to obtain relief of recalcitrant heel pain. They performed 68 operations in 60 patients and reported excellent results in 62, good in 2, and poor in 4. Jay and associates[89] performed calcaneal decompression for chronic heel pain through a lateral approach in four patients and achieved pain relief in three of the four patients. One patient had immediate relief. Two experienced relief from symptoms several weeks postoperatively. The fourth patient did not become pain free.

Kenzora[164] considered the painful heel syndrome an entrapment neuropathy. He described exploration of the nerve to the abductor digiti quinti and release of the nerve through a midline plantar incision of the heel. He submitted a preliminary report on six patients, none of whom were dissatisfied but had only a short follow-up. Two patients had temporary numbness along the medial aspect of the incision. He considered the procedure experimental.

Satku and colleagues [165] described heel pain produced by an unusual cause—that of an osteocartilaginous nodule within the heel. This was a glistening, circumscribed, lobulated, ovoid mass measuring 1.6 × 2.5 × 1.4 cm. The patient was relieved of pain with excision of the nodule.

Shaw and associates [166] called attention to the fact that heel pain can occur in patients with sarcoidosis. They reported seven cases in which sarcoid-related heel pain was a major characteristic of the sarcoid disease process and noted that bilateral heel pain can be a presenting symptom of sarcoidosis and can accompany or precede sarcoid arthritis.

Hoffman and Thul [167] reported two cases of fracture of the os calcis following surgical procedures for excision of the calcaneal exostosis and plantar fasciotomy. Lester and Buchanan [91] reported on 10 patients who underwent stripping of the plantar fascia and superficial plantar muscles from the calcaneus. These patients had been treated for an average of 12.4 months before surgery. They were followed for 24 months after surgery. Complete symptomatic relief was obtained in all patients, although hypoesthesia of the heel was present in five feet after the operation. Three patients were receiving workmen's compensation and returned to work within 16 weeks of surgery.

Endoscopic plantar fascia release has received significant attention in the past several years. Barrett and Day [78] described this technique as having the advantage of less tissue damage than open treatment. In a follow-up multicenter study of 652 procedures, they reported 62 complications in 53 patients but thought that it afforded satisfactory results. O'Malley and colleagues [168] reviewed the surgical results after endoscopic plantar fasciotomy in 16 patients (20 feet) with an average preoperative duration of symptoms of 4 years. Of the 20 feet, 9 had complete relief of pain, whereas symptoms were improved in 9 feet. One patient with bilateral symptoms had no relief in either foot. The average AOFAS hindfoot score improved from 62 to 80, a statistically significant difference. Unilateral patients did better than bilateral, with no bilateral patients reporting complete resolution of symptoms. There were no iatrogenic nerve injuries. On the basis of their review, they recommend endoscopic plantar fasciotomy as an alternative to open plantar fascial release for those patients with recalcitrant heel pain who fail conservative treatment.

Other authors have reported various complications from endoscopic plantar fascial release, including stress fractures, [169] pseudoanuerysm formation, [170] and recurrence of pain. [171] Because of the high incidence of lateral foot pain, it is recommended that only the medial two thirds of the plantar fascia be released.

Kitaoka and colleagues [1272] [1273] [1274] [1275] in a series of articles has shed light on the biomechanical risk to the foot from release of the plantar fascia. They have demonstrated that changes in displacement were more pronounced in unstable or destabilized feet. Their data suggest that operations involving fasciotomy affect arch stability and should not be performed in patients with evidence of concomitant pes planus deformity, because of the likelihood of further deformation. Flattening of the longitudinal arch occurred in their clinical series. Dynamic force-plate studies showed differences in peak vertical, fore-aft, and lateral-medial forces between patients and matched controls. More rapid progression of weight-bearing along the longitudinal axis of the foot during stance phase in patients indicated avoidance of heel loading.

Yu and colleagues [176] studied 17 patients (15 women, 2 men; age range 22 to 59 years; mean age, 40 years) with foot pain with a mean duration of 22 months after undergoing a fasciotomy. Each patient was instructed to localize the pain to a region of the foot; classify the pain as new onset, persistent, or recurrent; and characterize it as to the action that produced the greatest pain. MRIs were evaluated for abnormalities of the plantar fascia, perifascial soft tissues, tendons, and osseous structures. The plantar fascia appeared thick in all ankles (mean, 8.0 mm; range, 6 to 12 mm). A total of 25 symptomatic sites were assessed. An acute plantar fascia rupture explained plantar symptoms in 2 feet. In another 16 feet (12 with plantar heel pain and 4 with nonspecific heel pain), 6 had documentation of acute plantar fasciitis, and 9 demonstrated perifascial edema. Of the latter 9 feet, 5 demonstrated abnormalities of the posterior tibialis, peroneus longus, and peroneus brevis tendons. The pain localized to the medial arch in 6 feet; 5 feet had abnormalities of the posterior tibialis tendon; and 1 foot demonstrated edema in the flexor digitorum brevis muscle. The pain localized to the lateral midfoot in 1 foot, which had a cuboid stress fracture. The cause of foot pain in patients who had a plantar fasciotomy appeared to be multifactorial. Three likely causes of pain were identified: persistent or recurrent acute plantar fasciitis, pathology related to arch instability, and structural failure from overload.

Treatment of Athletes with Plantar Fasciitis

In 1986, Lutter [92] outlined the decision-making process in athletes with subcalcaneal pain. He described 182 patients with heel complaints related to sports injuries; most of the patients were runners (76%). About 20% of these patients required 3 to 4 months of conservative treatment before returning to sports activity. Five percent had chronic heel pain and did not recover within 9 to 12 months. For these, a surgical approach was considered. Lutter stated that the decision to operate on an athlete should be based on six specific tenets: (1) a correct diagnosis has been made, (2) about 12 months of conservative treatment have been tried, (3) electromyography and appropriate nerve blocks have been performed for diagnosis, (4) the surgeon has a thorough knowledge of the anatomy or performs a complete review, (5) the patient understands that even successful surgery may not allow him or her to return to high-performance athletics, and (6) correct and appropriately directed surgery is chosen. The procedure used in these patients depended on the preoperative diagnosis and varied from release of the nerves to release of the fascia to complete exploration of the posterior tibial nerve and its branches and release of the plantar fascia. Cycling or swimming was begun 2 weeks postoperatively. Gentle walk-dash-run training and a gradual escalation up to running was allowed about 6 weeks after surgery. Patients were asked to refrain from walking until they were pain free and had no tenderness. If pain occurred with increasing activity, the work-up was cut by 50% until the patient could tolerate the work-up without pain.

Shock-wave therapy has been examined in the treatment of chronic plantar fasciitis in the running athlete. Rompe and colleagues demonstrated that after 6 months of three weekly treatments with shock-wave therapy, the treatment group experienced greater relief than did the sham treatment group. [177]

Baxter and Thigpen [64] performed 34 operative procedures in 26 patients with recalcitrant heel pain. The procedure consisted of isolated neurolysis of the nerves supplying the abductor digiti quinti muscle as it passed beneath the abductor with release of the deep fascia of the abductor hallucis longus and removal of the heel spur if it impinged on or produced entrapment of the nerve. Among the 34 heels operated on, there were 32 good results and 2 poor results.

Clancy [2] treated patients with a medial heel wedge and flexible leather support, heel cord stretching, and rest for 6 to 12 weeks with a gradual return to running wearing the orthotic and the medial heel wedge for 10 weeks. In patients who failed to respond, surgery consisting of release of the plantar fascia and the fascia over the abductor hallucis longus was recommended. The 15 patients in whom surgery was performed returned to running within 8 to 10 weeks. D'Ambrosia and colleagues [1182] [1183] had success using anti-inflammatory medication, physical therapy, orthotic devices, and shoe modifications. Orthotic devices seemed to be the most useful part of the treatment. These orthotic devices were made of Vitrathene, Plastazote II, or Plastazote III.

Henricson and Westlin [88] described 11 heels in 10 athletes with chronic heel pain that was unrelieved by conservative therapy. The pain was due to compression of the calcaneal branches of the tibial nerve. There was entrapment of the anterior calcaneal branch where the nerve passed between the tight and rigid edges of the deep fascia of the abductor hallucis and the medial edge of the os calcis. Surgery consisted of identifying and releasing the tibial nerve and both calcaneal branches and releasing the deep fascia of the abductor hallucis. Follow-up for 58 months after surgery revealed that 10 of the 11 heels were asymptomatic. The patients had resumed athletic participation after an average of 5 weeks. It seems to us that both the nerve to the abductor digiti quinti and the medial calcaneal nerve were released.

Jørgensen [178] described three athletes with unusually soft and fat heel pads who were heel strikers. These athletes were treated successfully with external heel shock absorption pads. Jørgensen reported a diagnostic method for evaluating the shock-absorbing ability of the heel pad by using a visual compressible index calculated on the basis of radiographic films of the heel, loaded and unloaded by body weight.

According to Kwong and colleagues, [90] fasciitis is produced by an excessive amount or a prolonged duration of pronation. Temporary relief was obtained in his patients by the use of anti-inflammatory drugs and therapy. Long-term relief was obtained by achieving adequate control of pronation through the use of semirigid custom-molded orthotics that reduced plantar fascial strain by supporting the first metatarsal bone and controlling calcaneal position. These devices were used in conjunction with a firm posterior counter shoe.

Leach and coworkers [137] stated that most patients respond well to conservative therapy consisting of decreased activity, stretching, heel cups, and occasional local steroids. They described 15 competitive athletes in whom 16 operations were performed. Surgery consisted of release of the plantar fascia at the insertion of the os calcis, making the incision along the medial aspect of the heel. In one instance, the medial calcaneal nerve was involved in the inflammatory process. One patient returned to running at 6 weeks; most returned to running 9 weeks after surgery. Most patients continued to improve up to 6 months after the surgical procedure. Of the 15 operations, 14 were entirely successful in that the athletes returned to their previous level of activity. One failure occurred in a marathon runner who improved but was unable to train at the level he desired. There were no complications.

In 1984, McBryde [93] reported that in his running clinic, plantar fasciitis composed 9% of the total running disorders seen. The conservative (nonoperative) approach of McBryde and his group consisted of (1) ice massage for 2 minutes 4 to 6 times daily, including before and after runs; (2) heel cord stretching for 3 to 5 minutes 3 to 4 times daily; (3) posterior tibial and peroneal strengthening; (4) heel cushioning and control; and (5) anti-inflammatory medication. This regimen was usually successful in treating runners with plantar fasciitis who were seen within the first 8 weeks. In runners with symptoms lasting longer than 6 weeks, a period of absolute rest with casting was usually required. Orthoses were used. Five percent of the patients in the series underwent surgery, consisting of plantar fascial release through a short 1-inch longitudinal incision in the medial arch. All re-embarked on a successful running program 6 to 12 weeks after surgery. Overall, among the 100 patients with plantar fasciitis, 82 recovered with a conservative approach, 11 stopped running, 5 underwent surgery (all of whom returned to running), and 2 refused surgery and continued to be symptomatic.

Rask [95] reported a medial plantar neurapraxia that he termed *jogger's foot*. Three cases were reported in which there was probable entrapment of the medial plantar nerve behind the navicular tuberosity in the fibromuscular tunnel formed by the abductor hallucis; the inciting factor was eversion of the foot. All three patients were treated successfully with conservative measures, including change in running posture of the foot, anti-inflammatory medication, and proper footwear.

Sammarco [97] divided heel pain into three clinical classifications and provided a treatment algorithm for these conditions. First, calcaneodynia, produced by a stress fracture, was treated with a foot orthosis and decrease in running. Second, plantar fasciitis was treated with a foot orthosis, anti-inflammatory medication, and a flexibility program, plus occasional injections of corticosteroids. Recalcitrant plantar fasciitis (defined as symptoms lasting for more than 1 year) was considered for release of the plantar fascia and bone spur if one was present. Finally, calcaneodynia involving entrapment of the medial calcaneal nerve or the nerve to the abductor digiti quinti was diagnosed by Tinel's sign and treated with an orthosis or release of the nerve.

Snider and colleagues [99] reported 11 operations for plantar fascial release for chronic fasciitis in nine distance runners who had had symptoms for an average of 20 months and had not responded to nonsurgical treatment. The results of the operations were excellent in 10 feet and good in one foot, with an average follow-up time of 25 months. Eight of nine patients returned to their desired level of full training at an average time of 4.5 months.

In 2004, Saxena [179] reported on 16 athletes with intractable heel pain who had failed conservative care. Most of these athletes were runners. These patients were treated surgically with a uniportal endoscopic plantar fasciotomy. Saxena found that runners were able to return to athletic activity on average 2.6 months after surgery. Five poor results were found in patients with a body mass index of greater than 27.

Treatment of Children

Calcaneal apophysitis (Sever's disease) is a common cause of heel pain in the young child. Micheli and Ireland [180] reported calcaneal apophysitis in 137 heels in 85 children, both heels being affected in 61% of the patients. Soft Plastazote orthotics or heel cups were used in 98% of the patients in combination with proper athletic footwear. Physical therapy consisted of lower extremity stretching and ankle dorsiflexion strengthening. All patients improved and were able to return to the sport of their choice 2 months after the diagnosis. Two patients had recurrent difficulty.

Sullivan [181] reported that radiographic irregularity of the calcaneal apophysis is the rule rather than the exception. He found no evidence that treatment altered the radiographic picture. Because there are a variety of conditions that cause pain in the heel, he believes that Sever's disease might not even be a true entity. This author notes that differentiation can be made on the basis of location of the pain, the pain of Achilles tendinitis being proximal to the medial bursa and the pain of calcaneal apophysitis being along the distal portion of the posterior part of the os calcis.

In summary, conservative treatment consisting of anti-inflammatory medications, orthoses, heel cups (Fig. 25F-24), injections, physical therapy, and decreased activity level is effective in 95% of cases of subcalcaneal pain syndrome. In patients who do not respond to an adequate trial of these conservative measures, surgery consisting of release of the plantar fascia or release of the nerve to the abductor digiti quinti or the sensory medial calcaneal nerve can be performed with an expectation of good results. Heel spur, if present, is removed. However, problems with decreased sensation of the heel and persistent pain have been reported.

AUTHORS' PREFERRED METHOD OF TREATMENT

Plantar fasciitis arising at the medial tuberosity probably represents a traction periostitis with degeneration and tears of the plantar fascia and subsequent secondary involvement of the adjacent structures such as the medial calcaneal nerve and the nerve to the abductor digiti quinti. Occasionally, primary entrapment of the nerve of the abductor digiti quinti and the sensory branch of the medial calcaneal nerve may occur. A calcaneal spur is present in 50% of the cases and may be part of the inflammatory process. In longstanding cases, the chronic inflammation and traction on the insertion of the plantar fascia may result in a stress fracture of the os calcis, although this is more common in the older patient. An accurate diagnosis of the etiology of the pain and any concomitant conditions such as chronic heel fat pad atrophy, cavus foot deformity, contraction of the Achilles tendon, or nerve entrapment must be determined because they will affect both the prognosis and treatment modalities employed.

At the first visit, we recommend a discussion with the patient to explain the nature of this condition. We tell them the good news is that it almost always gets better without the need for surgery. The bad news is that it usually does not get better quickly. In our initial consultation, we explain that we expect it to take at least 6 to 9 months to resolve completely and sometimes up to year. It is important for both the physician and the patient to set this time frame early on to decrease the frustration often associated with this diagnosis.

The patient with subcalcaneal pain syndrome and associated proximal plantar fasciitis is evaluated to determine which foot type is present and whether there is any associated abnormality of the lower extremity or body. If there is none, I begin treatment consisting of anti-inflammatory medication and an orthotic device designed to cushion the heel and relieve the pressure on the tender area of the heel. There are many types of over-the-counter heel cups and cushioning devices that may be used, but we generally prescribe viscoelastic silicone heel pads and ask the patient to use well-cushioned shoewear such as a running shoe. Activities are restricted according to the patient's symptomatic tolerance.

The mainstay of treatment is to stretch both the Achilles and plantar fascia. Physical therapy consisting of stretching, strengthening, ultrasound, and at times, iontophoresis and phonophoresis may be used. We stress to the patient that following up with a daily home program of stretching is essential. If the condition is not responsive to this regimen within the first 6 weeks or has been present for more than 3 months, a night splint with the foot maintained in dorsiflexion is prescribed. We explain to the patient that we are treating pain on the bottom of their foot while we are attempting to allow them to keep ambulating on the foot. This is similar to trying to fill up a bath tub with the plug out of the drain. The only way it works is if more water is going in than is coming out. Therefore we use multiple modalities at once to try to diminish the inflammation more rapidly than we are creating it.

Cross-training activity is important. The patient should avoid repetitive impact activities such as running or treadmill and cross-train with a bike or elliptical trainer. These assist in maintaining conditioning and increasing flexibility while avoiding cyclic loading.

If this does not suffice after a period of 3 months and the patient's symptoms are severe enough, complete rest in a short leg weight-bearing cast for 4 weeks is considered. We explain this is like plugging the drain in the tub analogy. Once the cast is removed, the protocol is restarted.

The exception to this is the patient with severe pain to medial-lateral compression of the calcaneus at the level of the insertion of the plantar fascia that is greater than pain to palpation at the insertion on the calcaneus. This is more common in the elderly patient and often indicates a stress fracture. In these patients, a bone scan is ordered before starting therapy, and if it is positive for stress fracture, casting will be started.

Once the patient has become asymptomatic without tenderness and has maintained this status for 4 to 6 weeks, a gradual increase in activity may be allowed. The orthotic is continued for several months. After several months, the orthotic is discontinued unless the patient has a biomechanical abnormality of the foot such as a flatfoot or cavus deformity. If the patient has a flatfoot deformity, a device designed to correct the biomechanical abnormality, support the foot, and prevent the abnormal biomechanical stresses along the plantar fascia and the medial side of the heel is used. If the patient has a cavus deformity, a soft orthotic designed to decrease the shock and increase the weight-bearing area may be used indefinitely depending on the patient's symptoms. When a patient does not have a normal foot and has had a significant episode of subcalcaneal pain requiring treatment, I suggest that the orthotic be continued permanently. Although the over-the-counter type of heel cup can be used initially to try to provide some symptomatic relief, in a patient with a true biomechanical abnormality, a specific orthosis should be used.

In our practice, we generally avoid steroid injection to prevent any subsequent loss of fat pad. Although reasonable results have been obtained with injection, we believe that fat pad atrophy is an irreversible complication with long-term negative consequences. We use cast immobilization and NSAIDs instead.

If the patient does not respond to this conservative regimen, has an injury or impairment sufficient to prevent the performance of the desired activity, and demonstrates no other abnormality or systemic cause of the pain, I consider surgical intervention after 9 to 12 months of therapy. It has been our experience that surgery is rarely required unless there is an associated nerve lesion. In the athlete, we attempt to perform the least amount of surgery commensurate with a probably good result. In the athlete with a recalcitrant subcalcaneal pain syndrome who desires to continue athletic activity, the exact site of the abnormality is evaluated carefully by means of differential blocks using a long-acting anesthetic such as bupivacaine hydrochloride. This allows more precise localization of the exact area of pathology. We attempt to rule out tarsal tunnel syndrome and to define whether the abnormality lies along the medial tuberosity or is produced by the sensory medial calcaneal nerve or the nerve to the abductor digiti quinti.

Before surgery, nerve conduction and electromyographic studies are considered in patients in whom a tarsal tunnel syndrome is possible. Laboratory studies are done to exclude systemic arthritis or spondyloarthropathies. Bone scans with technetium-99m may be considered if one suspects a fatigue fracture or if the exact location of the pain is not clear.

We approach the area through a medial oblique incision along the heel as described by Schon (Fig. 25F-25). [182] Loupe magnification may be used. The sensory branch of the medial calcaneal nerve is located, inspected, and preserved. If there is entrapment of the medial calcaneal nerve as it comes through the fascia, this is explored and released. The central slip of the plantar fascia is released at its origin. The spur is removed using a small rongeur after stripping the muscles. The deep fascia of the abductor hallucis and the fascia of the quadratus plantae are divided to release the nerve to the abductor digiti quinti as it passes laterally. The wound is irrigated and closed with several plain subcutaneous and interrupted mattress sutures. The patient is placed in a short leg cast and kept non–weight-bearing for 4 to 7 days. Weight-bearing in a cast or an over-the-counter brace is maintained for 14 more days. At 3 weeks, weight-bearing with a shoe is permitted. Running is started at 6 to 12 weeks, and activity following this is allowed as tolerated. If the patient has a biomechanical foot abnormality, an orthotic device is used postoperatively.

In the patient who has symptoms that may involve the posterior tibial nerve as well as pathology of the medial tuberosity, or had undergone recurrent or failed surgery, a more extensive operation is considered on the understanding that whereas it may relieve the pain, the operation may produce more morbidity with a decreased chance of recovery. The operation for complex, resistant, or recurrent pain along the medial side of the heel consists of exploration of the posterior tibial nerve from the medial side of the ankle to the point at which it exits through the foramina of the abductor muscles and exploration and release of the medial calcaneal nerve and the nerve to the abductor digiti quinti along with release of the central portion of the plantar fascia and excision of the heel spur, if present (see Fig. 25F-25). The patient is kept non–weight-bearing for 2 weeks and can then bear weight in a short leg cast for 2 more weeks; increased activity is started at 12 weeks. This operation is used only for patients with recalcitrant conditions. It carries with it the expectation that the patient will probably, but not certainly, be able to return to his or her preinjury status.

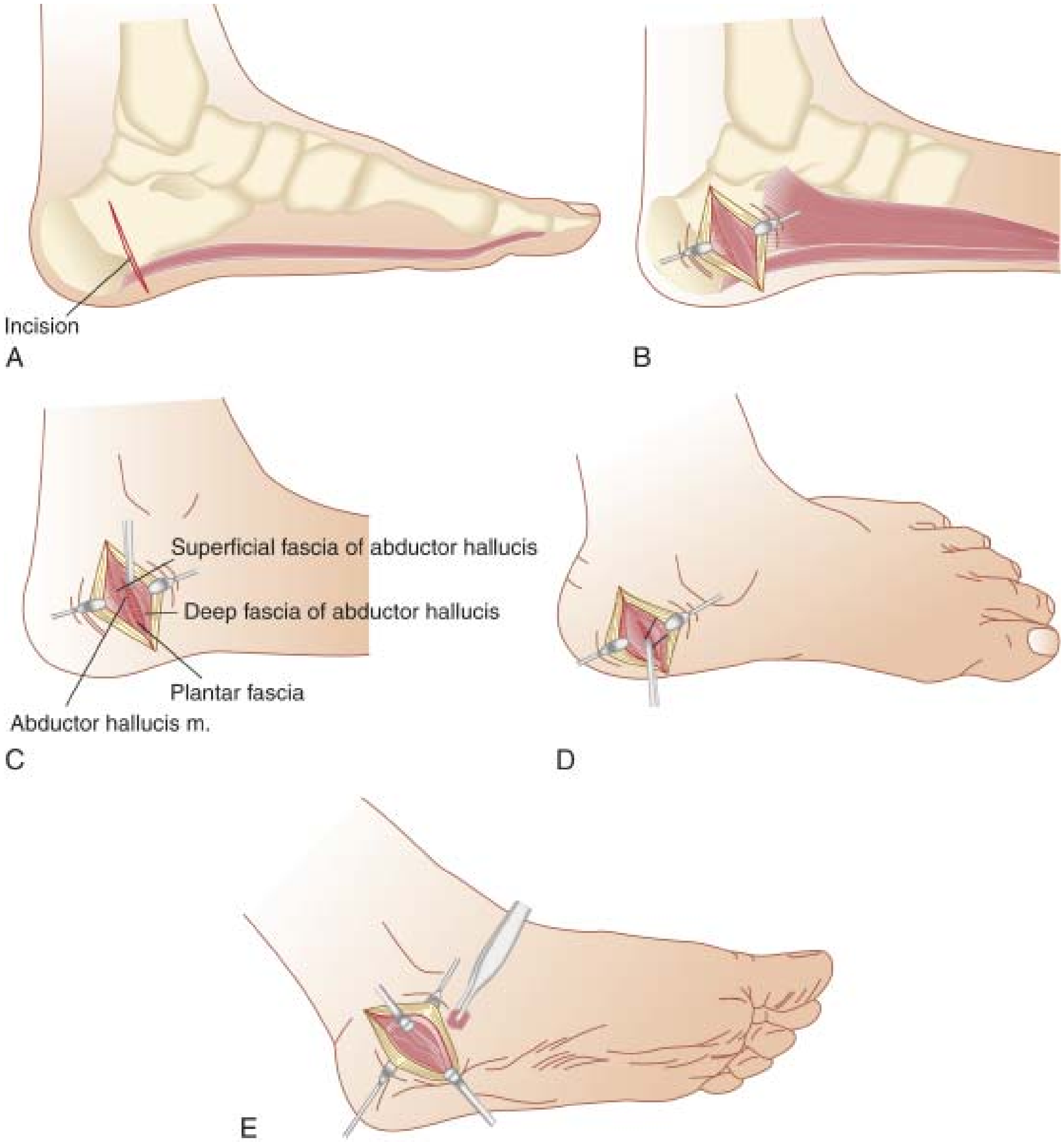

Figure 25F-25 Plantar fascia and nerve release. **A,** Incision. **B,** Release of the abductor hallucis muscle. **C,** Abductor hallucis muscle is reflected proximally. **D,** Abductor hallucis is retracted distally. **E,** Resection of small medial portion of the plantar fascia.

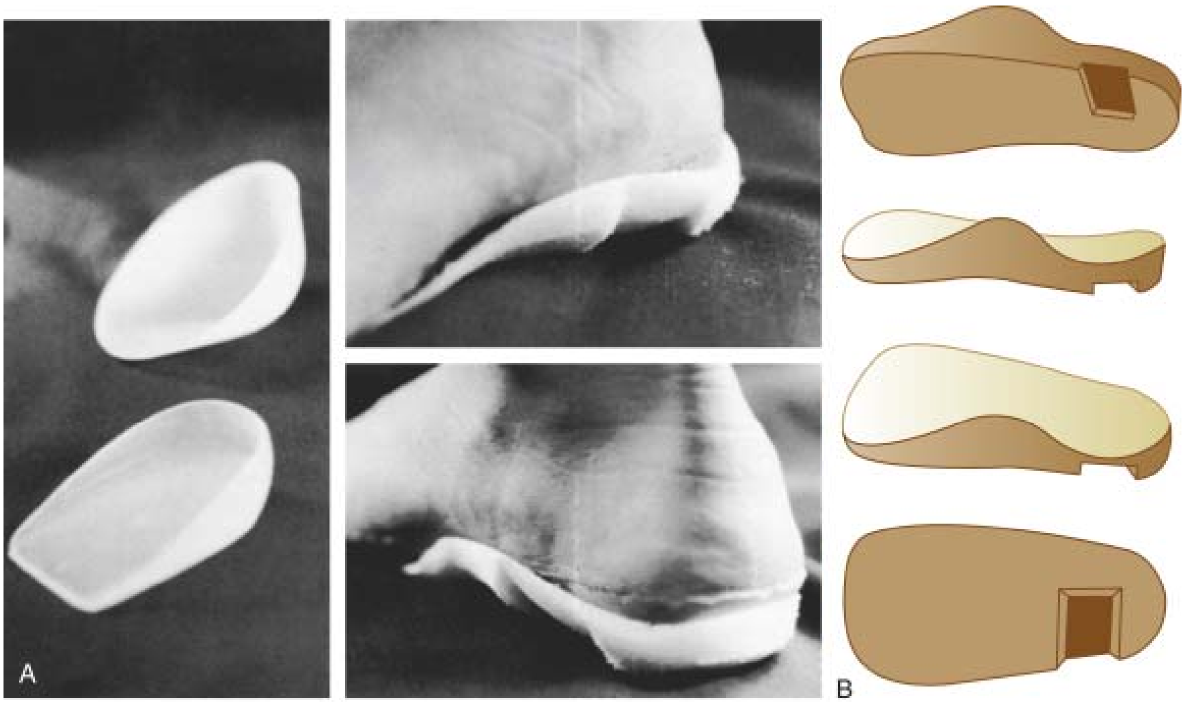

Figure 25F-24 **A,** Over-the-counter heel cups designed to compress the fat pad and cushion the heel. **B,** Custom orthoses used for treatment of subcalcaneal pain syndrome. These may be made of many types of material. Consistent features are support of the arch, presence of cushioning material, recess for area of pain beneath the heel, and slight medial elevation.

Criteria for Return to Sports Participation

Because plantar fasciitis associated with pain in the medial tuberosity in the athlete may be associated with a variety of presentations and degrees of severity and does not produce any significant structural effect other than alteration in form, activity may be allowed within the patient's symptomatic tolerance, although this varies greatly according to the pain component and the athlete's restriction. The exception to this is if the patient is developing a stress fracture. Once the pain has subsided with conservative treatment and no tenderness is present, gradually increasing activity is allowed using an orthotic within the shoe. If symptoms do not recur with the specific activity desired, full activity is allowed.

After surgical intervention, the patient is allowed increased activity when the symptoms of pain with activity and acute tenderness have resolved. This usually occurs between 6 and 12 weeks after surgery.

CRITICAL POINTS

RETROCALCANEAL BURSITIS

- Retrocalcaneal pain syndrome is commonly associated with the high-arched cavus foot and the varus heel.
- It is a condition characterized by inflammation of the retrocalcaneal bursa, the Achilles tendon just above its insertion, and at times the tissue between the Achilles tendon and the skin.
- Steroid injections should be made into the retrocalcaneal area, not into the tendon.
- It is generally managed by conservative measures consisting of anti-inflammatory medication, decreased activity, padding to prevent pressure on the affected area, orthoses or heel lifts, and strengthening and stretching exercises.
- Surgery generally consists of excision of the exostosis and the retrocalcaneal bursa and at times the adventitious bursa, if it is present, and correction of the Achilles tendon pathology with tendon transfer if necessary.

PLANTAR FASCIITIS

- Conservative treatment consisting of anti-inflammatory medications, orthoses, heel cups, injections, physical therapy, and decreased activity level is effective in 95% of cases of subcalcaneal pain syndrome.
- Surgery consists of release of the plantar fascia or release of the nerve to the abductor digiti quinti or the sensory medial calcaneal nerve.

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SECTION G Entrapment Neuropathies of the Foot

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An entrapment neuropathy results from localized damage or inflammation of a peripheral nerve, which may be due to an injury such as direct trauma or a mechanical irritation such as impingement against an anatomic structure. This entrapment may result in direct axonal compression or vascular changes. Generally, the common factor in an entrapment neuropathy is a nerve passing through a rigid compartment, through a fibrous or fibro-osseous canal in which the nerve changes direction, or over a bony prominence.

The main clinical complaint of patients with entrapment neuropathy is pain or dysesthesia. Depending on the nerve involved, the pain may be worse at night or at rest, or at other times pain may be activity related. Retrograde pain, known as the *Valleix phenomenon,* may be a prominent feature of the patient's complaint. When a purely sensory nerve is involved, a rather clear distribution of pain can be elicited from the patient, whereas compression of a motor nerve gives rise to a more diffuse type of pain. Occasionally, atrophy of the innervated muscles may occur. In all cases of entrapment neuropathies, it is imperative to elicit a careful history from the patient, carry out a physical examination, and obtain appropriate electrodiagnostic studies.

TARSAL TUNNEL SYNDROME

The tarsal tunnel syndrome in its basic form is an entrapment of the posterior tibial nerve within the tarsal canal. It was first described by Keck and Lam in 1962. Interestingly, some contemporaries of Lam believed tarsal tunnel syndrome to occur exclusively in jockeys. Currently there is no associated occupational risk attributed to the development of tarsal tunnel syndrome. [1287] [1288] It may involve only one of the terminal branches distal to the tarsal canal. The tarsal canal is located behind the medial malleolus and becomes the tarsal tunnel as the flexor retinaculum passes over the structures, creating a closed compartment (Fig. 25G-1). Distally, at about the level of the medial malleolus, the posterior tibial nerve divides into its terminal branches, giving rise to the medial plantar nerve, the lateral plantar nerve, and the medial calcaneal branches.

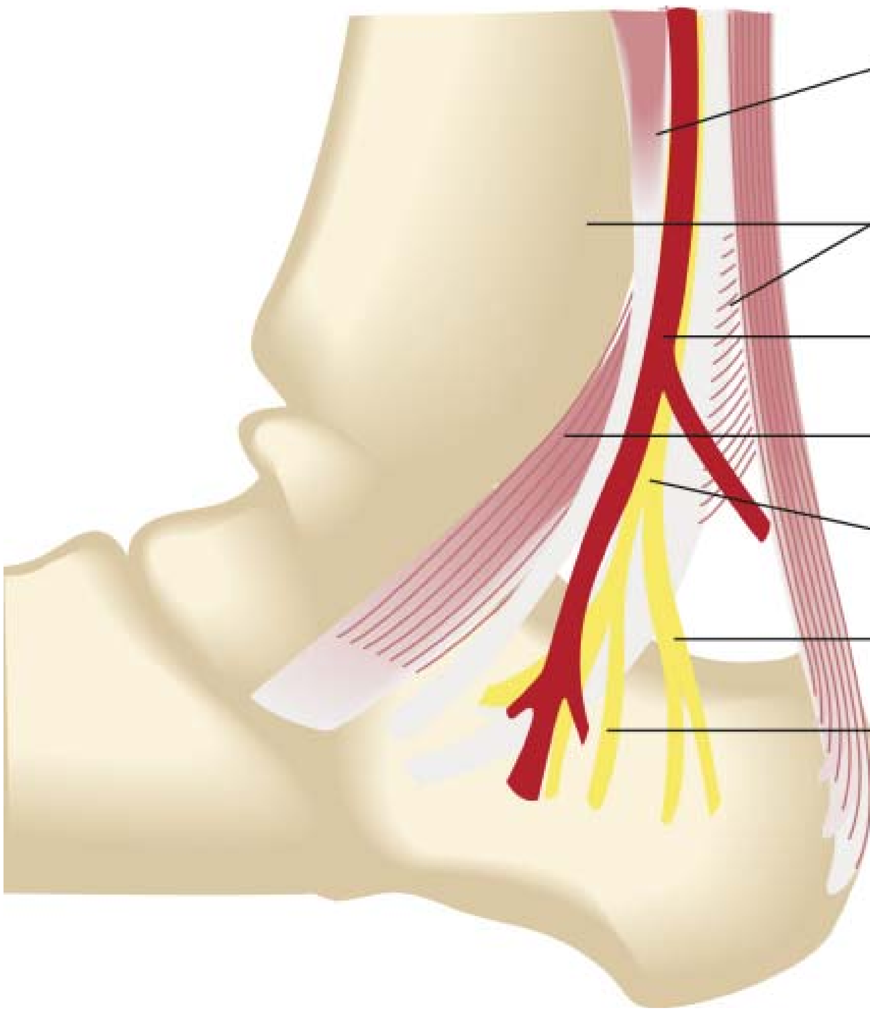

Figure 25G-1 Tarsal tunnel.

Diagnosis of the tarsal tunnel syndrome is a specific one that is made by correlating the patient's history, the physical findings, and electrodiagnostic studies.[3] If the findings on all three of these studies are not positive, the possibility of an entity other than the tarsal tunnel syndrome should be considered carefully.

Clinical Symptoms

Most patients with tarsal tunnel syndrome complain of a poorly defined burning, tingling, or numb feeling on the plantar aspect of the foot (Fig. 25G-2). At times, this pattern of pain may be localized to one of the three terminal branches of the posterior tibial nerve rather than the entire nerve. Generally, the pain is aggravated by activity and relieved by rest, but sometimes patients note that the symptoms are most bothersome in bed at night; this type of pain can be relieved by getting up, moving around, and massaging the foot. In our experience, younger patients who present with this problem complain of activity-related symptoms.

Figure 25G-2 Topographic areas of pain based on diagnosis.

Etiology

The cause of tarsal tunnel syndrome is idiopathic in about 50% of cases, and in the other 50%, a specific cause can be identified. The most commonly identified cause is a space-occupying lesion such as a synovial cyst, [4] ganglion protruding from a tendon sheath, [1291] [1292] lipoma, [3] neurilemoma, [7] venous varicosities, [1289] [1294] tenosynovitis, [9] severe pronation or valgus hindfoot deformity, [1291] [1296] trauma resulting in a fracture of the distal tibia or calcaneus, [3] or occasionally a severe ankle sprain. [3]

Diagnosis

Physical Examination

Examination of the patient with a suspected tarsal tunnel syndrome is important. Initially, the patient is examined in the standing position to evaluate the overall posture of the foot and the presence of edema, venous varicosities, or thickening around the medial aspect of the ankle or heel. The range of motion of the ankle and of the subtalar and transverse tarsal joints is evaluated. The physician then gently taps along the course of the posterior tibial nerve starting above the malleolus and passing distally below the malleolus along each terminal branch of the nerve. As this area is percussed, an attempt is made to elicit tingling along the course of the posterior tibial nerve or its terminal branches to identify the site of possible pathology. Careful palpation along the tendon sheaths is important because at times a cyst or ganglion may arise, placing pressure on the posterior tibial nerve or one of its terminal branches. A sensory examination on the plantar aspect of the foot is made, and the motor function to the toes is evaluated.

Electrodiagnostic Studies

In the patient in whom tarsal tunnel syndrome is suspected, electrodiagnostic studies should be carried out. [1289] [1297] The studies should include conduction velocities of the peroneal nerve to rule out the possibility of a peripheral neuropathy. The terminal latencies of the medial plantar nerve to the abductor hallucis and the lateral plantar nerve to the abductor digiti quinti are determined. As a general rule, the terminal latency of the medial plantar nerve to the abductor hallucis should be less than 6.2 msec, and that of the lateral plantar nerve to the abductor digiti quinti should be less than 7 msec. The presence of fibrillation in the abductor hallucis and the abductor digiti quinti is a late sign, and its absence does not necessarily rule out tarsal tunnel syndrome. Variation tends to occur between electromyographers, and the terminal latencies should be compared with the standard of the electromyographer carrying out the studies. [1298] [1299] Evaluation of the sensory components of the posterior tibial nerve likewise may be of benefit. [1300] [1301] There are conflicting reports among electromyographers about precisely which is the most important examination in determining the presence of an abnormality.

Differential Diagnosis

Box 25G-1 lists remote, intraneural, and extraneural causes.

BOX 25G-1

DIFFERENTIAL DIAGNOSIS OF TARSAL TUNNEL SYNDROME

Remote Causes

- Interdigital neuroma
- Intervertebral disc degeneration
- Plantar fasciitis

Intraneural Causes

- Peripheral neuritis
- Peripheral vascular disease
- Diabetic neuropathy
- Leprosy
- Neurilemoma

Extraneural Causes

- Ganglion
- Nerve tethering
- Blunt trauma
- Valgus of the hindfoot
- Rheumatoid arthritis with tenosynovitis
- Venous varicosities
- Hypertrophy of the abductor hallucis muscle origin
- Lipoma
- Bony impingement secondary to a previous fracture

Treatment

Conservative Management

Conservative management may be of benefit, particularly if the cause is thought to be postural, in which case an orthotic device may be used. If there is significant swelling of the extremity, this should be brought under control. The use of nonsteroidal anti-inflammatory medication or, occasionally, an injection of a steroid preparation into the area of the tarsal tunnel may be beneficial. At times, immobilization in a short leg cast and the use of a polypropylene ankle-foot orthosis may bring relief. If the problem is thought to be due to mechanical pressure on the nerve, usually conservative measures are not beneficial.

Surgical Management

Decompression of the posterior tibial nerve and its terminal branches should be undertaken through a longitudinal incision, which starts about 20 cm proximal to the medial malleolus and 3 to 4 cm behind the posterior border of the tibia and extends distally behind the medial malleolus and along the medial aspect of the foot. The direction of the incision below the malleolus depends on the area that needs to be explored. If most of the symptom complex involves the medial plantar nerve, the incision is brought along the dorsal margin of the abductor hallucis muscle, or if the symptoms appear to involve mainly the lateral plantar nerve, the incision is carried slightly more plantarward along the origin of the abductor hallucis muscle. The incision is developed down to the deep fascia of the abductor. Care is taken to identify and cauterize all bleeders.

The investing retinaculum is opened behind the posterior tibial tendon sheath, and usually the posterior tibial nerve lies just posterior to the flexor digitorum longus tendon. Occasionally, the flexor digitorum longus tendon is in its own tendon sheath; then one must open up the next tendon sheath posteriorly to identify the posterior tibial nerve. The nerve always is identified proximally, which makes the dissection easier as one proceeds distally toward the medial malleolus. As one reaches the area of the medial malleolus, the vascular leash, which accompanies the posterior tibial nerve, crosses over the nerve, making the dissection more difficult. At times, it is almost easier to identify the medial plantar nerve distal to the malleolus and work in a retrograde fashion, allowing identification of the medial plantar nerve along its entire course.

One follows the medial plantar nerve distally past the talonavicular joint, after which it passes through a fibro-osseous tunnel onto the plantar aspect of the foot. It is important that a clamp be passed through this tunnel to ensure that no entrapment is present. If entrapment does appear to be present, the tunnel should be opened.

Returning proximally, the lateral plantar nerve carefully is identified posterior to the medial plantar nerve (Fig. 25G-3).

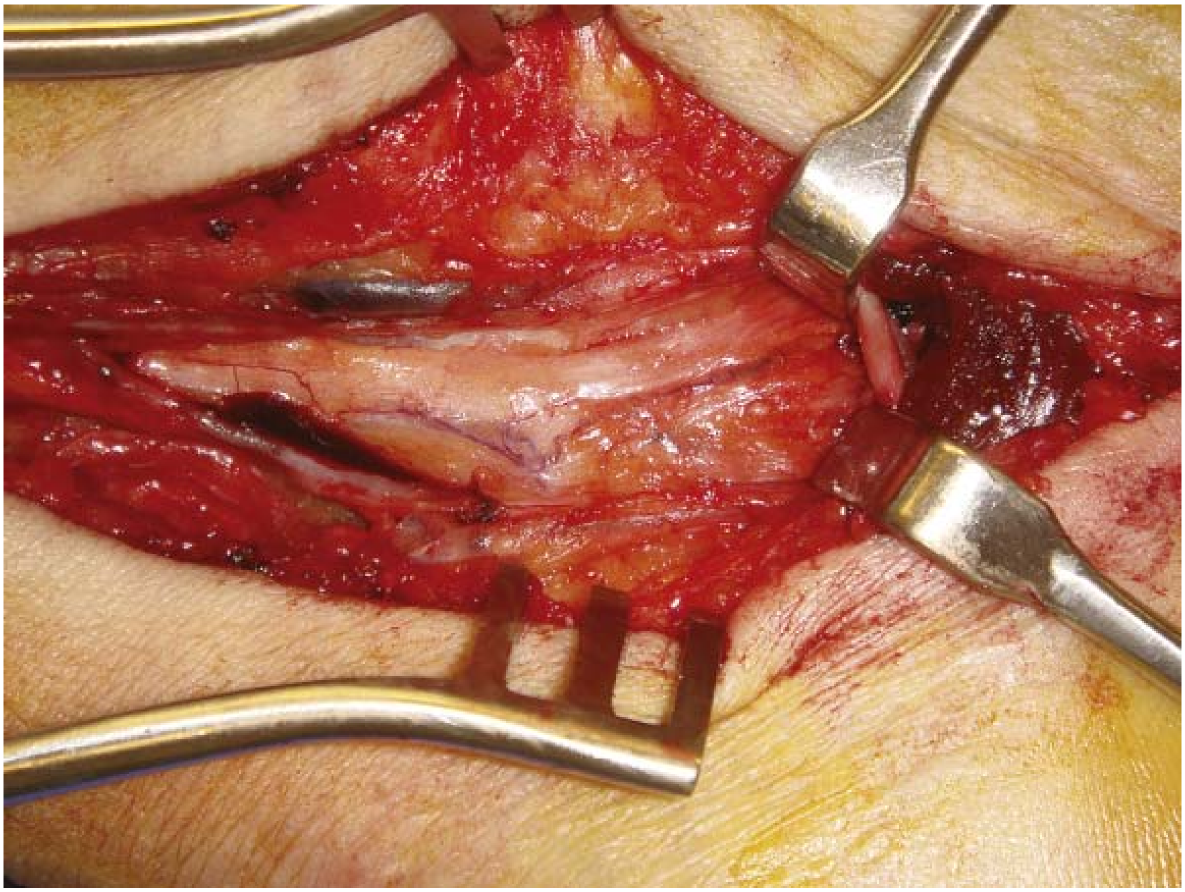

Figure 25G-3 Lateral plantar nerve.

Generally, the medial calcaneal branches come off the posterior aspect of the lateral plantar nerve at varying heights, usually starting at about the level of the medial malleolus, but at times they may branch off more proximally. Because of this, the dissection should be carried out along the anterior aspect of the lateral plantar nerve to avoid injury to the medial calcaneal branches.

The lateral plantar nerve is traced distally behind the vascular leash, after which it passes down beneath the origin of the abductor hallucis. At times, it is difficult to dissect out the lateral plantar nerve, but in patients in whom there is a positive Tinel's sign over the area of the nerve, it is imperative to take down the abductor origin sufficiently to allow the entire course of the lateral plantar nerve to be identified. Behind the abductor hallucis, the lateral plantar nerve passes through several firm fibro-osseous tunnels confluent with the deep fascia of the abductor hallucis muscle, each of which needs to be identified by blunt dissection. If there is any evidence of entrapment, the tunnel needs to be released.

The medial calcaneal branch should be identified as it passes along the posterior aspect of the lateral plantar nerve, but it does not need to be dissected out unless the symptoms point to entrapment. As this dissection is carried out, if any ganglion or cystlike structure appears to be arising from a tendon sheath, it must be identified and excised. This structure could be the main cause of the patient's symptoms and should be looked for carefully, particularly if there is a localized Tinel's sign preoperatively over one of the terminal branches below the level of the malleolus.

After the dissection has been completed, the tourniquet is released, and all bleeding is brought under control. The wound is closed in a routine manner, and a compression dressing is applied. Postoperatively, the patient is kept non–weight-bearing for 3 weeks, after which progressive weight-bearing is begun as tolerated. Swelling should be controlled with an elastic stocking. Impact-type activities are not allowed for at least 2 months and then only as tolerated.

Clinical Results

The results after release of the tarsal tunnel are variable. The best results occur in patients who have a mass-occupying lesion. In this group, a satisfaction rate of about 90% can be achieved. The remainder have about a 70% satisfaction rate. Many patients note that the pain or dysesthesias still are present but at a significantly reduced level. The success of revision tarsal tunnel surgery in the case of recurrent symptoms largely depends on the adequacy of the original release. Skalley and colleagues reviewed 12 patients (13 feet) who had failed initial tarsal tunnel release. [6] Three groups of patients emerged from their analysis. First, patients who failed the first procedure because of entrapment of the tibial nerve in scar but had an adequate release did not do well after their revision surgery. Individuals with scar entrapment and an inadequate release had mixed results. Finally, patients whose symptoms were purely due to an inadequate release did well after revision surgery. The overall picture for revision tarsal tunnel surgery is bleak, with only a small percentage of patients finding any real relief of their symptoms. [6]

ENTRAPMENT OF THE MOTOR BRANCH TO THE ABDUCTOR DIGITI QUINTI

Entrapment of the nerve to the abductor digiti quinti has been identified as a source of heel pain. [1296] [1302] [1303] [1304] [1305] Although it is not a common cause of heel pain, this nerve entrapment is something that should be sought carefully, particularly in the patient who presents with refractory pain around the heel.

Clinical Symptoms

Patients with this entrapment neuropathy complain of a vague burning pain around the heel pad area. The pain usually is poorly localized and rarely radiates out to the forepart of the foot or proximally in the heel. Generally, the symptoms are aggravated by activity and relieved by rest.

Etiology

The cause is believed to be the result of entrapment of the nerve to the abductor digiti quinti muscle (Fig. 25G-4), which usually arises as the first branch of the lateral plantar nerve. The entrapment occurs as this nerve passes beneath the plantar aspect of the deep investing fascia of the abductor hallucis muscle as it courses laterally across the foot. The nerve, after passing the abductor hallucis, passes along the calcaneus, lying between the long plantar ligament, which is dorsal to it, and the plantar fascia, which is plantar to it. It lies adjacent to the plantar tuberosity of the calcaneus. Continuing laterally, the nerve terminates in the muscle mass of the abductor digiti quinti. The entrapment of the nerve usually occurs beneath the abductor fascia or, owing to its proximity to the calcaneal spur, secondary to inflammation, trauma, or healing of a stress fracture.

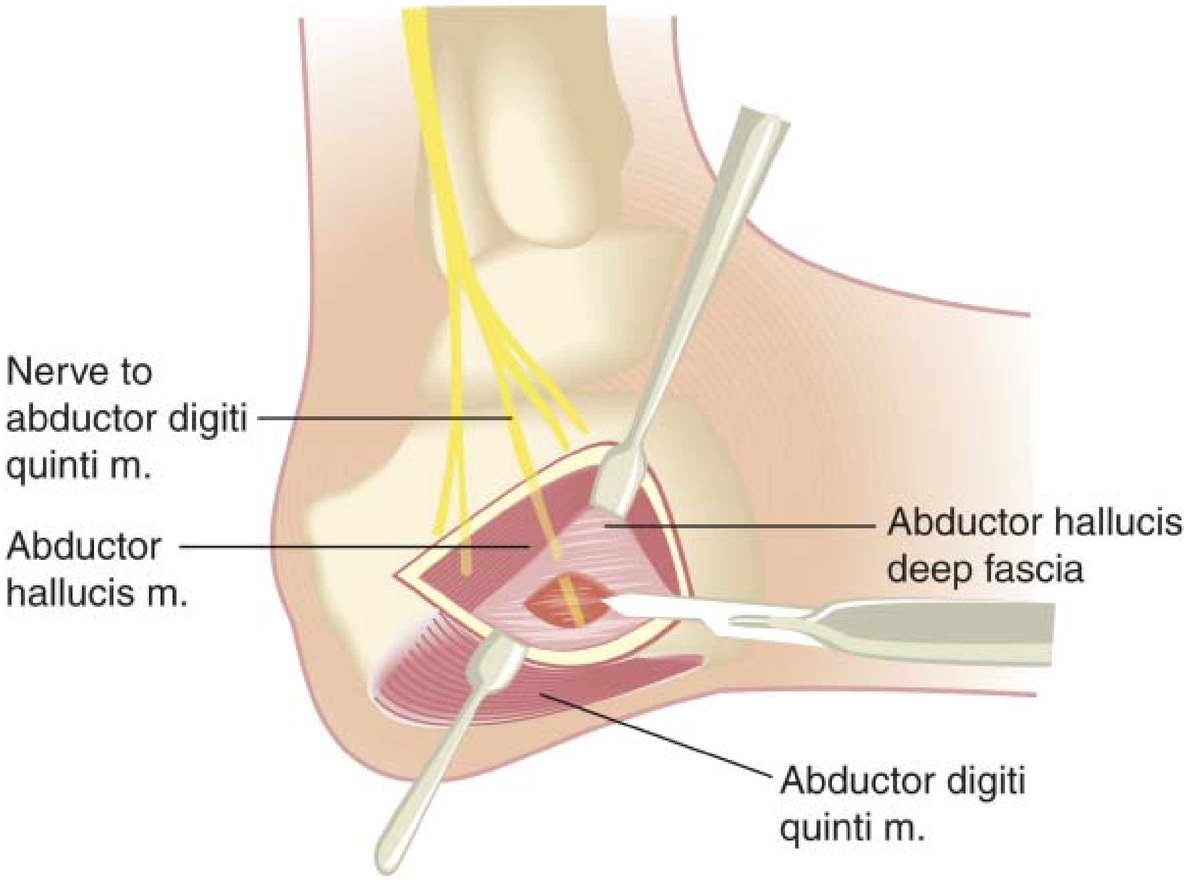

Figure 25G-4 Nerve to the abductor digiti quinti.

Diagnosis

Physical Examination

Pressure over the medial and slightly plantar aspect of the abductor hallucis muscle reproduces the patient's pain. Occasionally, a positive Tinel's sign can be elicited in the area. The patient with this condition usually needs to be evaluated on several occasions to be sure that the symptom complex is reproducible. Generally, electrodiagnostic studies are not useful in defining this problem.

Differential Diagnosis

The differential diagnosis in patients with this entrapment includes:

- Plantar fasciitis
- Fasciitis or tendinitis of the origin of the abductor hallucis muscle
- Periostitis
- Stress fracture of the calcaneus
- Tarsal tunnel syndrome
- Systemic arthritides
- Mechanical foot problem

Treatment

Conservative Management

Conservative management consists of the use of nonsteroidal anti-inflammatory medications, local steroid injection, an orthotic device to attempt to keep the stress off the involved area, and occasionally, immobilization of the foot in a short leg cast.

Surgical Treatment

In patients who fail to respond to conservative management, operative release can be considered. [16] The release is carried out through a 5-cm incision made along the upper border of the heel pad, with care taken to preserve the sensory branches to the calcaneus. The investing fascia over the abductor hallucis muscle is split, and the muscle is retracted dorsally. The deep layer of the investing fascia is released to observe the lateral plantar nerve. The branch to the abductor digiti quinti can be identified as it passes laterally. The nerve is traced laterally, and a hemostat is placed along the course of the nerve to ensure that an entrapment is not present as it passes adjacent to the calcaneal tuberosity. If there appears to be any evidence of pressure against the nerve from the medial half of the plantar fascia or the calcaneal tuberosity, the former is released, and the latter is excised. Postoperatively, a compression dressing is used, and weight-bearing is begun when it can be tolerated.

Clinical Results

Baxter and Thigpen[16] reported on 34 procedures in 26 patients, of which good results were obtained in 32 cases. Poor results were reported in 2 cases. Although satisfactory results have been obtained with this procedure, it is imperative to make a precise diagnosis; otherwise, satisfactory resolution of the problem cannot occur. There are many causes of heel pain, and entrapment of the nerve to the abductor digiti quinti is a cause of heel pain in only a few isolated instances.

SURAL NERVE INJURY

An entrapment of the sural nerve, which is a continuation of the tibial nerve formed by the communicating branch of the common peroneal nerve and the medial sural cutaneous nerve, is not common. The sural nerve is accompanied by the lesser saphenous vein behind the lateral malleolus, where the nerve usually divides into anterior and posterior branches. The nerve supplies sensation to the back of the leg, the lateral aspect of the heel, the lateral border of the foot, the fifth toe, and sometimes the lateral portion of the fourth toe (Fig. 25G-5). [20]

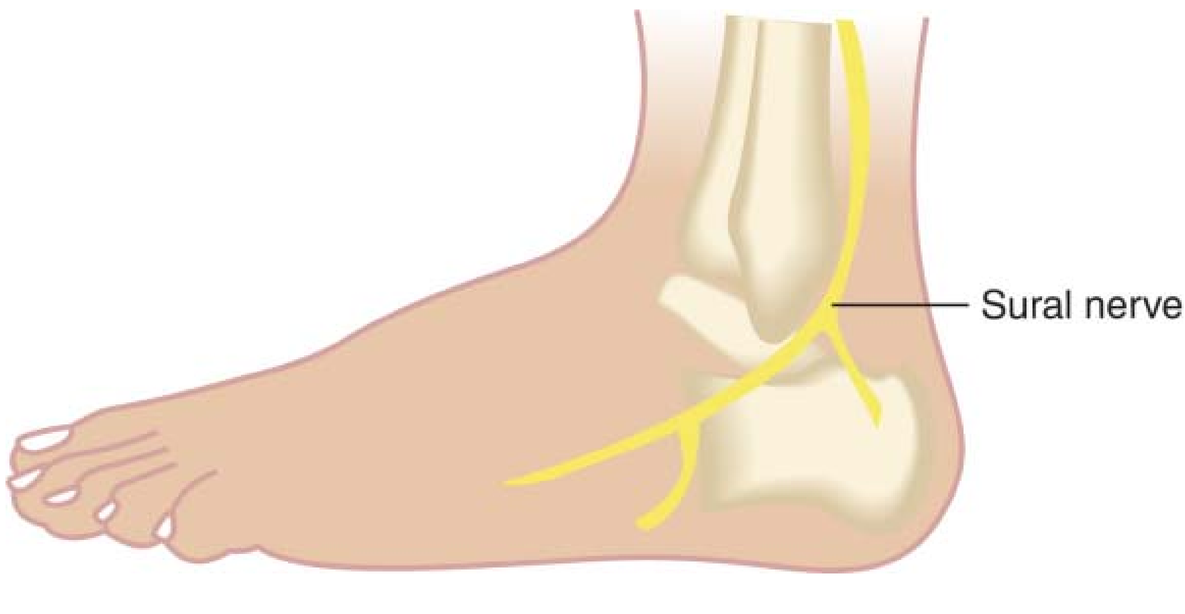

Figure 25G-5 Anatomy of the sural nerve.

A primary entrapment of this nerve is unusual because it is surrounded by adequate fatty soft tissue throughout its course and does not pass through any structures that result in constriction. It is damaged most frequently secondary to extrinsic factors, such as trauma or surgery. [1307] [1308] [1309] [1310]

Clinical Symptoms

Patients usually complain of a burning pain or numbness along the distribution of the sural nerve or its terminal branches. At times, the symptoms are brought about by wearing a shoe or boot that places pressure against the involved area.

Etiology

Although external pressure might be applied to the nerve, symptoms are unusual because even if a mass were present, the nerve is surrounded by so much soft tissue that sufficient pressure rarely can be applied to the nerve to bring about an entrapment. After a crush injury, however, the nerve can become entrapped in fibrous tissue. After surgical procedures about the malleolus or hindfoot area, the sural nerve can be entrapped either proximal to the malleolus or distally, after it divides into two branches.

Diagnosis

Physical Examination

The physical findings consist of tenderness along the course of the sural nerve along with tingling when the nerve is percussed in the involved area. One should start by examining the nerve proximal to the malleolus, then proceed down behind the malleolus and along its two terminal branches. Numbness can be detected distal to the area of the entrapment, or occasionally dysesthesias may be present if there is a neuroma in situ. Electrodiagnostic studies are not necessary to confirm the diagnosis of a sural nerve entrapment.

Treatment

Conservative Management

Conservative management consists in attempting to relieve the pressure on the involved area by padding or changing the type of boot or shoe that is worn. Occasionally, a steroid injection into the area of maximal tenderness may be beneficial.

Surgical Treatment

If a specific neuroma is identified, the area can be explored surgically, resecting the nerve back into an area of adequate soft tissue and attempting to bury it beneath the structures so that when the traumatic neuroma forms again, it will be, ideally, in an area that will not be subjected to pressure. Sometimes, however, even after successful excision of the neuroma, it may reform and cause the patient difficulty.

When the neuroma occurs above the level of the malleolus, the nerve should be traced proximally, resected, and buried in the anterior aspect of the gastrocnemius-soleus muscle so that it is not subjected to pressure from boots.

Postoperatively, the foot is placed in a compression dressing until the soft tissue heals and then weight-bearing is permitted as tolerated. Swelling should be controlled with an elastic stocking.

Clinical Results

Generally, resection of the neuroma results in satisfactory resolution of the problem. If a recurrent neuroma develops, a second operative procedure may be necessary.

DEEP PERONEAL NERVE ENTRAPMENT (ANTERIOR TARSAL TUNNEL SYNDROME)

The deep peroneal nerve may become entrapped at several sites as it passes distally (Fig. 25G-6). As it passes over the ankle joint beneath the inferior extensor retinaculum just lateral to the extensor hallucis longus tendon, it divides into medial and lateral branches. The lateral branch innervates the extensor digitorum brevis muscle and the surrounding joints. The medial branch passes with the dorsalis pedis artery and distally supplies the web space between the first and second toes. Compression against the nerve may occur at the ankle, as it passes over the talonavicular joint, and at the tarsometatarsal joints.

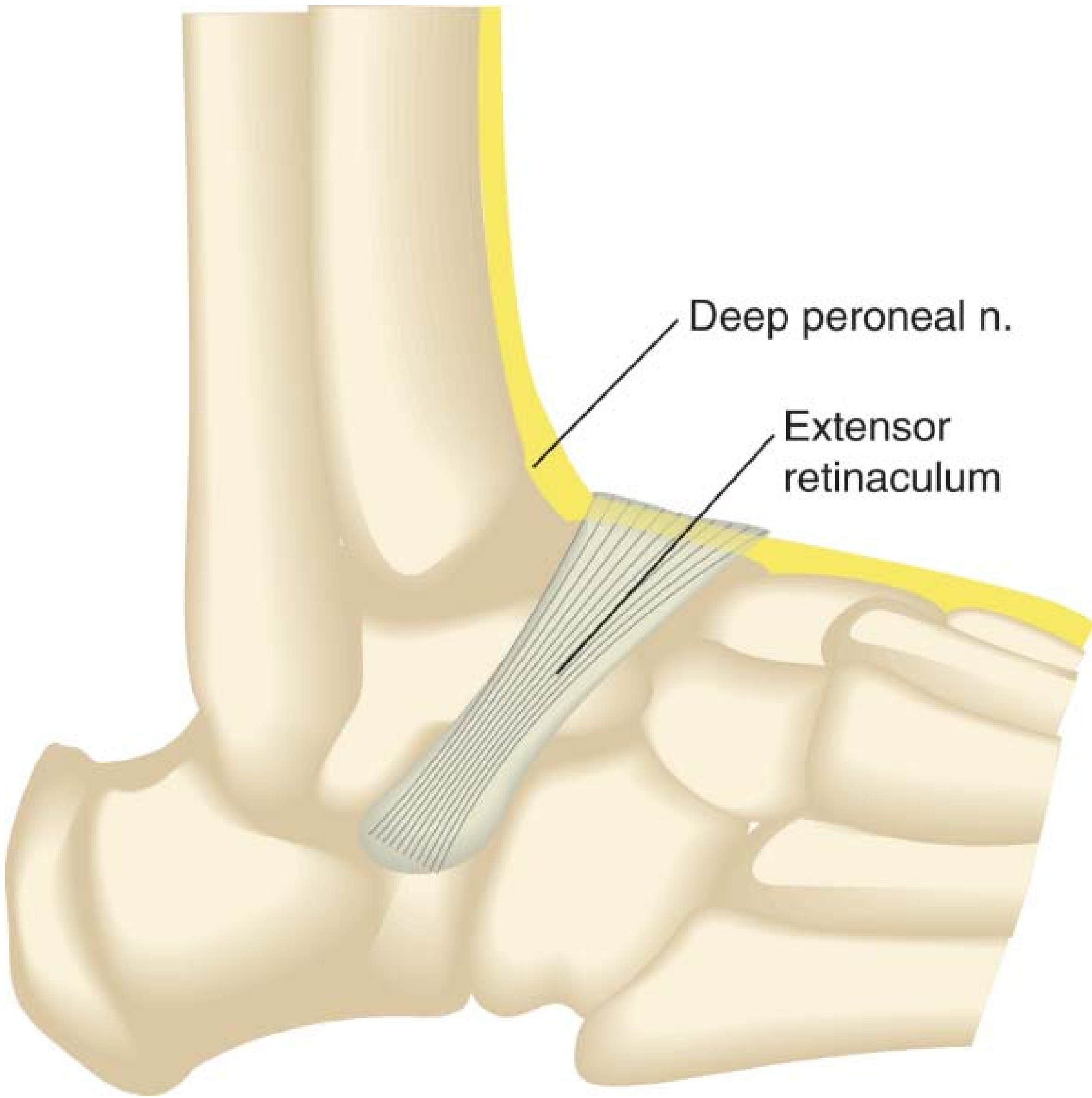

Figure 25G-6 Deep peroneal nerve entrapment beneath the retinaculum.

Clinical Symptoms

The patient usually complains of a vague pain over the dorsomedial aspect of the foot with occasional radiation into the first web space. At times, the main complaint is numbness within this area. The symptom complex often is aggravated by activities but in some cases is more bothersome when the patient is in bed at night.

Examination may reveal sensory changes in the first web space as well as weakness of the extensor digitorum brevis muscle. In about 22% of limbs, the extensor digitorum brevis muscle is innervated by the accessory deep peroneal nerve, not the deep peroneal nerve. [1311] [1312] Percussion along the course of the nerve is important, and usually tingling is noted at the level of the entrapment. If a bone ridge is encountered and is thought to be the cause of the patient's problem, radiographic evaluation is essential to define the size of the exostosis.

Diagnosis

Electromyographic Studies

Electrodiagnostic studies can be useful. These studies show an increase in the distal motor latency of the deep peroneal nerve with normal conduction velocities proximal to the ankle. [1313] [1314] There are, however, no specific criteria for nerve latencies established for this location. There may be signs of chronic denervation in the extensor digitorum brevis muscle.

Differential Diagnosis

An L5 radiculopathy, as well as a neuropathy of the common or superficial peroneal nerve, may mimic entrapment of the deep peroneal nerve. The electrodiagnostic studies should differentiate these entities.

Treatment

Conservative Management

If the entrapment is over a bony prominence at the level of the tarsometatarsal joints or at the level of the talonavicular joint, padding the area or wearing a looser shoe may permit resolution of the symptoms. Occasionally, a steroid injection may be of some benefit. If the impingement is mainly mechanical in nature, surgery is usually necessary.

Surgical Management

Surgical release of the nerve depends on the site of the entrapment. If the symptoms are located at the level of the extensor retinaculum, this area can be released easily through a small anterior incision. If the entrapment is secondary to bony spurs over the anterior aspect of the ankle or the talonavicular joint or at the level of the tarsometatarsal joints, the nerve should be exposed carefully over the involved area; the exostosis is then exposed and excised carefully. Postoperatively, the patient is placed in a compression dressing with weight-bearing as tolerated and is permitted to return to activities as tolerated.

Clinical Results

Generally, release of an entrapment of the deep peroneal nerve is successful. Caution must be exercised, however, because the nerve can be damaged while trying to release it. Occasionally, the exostosis may re-form, resulting in further difficulty for the patient.

SUPERFICIAL PERONEAL NERVE ENTRAPMENT

Entrapment of the superficial branch of the common peroneal nerve may occur where it exits through the sharp fascial edges in the distal anterolateral aspect of the leg about 10 to 15 cm proximal to the lateral malleolus (Figs. 25G-7 and 25G-8 [0925] [0930]). [1289] [1315] [1316] [1317] After exiting through the fascia, the nerve divides into its two terminal branches, forming the medial dorsal cutaneous and the intermediate dorsal cutaneous nerves. These nerves provide sensation to the dorsum of the foot, with the exception of the first web space, which is innervated by the superficial portion of the deep peroneal nerve.

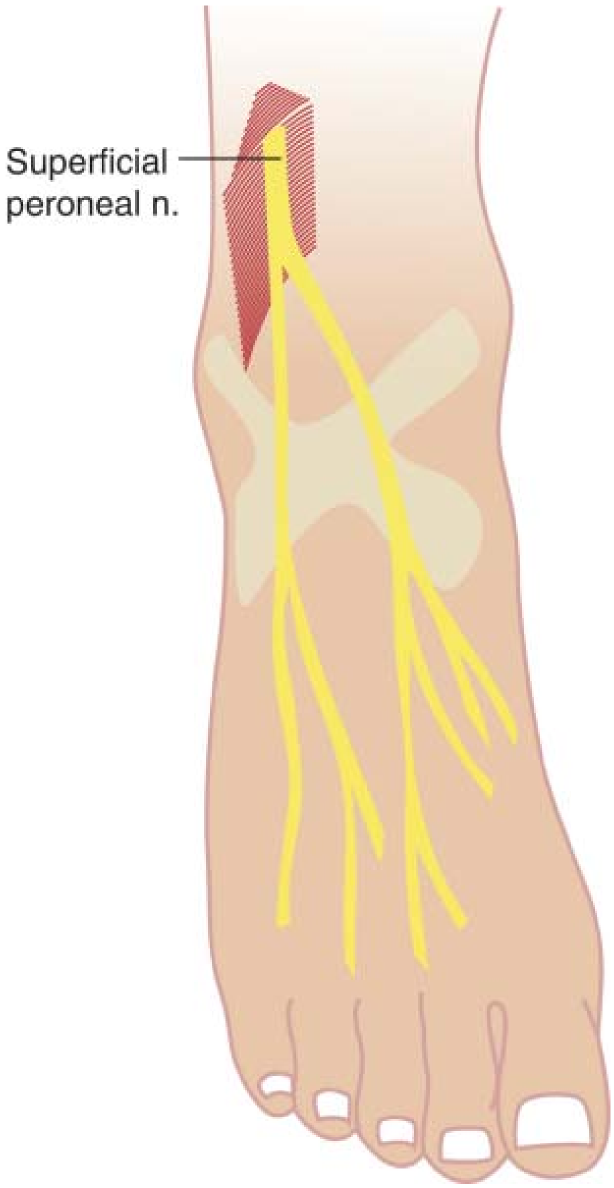

Figure 25G-7 Entrapment of the superficial peroneal nerve as it exits from the deep fascia of the leg.

Figure 25G-8 Cadaveric specimen: superficial peroneal nerve exiting hiatus.

Clinical Symptoms

The patient usually complains of numbness and tingling over the dorsal aspect of the foot and ankle, but the first web space is not involved. The symptoms may be stimulated by sporting activities, but the condition occurs in the nonathlete as well. If the entrapment is due to a fascial defect with resultant muscle herniation, the patient may note a fullness or swelling over the anterolateral aspect of the leg where the nerve exits through the fascia.

Etiology

The cause frequently is undetermined, but sometimes the condition is caused by a fascial defect with secondary muscle herniation, which results in tenting of the superficial peroneal nerve. [32] At times, compression from an external source, such as a lipoma or occasionally a bony callus resulting from a tibial fracture, may place pressure on the nerve. [1315] [1317] [1318]

Several studies have demonstrated nerve conduction abnormalities of the distal peroneal nerve, superficial peroneal nerve, and common peroneal nerve after inversion injury of the ankle. [33] A recent biomechanical study showed the magnitude of shear force generated across the superficial peroneal nerve during an inversion injury to be within the range necessary to structurally alter the nerve. [34]

Diagnosis

Physical Findings

The physical examination usually brings out the cause of the problem. Tenderness or irritability of the nerve to percussion is evident where it exits through the fascial defect. A muscle herniation or an underlying lipoma or callus usually can be palpated. Frequently, there is some sensory loss over the distribution of the nerve around the anterior aspect of the foot, sparing the first web space. Resisted dorsiflexion of the ankle joint may show the muscle herniation through the fascial defect.

Electromyographic Studies

Electrodiagnostic studies are rarely of any benefit in the diagnosis of superficial peroneal nerve injury or entrapment.

Differential Diagnosis

Superficial peroneal nerve entrapment may mimic a common peroneal nerve entrapment or possibly an L5 radiculopathy. The symptoms of a chronic ankle sprain or instability can mimic this condition, but with a careful history and examination, these usually can be differentiated.

Treatment

Conservative Management

Occasionally, a steroid injection or a diminished level of activity may be beneficial. If symptoms persist, however, and particularly if a muscle herniation or mass is present beneath the nerve, surgical release is indicated.

Surgical Treatment

The site of the entrapment is identified through a longitudinal incision, and the fascia over the anterior compartment is released. It is important to carry out an adequate release so that a muscle herniation does not result from the surgical decompression. If a mass is present, such as a lipoma or callus, this should be excised. Postoperatively, a compression dressing is used until the soft tissues heal, at which time activities can be resumed as tolerated.

Clinical Results

Generally, surgical decompression of the nerve results in satisfactory relief for the patient. One must be cautious, however, not to damage the nerve inadvertently when the decompression is carried out.

CRITICAL POINTS

- The most common error in surgical release of the tarsal tunnel is to make too short an incision and inadequately release the nerves distally as they pass underneath the abductor fascia.
- Surgical decompression is dramatically more reliable for tarsal tunnel syndrome due to mass-occupying lesions in the hindfoot than that due to idiopathic causes.
- Entrapment of the nerve to the abductor digiti quinti is an important component of the differential diagnosis of heel pain and may coexist with plantar fasciitis.
- Sural nerve injury due to extrinsic causes or iatrogenic surgical exposure is among the most common nerve problems encountered in sports medicine practice. Refractory cases are best dealt with by proximal resection and burial rather than neurolysis.
- Traction neuritis of the superficial peroneal nerve is an underappreciated source of additional pain in cases of acute or chronic ankle instability. Surgical release can be performed, but the neuritis almost always resolves following surgical stabilization of the ankle.

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SECTION H Conditions of the Forefoot

Sandra E. Klein

HALLUX VALGUS

Hallux valgus is a static subluxation of the first metatarsophalangeal (MTP) joint characterized by lateral or valgus deviation of the great toe and medial or varus deviation of the first metatarsal. [1] Occasionally, pronation or rotation of the hallux occurs with severe deformity. A hallux valgus deformity can also occur with lateral deviation of the distal articular surface of the first metatarsal. This anatomic variation is seen most commonly in juvenile hallux valgus. [1332] [1333] [1334]

Hallux valgus is found almost exclusively in shoe-wearing societies, although it has been noted in primitive societies that do not wear shoes. [1335] [1336] A higher incidence in females has also been reported in the literature. [1337] [1338] [1339] Many factors other than shoewear have been investigated for a causal or contributing relationship to hallux valgus deformity, including genetic predisposition, [1332] [1339] [1340] [1341] pes planus, [1339] [1342] first metatarsocuneiform hypermobility, ligamentous laxity, [1335] [1343] [1344] [1345] and Achilles contracture. [1335] [1343] [1346] [1347] In the athletic population, hyperextension injuries or acute dislocation of the first MTP joint may predispose to the development of a hallux valgus deformity.

A symptomatic hallux valgus deformity in a high-performance athlete presents a special problem for the treating physician. The increased stress manifested by running or jogging can affect the function of the foot significantly. [18] The increased force generated in the forefoot can approach 250% of body weight with running compared with 80% with walking. Lillich and Baxter [18] noted an increased range of motion of the joints of the lower extremity and an alteration in phasic activity of the muscles of the lower extremity with running activity. These factors must be considered when evaluating a hallux valgus deformity in an athlete. [19]

Anatomy and Biomechanics

The MTP joint of the hallux is differentiated from the lesser toes by the sesamoid mechanism as well as by specific intrinsic muscles that stabilize the great toe and provide motor strength. [20] The first metatarsal head has a rounded cartilage-covered surface that articulates with the concave base of the proximal phalanx. There is significant variation in the joint surfaces of the first MTP articulation. A rounded MTP articulation is more prone to the development of hallux valgus; a flattened or chevron-shaped MTP articulation is a stable configuration and is less prone to progressive development of hallux valgus (Fig. 25H-1). [1]

Figure 25H-1 **A,** A rounded metatarsal head is at risk for subluxation of the metatarsophalangeal joint. **B,** A flat metatarsophalangeal articulation is more resistant to subluxation. *(© M. J. Coughlin. Used by permission.)*

The first MTP joint is stabilized on the plantar medial and plantar lateral aspects by the collateral ligaments and sesamoid ligaments (Fig. 25H-2 **A**). The fan-shaped collateral ligaments originate from the medial and lateral epicondyles and course in a distal-plantar direction, inserting on the medial and lateral aspect of the base of the proximal phalanx. The collateral ligaments interdigitate with the sesamoidal ligaments, which originate in the epicondylar region of the metatarsal head and insert into the margins of each sesamoid as well as the plantar plate. Although the plantar medial and plantar lateral aspects of the MTP joint are stabilized by a thick capsular structure, the dorsal aspect is supported by the relatively thin extensor hood (see Fig. 25H-2 **B**). With pronation of the foot or with progressive subluxation of the first MTP joint, increased pressure is placed on the weaker dorsal medial capsule, which becomes stretched and attenuated with time (Fig. 25H-3).

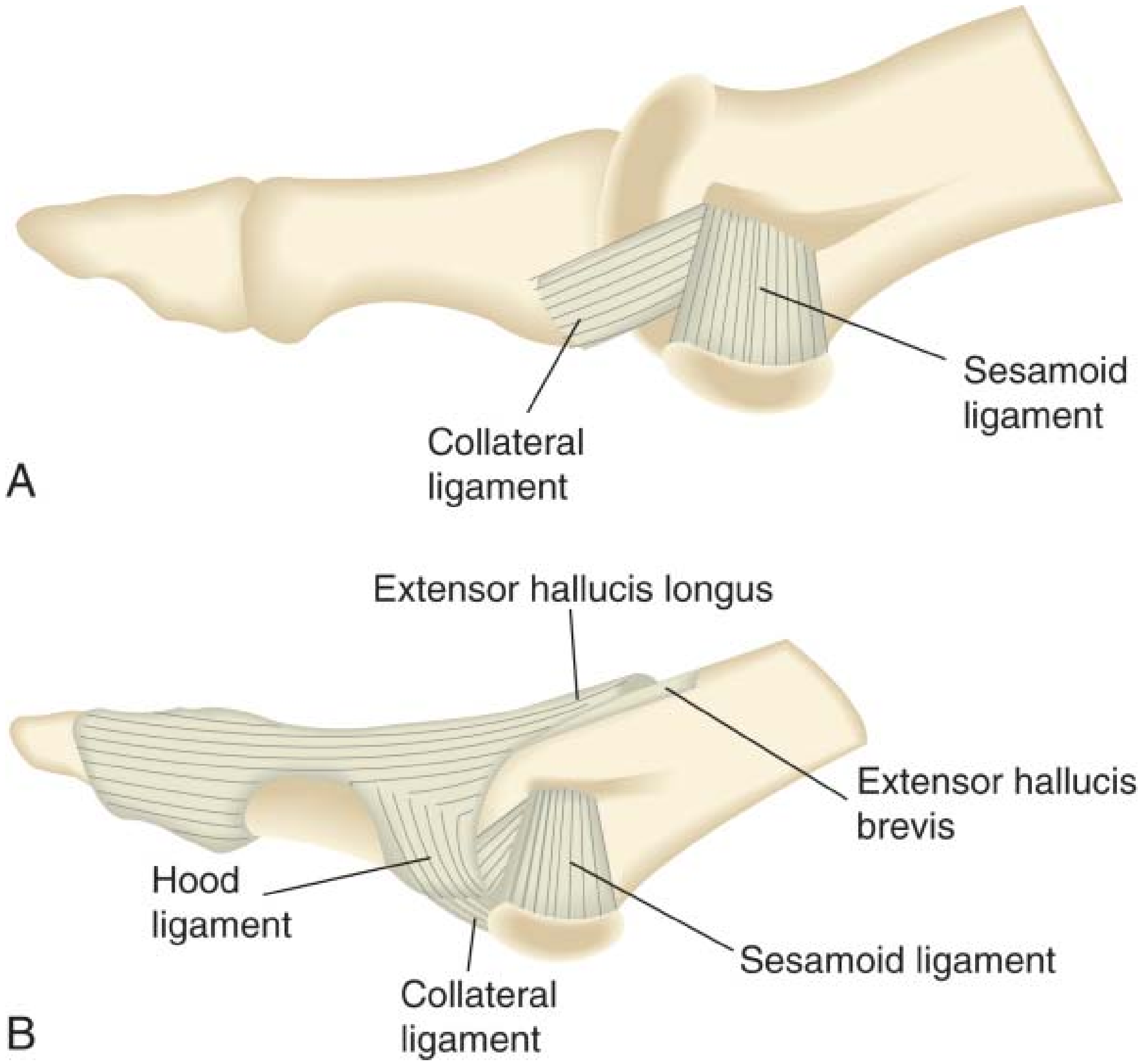

Figure 25H-2 **A,** The collateral ligaments and the sesamoid ligaments of the first metatarsophalangeal joint. **B,** The extensor hallucis longus inserts into the hood ligament.

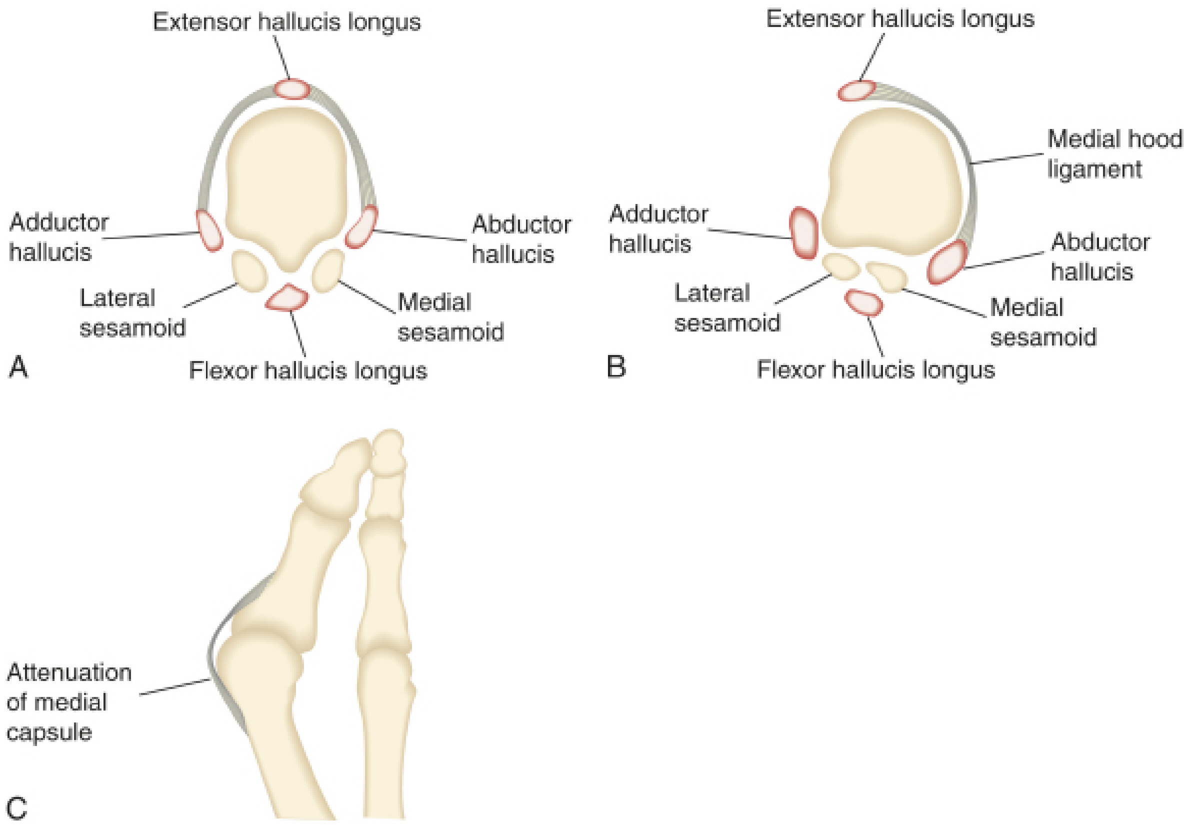

Figure 25H-3 A-C, With subluxation of the metatarsophalangeal joint, the dorsal capsule and the hood ligament become attenuated.

The intrinsic musculature of the first ray is composed of several distinct tendinous structures. The two tendons of the flexor hallucis brevis, located directly plantarward, insert into the medial and lateral sesamoids. The sesamoids articulate on their dorsal surfaces with medial and lateral facets on the plantar aspect of the first metatarsal head (Fig. 25H-4 **A**). The intersesamoidal ridge (the crista) (see Fig. 25H-4 B) separates these facets and affords stability to the metatarsal-sesamoid articulation. Distally, the sesamoids are attached to the fibrous plantar plate, which inserts onto the plantar base of the proximal phalanx. The abductor hallucis, located on the plantar medial aspect, inserts onto the plantar medial base of the proximal phalanx. The adductor hallucis, located on the plantar lateral aspect, inserts onto the plantar lateral base of the proximal phalanx. There are *no* tendinous insertions onto the metatarsal head; the control of the metatarsal is maintained by the capsuloligamentous complex and the intrinsic muscles that insert onto the plantar half of the proximal phalanx. As a hallux valgus deformity progresses, the ligaments and intrinsic musculature of the first ray play a significant role in the orientation of the MTP joint.

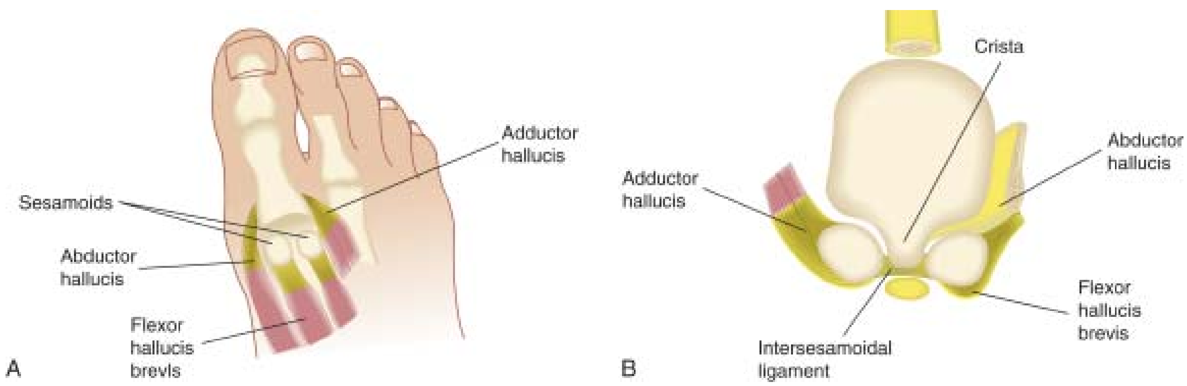

Figure 25H-4 A, The sesamoids are contained within the double tendon of the flexor hallucis brevis. **B,** Cross section through the metatarsophalangeal joint shows relationship of sesamoids and extrinsic muscles.

The extensor hallucis longus, located on the dorsal aspect of the hallux, is stabilized medially and laterally by the extensor hood (see Fig. 25H-2 **B**); it inserts onto the dorsal base of the distal phalanx, whereas the extensor hallucis brevis inserts onto the base of the proximal phalanx. The tendon of the flexor hallucis longus, contained within a fibrous tendon sheath on the plantar aspect of the sesamoid complex, inserts onto the plantar base of the distal phalanx.

Specific angular measurements on a weight-bearing anteroposterior radiograph quantify the characteristics of a hallux valgus deformity: the hallux valgus angle, 1-2 intermetatarsal angle, hallux interphalangeal angle, and distal metatarsal articular angle (Table 25H-1 ; Fig. 25H-5). [1334] [1340] [1346] [1351] [1352] [1353]

TABLE 25H-1 -- Angular Measurements in Hallux Valgus Deformity

Measured Angle	Radiographic Parameters	Normal Values
1,2 Intermetatarsal angle [1340] [1346] [1351]	Angle between the axes of the first and second metatarsals (see Fig. 25H-5)	Less than 9 degrees
Hallux valgus angle [1340] [1346] [1351]	Angle between the axes of the first metatarsal and the proximal phalanx (see Fig. 25H-5)	Less than 15 degrees
Hallux interphalangeal angle [22]	Angle between the axis of the proximal phalanx and an axis drawn through the center of the base of the distal phalanx and the tip of the distal phalanx	Less than 10 degrees
Distal metatarsal articular angle [1334] [1353]	Angle between the axis of the first metatarsal and a perpendicular to a line defining the lateral slope of the distal metatarsal articular surface	Less than 6 degrees of lateral inclination

Figure 25H-5 The 1-2 intermetatarsal angle is measured by lines bisecting the axis of the first and second metatarsals. The hallux valgus angle is the angle subtended by the axes of the proximal phalanx and the first metatarsal.

The first metatarsocuneiform joint plays an integral role in the stability of the first metatarsal. Increased obliquity of the first metatarsocuneiform joint increases the magnitude of the metatarsus primus varus (Fig. 25H-6), although routine radiographic imaging of this joint is often imprecise. A horizontal orientation of the metatarsocuneiform joint appears to be a stable configuration and resists deformity, whereas a curved metatarsocuneiform joint appears to be more flexible, allowing medial metatarsal deviation as the hallux valgus deformity increases. [1]

Figure 25H-6 Metatarsocuneiform orientation. **A,** A straight orientation of the metatarsocuneiform joint is stable. **B,** A curved metatarsocuneiform articulation is more flexible and may be at risk for deviation with an increased intermetatarsal angle. **C,** An oblique orientation of the metatarsocuneiform joint is often resistant to correction. An osteotomy may be necessary in this situation.

Congruency of the first MTP joint is also assessed. In a congruent joint (Fig. 25H-7 **A**), the center of the metatarsal articular surface corresponds to the center of the articular surface of the proximal phalanx. In contrast, with an incongruent articulation, there is subluxation of the MTP joint, that is, the base of the proximal phalanx is deviated laterally in relation to the articular surface of the first metatarsal, resulting in an uncovering of the metatarsal head (see Fig. 25H-7 **B**). [3] Piggott, [24] in a study of congruous and incongruous first MTP joints with hallux valgus, found that a congruous joint (with or without an increased distal metatarsal articular angle) was a stable articulation. A hallux valgus deformity in this situation does not increase with time. With an incongruous MTP joint, there is a significant risk for further progression of the deformity.

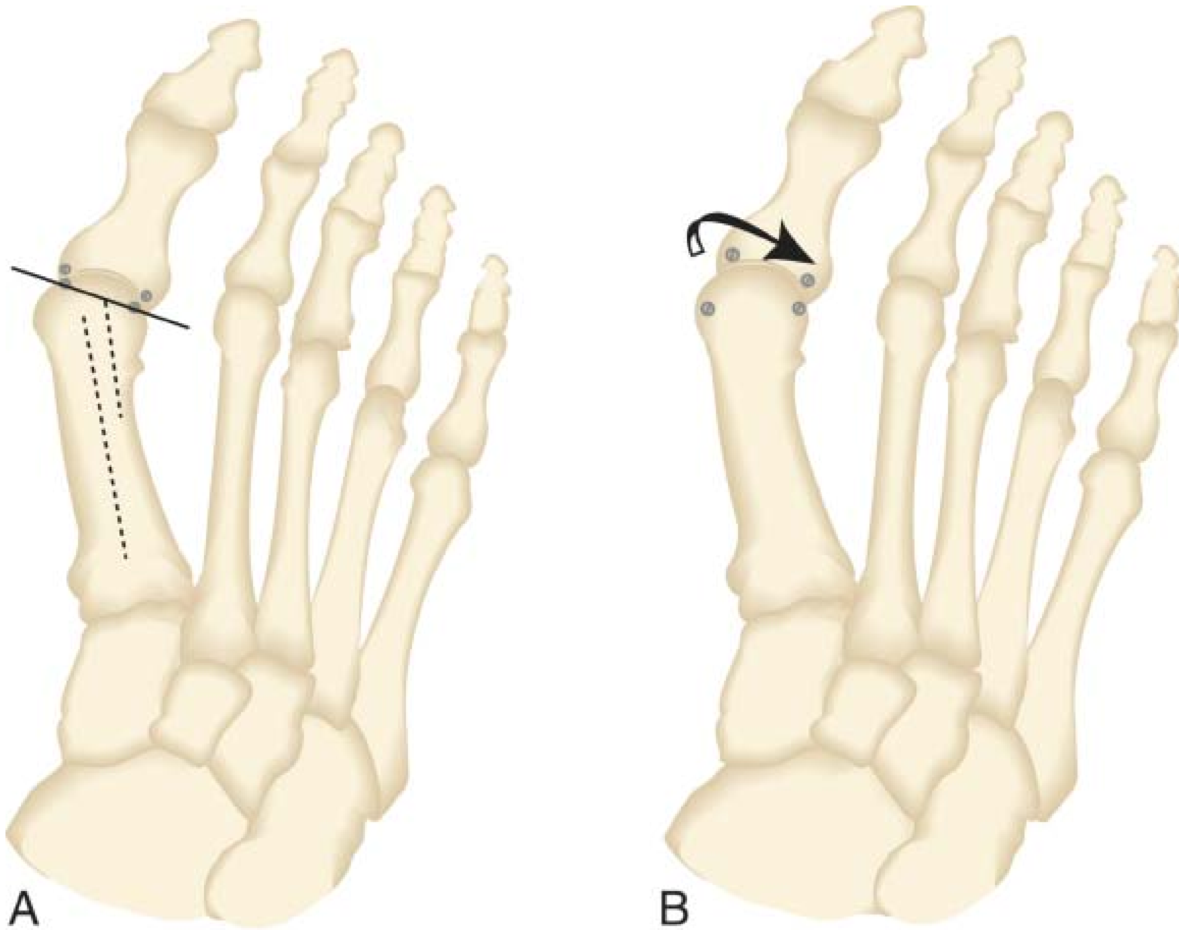

Figure 25H-7 **A,** The metatarsal articular orientation. The distal metatarsal articular angle is deviated in a lateral direction. **B,** A subluxated metatarsophalangeal joint. In this noncongruent joint, the articular surface of the phalanx subluxates laterally on the metatarsal articular surface.

The dynamic development of a hallux valgus deformity is characterized by changes at the MTP, metatarsocuneiform, and metatarsosesamoid articulations. As hallux valgus develops, the proximal phalanx is displaced in a valgus or lateral direction and often is pronated on the head of the first metatarsal. [25] The insertion of the adductor hallucis plays an integral role in the subsequent deformity. The adductor hallucis insertion onto the lateral sesamoid anchors the sesamoid complex to the lesser metatarsals so that they cannot drift medially with the first metatarsal (Fig. 25H-8). As the hallux is forced in a lateral direction by extrinsic forces, the base of the proximal phalanx pushes the metatarsal head medially and attenuates the medial capsule. The insertion of the adductor hallucis onto the plantar lateral base of the proximal phalanx and lateral sesamoid exerts not only a lateral force on these structures but also a rotational force on the proximal phalanx. As the metatarsal drifts medially, the proximal phalanx rotates along the longitudinal axis of the adductor hallucis, the pivot point being the point of insertion of the adductor. As the great toe displaces laterally, the intrinsic plantar cuff (consisting of the abductor hallucis, the flexor hallucis brevis, and the adductor hallucis) (Fig. 25H-9) rotates in relation to the metatarsal head, exposing the thin dorsal medial capsule to further deforming forces.

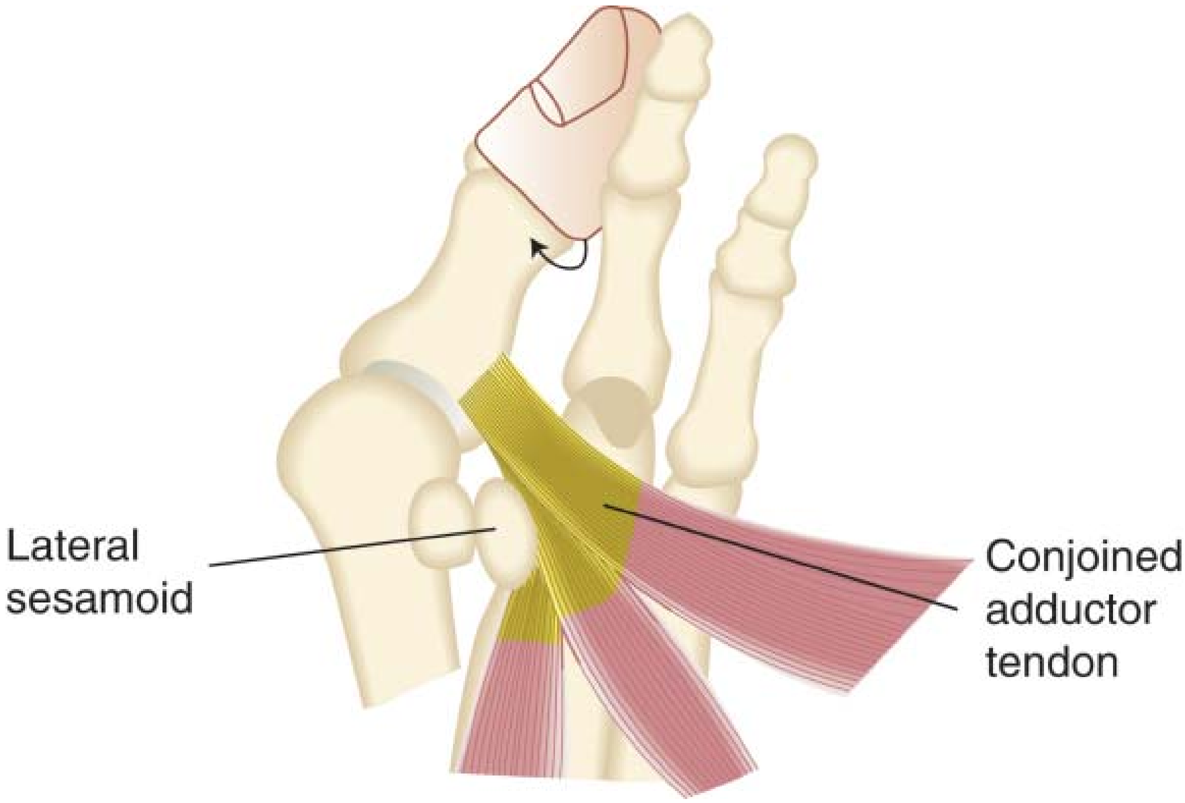

Figure 25H-8 The adductor hallucis anchors the lateral sesamoid and the proximal phalanx. When subluxation of the metatarsophalangeal joint occurs, the toe may rotate or pronate owing to insertion of the conjoined adductor tendon.

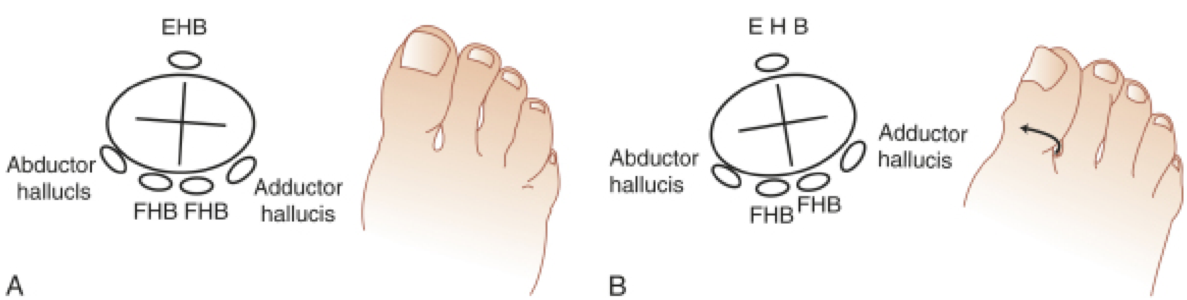

Figure 25H-9 **A,** Schematic representation of the intrinsic muscles surrounding the first metatarsal head. Normal articulation. **B,** With valgus deviation, the intrinsic muscles rotate. The abductor hallucis assumes a more plantar position, and the adductor hallucis assumes a more lateral orientation. EHB, extensor hallucis brevis; FHB, flexor hallucis brevis.

On the plantar aspect, the sesamoid complex displaces in relation to the first metatarsal head. The sesamoid mechanism, owing to the adductor hallucis insertion, retains its relationship to the lesser metatarsals, whereas the first metatarsal literally slides off the sesamoid complex. Although the term *sesamoid subluxation* is used to describe this progressive deformity, it is the first metatarsal that is displaced in relation to the sesamoids and lesser metatarsals. As this displacement occurs, the intersesamoidal ridge is smoothed out gradually (Fig. 25H-10) until it affords no resistance to further deformity. In severe hallux valgus deformities, as the first metatarsal displaces medially off the sesamoid mechanism, the extensor hallucis longus displaces into the first intermetatarsal space. As a result, contraction of the extensor hallucis longus not only extends the toe but also adducts the toe. The abductor hallucis, having assumed a more plantar position in relation to the first metatarsal, loses its splinting effect on the first metatarsal head. The lateral sesamoid may come to lie on the lateral aspect of the first metatarsal head and on occasion lies vertically above the medial sesamoid. The medial sesamoid may articulate with the lateral facet of the first metatarsal.

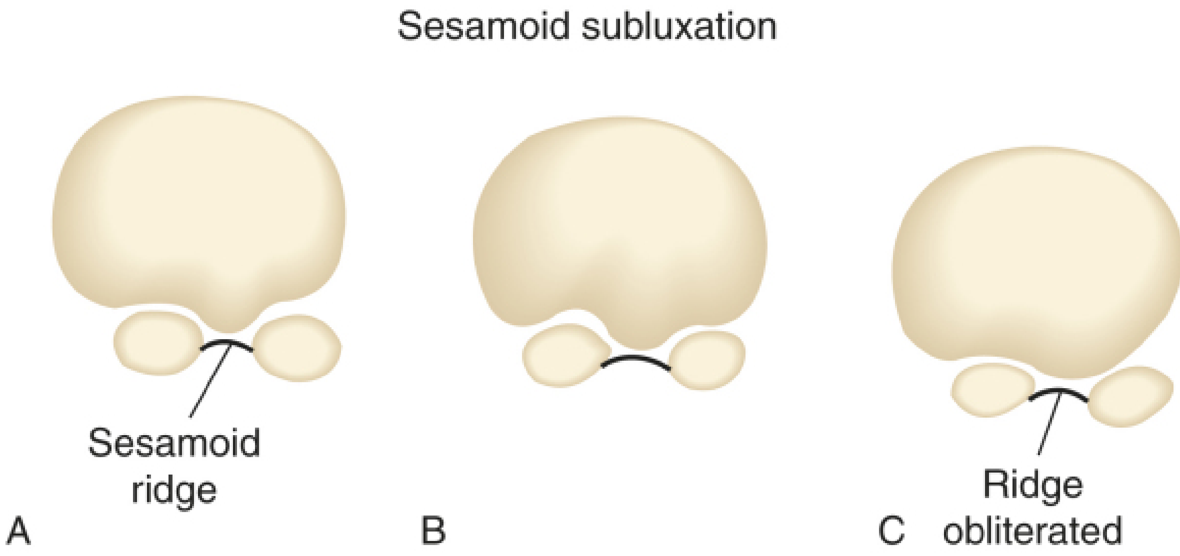

Figure 25H-10 **A,** Schematic representation shows sesamoid view with normal crista. **B,** Schematic representation of a patient with hallux valgus shows moderate obliteration of crista. **C,** Complete obliteration of crista with sesamoid subluxation.

Running places higher demands on the foot than walking. [1348] [1356] [1357] Various athletic activities make varying demands on the foot and ankle. A sprinter may require extreme range of motion in dorsiflexion and plantar flexion at the first MTP joint, in contrast to a middle-distance or long-distance runner. [18] Analysis of the particular sports avocation, training techniques, strength requirements of the first ray, and range of motion needed by the first MTP joint plays an important role in the assessment of the functional biomechanics of the forefoot. This careful evaluation helps the practitioner to develop a plan for the symptomatic athlete.

Although much attention has been paid to the bunion or medial eminence, it is most often the increased 1-2 intermetatarsal angle coupled with lateral deviation of the great toe that creates a prominent and painful medial border of the first metatarsal head. At times, the medial eminence may become hypertrophied and symptomatic; however, this is uncommon. With progressive subluxation, a groove or sagittal sulcus (Fig. 25H-11) develops on the first metatarsal head, delineating the articular surface from the medial eminence. Although this anatomic landmark delineates the border of the metatarsal articular surface, its location is variable and depends on the severity of the deformity. Sometimes the sagittal sulcus is located in the center of the metatarsal head. It is important not to use the sagittal sulcus as a landmark for the medial eminence resection because in severe deformities an excessive amount of metatarsal head is removed. In mild and moderate cases of hallux valgus, attention must be directed to the location of the sagittal sulcus on the anteroposterior radiograph to determine whether resection of the medial eminence should be performed through the center of the sagittal sulcus or medial or lateral to the center of the sagittal sulcus.

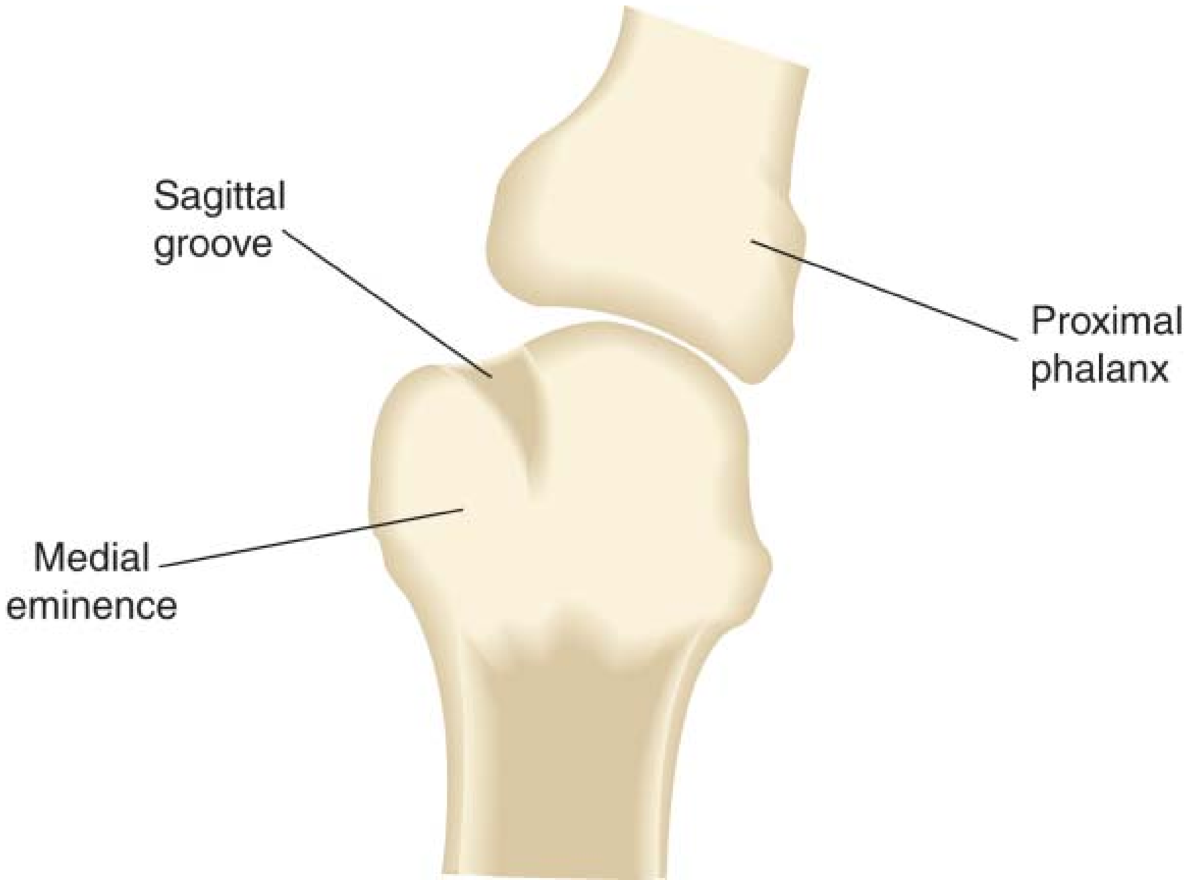

Figure 25H-11 The sagittal sulcus is created at the medial margin of the articular surface and is an osteophyte delineating the border of the medial eminence.

Classification

Hallux valgus deformities are typically classified as mild, moderate, or severe. [1] Classification is used as a general guideline in the decision-making process when treating a hallux valgus deformity (Box 25H-1).

BOX 25H-1

CLASSIFICATION OF HALLUX VALGUS DEFORMITIES

Mild Deformity

- Hallux valgus angle < 20 degrees
- Hallux valgus interphalangeus may contribute to deformity
- First metatarsophalangeal joint often congruent
- 1-2 intermetatarsal angle < 11 degrees

Moderate Deformity

- Hallux valgus angle 20-40 degrees
- 1-2 intermetatarsal angle 11-16 degrees
- Subluxation of first metatarsophalangeal joint (unless distal metatarsal articular angle is abnormal)
- Hallux may be pronated

Severe Deformity

- Hallux valgus angle > 40 degrees
- 1-2 intermetatarsal angle greater than 16 degrees
- Subluxation of metatarsophalangeal joint
- Hallux typically pronated

Evaluation

Clinical Presentation and History

The major subjective complaint of a patient with hallux valgus is pain over the medial eminence. At times, especially with repetitive athletic activities such as running, blistering of the skin or development of an inflamed bursa overlying the medial eminence may occur. Pressure from a shoe may cause compression of the dorsal medial sensory nerve to the hallux, causing either neuritic pain or numbness. With a severe deformity, diminished weight-bearing capacity of the first ray may lead to lateral metatarsalgia with development of an intractable plantar keratosis beneath the lesser metatarsal heads. Runners with hallux valgus may develop a callus on the medial border of either the great toe or the medial eminence (Box 25H-2).

BOX 25H-2

TYPICAL FINDINGS IN HALLUX VALGUS DEFORMITIES

History

- Medial eminence pain
- Metatarsophalangeal joint pain
- Bursal or skin irritation
- Callus formation

Physical Examination

- Medial deviation of the first metatarsal
- Lateral deviation of the hallux
- Pronation of the hallux

Radiographic Examination

- Increased 1-2 intermetatarsal, hallux valgus, or hallux interphalangeal angles
- Increased distal metatarsal articular angle
- Congruent versus incongruent first metatarsophalangeal joint

Physical Examination

Evaluation of the athlete with hallux valgus begins with an examination of the patient while standing. Often, deformities are accentuated with weight-bearing. Assessment of postural abnormalities such as pes planus, pes cavus, and a contracted Achilles tendon; neuromuscular abnormalities; and hyperelasticity associated with a collagen deficiency syndrome is important. The hallux is evaluated for lateral deviation and pronation. Medial deviation of the first metatarsal may also be noted on physical examination. A vascular evaluation should assess the presence or absence of pulses. Doppler evaluation may assist if vascular integrity is in doubt. Neurologic evaluation is important for assessment of sensation, motor strength and control, and gait abnormalities. Assessment of active and passive range of motion is important in an athlete.[28] Often bunion surgery may be associated with diminished first MTP joint range of motion postoperatively, and quantitation of preoperative range of motion may help in developing a plan of treatment. The mobility of the first metatarsocuneiform joint is evaluated. [1359] [1360] Skin changes such as inflammation of the medial eminence, callus formation, or intractable plantar keratoses are noted. The magnitude and rigidity of a pes planus deformity may influence preoperative or postoperative orthotic management (see Box 25H-2).

Imaging

Standard radiographic views include standing anteroposterior, oblique, lateral, and sesamoid views. The magnitudes of the 1-2 intermetatarsal angle and the hallux valgus angle help to quantitate the severity of the deformity. Analysis of the MTP joint for congruity, joint subluxation, and the presence of degenerative arthritis or hallux rigidus is indicated. The metatarsocuneiform joint articulation, although often difficult to assess radiographically, should be analyzed as well. The radiographs should be inspected for other lesser toe abnormalities that may be associated with the hallux valgus deformity (see Box 25H-2).

Treatment Options

Nonoperative

For the athletically active person, conservative care is most important. Often pain and blistering can be reduced by diminishing friction over the medial eminence. Evaluation of the athlete’s footwear is important. Modification of shoes by stretching constricting areas or relieving pressure areas may relieve an athlete’s symptoms completely. Wider footwear with a roomy toe box may afford significant reduction in discomfort related to static forefoot abnormalities. The use of pads, arch supports, and various insoles may assist in maintaining an athlete’s activity.

The overall philosophy of treatment of an athlete with a hallux valgus deformity should be nonoperative until pain or discomfort forces the athlete to make significant modifications in athletic activity. As Lutter stated, [19] “The stiffness potential occurring after an operative procedure is potentially more harmful to a running career than pain.” If, despite conservative care and modifications of athletic activity, significant discomfort still exists, operative intervention may be considered. Operations that lead to significant alterations in the weight-bearing pattern or that are associated with significant restriction of motion postoperatively should be avoided whenever possible. A patient should be counseled about the risks, complications, and expected outcomes. Unreasonable patient expectations about postoperative athletic activities are discussed before surgery. As Lutter noted, [19] “Surgery is not contraindicated, but a runner must be willing to trade relief of pain for a lower ability to run.”

Operative

Many surgical repairs have been devised to correct the hallux valgus deformity. The huge number of operative procedures available makes it clear that no one operation can suffice for every deformity. Various anatomic abnormalities, different pathologic entities, and varying degrees of deformity make it imperative that a surgeon have several methods of hallux valgus repair within his or her surgical repertoire. The surgical procedure chosen must address the various anatomic abnormalities that may be present (Box 25H-3). The preoperative assessment of the patient is important to establish which significant pathologic components are present and to select the most appropriate procedure for a particular bunion deformity (Fig. 25H-12).

BOX 25H-3

ANATOMIC FACTORS ADDRESSED IN SURGICAL TREATMENT OF HALLUX VALGUS

- Prominent medial eminence
- Valgus angulation of proximal phalanx
- Increased 1-2 intermetatarsal angle
- Sesamoid subluxation
- Pronation of great toe
- Increased distal metatarsal articular angle (congruent first metatarsophalangeal joint)

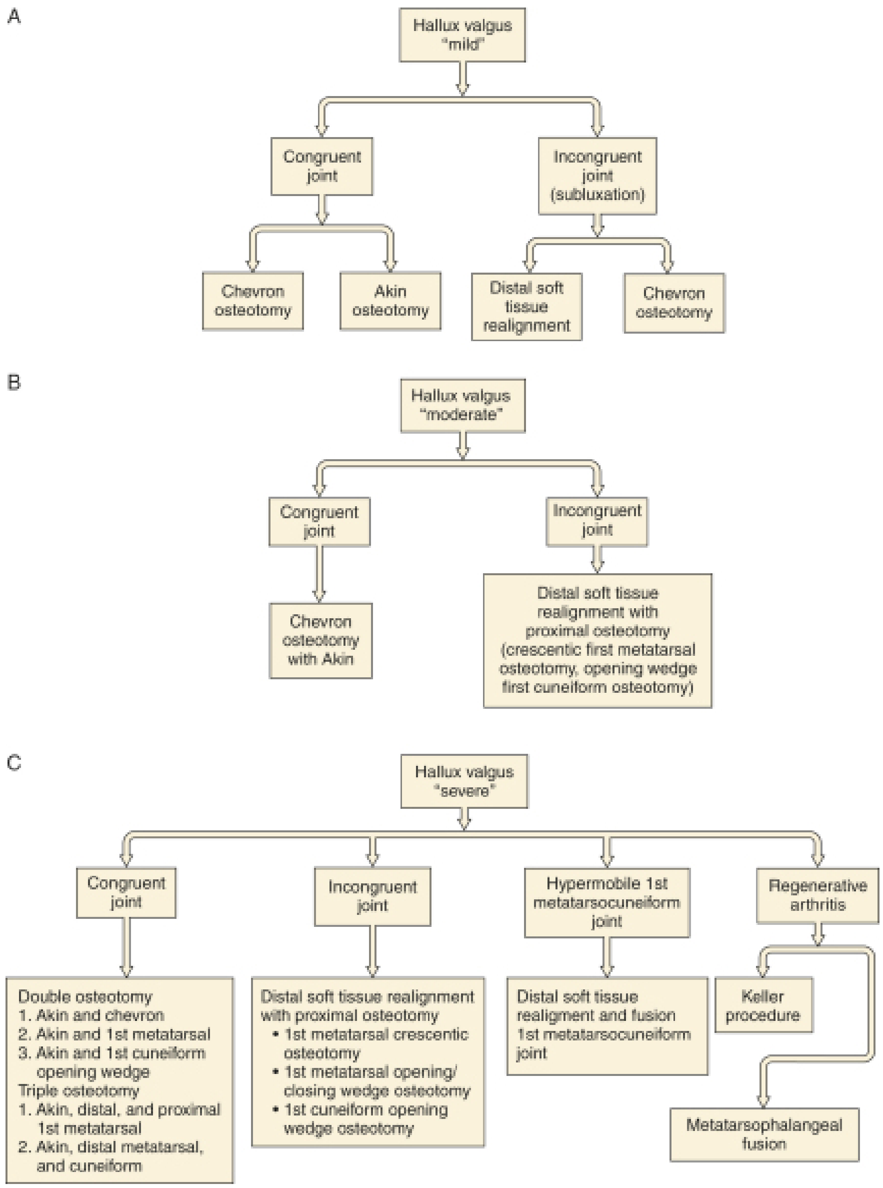

Figure 25H-12 **A,** Algorithm for treatment of mild hallux valgus deformity. **B,** Algorithm for treatment of moderate hallux valgus deformity. **C,** Algorithm for treatment of severe hallux valgus deformity.

The objective of this discussion is to describe pertinent operative procedures that may be considered for the athlete. Just as various operations address mild, moderate, and severe hallux valgus deformities, there are a wide variety of requirements and abilities in athletes (from walkers and joggers to high-performance sprinters, dancers, and professional athletes). A careful decision-making process is important to select the appropriate procedure for the symptomatic athlete (Box 25H-4 ; see Fig. 25H-12).

BOX 25H-4

COMMON SURGICAL PROCEDURES TO CORRECT HALLUX VALGUS DEFORMITY

Akin procedure

- Proximal phalangeal osteotomy, medial eminence resection, medial soft tissue reefing

Distal Soft Tissue Realignment (modified McBride procedure)

- Medial eminence resection, lateral capsule and adductor hallucis release, medial soft tissue reefing

Distal metatarsal osteotomy (chevron procedure)

- Distal metatarsal chevron osteotomy, medial eminence resection, medial soft tissue reefing

Proximal metatarsal osteotomy with distal soft tissue realignment

- Medial eminence resection, lateral capsule and adductor hallucis release, medial soft tissue reefing, proximal first metatarsal osteotomy

Combined multiple first ray osteotomies

- Phalangeal osteotomy, distal and proximal first metatarsal osteotomies, cuneiform osteotomy

Salvage procedures (not indicated as primary procedures in the athlete)

- First metatarsophalangeal joint arthrodesis, Keller resection arthroplasty

Akin Procedure

The Akin [1361] [1362] [1363] [1364] procedure involves a medial eminence resection and medial capsular reefing combined with a medial closing wedge osteotomy of the proximal phalanx (Box 25H-5 ; Fig. 25H-13). A correction of an increased 1-2 intermetatarsal angle cannot be achieved with this procedure. Indications for its use include hallux valgus interphalangeus, mild hallux valgus without significant metatarsus primus varus, and recurrent hallux valgus of a mild nature (Fig. 25H-14). In the presence of a congruent MTP joint, a proximal phalangeal osteotomy can be combined with a metatarsal osteotomy without significantly altering MTP joint congruity.

BOX 25H-5

AKIN PROCEDURE TECHNIQUE

- Make a medial longitudinal incision over the medial eminence.
- Protect the dorsal and plantar digital nerves within the skin flap.
- Create an L-shaped distally based capsular flap.
- Resect the medial eminence with an oscillating saw starting toward the medial aspect of the sagittal sulcus.
- Make the phalangeal osteotomy in the proximal metaphyseal region (see Fig. 25H-13).
- Cut and remove a small medially based wedge of bone.
- Score the lateral cortex with the saw and close the osteotomy.
- Stabilize the osteotomy with one or two Kirschner wires placed obliquely.
- Repair the medial capsule to the surrounding metatarsophalangeal joint capsule and periosteum.
- If necessary, place a drill hole in the medial metaphyseal cortex to suture the capsule flap to bone.

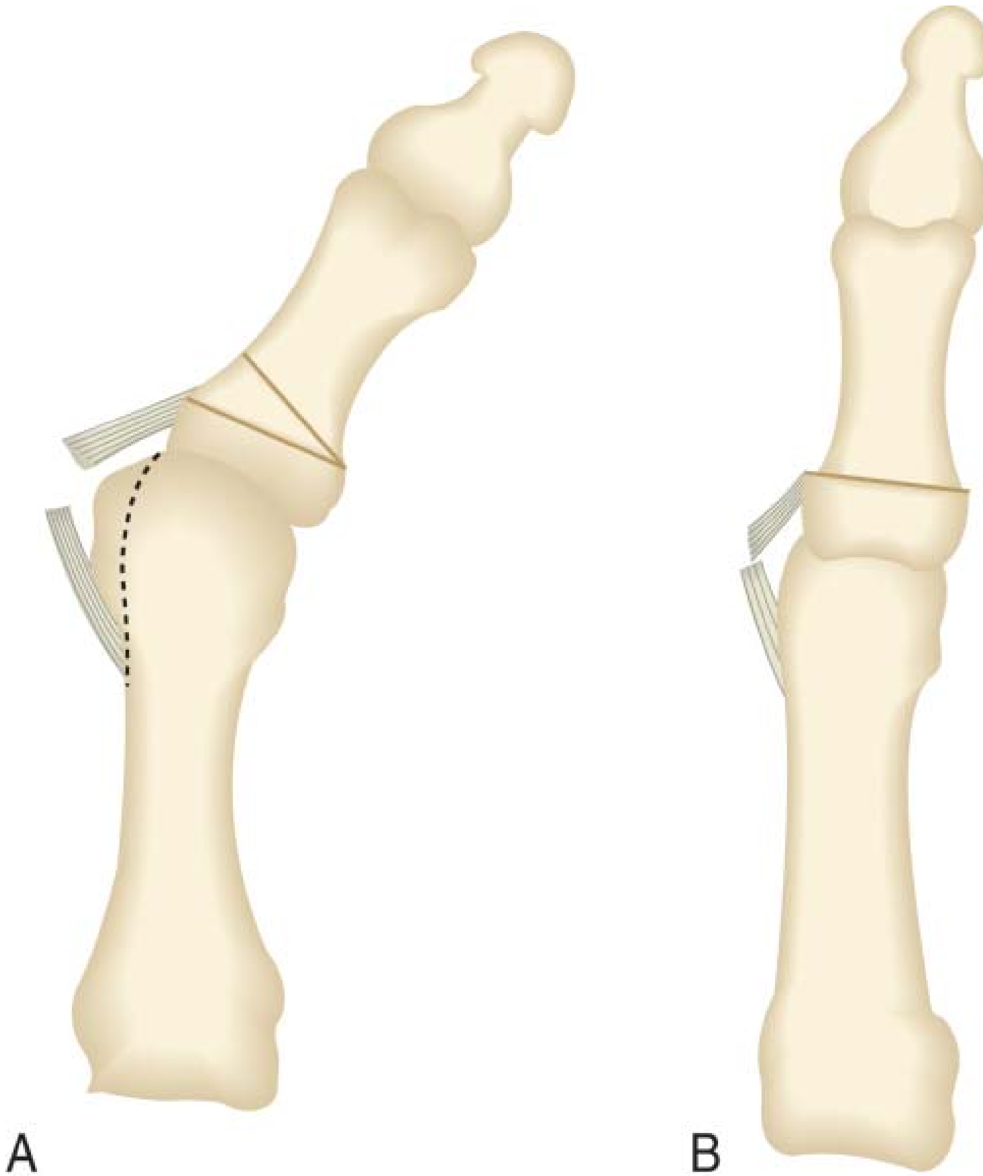

Figure 25H-13 A, Location of osteotomy of proximal phalanx. **B,** After closure of osteotomy site.

Figure 25H-14 **A,** Preoperative radiograph shows hallux valgus interphalangeus deformity. **B,** After Akin phalangeal osteotomy, adequate alignment is maintained.

Chevron Procedure

The chevron osteotomy, as described by Johnson and colleagues [35] and others, [1340] [1366] is indicated for a mild-to-moderate hallux valgus deformity with a hallux valgus angle of less than 30 degrees, an intermetatarsal angle of less than 15 degrees, and no pronation of the hallux. Because the chevron osteotomy achieves an extra-articular correction, it may be used to correct a congruous deformity as well as an incongruous or subluxated MTP joint (Box 25H-6 ; Fig. 25H-15). Neither the lateral capsule nor the conjoined tendon is released. Although release of these structures may allow a greater correction, [36] Mann [37] and others [1368] [1369] have reported several cases of avascular necrosis of the metatarsal head after a chevron procedure (Fig. 25H-16). Although several authors have not found an increased incidence of avascular necrosis after a lateral MTP release, [1365] [1366] [1370] [1371] an extensive lateral release may place a patient at greater risk for avascular necrosis of the metatarsal head. [1372] [1373]

BOX 25H-6

CHEVRON PROCEDURE TECHNIQUE

- Make a medial longitudinal incision over medial eminence from the midportion of the proximal phalanx extending proximally 5 cm (see Fig. 25H-15 A).
- Protect the dorsal and plantar digital nerves within the skin flap.
- Create an L-shaped distally based capsular flap.
- Resect the medial eminence with an oscillating saw starting toward the medial aspect of the sagittal sulcus cutting in line with the medial border of the foot.
- Place a horizontal drill hole in the center of the metatarsal head to mark the apex of the osteotomy.

- Make a horizontal osteotomy from medial to lateral at an angle of approximately 60 degrees (Fig. 25H-15 B).
- Avoid dissection of the lateral aspect of the metatarsophalangeal joint to reduce the risk for avascular necrosis of the first metatarsal head (Fig. 25H-16).
- Displace the osteotomy one third of the metatarsal width (Fig. 25H-15 C, D).
- Stabilize the osteotomy with one 0.062 Kirschner wire placed obliquely.
- Resect the remaining metaphyseal flare.
- Repair the medial capsule to the surrounding metatarsophalangeal capsule and periosteum.
- If necessary, place a drill hole in the medial metaphyseal cortex to suture the capsule flap to bone.

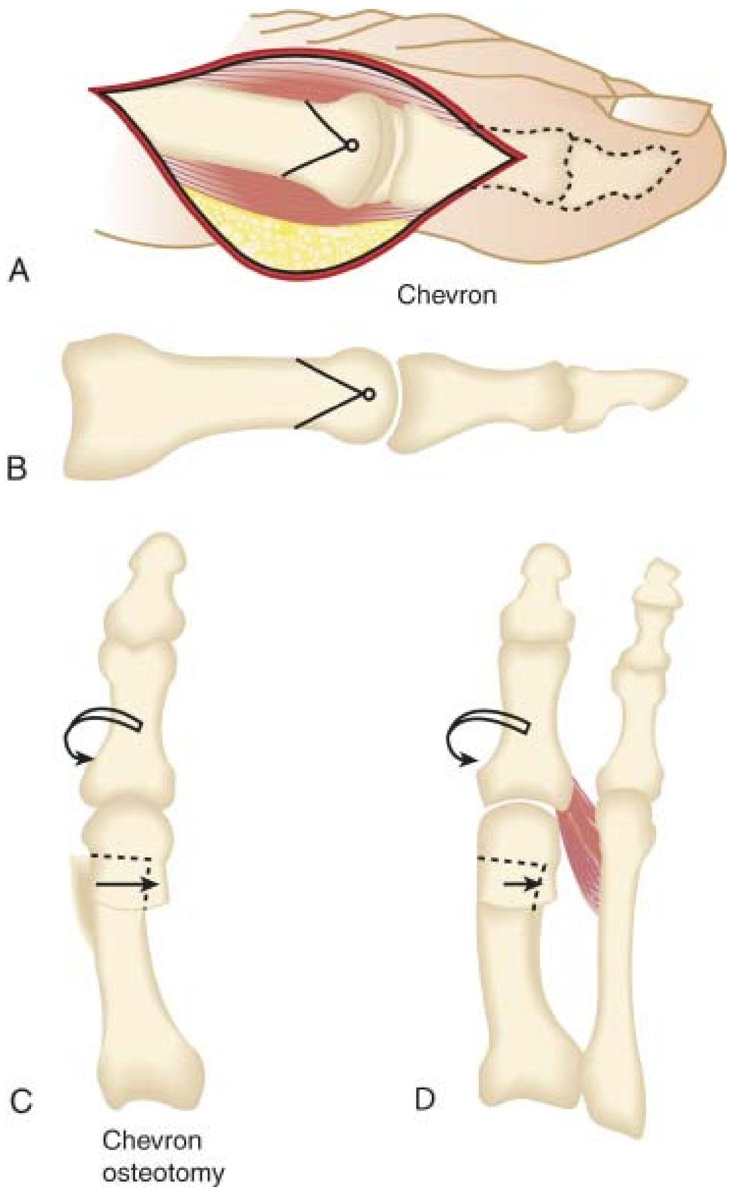

Figure 25H-15 A, Proposed chevron osteotomy of first metatarsal. **B,** A V-shaped osteotomy at about a 60-degree angle based proximally is centered in the metaphyseal region. **C** and **D,** The osteotomy is displaced laterally, and the remaining medial eminence is resected.

Figure 25H-16 A, Postoperative radiograph shows avascular necrosis after a distal metatarsal osteotomy. **B,** Computed tomographic scan shows cystic degeneration consistent with avascular necrosis of the first metatarsal head.

Distal Soft Tissue Realignment

The distal soft tissue procedure has been used to correct mild-to-moderate hallux valgus deformities. [1345] [1374] [1375] The description of this technique as a distal soft tissue realignment is meant to clarify the fact that this is an intra-articular repair that does not use an osteotomy to achieve realignment. Although an osteotomy may be combined with this procedure to achieve an adequate repair, a distal soft tissue repair, when indicated, may be used by itself to correct a mild-to-moderate deformity (Box 25H-7 ; Figs. 25H-17 and 25H-18 [1015] [1020]).

BOX 25H-7

DISTAL SOFT TISSUE REALIGNMENT TECHNIQUE

Lateral Release

- Make a dorsal longitudinal incision in the first intermetatarsal web space about 3 cm in length.
- Retract the first and second metatarsals to expose the tendon of the adductor hallucis.
- Insert the scalpel blade into the interval between the fibular sesamoid plantarly and the metatarsal head dorsally.
- Direct the blade distally along the adductor tendon until it strikes the base of the proximal phalanx.
- Turn the blade laterally against the adductor tendon and release it from the base of the proximal phalanx.
- Bring the knife proximally to dissect the tendon from the lateral sesamoid.
- Reposition the retractors to expose the transverse intermetatarsal ligament.

- Transect the intermetatarsal ligament, taking care not to injure the common digital nerve lying directly below the ligament.
- Perforate the lateral capsule with several puncture incisions.
- Stretch the hallux into varus to tear the remaining lateral capsule.
- Approximate the first and second MTP capsules with three interrupted sutures incorporating the adductor tendon into this repair.
- Tie these sutures after the medial plication is complete.
- Compress the transverse metatarsal arch when tying the sutures to approximate the first and second metatarsal heads.

Medial Eminence Resection and Medial Plication

- Make a medial longitudinal incision directly over the medial eminence (see Fig. 25H-17 A).
- Protect the dorsal and plantar digital nerves within the skin flap.
- Make a vertical capsular incision with a No. 11 blade about 2 to 3 mm proximal to the base of the proximal phalanx.
- Make a second parallel incision more proximal to the first (4 to 8 mm depending on the severity of the deformity).
- Connect the two capsular incisions dorsally by an inverted-V 5-10 mm medial to the extensor hallucis longus tendon.
- Connect the two capsular incisions plantarly by a V, keeping the blade inside the joint to prevent damage to the plantar medial cutaneous nerve.
- Incise the capsule along its dorsomedial aspect to create a proximally and plantarly based flap.
- Resect the medial eminence in line with the medial diaphyseal cortex of the first metatarsal shaft starting about 2 mm medial to the sagittal sulcus (see Fig. 25H-17 B).
- Reef the medial capsule with interrupted sutures holding the hallux in appropriate alignment (neutral varus-valgus and neutral rotation).
- Excise additional capsule if needed to improve the correction.

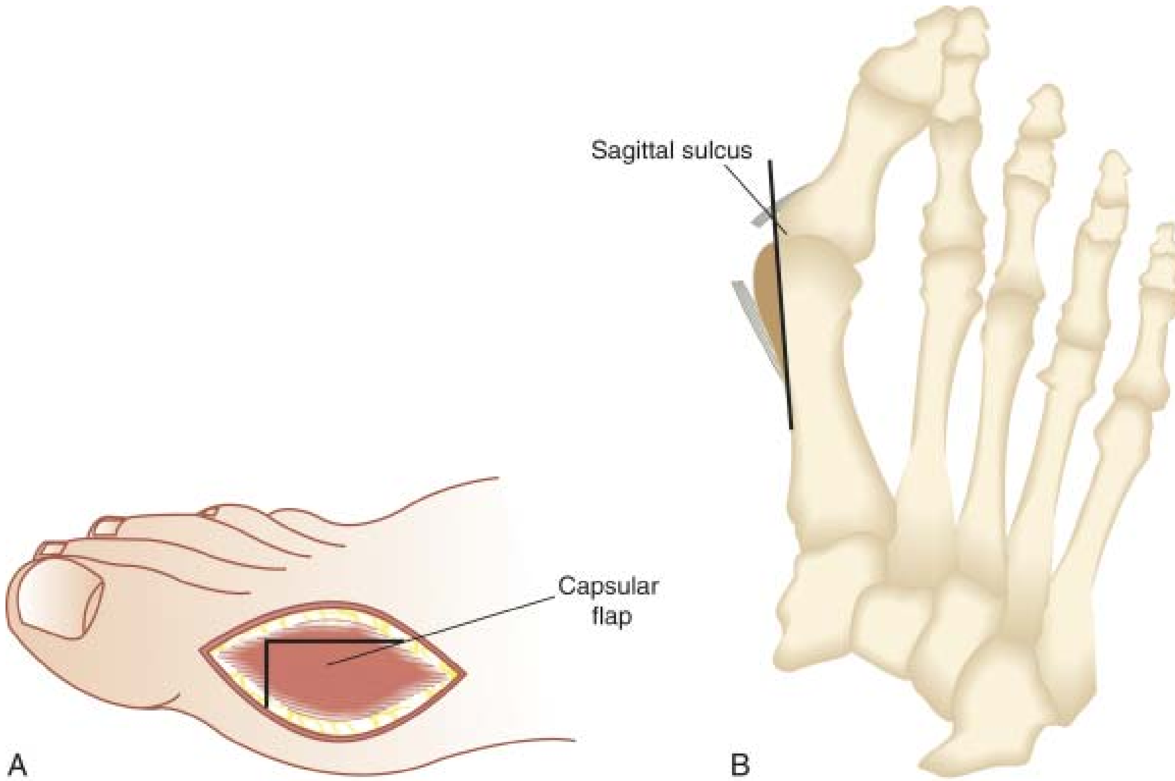

Figure 25H-17 A, A proximally based, L-shaped capsular flap is used to expose the medial eminence. **B,** The medial eminence is resected along a line parallel with the long axis of the medial cortex of the first metatarsal.

Figure 25H-18 **A,** Preoperative radiograph. **B,** Postoperative radiograph after distal soft tissue realignment. *(© M. J. Coughlin. Used by permission.)*

Proximal First Metatarsal Osteotomy

A proximal metatarsal osteotomy that corrects an *increased* 1-2 intermetatarsal angle can be combined with a distal soft tissue reconstruction that realigns the first MTP joint. As a rule, a proximal osteotomy is indicated if the 1-2 intermetatarsal angle is greater than or equal to 15 degrees, if the hallux valgus angle is greater than or equal to 30 to 40 degrees, or if after a distal soft tissue realignment an adequate correction of the 1-2 intermetatarsal angle cannot be obtained. Sometimes a more severe hallux valgus deformity (>40 degrees) with an increased 1-2 intermetatarsal angle can be realigned adequately without an osteotomy; sometimes a moderate hallux valgus deformity (20 to 40 degrees) cannot be corrected adequately without a first metatarsal osteotomy. Although an opening or closing wedge osteotomy can be performed, significant lengthening or shortening of the first ray usually is not desirable. For this reason, a proximal crescentic osteotomy frequently is used. [1331] [1376] [1377] This osteotomy is performed in the proximal first metatarsal metaphysis, an area that provides a broad, stable cancellous surface that allows fairly rapid healing. A distal soft tissue realignment is typically performed first in conjunction with the proximal first metatarsal osteotomy. Intraoperative radiographs are taken to evaluate the correction of the 1-2 intermetatarsal angle and the internal fixation. Internal fixation can be removed easily under local anesthesia 6 weeks after surgery (Box 25H-8 ; Fig. 25H-19).

BOX 25H-8

PROXIMAL FIRST METATARSAL OSTEOTOMY TECHNIQUE

- Make a dorsal 3-cm longitudinal incision over the proximal first metatarsal (see Fig. 25H-19 A).

- Dissect down to the subperiosteal level to expose the proximal metatarsal shaft (along the medial aspect of the extensor hallucis longus tendon).
- Identify the metatarsocuneiform joint.
- Perform the crescentic osteotomy with the concave aspect directed proximally 1 cm distal to the metatarsocuneiform joint (see Fig. 25H-19 B).
- Orient the plane of the osteotomy halfway between the perpendicular plane to the first metatarsal shaft and the floor.
- Displace the osteotomy 2 mm laterally.
- Fix the osteotomy with a single 0.062 Kirschner wire and a single compression screw (see Fig. 25H-19 C, D, and E).
- Bend the Kirschner wire, keeping it subcutaneous after closure.

Figure 25H-19 **A,** For a proximal first metatarsal osteotomy, a dorsal incision is made over the first metatarsal. **B,** A curved saw blade is used to create a crescentic osteotomy. **C,** Preoperative radiograph. **D,** Postoperative radiograph after a first metatarsal osteotomy. **E,** Lateral postoperative radiograph shows internal fixation within the first metatarsal and not crossing the metatarsocuneiform joint. *(© M. J. Coughlin. Used by permission.)*

Combined Multiple First Ray Osteotomies

In the presence of a congruent first MTP joint associated with a hallux valgus deformity, a distal soft tissue realignment is contraindicated because it would create an incongruent MTP joint. [1331] [1332] [1333] An incongruent joint is at risk for later degenerative arthritis or recurrence of deformity. When a hallux valgus deformity occurs in a patient with a congruent first MTP joint, an extra-articular MTP joint correction with periarticular osteotomies (Akin, distal metatarsal, cuneiform) is indicated. The magnitude of the distal metatarsal articular angle determines the necessity of multiple first ray osteotomies.

An Akin phalangeal osteotomy [1331] [1332] [1361] [1378] decreases phalangeal angulation associated with an increased proximal phalangeal articular angle; a first ray osteotomy (proximal first metatarsal osteotomy) [1342] [1379] or cuneiform osteotomy [1332] [1380] may correct an increased intermetatarsal angle; occasionally, an increased distal metatarsal articular angle may necessitate a medial closing wedge distal first metatarsal osteotomy. [1381] [1382] [1383] When a proximal first metatarsal osteotomy is performed in conjunction with a distal first metatarsal osteotomy, care must be taken to avoid excessive soft tissue stripping, which may devascularize the first metatarsal. Alternatively, an opening wedge cuneiform osteotomy may be performed (Fig. 25H-20). Mitchell and Baxter [54] recommended a combined chevron osteotomy and phalangeal osteotomy.

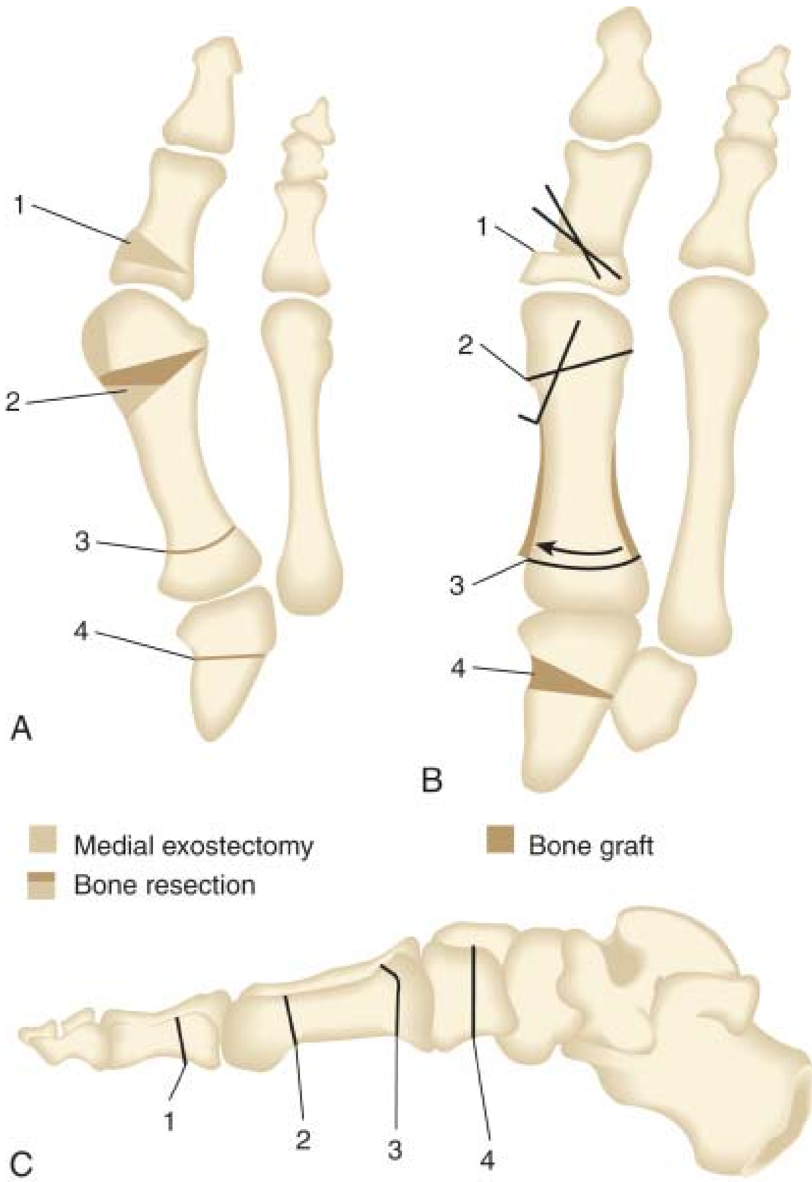

Figure 25H-20 Schematic diagram of double and triple first ray osteotomies. (**A** and **B** are anteroposterior views; **C** is a lateral view.) 1, Phalangeal osteotomy (closing wedge); 2, distal metatarsal osteotomies (closing wedge); 3, crescentic proximal first metatarsal osteotomy; 4, cuneiform osteotomy (opening wedge). *(From Coughlin M, Carlson R: Treatment of hallux valgus with an increased distal metatarsal articular angle: Evaluation of double and triple first ray osteotomies. Foot Ankle Int 20:765, 1999.)*

Richardson and coworkers [23] stated that the average distal metatarsal articular angle in normal feet was 6 degrees. As this angle increases, the magnitude of a congruent hallux valgus deformity increases. [2] Piggott [24] noted that 9% of adults with a hallux valgus deformity had a congruous MTP joint. Coughlin found this to occur in 46% of juveniles with hallux valgus and 37% of adult men with hallux valgus. [55] Coughlin and Carlson [3] reported a 2% incidence of congruent hallux valgus deformities in a large series of adults requiring hallux valgus surgery.

Salvage Procedures

Excisional arthroplasty, [56] placement of MTP joint implants, and MTP arthrodesis all are techniques that must be considered salvage procedures. Although Cleveland and Winant [8] and Jordan and Brodsky [57] found that the Keller procedure produced acceptable results, most authors agree that the procedure is associated with multiple complications. The decreased weight-bearing capacity of the first metatarsal, [58] the increased incidence of metatarsalgia, the cock-up deformity in the hallux, and the decreased strength and loss of stability created by disruption of the plantar aponeurosis and intrinsic musculature of the first ray make it unlikely that the Keller procedure would result in useful improvement for the symptomatic athlete with hallux valgus. As a salvage procedure in older patients, this procedure has merit, but in young and middle-aged athletes, strength and weight-bearing capacity of the first ray are functions that should be preserved if possible.

The use of a silicone hemiarthroplasty or double-stem total joint replacement is contraindicated in an athlete because the decreased weight-bearing capacity of the first ray after joint implantation may lead to lateral transfer metatarsalgia. The increased stress placed on the first MTP joint by a jogger or runner creates the potential for early failure of the implant.

Arthrodesis of the first MTP joint for severe hallux valgus and hallux rigidus is probably the best alternative of all these salvage procedures in an athlete. Arthrodesis leaves the athlete with increased rigidity of the forefoot, which leads to early lift-off [59] with walking and running and to potentially decreased function.

Although these salvage procedures should be part of the surgical armamentarium of an orthopaedic foot surgeon, their applicability in an athletically active individual is limited. The primary procedures discussed earlier have lower postoperative morbidity than these salvage procedures and are preferable when surgery is indicated for an athlete with a symptomatic hallux valgus deformity.

Weighing the Evidence

Akin Procedure

A phalangeal osteotomy as described by Akin [31] produces slight, if any, correction of the 1-2 intermetatarsal angle. [1390] [1391] Plattner and Van Manen [61] reported on a series of 22 patients who had undergone an Akin procedure. An initial correction of the hallux valgus angle of 13 degrees decreased to a correction of only 6 degrees at an average 4.5 years of follow-up. Seelenfreund and associates [32] reported a 16% recurrence (8 of 50 patients), and Goldberg and colleagues [60] reported a 21% recurrence (75 of 351 patients) (Fig. 25H-21). Internal fixation to stabilize a phalangeal osteotomy site is preferred, and nonunion is uncommon. [32] Although Colloff and Weitz [62] used a lateral MTP capsular release, this technique may devascularize the proximal phalangeal fragment and is not recommended. Other complications reported after the Akin procedure include a poor cosmetic appearance [60] and a high level of subjective postoperative patient dissatisfaction. [1390] [1391] Goldberg and associates [60] concluded that a phalangeal osteotomy as an isolated procedure in treatment of a hallux valgus deformity "does not have a sound biomechanical basis and should not be performed as an isolated procedure." Plattner and Van Manen [61] recommended this procedure for hallux valgus interphalangeus and not for a subluxated hallux valgus deformity (see Fig. 25H-14). Mitchell and Baxter [54] and Colloff and Weitz [62] suggested that a phalangeal osteotomy may be used in combination with a proximal repair to gain increased correction.

Figure 25H-21 **A,** Immediate postoperative radiograph shows correction with an Akin phalangeal osteotomy and medial eminence resection. **B,** One year after surgery, a recurrent deformity has developed with an increase in the hallux valgus angle. **C,** Ten years after surgery, further progression of deformity is noted.

Chevron Procedure

Some authors have recommended the chevron procedure for mild-to-moderate hallux valgus deformities, [1340] [1355] [1366] and Lillich and Baxter [18] reported the use of the chevron procedure in two elite female middle-distance and marathon runners. The rationale for using this procedure was that toe-off power could be maintained, and range of motion would not be altered significantly in this extra-articular type of repair. Lillich and Baxter [18] further stated that the stable nature of this osteotomy would make displacement unlikely, and the possibility of a transfer keratotic lesion is avoided.

Johnson and colleagues [35] and others [1366] [1371] [1372] [1393] [1394] [1395] noted a high level of excellent results with the chevron procedure, with a reported average correction of the hallux valgus angle of 12 to 13 degrees and an average correction of the 1-2 intermetatarsal angle of 4 to 5 degrees. Because of the limited correction of the hallux valgus angle offered by the chevron procedure, it should be reserved for mild and low-moderate deformities. Extension of the indications for this procedure to more severe deformities appears to increase the risk for recurrence, patient dissatisfaction, and complications. Meier and Kenzora [39] reported on 50 patients (72 feet) after a distal metatarsal osteotomy and noted a 74% satisfaction rate when the preoperative 1-2 intermetatarsal angle was greater than 12 degrees and a 94% satisfaction rate when the 1-2 intermetatarsal angle was 12 degrees or less. The most frequent complication associated with the chevron procedure is undercorrection or recurrence, which varies from 10% to 14%. [1366] [1393] [1395] Recurrent hallux valgus may develop when the indications for the chevron procedure are expanded to more severe deformities. Loss of correction can occur because of inadequate fixation or slippage at the osteotomy site. Shortening may occur as a result of excessive bone loss. [1366] [1367] [1369] [1363] Klosok and coworkers [40] reported postoperative transfer lesions in 12% of cases.

Distal Soft Tissue Realignment

Although Silver [66] recommended resection of the medial eminence and a lateral soft tissue release, he did not report on the results of this procedure. Later, Kitaoka and associates, [67] in reporting on the Mayo Clinic experience after simple bunionectomy and medial capsulorrhaphy with or without lateral capsulotomy, noted at an average 4.8-year follow-up that the hallux valgus angle had increased 4.8 degrees from the preoperative deformity, and the 1-2 intermetatarsal angle had increased almost 2 degrees. Of the feet that had undergone a bunionectomy without a lateral capsulotomy, 29% underwent reoperation at 5 years. A failure rate of 24% for the entire group was reported. Bonney and Macnab [7] reported generally poor results after simple exostectomy. Of their patients, 37% underwent additional treatment, and the authors concluded that the only indication for a simple bunionectomy is a large medial eminence that is the sole cause of symptoms in a patient whose general medical condition contraindicates an extensive procedure and to whom the postoperative appearance is unimportant.

Meyer and colleagues [68] reported the results of the modified McBride procedure in 21 women joggers who had a symptomatic hallux valgus deformity. These authors reported a successful correction if the preoperative 1-2 intermetatarsal angle was less than or equal to 14 degrees and the hallux valgus angle was less than 50 degrees. They reported an average overall hallux valgus correction of 20 degrees and an average correction of the 1-2 intermetatarsal angle of 4.2 degrees. Of 21 patients, 19 (90%) reported significant improvement postoperatively. Two thirds of the patients resumed jogging 3 months after surgery, and 19 of 21 patients were involved in athletic activities 6 months after surgery.

Mann and Coughlin, [13] in a review of the results of 100 McBride procedures, reported an average correction of the hallux valgus angle of 14.8 degrees and an average correction of the 1-2 intermetatarsal angle of 5.2 degrees. Mann and Coughlin [13] recommended that if more than 20 degrees of correction of the hallux valgus angle was indicated, the procedure should be combined with a first metatarsal osteotomy.

The limitations of a McBride or distal soft tissue reconstruction are substantial. Mann and Pfeffinger [69] observed that a severe hallux valgus deformity was not corrected adequately by a distal soft tissue reconstruction in half of the cases. The indication for this procedure is a subluxated hallux valgus deformity of less than 30 degrees with a 1-2 intermetatarsal angle of less than 15 degrees.

One of the most significant complications of the distal soft tissue procedure is a postoperative hallux varus deformity (Fig. 25H-22). Mann and Coughlin [13] reported hallux varus to occur in 11% of patients, although a severe deformity occurred in only 4%. Mann and Pfeffinger [69] reported a higher incidence of hallux varus deformities after attempted correction of severe deformities.

Figure 25H-22 Hallux valgus deformity (overcorrection) caused by excessive medial eminence resection.

Proximal First Metatarsal Osteotomy

A high satisfaction rate has been reported with the combined procedure of distal soft tissue reconstruction and proximal first metatarsal osteotomy (78% to 93%). [1400] [1401] [1402] [1403] [1404] [1405] The average correction of the hallux valgus angle has been reported consistently to be 23 to 24 degrees. [1332] [1336] [1401] [1405] The magnitude of improvement achieved is directly proportional to the severity of the preoperative hallux valgus deformity. A crescentic osteotomy is preferred [1331] [1377] because it results in minimal shortening of the first metatarsal. Lengthening of the first metatarsal with an opening wedge osteotomy may lead to instability at the osteotomy site, and a closing wedge osteotomy may lead to lateral metatarsalgia as a result of first ray shortening.

Complications associated with this procedure include overcorrection or hallux varus, undercorrection or recurrence, lateral metatarsalgia, and delayed union or malunion. Overcorrection often has been associated with lateral sesamoidectomy, and Simmonds [74] and Mann and Coughlin [13] have recommended that a lateral sesamoidectomy should be avoided. Retention of the lateral sesamoid has decreased the prevalence of hallux varus in one series from 11% [13] to 8%. [69] Often hallux varus deformities less than 10 degrees are asymptomatic and are associated with a satisfactory result.

Combined Multiple First Ray Osteotomies

Funk and Wells [51] and others [1342] [1378] [1382] [1383] reported success with distal metatarsal osteotomies and double osteotomies. Funk and Wells [51] reported an average correction of the 1-2 intermetatarsal angle of 7.2 degrees. Coughlin and Carlson [3] reported on the use of double and triple osteotomies in the treatment of adolescent hallux valgus deformities in 18 patients (21 feet) with a hallux valgus deformity characterized by an increased distal metatarsal articular angle, who underwent either double or triple first ray periarticular osteotomies. The average hallux valgus correction measured 23 degrees, and the average 1-2 intermetatarsal angle correction was 9 degrees. The distal metatarsal articular angle averaged 23 degrees preoperatively and was corrected to an average of 9 degrees postoperatively. Peterson and Newman [49] reported a similar correction of the hallux valgus angle and the 1-2 intermetatarsal angle.

Complications associated with multiple metatarsal osteotomies include loss of correction, loss of fixation, malunion, avascular necrosis, and degenerative arthritis of either the interphalangeal or MTP joint. These techniques are difficult and should be reserved for the occasional case of a hallux valgus deformity associated with a congruent joint with an increased distal metatarsal articular angle of greater than 15 degrees (Fig. 25H-23).

AUTHOR'S PREFERRED METHOD

In general, it is preferable to treat most high-level athletes symptomatically until their desire for high-level competition wanes. If the hallux valgus deformity continues to be symptomatic, surgery can be performed at that time. When surgery is contemplated, careful analysis of the components of the hallux valgus deformity is essential in the decision-making process to ensure the appropriate choice of hallux valgus repair (Box 25H-9).

For a mild hallux deformity (0 to 20 degrees), usually conservative care suffices. An Akin procedure or a chevron repair is used when indicated. Because these two repairs are extra-articular, they can be carried out in a patient with a congruent (increased distal metatarsal articular angle) or subluxated MTP joint. For a low-moderate deformity (a hallux valgus angle of 20 to 30 degrees), a chevron procedure or a distal soft tissue realignment is the procedure of choice. The chevron procedure may be used to correct a congruent or subluxated MTP joint, but distal soft tissue realignment can be used only in a patient with a noncongruent or subluxated joint.

For a high-moderate deformity (a hallux valgus angle of 30 to 40 degrees), distal soft tissue repair with or without a proximal first metatarsal osteotomy appears to offer the best chance for successful hallux valgus repair in the presence of a subluxated first MTP joint. For a congruent hallux valgus deformity, periarticular osteotomies are indicated in the presence of an increased distal metatarsal articular angle. An intra-articular correction of a congruent MTP joint with hallux valgus places the repair at high risk for recurrence, degenerative joint disease, or at least restricted range of motion. Adding an osteotomy increases the postoperative morbidity and recovery time in an athlete. When a more severe hallux valgus deformity is present, the magnitude of the surgical procedure increases, and postoperative swelling, reduced MTP joint range of motion, and reduced strength become more likely. For these reasons, patient counseling is especially advocated in this group. A change in athletic activity, substituting activities such as bicycling and other nonimpact activities for running, may reduce symptoms significantly in an athlete with hallux valgus.

BOX 25H-9

PRIMARY GOALS OF HALLUX VALGUS CORRECTION SURGERY

- Maintain a flexible metatarsophalangeal joint with as normal range of motion as possible.
- Restore normal weight-bearing pattern in the forefoot.
- Correct the deformity without producing residual disability.
- Allow a reasonable route of salvage if a complication develops.

Figure 25H-23 **A,** Preoperative radiograph shows hallux valgus angle of 33 degrees, intermetatarsal angle of 18 degrees, and distal metatarsal articular angle (DMAA) of 25 degrees. **B,** Postoperative radiograph shows proximal crescentic first metatarsal osteotomy, distal first metatarsal closing wedge osteotomy, and phalangeal closing wedge osteotomy. **C,** Lateral radiograph shows periarticular osteotomies. It is likely that the screw and pin did cross the metatarsocuneiform joint. Hardware typically is removed 6 weeks after surgery, as shown in **D,** Radiograph at 3 years' follow-up shows hallux valgus angle of 16 degrees, intermetatarsal angle of 13 degrees, and DMAA of 11 degrees. *(From Coughlin M, Carlson R: Treatment of hallux valgus with an increased distal metatarsal articular angle: Evaluation of double and triple first ray osteotomies. Foot Ankle Int 20:767, 1999.)*

Postoperative Prescription, Outcomes Measurement, and Potential Complications

A soft compression dressing is applied at surgery, holding the toe in appropriate alignment, and is changed weekly in the first 6 to 8 weeks for most hallux valgus correction procedures. A postoperative shoe is appropriate for most realignment procedures for the first 6 weeks. When multiple first ray osteotomies are performed, a short leg cast is recommended. A cast may also be used in the case of a proximal first metatarsal osteotomy, but is not required. Weight-bearing is allowed mostly on the heel in the first 6 weeks after surgery. Non–weight-bearing in the cast is preferred for multiple first ray osteotomies. Kirschner wires used to fix Akin or chevron osteotomies are removed 4 to 6 weeks after surgery.

At 6 weeks, the patient is transitioned to a stiff-soled sandal and then gradually into a supportive athletic shoe as pain and swelling allow. Range of motion exercises are started between 3 and 6 weeks. Aggressive walking activity can be started 6 weeks after surgery if the patient is progressing well. A comfortable, supportive athletic shoe with a roomy toe box and a spacer between the first and second toes protects the athlete as he or she returns to activities. Activity is increased as tolerated, with expected return to jogging at 2 to 3 months, running at 3 to 4 months, and full return to sport at 3 to 6 months. Distal soft tissue realignment requires a longer return to full activity to maintain the alignment of the hallux. Full healing of the proximal first metatarsal osteotomy or multiple first ray osteotomies occurs at about 6 to 8 weeks. Advancing range of motion and more aggressive walking should be delayed until osteotomy sites are completely healed. The most common complication following hallux valgus surgery is undercorrection or recurrence of the deformity (Box 25H-10).

BOX 25H-10

COMPLICATIONS AFTER HALLUX VALGUS SURGERY

- Undercorrection or recurrent deformity
- Hallux varus
- Malunion or nonunion of the osteotomy site
- Avascular necrosis of the metatarsal head
- Lateral metatarsalgia or transfer lesions
- Degenerative arthritis in the hallux metatarsophalangeal joint or interphalangeal joint

Criteria for Return to Play

With any hallux valgus correction, return to play occurs when full healing of osteotomies is noted on radiographs and the athlete regains adequate range of motion, strength, and endurance to return to sport. Lillich and Baxter [18] stressed the importance of a precise operative technique with minimal joint dissection. They allowed running 7 weeks after surgery in a loose-fitting shoe with a toe spacer placed between the hallux and second toe. Running speed and duration were increased gradually, with a return to full activity achieved at 12 weeks after surgery. In many cases, full return to sport may not occur until 6 months after surgery (Box 25H-11).

BOX 25H-11

CRITERIA FOR RETURN TO PLAY AFTER HALLUX VALGUS SURGERY

- Full healing of osteotomies on radiographs
- Adequate range of motion, strength and endurance
- Running 7 weeks postoperatively in a loose-fitting shoe with a toe spacer placed between the hallux and second toe
- Full return to sport 3 to 6 months postoperatively

Special Populations

A symptomatic hallux valgus deformity presents a special problem to the treating physician. Although, in general, conservative methods should be used in all athletes, a high-performance athlete requires more deliberate treatment. Hamilton (quoted by Lillich and Baxter [18]) stated that bunionectomies should be avoided in sprinters and ballet dancers. Restricted dorsiflexion and plantar flexion may interfere with a dancer's functional ability. Likewise, the need for dorsiflexion as well as strength in push-off makes sprinters poor candidates for a hallux valgus repair. Middle- and long-distance runners appear to have less need for excessive dorsiflexion and push-off than sprinters. It is wise to treat most high-level athletes symptomatically until their desire for high-level competition wanes; if the hallux valgus deformity continues to be symptomatic, surgery can be performed at that time.

TURF TOE

MTP joint injuries are commonly the result of a hyperextension injury and have become an increasing source of forefoot pain and dysfunction in professional athletes. [1406] [1407] [1408] [1409] [1410] Occasionally, these injuries are characterized by a significant delay in return to sporting activities. [76] Bowers and Martin [81] initially described a syndrome in which "a sprain of the plantar capsuloligamentous complex of the great toe of the MTP joint" occurred. Bowers and Martin [81] reported on 27 such injuries of the foot in football players and coined the term *turf toe* because of the frequency with which the injury occurred on artificial playing surfaces as opposed to natural grass. Clanton and associates [76] reported on 62 MTP joint injuries in 53 collegiate football players, all of whom sustained injuries on artificial playing surfaces.

Severe trauma to the MTP joint of the hallux is relatively uncommon. Jahss, [82] Giannikas and coworkers, [83] DeLee, [84] and Rodeo and colleagues [85] reported on fracture-dislocations and dislocation of the first MTP joint after hyperextension injuries. Usually these injuries are associated with a significant dissipation of energy, and sesamoid fractures may occur. Hyperextension injuries [1407] [1411] [1416] [1417] to the hallux and lesser MTP joints have been linked to flexible shoewear [88] and artificial playing surfaces [1407] [1411] [1416] [1417] and in certain cases may have long-term sequelae for the symptomatic athlete.

Anatomy and Biomechanics

The first MTP joint is a relatively shallow articulation between the convex metatarsal head and concave base of the proximal phalanx. [89] Joint stability is primarily provided by the capsuloligamentous structures and enhanced by musculotendinous and bony structures that surround the joint (Box 25H-12). The sesamoids lie within the flexor hallucis brevis tendon (Fig. 25H-4 **A**) and articulate with the medial and lateral plantar facets of the first metatarsal head (see Fig. 25H-4 **B**). The tendons of the abductor hallucis and adductor hallucis contribute to the joint capsule medially and laterally and insert on medial and lateral borders of the sesamoids as well as onto the base of the proximal phalanx. These structures provide plantar stability along with the plantar plate, a thick fibrous structure extending from a loose attachment to the metatarsal neck to a firm attachment to the base of the proximal phalanx. The medial and lateral collateral ligaments interdigitate with the sesamoid ligaments (Fig. 25H-2 **A**) and contribute to the medial and lateral stability of the MTP joint. Dorsally, the extensor hallucis longus, extensor hallucis brevis, and hood ligaments (see Fig. 25H-2 **B**) of the extensor expansion form the major capsular stabilizing structures. However, the dorsal structures are weak relative to the plantar supporting structures.

BOX 25H-12

STABILIZING STRUCTURES OF THE FIRST METATARSOPHALANGEAL JOINT

Joint Capsule

Ligaments

- Plantar plate
- Medial and lateral collateral ligaments
- Sesamoid ligaments

Tendons

- Flexor hallucis brevis
- Adductor hallucis
- Abductor hallucis

Bone

- Medial and lateral sesamoids

Motion at the MTP joint occurs by means of a sliding action along the joint surface. Joseph [90] reported that normal active extension of the MTP joint approximates 80 degrees (Fig. 25H-24). Clanton and coworkers [1406] [1421] [1422] and others [81] reported that an average of 60 degrees of dorsiflexion can be expected with normal gait. As the phalanx approaches the upper range of dorsiflexion excursion, this sliding action is replaced by axial compression forces on the articular surfaces of both the metatarsal head and base of the proximal phalanx, which can lead to joint injury. Stiff sole shoes diminish MTP dorsiflexion by up to 30 degrees without causing any noticeable impairment of gait (Fig. 25H-25).

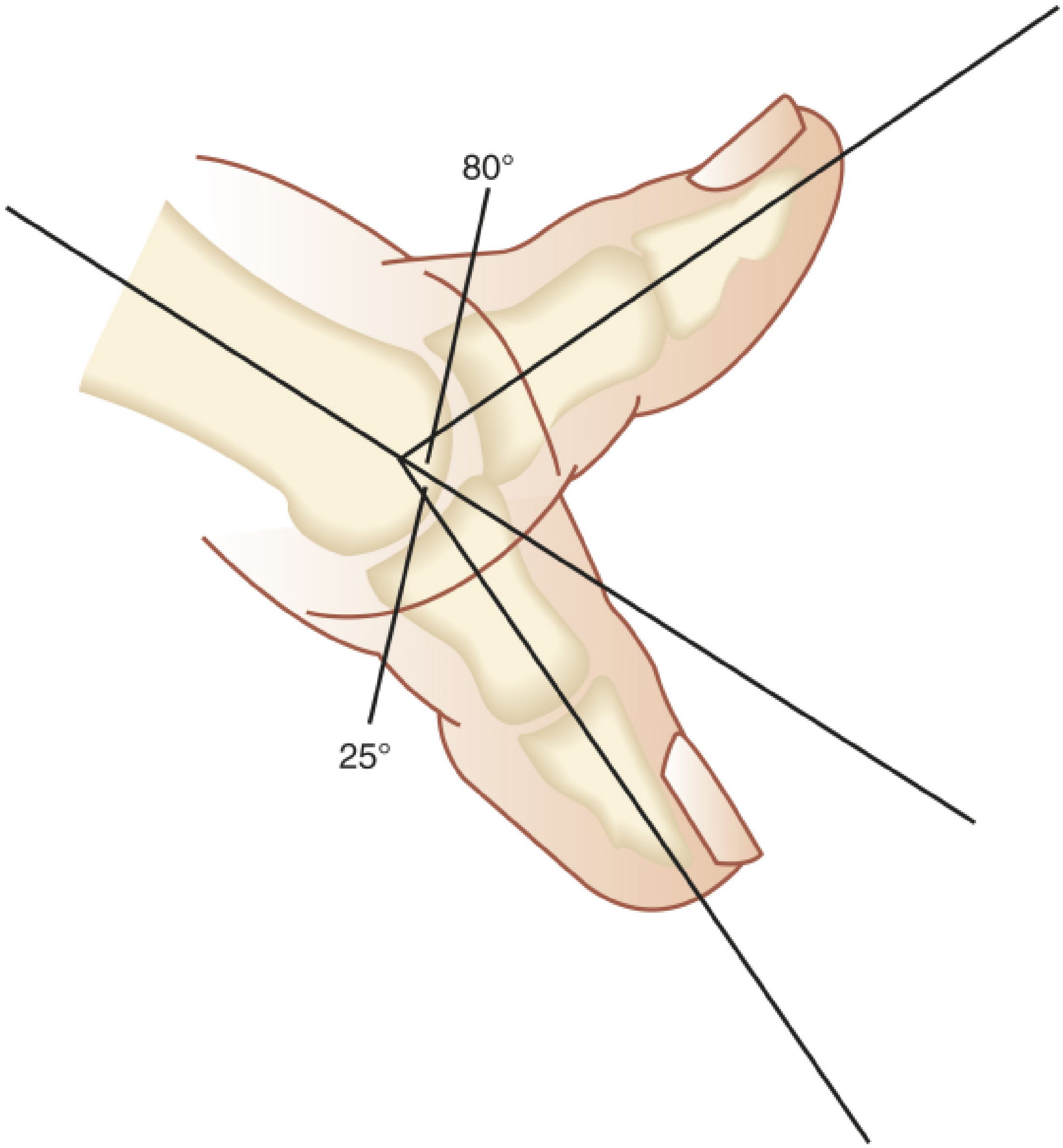

Figure 25H-24 Normal active extension of the first metatarsophalangeal joint averages 80 degrees. Active plantar flexion averages 20 to 25 degrees. This excursion may decrease with advancing age.

Figure 25H-25 A shoe with a flexible insole affords little protection for a hyperextension injury. *(From Clanton TO, Butler JE, Eggert A: Injuries to the metatarsophalangeal joints in athletes. Foot Ankle 7:162-176, 1986. © American Orthopaedic Foot and Ankle Society, 1986.)*

The primary mechanism of injury is forced hyperextension of the hallux MTP joint, [1406] [1409] [1411] [1421] often with an axial load to the heel. [77] The resulting injury to the plantar structures of the joint ranges from a mild sprain to frank dislocation of the joint. The plantar plate frequently tears at the weakest point, the neck of the first metatarsal. However, the plantar structures can also be disrupted distal to the sesamoids, through a bipartite sesamoid or with sesamoid fracture. With further dorsiflexion of the joint, an impaction injury to the joint surface can occur. A valgus force can be associated with the injury causing disruption of the plantar medial capsuloligamentous structures or the tibial sesamoid. Less frequently, a first MTP joint sprain is the result of a hyperflexion injury. [77]

Classification

Rodeo and coworkers [85] proposed a classification of first MTP joint injury (Box 25H-13). Clanton [1406] [1421] [1422] proposed a classification for more acute injuries (Table 25H-2).

BOX 25H-13

CLASSIFICATION OF FIRST METATARSOPHALANGEAL JOINT INJURIES

Grade 1: Acute sprain of the first metatarsophalangeal (MTP) joint plantar capsule

- Localized tenderness, swelling, and pain with dorsiflexion

- Normal radiographs
- Conservative treatment

Grade 2: Acute sprain of the first MTP joint with significant plantar capsule disruption

- Ecchymosis, painful dorsiflexion, and loss of motion.
- Diastasis of a partite sesamoid or joint instability on radiographs
- No degenerative first MTP joint changes
- Conservative or surgical treatment

Grade 3: Chronic symptoms involving the first MTP joint resulting from previous injury

- Loss of motion
- Degenerative joint disease, hallux rigidus, or malalignment on radiograph
- Treatment is often surgical

TABLE 25H-2 -- Acute First Metatarsophalangeal Joint Injury

Grade	Objective Findings	Activity Level	Pathology	Treatment
1	Localized plantar or medial tenderness Minimal swelling No ecchymosis	Continued athletic participation	Stretching of the capsuloligamentous complex	Symptomatic
2	More diffuse and intense tenderness Mild to moderate swelling Mild to moderate ecchymosis Painful and restricted range of motion	Loss of playing time for 3-14 days	Partial tear of the capsuloligamentous complex	Walking boot and crutches as needed
3	Severe and diffuse tenderness Marked swelling Moderate to severe ecchymosis Range of motion painful and limited	Loss of playing time for 2-6 weeks	Tear of the capsuloligamentous complex Articular cartilage and subchondral bone injury Possibility of sesamoid fracture or separation of bipartite sesamoid Possibility of dislocated first metatarsophalangeal joint with spontaneous reduction	Long-term immobilization in boot or cast versus surgical repair

Evaluation

Clinical Presentation and History

Both clinical history and physical examination are directed toward identifying the severity and location of the injury. The history elicited from the athlete gives some indication of the mechanism of the injury. Players may complain of localized pain, swelling, and pain with range of motion and ambulation. The patient history should also include the type of footwear used and the playing surface involved.

Physical Examination

On physical examination, periarticular swelling and ecchymosis are typically present. Mild injuries present with plantar or plantar medial tenderness. Dorsal tenderness is elicited in more severe capsular disruption. In the most severe injuries, fracture-dislocation of the joint can occur with obvious joint deformity. A palpable plantar defect may be noted, indicating a partial or complete disruption of the plantar plate–sesamoid mechanism. [93]

The athlete may present with an antalgic gait, attempting to walk with the limb in external rotation or the foot everted to minimize MTP joint excursion. Push-off is impaired with running, and it may be difficult to crouch with the MTP joint extended. [81] Range of motion and maneuvers to load the joint will also elicit discomfort on physical examination. [81] Decreases in range of motion can be observed in both dorsiflexion and plantar flexion. Weakness of the MTP joint in plantar flexion may be observed (Box 25H-14).

BOX 25H-14

TYPICAL FINDINGS IN TURF TOE INJURIES

Physical Examination

- First metatarsophalangeal (MTP) joint swelling
- Ecchymosis adjacent to the area of capsular injury
- Plantar tenderness at the MTP joint
- Pain with passive MTP joint dorsiflexion
- Pain with joint loading: walking, push-off, crouching with the MTP joint extended
- Decreased dorsiflexion of the MTP joint

Radiographic Examination

- Soft tissue swelling
- Small periarticular bony avulsions
- Intra-articular loose bodies
- Diastasis of bipartite sesamoid
- Sesamoid fracture
- Migration of sesamoids

Imaging

Weight-bearing anteroposterior and lateral radiographs, as well as oblique views, are helpful in evaluating first MTP dorsiflexion injuries. Bilateral weight-bearing anteroposterior views are helpful to identify proximal migration of the sesamoids. A forced dorsiflexion lateral view assists in the diagnosis of distal sesamoid migration and diastasis of a bipartite or fractured sesamoid. In general, initial radiographs typically reveal only soft tissue swelling. [1406] [1411] [1422] However, radiographs should be reviewed thoroughly for more subtle findings associated with these injuries (see Box 25H-14 ; Fig. 25H-26 **A**). When a compression fracture of the metatarsal head has occurred, intra-articular loose bodies may be apparent (see Fig. 25H-26 **B**). Chondrolysis of the joint space can occur with time, and Clanton and colleagues [76] have also reported cystic changes in the metatarsal head denoting progressive degenerative changes.

Figure 25H-26 **A,** A fleck of bone may indicate ligamentous or capsular disruption. **B,** Intra-articular loose bodies (*arrow*) may occur after turf toe injury. **C,** A bone scan may show increased uptake even when radiographs still appear normal. *(From Clanton TO, Butler JE, Eggert A: Injuries to the metatarsophalangeal joint in athletes. Foot Ankle 7:162-176, 1986. © American Orthopaedic Foot and Ankle Society, 1986.)*

Bone scan can be helpful in distinguishing a bipartite sesamoid from an acute fracture (Fig. 25H-26 **C**). Magnetic resonance imaging (MRI) can be used to evaluate the presence and extent of capsular or plantar plate disruption. In addition, MRI will reveal osseous or articular damage in the presence of normal radiographs (Fig. 25H-27). [94]

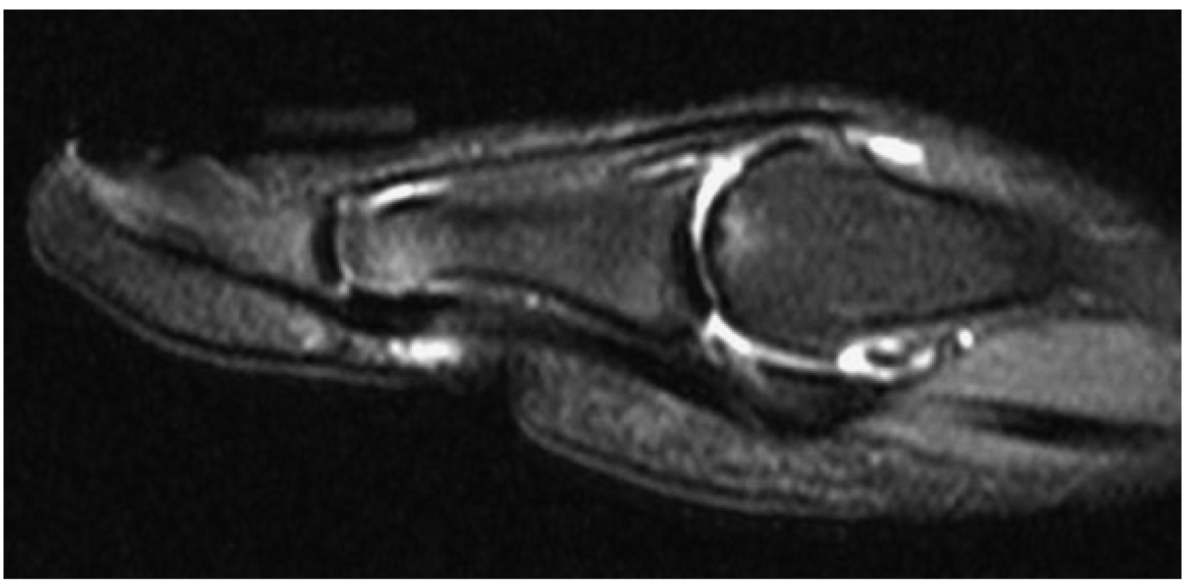

Figure 25H-27 Magnetic resonance image of the first metatarsophalangeal joint may reveal osseous or articular cartilage abnormalities.

Treatment Options

Nonoperative

For grade 1 injuries, ice, compression, and nonsteroidal anti-inflammatory drugs may be used. [1406] [1411] [1421] [1422] The patient can continue to participate in athletics if pain is minimal. Taping of the toe helps compress the joint and limit motion. Inserting a rigid forefoot insole [1406] [1411] [1421] [1422] to stiffen the shoe may decrease discomfort and help to reduce the incidence of recurrent injury.

Similar nonoperative methods are employed for grade 2 injuries. However, activity should be restricted in athletes with more severe injuries and significant discomfort. Addition of a firm insole prevents dorsiflexion strain to the toe (Fig. 25H-28). Typically, refraining from athletic activity for 1 to 2 weeks allows significant improvement.

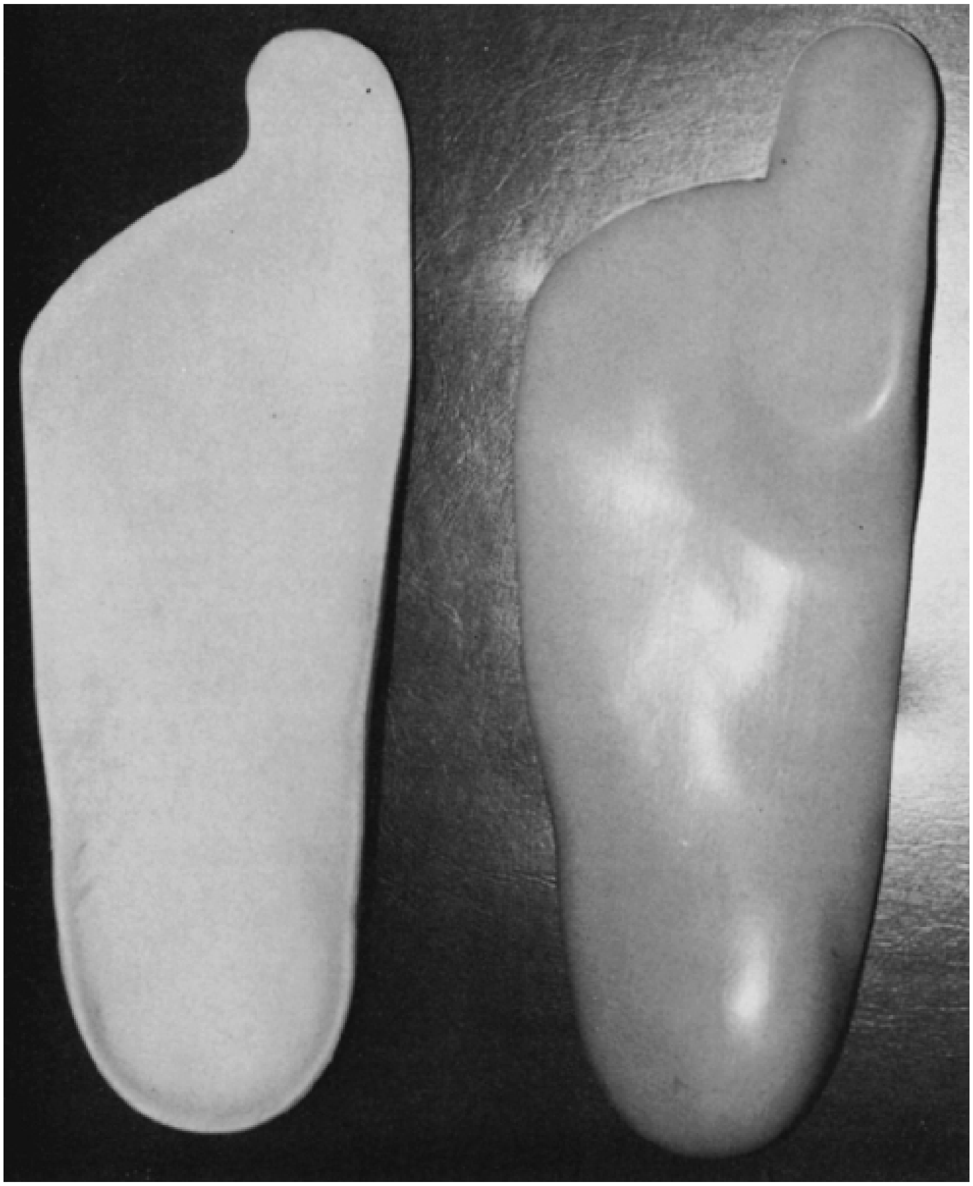

Figure 25H-28 Use of a firm insole that restricts forefoot motion may prevent injury and may diminish symptoms after injury. *(From Clanton TO, Butler JE, Eggert A: Injuries to the metatarsophalangeal joints in athletes. Foot Ankle 7:162-176, 1986. © American Orthopaedic Foot and Ankle Society, 1986.)*

For grade 3 injuries, immobilization and a period of restricted weight-bearing is usually required in addition to ice, elevation, and compression. Weight-bearing is initiated within the limits of pain. Mobilizing the joint as the patient is able to tolerate assists in achieving more normal range of motion. The athlete may progress weight-bearing when gait becomes less painful and gradually increase speed and activity level. Cutting maneuvers are initiated last within the limits of pain. Gradual return to sporting activities depends on the resolution of symptoms. [1406] [1421] [1422] Intra-articular injections of steroids disguise symptoms or give temporary relief only and are generally discouraged. [1406] [1407] [1421] [1422]

When joint dislocation is present (Fig. 25H-29), prompt reduction should be performed. Reduction is performed by hyperextending the joint with concurrent longitudinal traction and plantar pressure on the base of the proximal phalanx. If the joint is irreducible, operative intervention is required (Box 25H-15). [84]

Figure 25H-29 A-D, Fracture-dislocation of the metatarsophalangeal-sesamoid joint (grade 3 injury).

BOX 25H-15

TREATMENT OPTIONS FOR FIRST METATARSOPHALANGEAL JOINT INJURIES

Nonoperative

- Immobilization
- Restricted weight-bearing
- Taping of the toe

- Rigid forefoot insole

Operative

- Plantar plate and capsular repair
- Partial or full sesamoid excision
- Joint débridement
- Correction of traumatic hallux valgus
- Cheilectomy
- First metatarsophalangeal arthrodesis

Operative

Operative treatment is considered in more severe injuries with joint instability, diastasis of bipartite sesamoid or sesamoid fracture, retraction of sesamoids, traumatic hallux valgus, or presence of a loose body or chondral injury. Operative intervention is rarely necessary in acute injuries; management is usually centered on conservative methods. After defining a capsular injury, an open repair occasionally is performed, especially in the presence of a partial or complete plantar plate injury.[77] Coker and associates[77] recommended operative repair for these injuries. When a sesamoid injury (fracture, osteonecrosis) occurred and did not heal successfully, Coughlin[93] believed that surgical excision may be warranted. Open débridement of the joint may be required when intra-articular injury occurs. If hallux rigidus develops, cheilectomy or arthrodesis is considered (see Box 25H-15).

With a progressive hallux valgus deformity, it is likely that disruption of the medial or plantar medial capsule has occurred. Conservative treatment in the competitive athlete is warranted initially; however, with progression of pain and deformity, surgical repair is often necessary. Roomy footwear and the use of orthotic devices are preferred to surgical intervention if the progression of the deformity is insidious and asymptomatic. Operative treatment of hallux valgus in the competitive athlete can be associated with restricted range of motion, which may limit running activity significantly.

Weighing the Evidence

Although there are few documented studies of athletes with MTP joint injuries, Rodeo and colleagues[79] evaluated professional football players. Of players surveyed, 57% (38 players) reported symptoms of turf toe. Although this incidence was comparable to that seen in players from other teams whose home field was a natural grass surface, 84% of the players in this series reported that their initial injury occurred on artificial turf. Other factors that predispose an individual to injury include increasing age of the player, number of years in professional football,[78] increased range of ankle dorsiflexion,[78] pes planus, and decreased preinjury MTP range of motion.[76]

The classic mechanism of injury is a forced hyperextension injury to the first MTP joint. [1406] [1409] [1411] [1421] Coker and associates[77] described this as an axial loading of the foot with the MTP joint in hyperextension. Dorsiflexion in excess of a normal range of motion can lead to varying degrees of soft tissue capsular disruption, plantar plate rupture, or injury to the articular cartilage and subchondral bone. Only 35 of 51 patients in Clanton's series could recall the mechanism of injury[76]; however, 32 of the 35 patients (92%) could recall and describe a hyperextension injury. Rodeo and associates[79] reported that 85% of the football players in their series sustained a hyperextension injury. Coker and associates[77] reported that the mechanism of injury could be hyperextension, hyperflexion (which is rare), or valgus, depending on the applied stress. Massari and coworkers[95] described a rare case of varus injury of the first MTP joint. Clanton and colleagues[76] hypothesized that more severe MTP joint injuries occur in players who have preexisting restricted motion of the first MTP joint. With continued forced dorsiflexion, capsular, ligamentous, or tendinous injury and axial compression of the joint surfaces occur sooner than they would in a joint with more excursion.

Although Garrick[88] noted an increased injury rate in players playing on artificial surfaces, Bowers and Martin[81] found a correlation between flexible shoes and a relatively hard artificial playing surface. The use of relatively lightweight, flexible shoes on artificial turf apparently predisposes the forefoot to injury. [1407] [1416] [1417] In contrast, on grass surfaces, the use of a conventional cleated shoe, which is much stiffer (a steel plate is incorporated into the sole for attachment of the cleat), limits forefoot excursion.[76] Clanton and colleagues[76] observed that the rate of injury on artificial turf could be reduced markedly by inserting a steel forefoot insole inside the players' shoes.

Depending on the magnitude of force, the position of the MTP joint, and the direction of force, various injuries to the MTP joint can occur. Although most often a hyperextension injury leads to symptoms of turf toe, Coker and associates [77] and Clanton and colleagues [76] noted that occasionally a plantar flexion injury is associated with a turf toe injury. Valgus stress can occur as well, either alone or in combination with other forces, leading to different injury patterns.

AUTHOR'S PREFERRED METHOD

Preseason physical examination helps identify which players are at high risk for turf toe. Players with less than 60 degrees of dorsiflexion at the MTP joint or with increased dorsiflexion at the ankle joint should be protected with a stiff-soled shoe or a stiff insole. These modalities may prevent MTP joint injury or reduce the magnitude of injury should it occur. An MRI scan should be obtained when there is suspicion of significant injury. In the high-performance athlete, open repair of partial and complete capsular, plantar plate, or sesamoid disruption may be indicated.

The use of appropriate footwear provides a great opportunity to reduce the incidence of first MTP joint injury in the athlete. Although turf toe can be a disabling injury that can bring about the premature conclusion of an athletic career, prophylactic shoewear modification and counseling athletes at risk can reduce the frequency and magnitude of this injury.

In the athlete with an acute grade 1 turf toe injury, ice, compression, nonsteroidal anti-inflammatory drugs (NSAIDs), and taping of the toe make continued sports participation possible. For more severe injuries, more aggressive physical therapy is necessary. In patients with grade 2 injuries, a firm forefoot insole is helpful to prevent further injury to the MTP joint of the great toe. Usually it is necessary to refrain from athletic activities for 1 to 2 weeks. With more severe injuries, such as grade 3 turf toe, an athlete may be unable to return to sports for the remainder of the season. Surgical intervention is considered in the presence of a partial or complete plantar plate disruption. This injury can occur with a dislocation or subluxation of the MTP joint or a severe articular or nonarticular injury.

Postoperative Prescription, Outcomes Measurement, and Potential Complications

Postoperative management must balance the soft tissue healing with early range of motion of the MTP joint. [96] Immobilization in a removable splint with restricted weight-bearing continues for 4 weeks. Seven to 10 days after surgery, passive range of motion is started. At 4 weeks, weight-bearing and active range of motion are initiated. Modified shoewear is allowed at 8 weeks with return to full sport at 3 to 4 months. After return to sport, the athlete continues shoe modifications to protect from excessive dorsiflexion.

After a turf toe injury, athletes can experience persistent pain with toe-off and stiffness of the first MTP joint. Degenerative joint disease can cause pain and restricted range of motion. Progressive hallux valgus deformity develops in cases of medial capsule injury. The development of chronic pain, restricted motion, and discomfort with running may herald the end of an athletic career.

Criteria for Return to Play

Return to sport in first MTP joint injuries is gradual and dependent on the resolution of symptoms. Shoe modifications with a stiff forefoot insole will decrease discomfort with return to activity. Recovery from an acute injury typically ranges from 2 to 6 weeks depending on the severity of the injury. In the case of an established turf toe, sporting activity can exacerbate the injury. Physical therapy, NSAIDs, and the use of a stiff insole may allow return to sports and continued athletic activity (Box 25H-16).

BOX 25H-16

CRITERIA FOR RETURN TO PLAY AFTER FIRST METATARSOPHALANGEAL JOINT INJURY

Nonoperative Treatment

- Return to sport is gradual.
- Recovery from an acute injury ranges from 2 to 6 weeks.
- A stiff forefoot insole minimizes discomfort with return to sport.

Operative Treatment

- Full return to sport is typically 3 to 4 months after surgery.

Special Populations

The preference of athletes for lightweight, flexible shoes that afford good traction but little structural support presents an increasing risk factor for competitive athletes. First MTP joint sprains were first noted by Bowers and Martin [81] in 1976 and associated with artificial playing surfaces and flexible footwear. Rodeo and colleagues [79] evaluated professional football players, and 45% (36 of 80 players surveyed) reported symptoms of a turf toe. When the players could recall the initial injury, 83% of players reported their injury occurred on artificial turf. Players with restricted motion at the MTP joint may sustain an injury with relatively less excursion of the hallux. Protection of the forefoot with an orthotic device can avoid more serious injury. [1406] [1421] [1422]

SESAMOID DYSFUNCTION

The first MTP joint is characterized by the two sesamoids, which play a significant role in the function of the great toe (Box 25H-17). [97] Sesamoid dysfunction is uncommon; however, it can occur with arthritis, [1427] [1428] [1429] [1430] [1431] [1432] trauma, [1427] [1430] [1433] [1434] [1435] [1436] [1437] [1438] [1439] [1440] [1441] [1442] [1443] [1444] osteochondritis, [1429] [1435] [1437] [1445] [1446] [1447] [1448] infection, [1449] [1450] [1451] [1452] [1453] [1454] [1455] or sesamoiditis. [1428] [1437] [1445] [1446] [1447] [1456] [1457]

BOX 25H-17

SESAMOID FUNCTION

- Protect the tendon of the flexor hallucis longus.
- Absorb most of the weight-bearing on the medial aspect of the forefoot.
- Increase the mechanical advantage of intrinsic musculature of the hallux.

Anatomy and Biomechanics

The sesamoids, contained within the double tendon of the flexor hallucis brevis (see Fig. 25H-4 A) on the plantar aspect, articulate dorsally with plantar facets on the first metatarsal head. These concave facets are separated by a crista or intersesamoidal ridge, which divides the medial and lateral metatarsal facets. This crista affords intrinsic stability to the sesamoid complex (Fig. 25H-30) and may be eroded or become obliterated when severe hallux valgus causes an insidious dislocation of the sesamoid complex. The sesamoids insert on the base of the proximal phalanx through the plantar plate, an extension of the tendons of the flexor hallucis brevis. Suspended from a sling-like mechanism composed of the sesamoid ligaments and the MTP collateral ligaments (Fig. 25H-2) on the medial and lateral aspects of the MTP joint, the sesamoids are stabilized superiorly by the plantar capsule and the plantar aponeurosis. The flexor hallucis brevis provides an active plantar flexion force at the MTP joint; through its insertion into the sesamoid mechanism, an increased mechanical advantage in plantar flexion is maintained. [97] The tendon of the flexor hallucis longus, which is protected in its tendon sheath by the medial and lateral sesamoids, provides a plantar flexion force to the interphalangeal joint of the great toe (Fig. 25H-4 B).

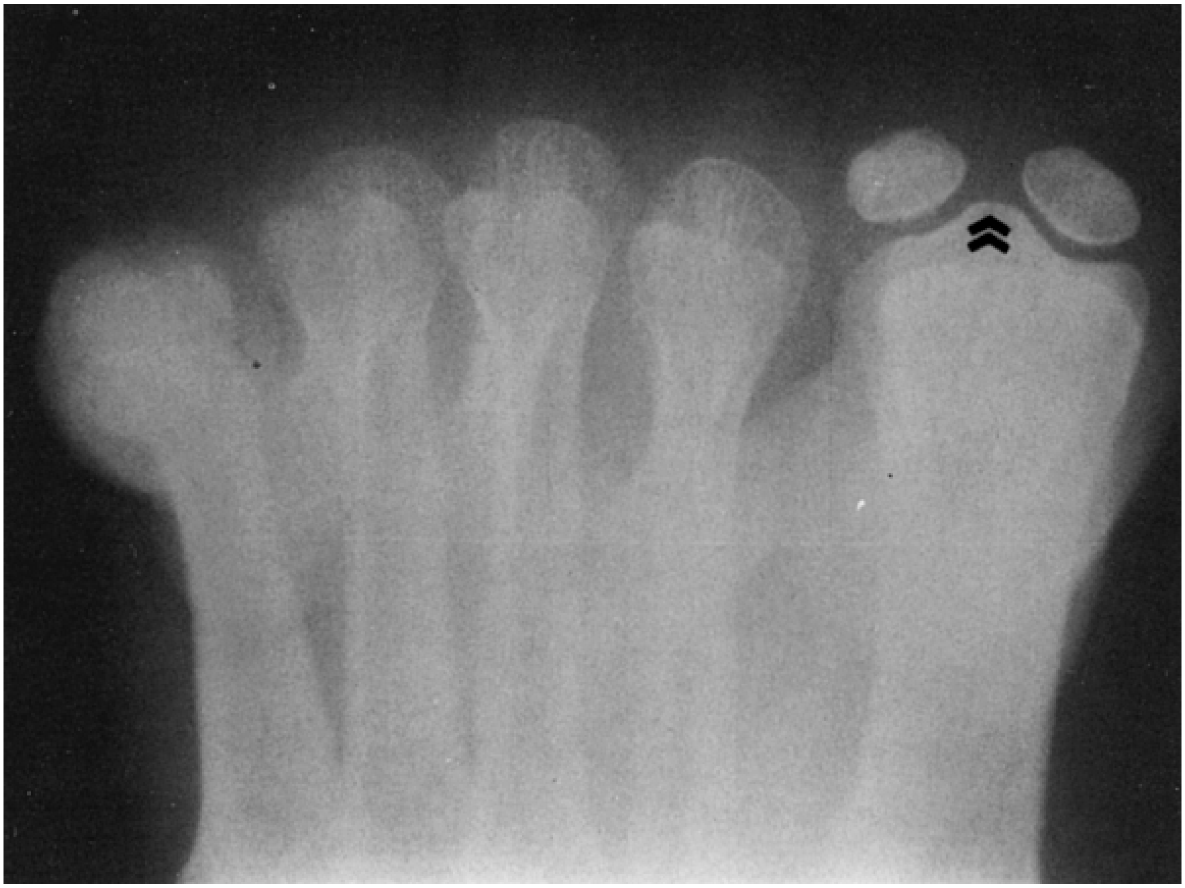

Figure 25H-30 The intersesamoidal ridge or crista is well seen on an axial radiograph. *(© M. J. Coughlin. Used by permission.)*

The abductor hallucis inserts onto the plantar medial base of the proximal phalanx and the medial sesamoid, providing a medial stabilizing force on the sesamoid mechanism. Similarly, the adductor hallucis tendon inserts onto the plantar lateral base of the proximal phalanx and the lateral sesamoid, providing a lateral stabilizing force.

Kewenter [100] reported that the medial sesamoid is located slightly more distal and is slightly larger than the lateral sesamoid. Orr [128] measured the tibial sesamoid to be 9 to 11 mm in width and 12 to 15 mm in length on average. The fibular sesamoid has an average width of 7 to 9 mm and an average length of 9 to 10 mm. Kewenter [100] reported that ossification of the hallucal sesamoids occurs between the ages of 7 and 10 years. Frequently, ossification of the sesamoids occurs from multiple centers; this may be the reason for the development of multipartite sesamoids.

Several authors have described the vascular supply of the sesamoids. [1459] [1460] Sobel and coworkers [129] determined that the primary vascular supply to the sesamoids enters proximally and plantarly. The distal blood supply is much more tenuous, contributing to delayed healing and nonunion of sesamoid fractures. Pretterklieber and Wanivenhaus [130] described three different types of arterial circulation (Box 25H-18). These authors concluded that the pattern of arterial supply of the sesamoids plays a role in the development of avascular necrosis.

BOX 25H-18

VASCULAR SUPPLY OF THE SESAMOIDS

- Type A: arterial circulation derived from the medial plantar artery and the plantar arch (52%)
- Type B: circulation predominantly from the plantar arch (24%)
- Type C: circulation from the medial plantar artery (24%)

With standing, the sesamoids are located slightly posterior to the metatarsal head. As the MTP joint dorsiflexes, the sesamoids are pulled distally, protecting the plantar surface of the first metatarsal head and absorbing the weight-bearing forces on the medial aspect of the forefoot (see Box 25H-17).

Classification

A classification system for sesamoid dysfunction has not been described. However, there are several different causes associated with sesamoid pain (Box 25H-19).

BOX 25H-19

COMMON CAUSES OF SESAMOID PAIN

- Sesamoid fracture (acute trauma or stress fracture)
- Bipartite sesamoid
- Nerve compression
- Osteochondritis and avascular necrosis
- Arthritis
- Sesamoiditis
- Intractable plantar keratosis
- Infection

Bipartite Sesamoid and Fracture

The incidence of bipartite sesamoids as well as their cause is a subject of significant controversy in the literature.[114] Dobas and Silvers[131] found a 19% incidence of bipartite sesamoids, whereas Kewenter[100] found a 31% incidence. Rowe[123] reported a 6% to 8% frequency of bipartite sesamoids and noted that 90% of these were bilateral. Dobas and Silvers[131] noted that 87% of the bipartite sesamoids in their series involved the tibial or medial sesamoid. Dobas and Silvers also noted that about 25% of the divided tibial sesamoids had an identical bipartite tibial sesamoid, and the remaining sesamoids were asymmetric in regard to division. Jahss[99] reported that the medial sesamoid has a 10 times greater incidence of bipartitism. Although Giannestras[132] stated that bipartite sesamoids are symmetrical, this finding has not been shown in other studies. The tibial sesamoid often is divided into two or more parts, whereas a lateral sesamoid rarely is divided into more than two fragments. Inge and Ferguson[97] reported a 10.7% incidence of bipartite sesamoids and noted that only 25% of these were bilateral. Of bipartite sesamoids that were bilateral, 85% showed asymmetric division.

Fracture of a sesamoid is usually due to either sudden loading of the forefoot, a fall onto the forefoot, or a crush injury.

Nerve Compression

The plantar lateral digital nerve and the plantar medial digital nerve are located adjacent to the lateral and medial sesamoids (Fig. 25H-31). Impingement of either one of these branches may be a source of pain in the area of the sesamoids. Helfet[133] noted compression of the plantar lateral cutaneous branch to the hallux. The plantar medial digital nerve may be compressed by the medial sesamoid in a similar fashion. Often this type of pain is difficult to differentiate from pain localized to a particular sesamoid.

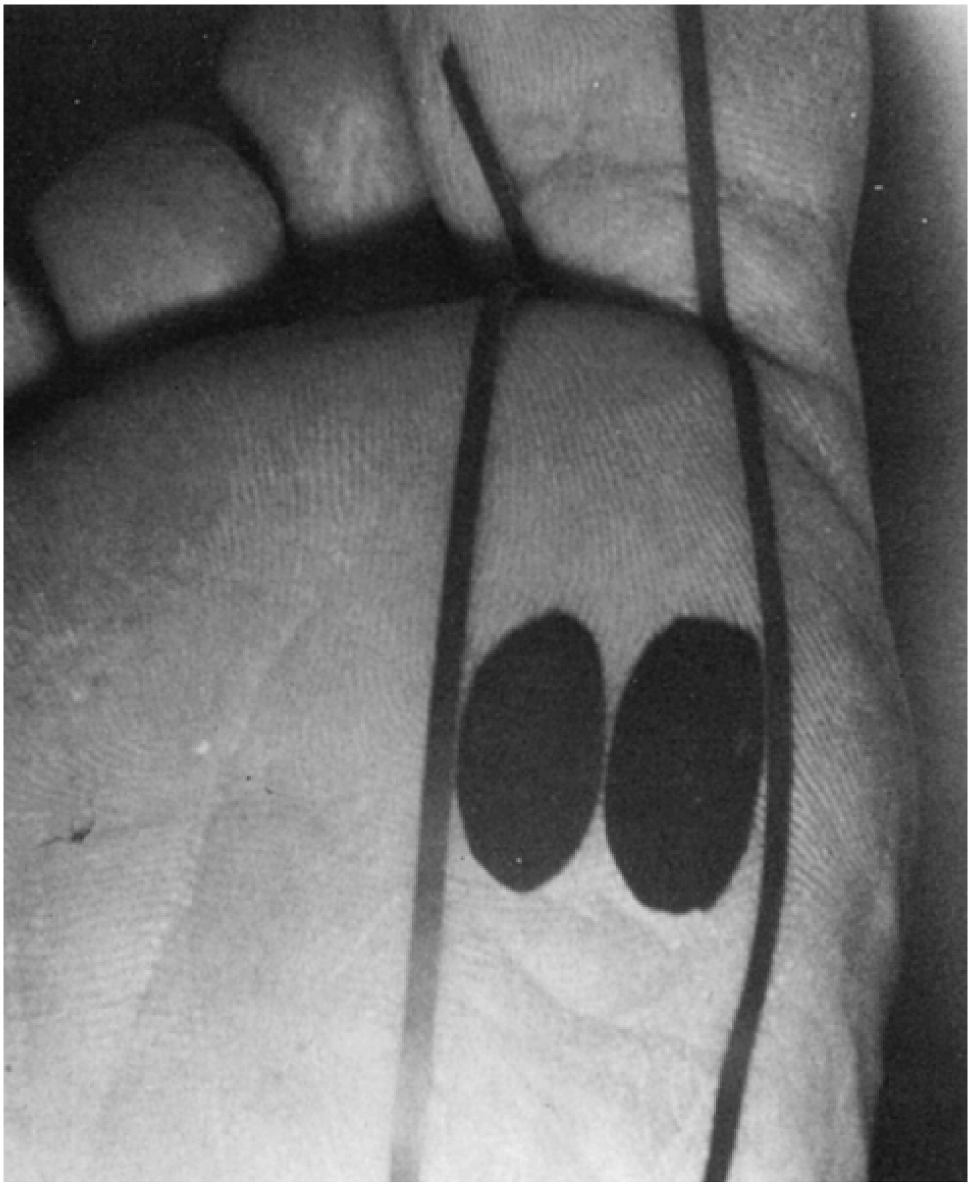

Figure 25H-31 The medial and lateral common digital nerves are found on the medial and lateral aspects of the respective sesamoids. They should be protected in any surgical excision. *(© M. J. Coughlin. Used by permission.)*

Osteochondritis and Avascular Necrosis

Osteochondritis of the sesamoid is characterized by tenderness on palpation, pain localized to the area of the involved sesamoid, and fragmentation or mottling of the involved sesamoid on radiographic evaluation. [1446] [1447] Although Renander [127] first described this condition in 1924, the cause of osteochondritis of the sesamoid is unknown. Osteochondritis appears to be infrequent, [116] and trauma frequently has been thought to be the cause of fragmentation. [98] Jahss [99] compared osteochondritis with osteonecrosis. Fleischli and Cheleuitte [134] and Julsrud [135] reported cases of osteonecrosis of the sesamoids. Kliman and associates [117] suggested that the cause of osteochondritis is a stress fracture and that the subsequent healing process is the cause of the fragmentation.

Arthritis

Osteoarthritis of the sesamoids has been reported [1427] [1428] [1429] [1430] [1431] [1432] in association with hallux valgus, hallux rigidus, and rheumatoid arthritis and as an isolated occurrence. Osteoarthritis of the sesamoids may be a natural progression of early sesamoiditis, chondromalacia, or localized sesamoid trauma. Scranton and Rutkowski [102] described erosion of the articular cartilage in what they termed *progressive sesamoid chondromalacia* that eventually led to surgical resection.

Sesamoiditis

The diagnosis of sesamoiditis is one of exclusion. Usually it is associated with trauma or repetitive stress and often occurs in young athletes. Apley [115] found remarkable similarity between the articular cartilage degeneration seen in sesamoiditis and that seen in chondromalacia of the patella, and he termed this condition *sesamoid chondromalacia.*

Intractable Plantar Keratosis

A keratotic lesion may develop beneath either sesamoid. [1466] [1467] When associated with a high-arched or cavus type of foot, a plantar flexed first ray may be the cause of the callus formation. A large diffuse callus beneath the metatarsal head is usually associated with a cavus foot. When a sesamoid is involved, there is usually a more localized or concentric lesion (Fig. 25H-32).

Figure 25H-32 An intractable plantar keratosis may develop beneath the tibial sesamoid. *(© M. J. Coughlin. Used by permission.)*

Evaluation

Clinical Presentation and History

In the history of a patient with a symptomatic sesamoid, the most frequent subjective symptoms are pain and discomfort in the toe-off phase of gait. Sesamoid dysfunction that restricts the range of motion of the MTP joint may lead to a pathologic gait pattern. An athlete may tend to evert the forefoot to decrease dorsiflexion excursion in the latter part of gait or may tend to toe-off prematurely to minimize dorsiflexion excursion of the hallux. In the case of sesamoid fracture, an athlete often walks on the lateral aspect of the foot to decrease weight-bearing on the sesamoid complex.

Physical Examination

Pain, warmth, swelling, decreased range of motion, and diminished strength are common findings on examination of patients with sesamoid dysfunction (Box 25H-20). Localized tenderness to palpation of the involved sesamoids with inflammation on the plantar aspect of the sesamoid mechanism can be present. Often, swelling of the MTP joint or synovitis is noted. In addition to restricted range of motion of the MTP joint, the patient may experience pain with forced dorsiflexion of the MTP joint or pain on localized palpation. A plantar keratosis beneath either the tibial of fibular sesamoid occasionally may accompany a symptomatic sesamoid.

BOX 25H-20

TYPICAL FINDINGS IN SESAMOID DYSFUNCTION

Physical Examination

- Pain on direct palpation of sesamoids
- Restricted range of motion at the first metatarsophalangeal (MTP) joint
- Pain with motion of the first MTP joint
- Swelling in the first MTP joint
- Diminished strength in plantar flexion or dorsiflexion
- Neuritic symptoms in cases of nerve compression

Radiographic Examination

- Radiographs frequently normal
- Sesamoid fracture (trauma) or fragmentation (osteochondritis)
- Increased uptake on bone scan

Deviation of the hallux either medially (hallux varus) or laterally (hallux valgus) can be associated with sesamoid dysfunction. Traumatic disruption of a sesamoid may cause progressive, insidious deviation of the great toe. A hallux valgus or hallux varus deformity can also occur after previous sesamoid resection.

A careful sensory examination may reveal neuritic symptoms or numbness secondary to digital nerve compression by either the tibial or fibular sesamoid. In this setting, a positive Tinel's sign may be elicited along the borders of the sesamoids. Patients may or may not note decreased sensation distal to the nerve compression.

Imaging

Routine anteroposterior and lateral radiographs may not provide sufficient information with which to evaluate the sesamoids (Fig. 25H-33 **A** and **B**). The tibial sesamoid is seen best on an oblique radiograph, with the MTP joint extended about 50 degrees (see Fig. 25H-33 **C**). The x-ray beam is directed 15 degrees cephalad from a lateral position and is centered over the first metatarsal head. The fibular sesamoid is seen best on a lateral oblique radiograph (see Fig. 25H-33 **D**), where it is shown in the first interspace between the first and second metatarsal heads. Often the most useful radiograph is the axial sesamoid view (see Fig. 25H-33 **E**). Fragmentation of a sesamoid in osteochondritis may be seen on the axial radiograph (Fig. 25H-34).

Figure 25H-33 A, Anteroposterior radiograph shows the sesamoids. **B,** Lateral radiograph shows sesamoids, although there is significant overlap that may conceal disease. **C,** An oblique radiograph shows the tibial sesamoid. **D,** A lateral oblique radiograph shows the fibular sesamoid. **E,** Axial view shows fracture of the lateral sesamoid. *(**A,** From Coughlin MJ: Sesamoids and accessory bones of the foot. In Mann RA, Coughlin MJ [eds]: Surgery of the Foot and Ankle, 6th ed. St. Louis, Mosby, 1993; **B-E,** © M. J. Coughlin. Used by permission.)*

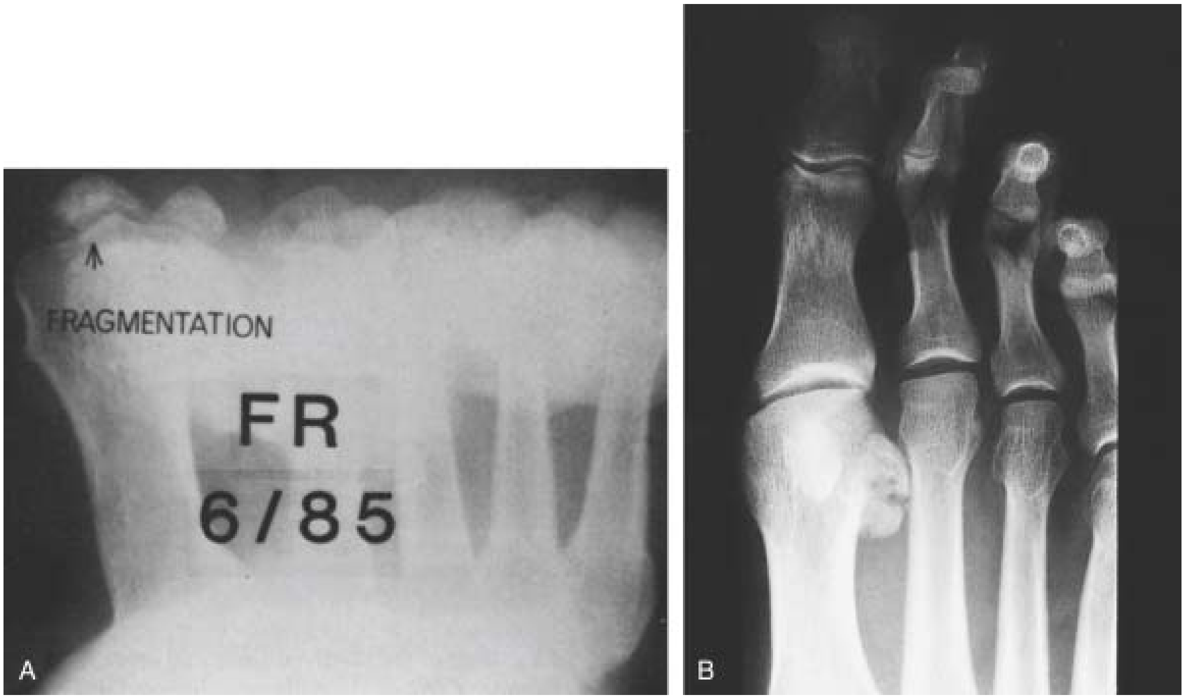

Figure 25H-34 **A,** Radiograph shows fragmentation or osteochondritis. **B,** Anteroposterior radiograph shows osteochondritis of lateral sesamoid. *(© M. J. Coughlin. Used by permission.)*

Radiographs are frequently normal despite subjective symptoms (sesamoiditis). In these cases, a bone scan may aid the diagnosis of sesamoid dysfunction. [1429] [1447] Increased uptake within the sesamoids may occur on bone scan before the development of any significant radiographic changes (Fig. 25H-35). MRI [138] is useful in visualizing bone and soft tissue abnormalities of the sesamoid mechanism (see Box 25H-20).

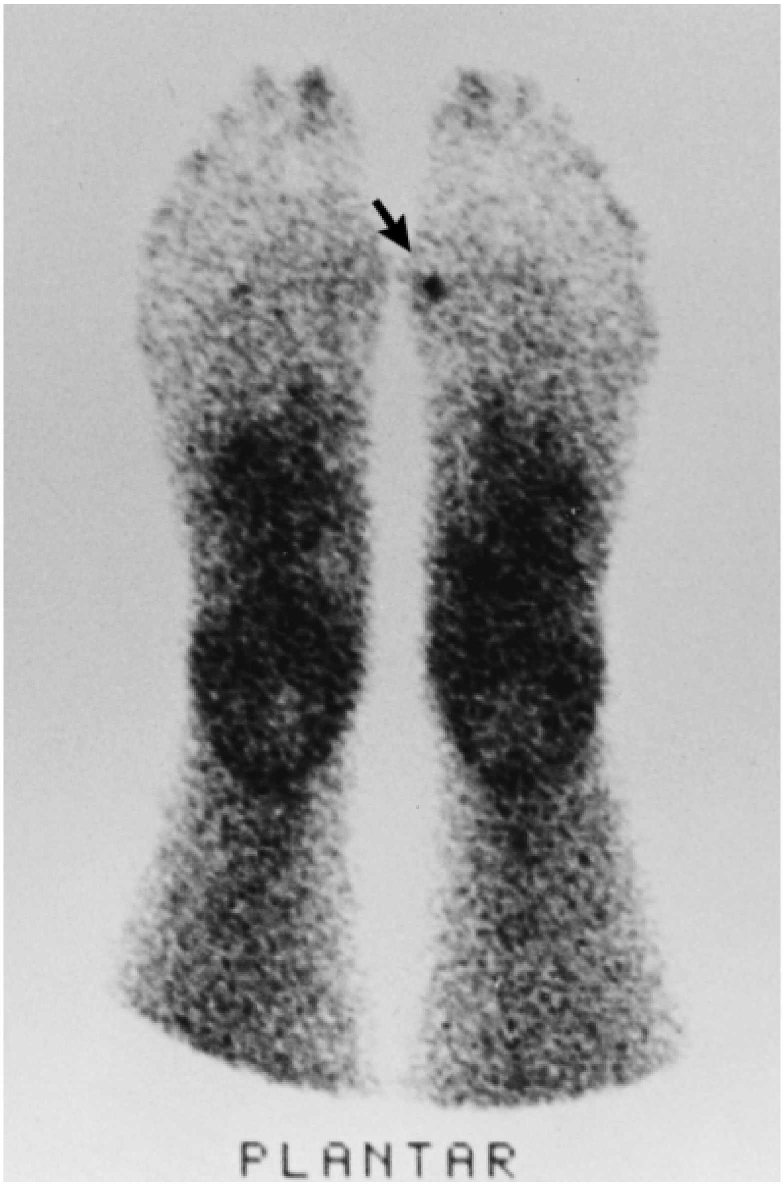

Figure 25H-35 The *arrow* marks increased uptake in the medial sesamoid consistent with osteochondritis.

Treatment Options

Nonoperative

Most sesamoid problems in the athlete can be treated effectively without surgery (Box 25H-21). Decreasing activity such as running and implementing other aerobic activity may diminish symptoms. Decreasing the heel height of shoes will diminish pressure over the sesamoids. A molded insole or a metatarsal arch support is helpful in decreasing plantar pressure in the sesamoid region for treatment of a fracture, sesamoiditis, or osteonecrosis. This type of support may also relieve the pressure of an intractable plantar keratosis (IPK). Patients can be instructed in shaving the callus, which may alleviate symptoms as well. Oral NSAIDs may give relief of pain associated with sesamoiditis, osteonecrosis, or arthritis. A steroid injection occasionally will help when there is localized inflammation or when sesamoiditis has occurred, but injections should be used judiciously. In the presence of a fracture or osteonecrosis, intra-articular injection is contraindicated. In patients with sesamoid arthritis, a stiff insole, a rocker-bottom sole, and metatarsal padding reduce metatarsal joint motion and can relieve pain with ambulation.

BOX 25H-21

Treatment Options for Sesamoid Dysfunction

Nonoperative

- Nonsteroidal anti-inflammatory drugs
- Decreased shoe heel height
- Molded insole or metatarsal arch support
- Shoe modifications (stiff insole, rocker sole)
- Shaving of a painful callus

Operative

- Sesamoidectomy (tibial or fibular)
- Sesamoid shaving
- Screw fixation of sesamoid fracture
- Autologous bone grafting

In the case of sesamoid fractures, a walking cast or taping of the hallux into a neutral position (Fig. 25H-36) with a stiff-soled shoe may be used. Typically, 6 weeks of immobilization and activity restriction will allow resolution of symptoms and fracture healing. After 6 weeks, the athlete may gradually return to activities with repeat radiographs at 3 to 4 months after injury to evaluate healing.

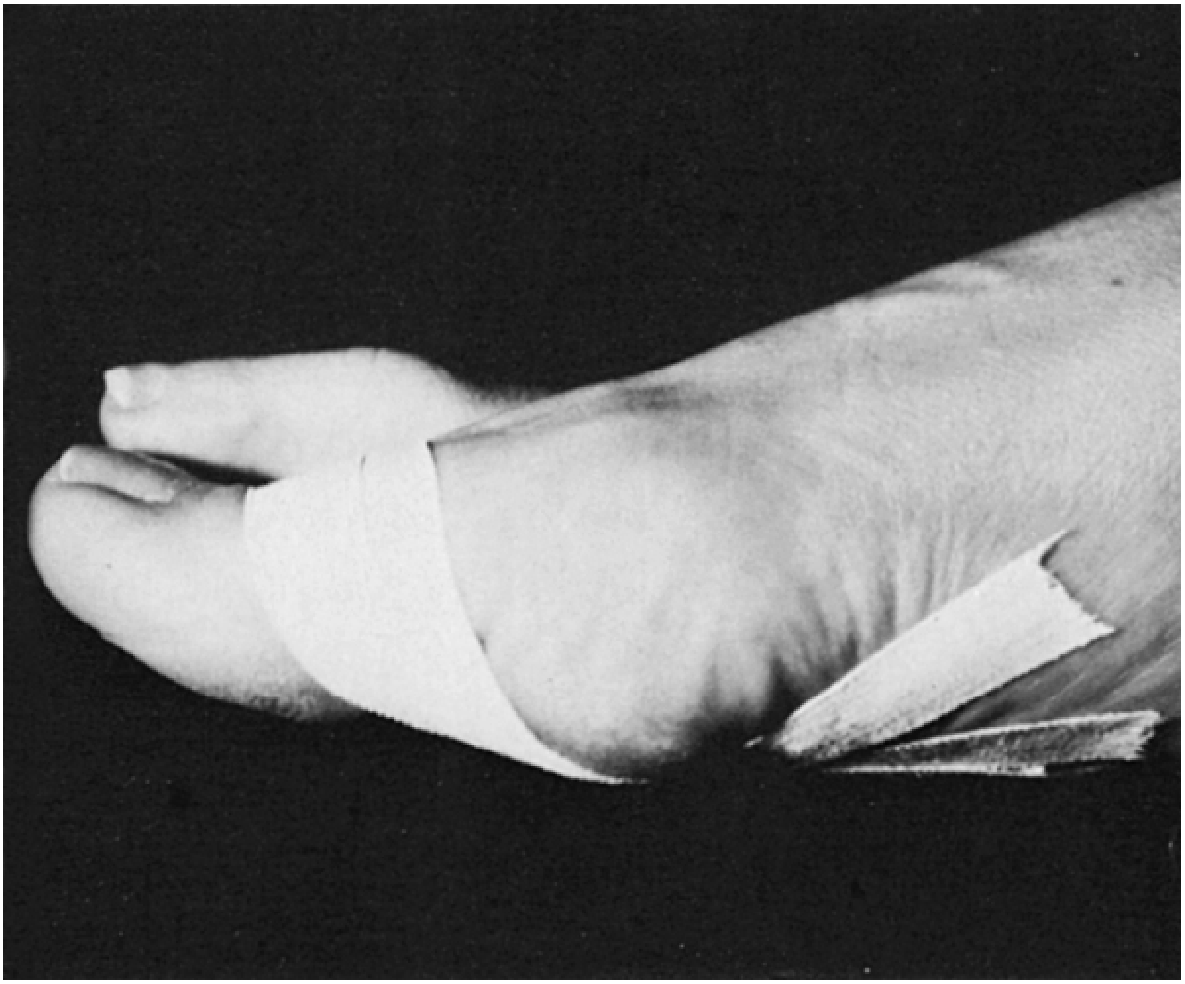

Figure 25H-36 Taping of the hallux minimizes dorsiflexion and may relieve symptoms. *(© M. J. Coughlin. Used by permission.)*

Operative

If conservative treatment has been unsuccessful, several surgical options are available depending on the pathology (see Box 25H-21). Although sesamoid abnormalities are infrequent, injury or degeneration of a sesamoid may lead to significant dysfunction of the MTP joint in athletes.

Scranton and Rutkowski [102] and Speed [139] described excision of both sesamoids. Scranton [102] advised a reapproximation of the surgical defect to minimize postoperative disability. Helal [98] and Inge and Ferguson [97] cautioned against combined medial and lateral sesamoid excision because postoperative clawing of the hallux may occur with dual sesamoid excision. Jahss [99] recommended repair of the defect in the flexor hallucis brevis after dual sesamoid excision on the theory that it was comparable to repairing the quadriceps mechanism after patellectomy. Occasionally, dual sesamoidectomy may be necessary (i.e., with infection); elective dual sesamoidectomy should be avoided because a claw-toe deformity may occur.

For isolated arthritis of either the medial or the lateral sesamoid, surgical excision may be performed. When both sesamoids are involved, however, a combined medial and lateral sesamoid excision is likely to destroy the insertion of the flexor digitorum brevis and may lead to eventual clawing of the hallux. [1427] [1429] Arthrodesis of the MTP joint may alleviate arthritic symptoms and provide a stable medial buttress to the first ray. Although pain may be relieved considerably with operative resection of an involved sesamoid, MTP joint motion may not improve when significant arthritis is present.

Depending on which sesamoid is to be resected, various surgical approaches are available. The lateral or fibular sesamoid is approached through either a dorsal or a plantar approach. Mann and Coughlin [136] advocated a dorsal lateral approach to resect the fibular sesamoid in patients with hallux valgus; they recommended this approach for isolated fibular sesamoid excision also. [137] The fibular sesamoid is resected through a dorsal first interspace incision (Box 25H-22 ; Fig. 25H-37). [1470] [1471] Inge and Ferguson [97] and others [1441] [1447] recommended a dorsal approach to avoid a painful plantar scar as well as injury to the plantar sensory nerve in the first interspace.

BOX 25H-22

FIBULAR SESAMOIDECTOMY, DORSAL APPROACH

- Make a dorsal incision over the first interspace.
- Retract the first and second metatarsals with a Weitlaner retractor to sublux the lateral sesamoid.
- Reflect the adductor hallucis off the lateral sesamoid.
- Protect the continuity of the adductor tendon (to prevent postoperative varus).
- Detach the intersesamoidal ligament.
- Excise the fibular sesamoid from the surrounding tissues.
- Confirm that the adductor hallucis and the flexor hallucis longus are still intact.

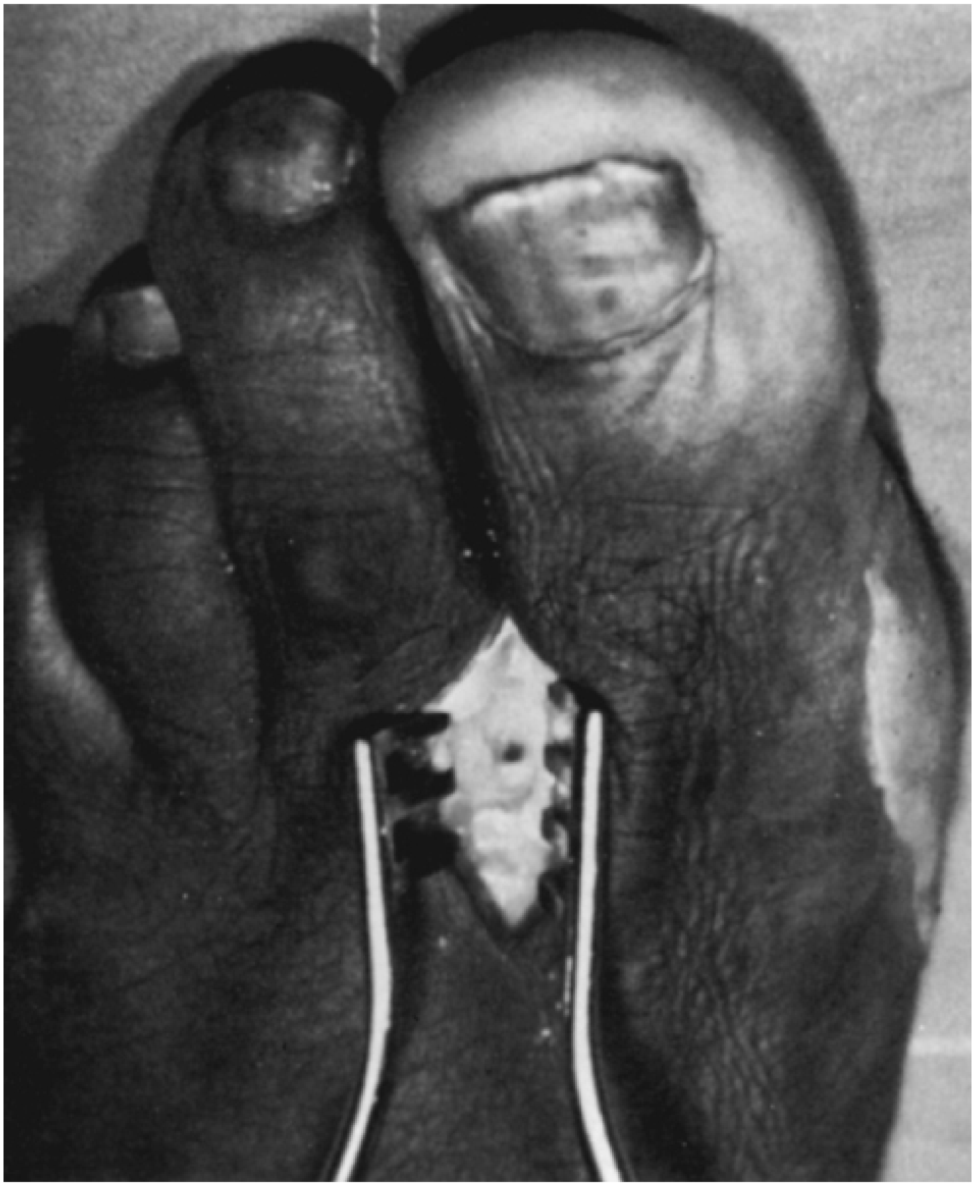

Figure 25H-37 The fibular sesamoid is exposed through a dorsal incision. *(© M. J. Coughlin. Used by permission.)*

If a plantar approach is used to resect the fibular sesamoid, an intermetatarsal incision is recommended (Box 25H-23 ; Fig. 25H-38). Van Hal and colleagues,[113] Helal,[98] and Jahss[99] recommended a longitudinal plantar incision adjacent to the fibular sesamoid. Jahss[99] found a dorsal approach for a sesamoidectomy difficult. If one wishes to approximate the surgical defect, a plantar approach is necessary.

BOX 25H-23

FIBULAR SESAMOIDECTOMY, PLANTAR APPROACH

- Make a longitudinal plantar incision in the first intermetatarsal space.
- Identify the plantar digital nerve adjacent to the lateral aspect of the hallux and the lateral sesamoid.
- Protect the plantar digital nerve.
- Detach the capsular fibers of the flexor hallucis brevis.
- Release the sesamoid proximally, laterally, and distally.

- Incise the intersesamoidal ligament.
- Remove the fibular sesamoid.
- Inspect the tendon of the flexor hallucis longus for continuity.
- Close the defect created by the sesamoid excision.

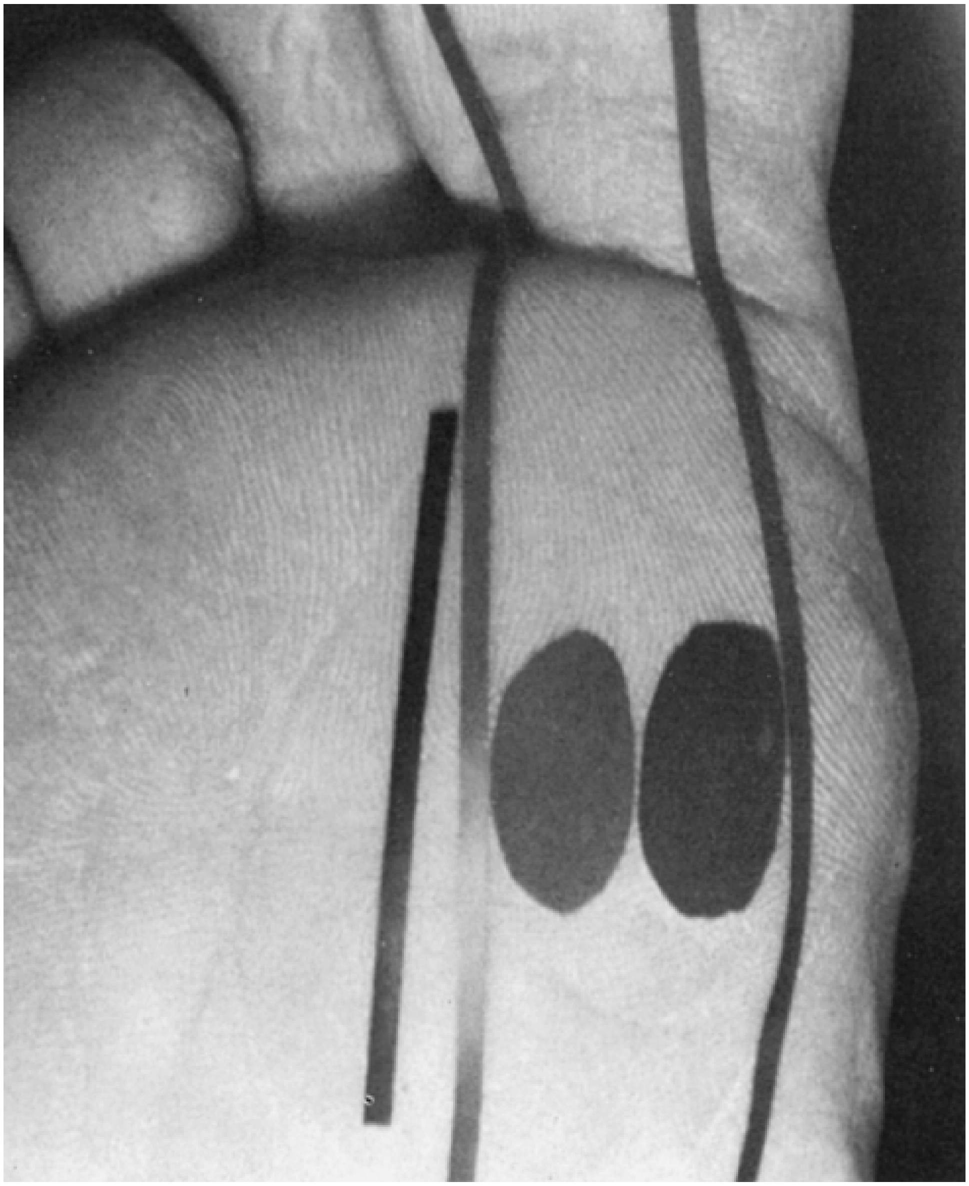

Figure 25H-38 A plantar incision to excise the lateral sesamoid should be located on the lateral aspect of the lateral sesamoid. *(© M. J. Coughlin. Used by permission.)*

The medial sesamoid may be resected by either a plantar medial incision (Fig. 25H-39 **A**) [1428] [1443] or a direct medial incision (Box 25H-24). [1427] [1429] [1447] [1456] Kliman and associates [117] advocated a direct medial approach to avoid the plantar digital nerves, which may be in close proximity to the surgical dissection through a medial plantar approach. Mann and coworkers [137] discouraged a plantar approach because of the possibility of developing a painful postoperative plantar scar.

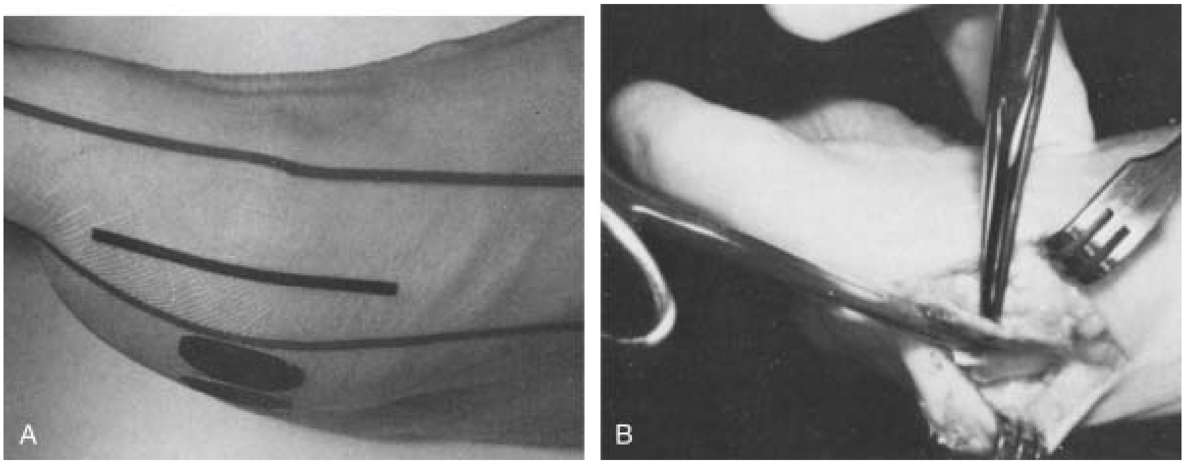

Figure 25H-39 **A**, Excision of the medial sesamoid through a plantar medial approach. **B**, Location of the medial plantar nerve. *(© M. J. Coughlin. Used by permission.)*

BOX 25H-24

TIBIAL SESAMOIDECTOMY

- Make either a plantar medial or direct medial incision.
- Identify the medial plantar cutaneous nerve on the medial aspect of the sesamoid (see Fig. 25H-39 A).
- Retract the nerve with the plantar skin flap to avoid postoperative neuroma.
- Incise the medial capsule to expose the articular surface of the sesamoid.
- Dissect the sesamoid free of surrounding tissues.
- Incise the intersesamoidal ligament (see Fig. 25H-39 B).
- Inspect the tendon of the flexor hallucis longus for continuity.
- Close the defect created by the sesamoid excision.

Although sesamoidectomy is occasionally necessary for IPKs, surgical shaving of the involved sesamoid may preserve function in the athlete and allow more rapid recovery (Box 25H-25 ; Fig. 25H-40). [1472] [1473] Resection of the plantar half of the medial sesamoid may reduce a prominent tibial sesamoid and allow resolution of a plantar keratotic lesion. Sesamoid shaving or resection in the presence of a plantar flexed first ray is associated with a high rate of recurrence of the keratotic lesion. [1472] [1473] In this situation, a closing wedge dorsiflexion first metatarsal osteotomy is preferable.

BOX 25H-25

SHAVING A PROMINENT TIBIAL SESAMOID

- Make either a plantar medial or direct medial incision.
- Identify the medial plantar cutaneous nerve on the medial aspect of the sesamoid (see Fig. 25H-40 A).
- Retract the nerve with the plantar skin flap to avoid postoperative neuroma
- Retract the plantar fat pad and protect the flexor hallucis longus
- Resect the plantar surface of the tibial sesamoid with a sagittal saw (see Fig. 25H-40 B).
- Bevel any remaining prominent edges of the sesamoid.

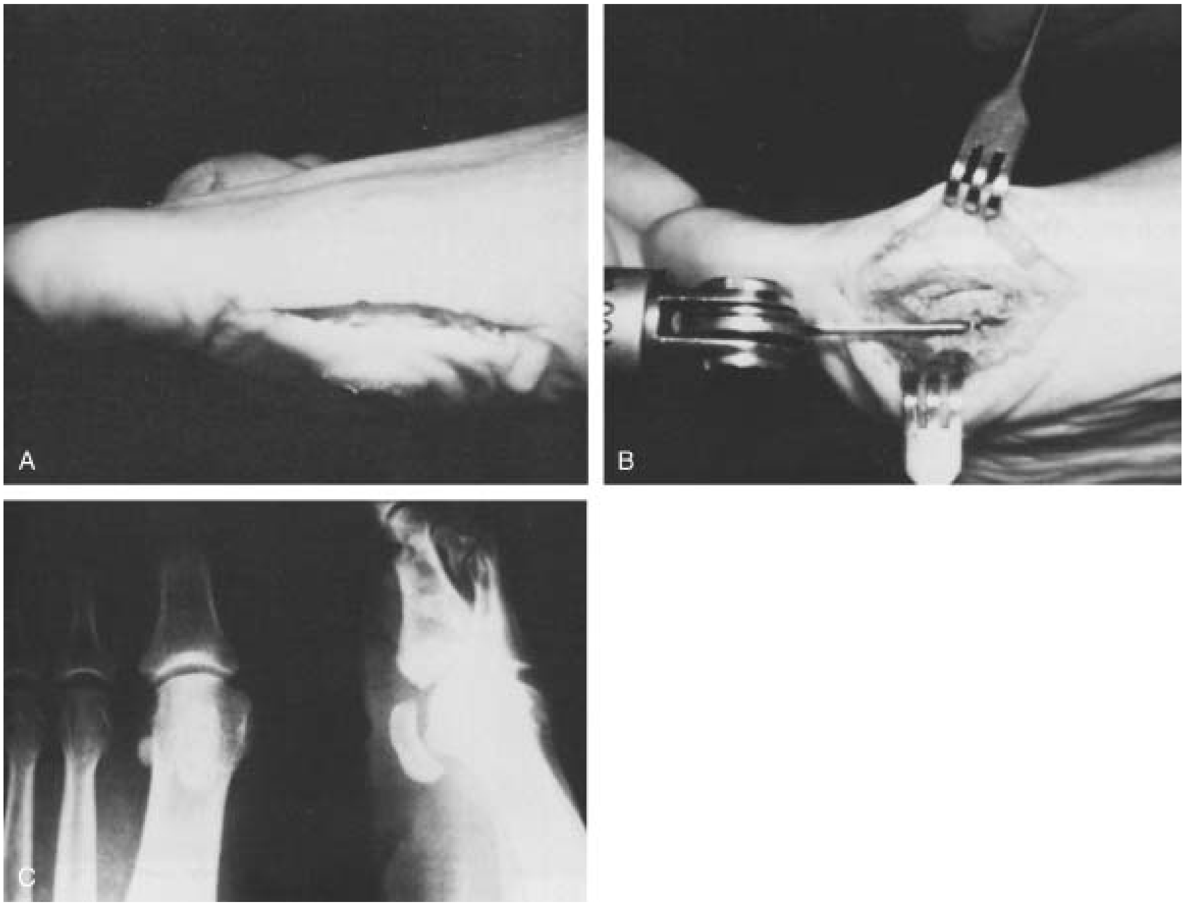

Figure 25H-40 **A**, A plantar medial incision is used to expose the medial sesamoid for shaving. **B**, Half of the tibial sesamoid is shaved to treat an intractable plantar keratosis. Care is taken to protect the flexor hallucis longus tendon. **C**, Radiograph after tibial shaving. *(© M. J. Coughlin. Used by permission.)*

Anderson and McBryde reported on a series of sesamoid fracture nonunions treated with autologous bone grafting with good results in a series of 21 patients. [144] Autologous bone grafting may offer an alternative to sesamoid excision in some individuals. Other authors have described screw fixation of sesamoid fractures. [145]

Weighing the Evidence

Surgical excision of the chronically painful sesamoid has been advocated by multiple authors. [1427] [1428] [1429] [1437] [1438] [1443] [1444] [1445] [1446] [1447] [1449] [1450] [1451] [1452] [1453] [1454] [1455] [1456] [1457] [1469] [1476] [1477] [1478] [1479] However, controversy exists about whether an acceptable result can be achieved after sesamoidectomy. Lee and colleagues [150] describe tibial sesamoidectomy as “safe and effective,” emphasizing meticulous surgical technique. Ilfeld and Rosen [116] reported excellent postoperative pain relief after surgical excision of the involved sesamoid in cases of sesamoiditis. Although Van Hal and colleagues [113] and others [1441] [1456] [1481] reported acceptable results after hallucal sesamoidectomy, Inge and Ferguson [97] reported significant dysfunction of the MTP joint after sesamoidectomy.

The incidence of hallux varus deformity after fibular sesamoidectomy was about 8% according to Mann and Coughlin in their review of the McBride procedure. After isolated fibular sesamoidectomy, Coughlin [146] and others [137] reported a 5% varus drift of the hallux.

Often residual pain exists after the removal of a diseased sesamoid. Zinman and colleagues [114] and others [1443] [1456] noted complete resolution of pain and resumption of normal activities. Inge and Ferguson [97] found that only 41% of their patients experienced complete relief of pain. Coughlin [146] and Mann and coworkers [137] reported complete relief of pain in only 50% of patients postoperatively.

Stiffness is a common complaint after sesamoidectomy. [1429] [1447] [1450] [1455] [1456] [1467] [1476] Coughlin [146] and

Mann and coworkers [137] noted that one third of patients had decreased MTP motion at long-term follow-up. Torgerson and Hammond [125] and Colwill [120] noted decreased range of motion preoperatively but did not mention restricted motion after surgery. Jahss [99] reported that surgical excision could lead to stiffness. Inge and Ferguson [97] reported that 58% had restricted range of motion. Both Mann and coworkers [137] and Coughlin [146] reported a 60% incidence of plantar flexion weakness. Diminution of strength of the flexor hallucis brevis was hypothesized by Van Hal and associates. [113] Inge and Ferguson [97] reported a 17% incidence of clawing in the hallux, but many of these patients had had dual sesamoid resection. Postoperative migration of the sesamoid has been noted to occur in 10% of cases. [1467] [1476] Nayfa and Sorto [151] quantitated this drift to average 6.2 degrees in either varus or valgus postoperatively.

Results of tibial sesamoid shaving routinely have been good. Aquino and colleagues [142] reported on 26 feet in which tibial sesamoid shaving was performed for an IPK. Satisfactory results were reported in 89%. This high success rate is probably due to the fact that the intrinsic musculature was not disrupted with this dissection. Minimal weakness was observed at the first MTP joint.

Mann and Wapner, [143] reporting on their results of 16 tibial sesamoid shavings, noted no functional limitations and good or excellent results in 94%. In one case, a recurrent callus developed. Mann and Wapner [143] observed that there is a possibility of fracture if an excessive resection is performed, although this did not occur in the series. A tibial sesamoid shaving is contraindicated in the presence of plantar flexion of the first ray greater than 8 degrees.

AUTHOR'S PREFERRED METHOD

Conservative treatment with shoe modifications and taping of the toe is preferred in most athletes with sesamoid dysfunction. For a diseased tibial or fibular sesamoid that is refractory to conservative care, surgical excision can be considered. The preferred method for a tibial sesamoid resection is a plantar medial approach. Repair of the surgical defect should be attempted. A fibular sesamoid may be excised through either a dorsal or plantar approach. In the presence of a hallux valgus deformity, the dorsal approach provides excellent visualization of the fibular sesamoid. The plantar approach is more direct in cases of normal alignment. With a plantar approach, a painful plantar scar can develop, and a patient should be informed of this before surgery. Care must be taken not to disrupt the lateral capsule of the first MTP joint or to disrupt the adductor hallucis brevis in order to maintain function and stability of the first MTP joint. Although resection of a diseased sesamoid often achieves acceptable results, the athlete can experience continued symptoms, restricted motion, or diminution of strength. An attempt should be made to avoid a dual sesamoid resection unless absolutely necessary.

Despite careful dissection and postoperative care, it is common for 50% of patients to have continued symptoms after surgical resection. About 50% of patients may note diminution of plantar flexion strength, and restricted range of motion may be experienced. Postoperative physical therapy exercises to rehabilitate plantar flexion strength and to achieve greater range of motion are helpful in the recovery of an athlete.

Postoperative Prescription, Outcomes Measurement, and Potential Complications

After sesamoid excision, a soft compression dressing is used, and the patient is allowed to ambulate in a postoperative shoe. The toe is taped into appropriate alignment for 6 to 8 weeks after surgery. After successful healing of the surgical incision, range-of-motion activities may be started in the athlete. Although postoperative mobilization of the MTP joint may be initiated within 2 to 3 weeks after surgery, it is still important to tape the toe in correct alignment to minimize postoperative drift. By 6 to 8 weeks, aggressive walking activity can be initiated, and by 8 to 10 weeks, jogging and running activity can be instituted.

Postoperative dressings and later taping are important to protect the hallux from either medial migration of the hallux (hallux varus) after a fibular sesamoid resection or development of hallux valgus after a medial sesamoid resection. Nayfa and Sorto [151] reported a 42% incidence of hallux valgus after medial sesamoidectomy. Mann and colleagues [137] reported a 5% incidence of valgus drift after tibial sesamoid resection.

Inge and Ferguson [97] found that impaired motion, deformity, and pain occurred in many patients after sesamoidectomy. The development of postoperative IPKs beneath a remaining sesamoid, [1466] [1467] valgus deviation after tibial sesamoidectomy, [1429] [1467] [1481] [1482] varus deviation after fibular sesamoidectomy, [1429] [1447] [1467] development of restricted motion in the MTP joint, [97] and weakening of plantar flexion strength of the MTP joint [97] all have been reported. Clawing of the hallux after a medial and lateral sesamoidectomy has been noted. [1427] [1432] The development of a postoperative neuroma after surgery may be more symptomatic than the patient's presurgical complaint.

Taping of the toe is usually not necessary after shaving of the tibial sesamoid because the MTP joint is not entered. After tibial sesamoid shaving, a soft dressing is used, and the patient is allowed to ambulate in a postoperative shoe for 3 weeks. Range of motion exercises are started as soon as wound healing is complete. Usually the patient can start aggressive walking the fourth week. Jogging and running are initiated 6 weeks after surgery. The athlete may gradually return to full activity as pain and swelling allow.

Criteria for Return to Play

Return to play after surgical intervention for sesamoid dysfunction is typically between 2 and 4 months after surgery. When shaving of the tibial sesamoid is performed, the athlete is allowed to resume running about 6 weeks after surgery. With excision of a sesamoid, running is delayed until 8 weeks after surgery, when taping of the toe is discontinued. Once the athlete is running without pain or swelling, gradual return to play is allowed (Box 25H-26).

BOX 25H-26

Criteria for Return to Play After Sesamoid Surgery

- Typically expected 2 to 4 months postoperatively
- Return to play earlier (2 months) with sesamoid shaving
- Athlete must be running with resolution of pain and swelling.

Special Populations

Sesamoid dysfunction has been reported in both dancers and gymnasts. [1483] [1484] Any sport that requires repetitive impact loading of the forefoot may place an athlete at increased risk.

INGROWN TOENAILS

Onychocryptosis, or ingrown toenail, occurs with great frequency in young athletes. Trauma such as stubbing the toe can lead to inflammation or infection of the toenail edge. [155] Cutting the toenails in a curved fashion and tearing them often leads to ingrowth with subsequent infection. As the toenail grows out, the corner of the advancing nail plate impinges on the lateral nail groove. [156] Tight-fitting shoes can cause increased pressure between the nail and the nail fold, leading to ingrowth of the toenail. [1486] [1487]

Anatomy and Biomechanics

The toenail, or the nail plate, is composed of several layers of dense overlapping keratinized cells. The nail plate consists of three layers, each originating from a different area of the nail unit. The thin dorsal layer is relatively stiff or brittle and covers the thicker middle layer. The deep layer is believed to be derived from the nail bed. [158] The nail is supported by the nail unit, an area of epithelial tissue that is divided into four components (Box 25H-27 ; Fig. 25H-41). [159]

BOX 25H-27

Components of the Nail Unit

- *Nail bed:* a roughened epithelial surface that consists of longitudinal grooves that interdigitate with corresponding grooves on the undersurface of the nail
- *Hyponychium:* smooth border of skin at the distal end of the nail bed, which forms a seal between the distal end of the toenail and the nail bed.
- *Proximal nail fold:* a complex structure that participates in the germination of the nail plate
- *Nail matrix:* main germinal area of the nail

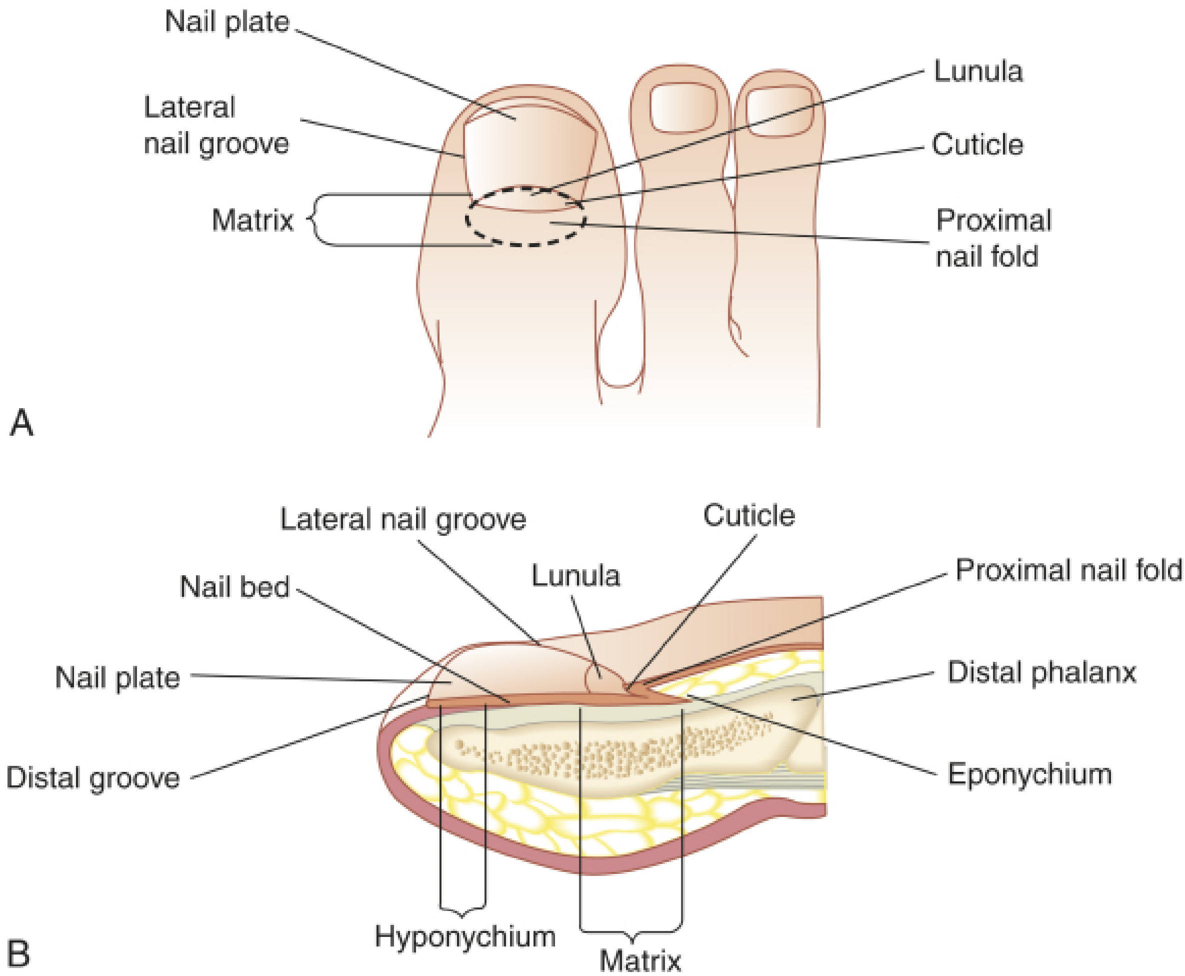

Figure 25H-41 **A,** Germinative layer of nail matrix. **B,** Cross section of toe shows nail bed, nail matrix, toenail, hyponychium, and eponychium.

The nail plate lies on the nail bed, a roughened epithelial surface that consists of longitudinal grooves that interdigitate with corresponding grooves on the undersurface of the toenail. This interdigitation creates a firm bonding of the nail plate to the nail bed. The nail bed is believed to contain some germinative cells that produce a portion of the nail plate. [1488] [1490] [1491] [1492] At the distal end of the nail bed, as the nail bed and nail plate separate, there is a smooth border of skin (the hyponychium), which forms a seal between the distal end of the nail and the nail bed.

On the tibial and fibular borders of the toenail, the nail plate is surrounded by epidermal skin folds called *lateral nail folds*. The base of the nail is covered by the *proximal nail fold*, a complex structure that participates in the germination of the nail plate. [162] The dorsal surface of the nail fold is composed of the skin on the dorsal surface of the toe. On the plantar surface of this fold, the eponychium forms a thin surface that attaches to the nail plate. The distal surface of the two components of the proximal nail fold forms the cuticle.

The nail matrix is the main germinal area for the toenail. The matrix extends from a point just distal to the lunula, as far laterally as the entire width of the nail plate [155] and 8 mm proximally to the edge of the cuticle. The matrix closely borders the insertion of the long extensor tendon. [158] Nail matrix is seen beneath the nail plate as the lunula, the opaque crescent-shaped area at the base of the toenail. Distally, the nail matrix is contiguous with the nail bed. The germinal matrix is covered by a small epidermal surface and does not have the epidermal ridges characteristic of the nail bed. At the proximal margin of the matrix, there appears to be a small area on the plantar surface of the proximal nail fold that contributes to nail plate growth. [1488] [1493] [1494]

The major area of matrix germination occurs between the apex of the matrix and the distal border of the lunula. The area covered by the proximal nail fold forms the thin dorsal layer of the nail plate. [158] The distal area of the matrix (the lunula) produces the thicker and softer area of the middle portion of the nail plate. [158] Microscopic toenail matrix contained within the nail folds and the distal nail bed [1490] [1491] produces a thin layer of the ventral toenail plate, a factor that occasionally causes postoperative recurrence. [1485] [1488] [1493]

Biomechanical imbalance may be a cause of ingrown toenails in athletes. Tight-fitting shoes causing pressure at either the medial or lateral nail folds can result in infection of the toenail margins of the great toe. Pronation deformity of the hindfoot can also cause pressure on the medial nail fold. Trauma to the great toe with formation of a subungual hematoma often leads to the loss of a toenail.

Classification

Heifetz[156] proposed a classification of ingrown toenails into three distinct stages (Box 25H-28).

BOX 25H-28

Classification of Ingrown Toenails

Stage I: Swelling and erythema occur along the lateral fold. The edge of the nail plate may be embedded in an irritated nail fold.

Stage II: Acute or active infection is characterized by increased pain. Drainage is present.

Stage III: With chronic infection, granulation tissue may develop in the lateral nail fold. Hypertrophy of the surrounding soft tissue is present.

Evaluation

Clinical Presentation and History

A careful history should be taken to elicit any familial traits or genetic factors that predispose an athlete to congenital nail deformities. Prior toenail problems may indicate extrinsic factors such as ill-fitting footwear, a problem related to the type of athletic activity, or other causes of toenail abnormalities. Recent stubbing or trauma to the toe may indicate the cause of toenail pathology. Changes in footwear or athletic shoes can also contribute to the development of toenail problems.

Physical Examination

In evaluating a toenail, physical examination of the entire foot and lower extremity is important. Palpation of arterial pulses and evaluation of the skin are required because vascular insufficiency may lead to toenail abnormalities. Examination of the skin also provides information about fungal infections, ulcerations, blisters, or other sources of skin lesions. The patient's gait should be observed to look for gait patterns that subject the toenails to undue pressure. The postural position of the foot may show pes planus, pes cavus, or other anatomic abnormalities.

Swelling and erythema may be present at the nail fold. In acute infection, purulent drainage is present, often with significant tenderness along the nail fold. Granulation tissue and hypertrophy of the surrounding soft tissue occur in chronic infections (Box 25H-29).

BOX 25H-29

Typical Findings in Ingrown Toenails

- Swelling
- Erythema
- Tenderness to palpation at the nail fold
- Purulent drainage in acute infection

Imaging

Radiographic evaluation of the involved toes is warranted in patients with recurrent toenail infections or toenail abnormalities. Evaluation of the distal phalanx by radiograph helps to rule out osteomyelitis in a chronically infected hallux. A subungual exostosis is best seen on a lateral radiograph and can be an explanation for chronic toenail elevation, ulceration, or infection.

Treatment Options

Nonoperative

Stage I lesions of the nail frequently respond to conservative care. Appropriate footwear is essential in preventing recurrence. Effective treatment requires elevation of the lateral toenail plate from the inflamed nail fold. A wisp of cotton is inserted carefully beneath the edge of the nail plate, taking care not to fracture the nail plate (Fig. 25H-42 **A**). Frequently a digital anesthetic block is required to reduce discomfort when the nail edge is elevated.

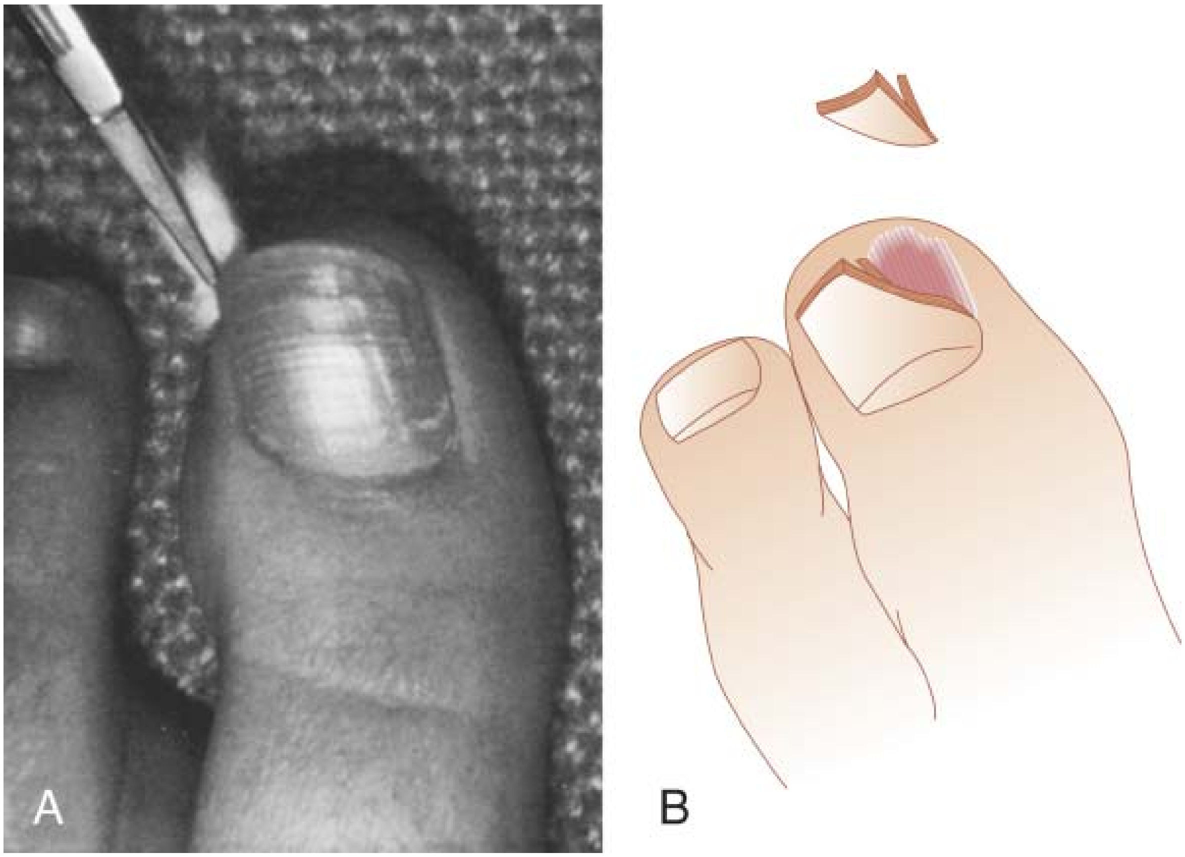

Figure 25H-42 **A,** Elevation of toenail with cotton placed under edge. **B,** Diagonal trimming of toenail edge relieves pressure on adjacent soft tissue. *(From Mann RA, Coughlin MJ: Video Textbook of Foot and Ankle Surgery. St. Louis, Medical Video Production, 1991, p 56.)*

When an adequate length of the nail plate has regrown, the patient is instructed to cut the nail in a transverse fashion. Tearing or picking a nail or cutting the nail in a curved fashion can result in recurrent infection. Diagonal trimming of a nail helps diminish the inflammation of an acute infection, but it is merely a temporary treatment and is often associated with recurrent infection as the nail plate grows (see Fig. 25H-42 **B**).

A subungual hematoma can result from a single traumatic episode or repetitive stress of the toe striking the end of the toe box in tennis, jogging, or other athletic activities. If acute pain develops, decompression of the hematoma by penetrating the nail plate with either a small nail drill or a hot paper clip alleviates discomfort significantly. As this nail detaches, a new nail grows beneath the old one. As the new nail grows distally, attention must be directed to the re-establishment of a new lateral nail fold so that an ingrown toenail does not develop. Painless discoloration underneath the toenail with no history of trauma may be an asymptomatic melanoma, and the toenail should be reinspected at a later date. In this situation, a biopsy is necessary to further evaluate the possibility of a subungual melanoma.

Operative

To be effective, treatment of ingrown toenails must be tailored to the individual patient. Heifetz[156] stated that only during stage I can conservative means be used to obtain a cure. When conservative care is unsuccessful or when acute (stage II) or chronic (stage III) infection occurs, more aggressive care is necessary (Box 25H-30). A digital anesthetic block is performed routinely before any toenail procedure (Fig. 25H-43). A small Penrose drain may be used as a tourniquet if needed.

BOX 25H-30

SURGICAL TREATMENT OF INGROWN TOENAILS

- Partial nail plate avulsion
- Complete nail plate avulsion
- Plastic nail wall reduction
- Partial onychectomy
- Complete onychectomy
- Syme amputation of the toe
- Phenol and alcohol matrixectomy

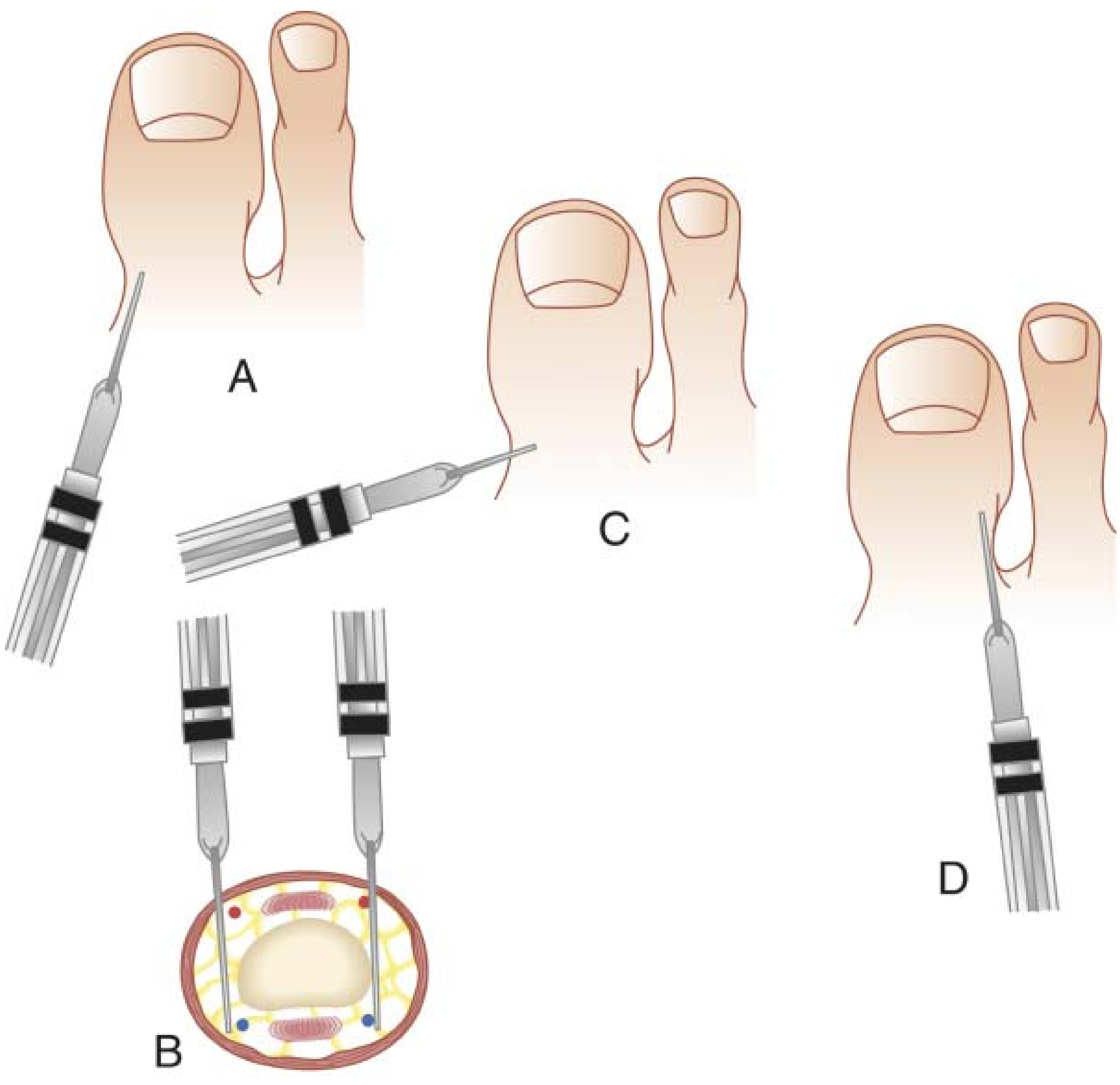

Figure 25H-43 Digital block of great toe. **A,** Injection of medial aspect of toe. **B,** Vertical injection techniques. **C,** The needle is turned in a horizontal direction. **D,** A final injection is used to anesthetize the lateral aspect of the toe.

Partial Nail Plate Avulsion

After elevating the outer edge of the nail plate proximally to the level of the cuticle, a scissors or small bone cutter is used to section the nail longitudinally. Only the outer edge of the nail is resected, removing as narrow a section of nail as possible. The toe should be examined closely to ensure that no spike of the nail plate remains in the lateral nail fold because this would continue to incite a foreign body reaction. After removal of the nail edge, the acute or chronic infection often subsides (Fig. 25H-44).

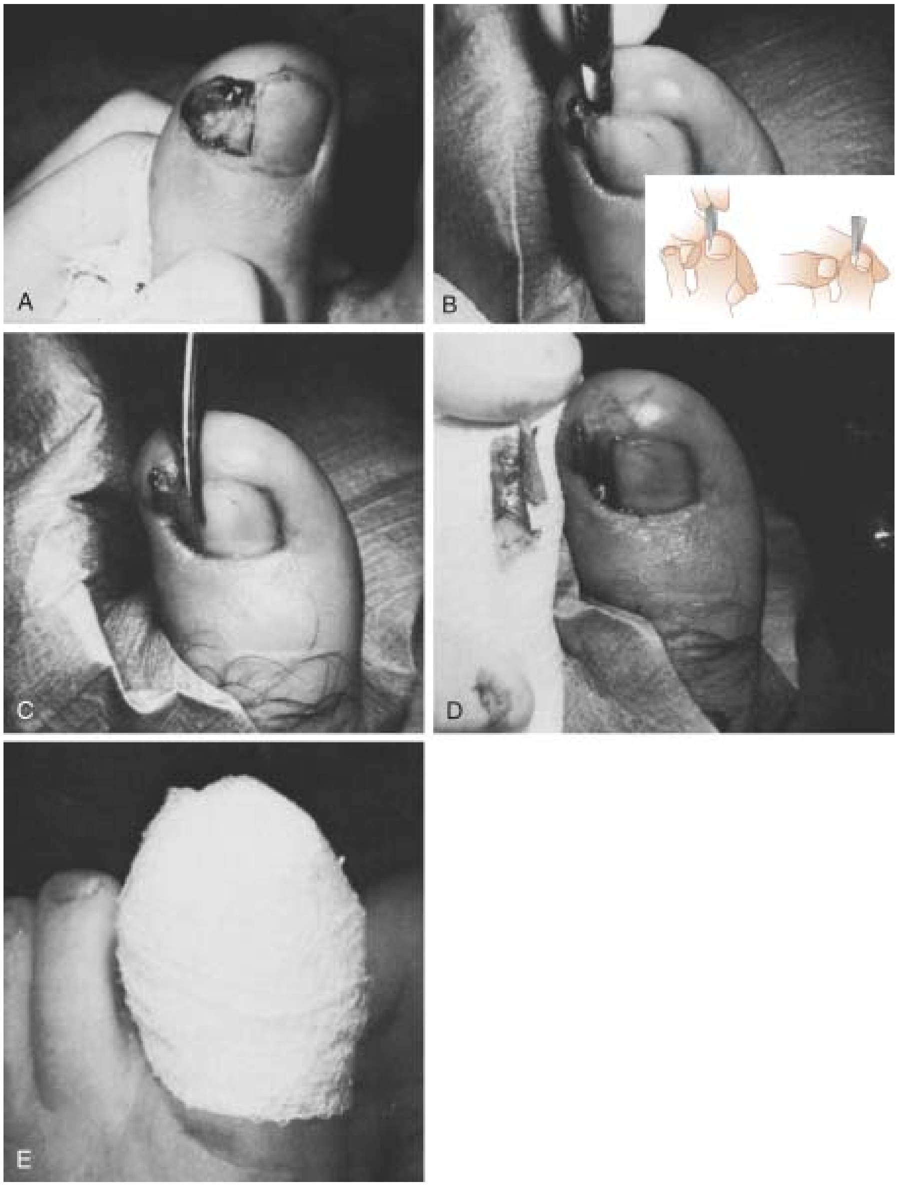

Figure 25H-44 Avulsion of edge of toenail. **A,** Stage III infection of medial edge of toenail with granulation tissue and purulence. **B,** Elevation of edge of toenail. **C,** Longitudinal sectioning of toenail. **D,** Avulsion of toenail edge. **E,** Typical postoperative compression dressing. *(From Mann RA, Coughlin MJ: Video Textbook of Foot and Ankle Surgery. St. Louis, Medical Video Production, 1991, pp 57, 61.)*

Complete Toenail Avulsion

When more extensive infection is present, a complete toenail avulsion may be necessary. After the nail plate is elevated proximally, the toenail is avulsed. Re-epithelialization of the nail bed occurs in 2 to 3 weeks. [155] As the nail plate regrows, the advancing edges must be elevated, or an ingrown toenail can develop (Fig. 25H-45). [165]

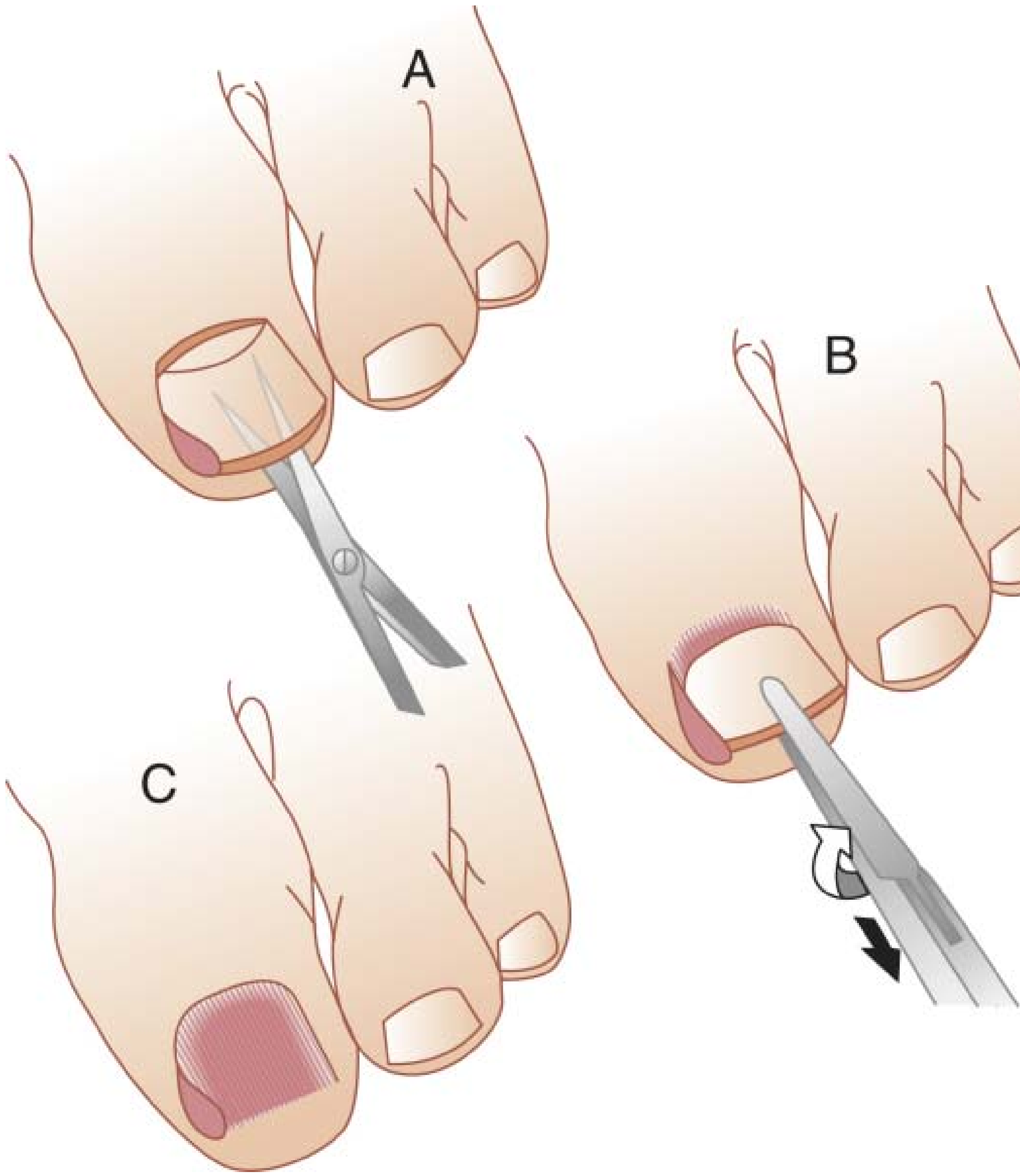

Figure 25H-45 Complete toenail avulsion for infection. **A,** Nail is elevated with sharp dissection. **B,** Nail is avulsed. **C,** Remaining nail bed and matrix after avulsion.

Plastic Nail Wall Reduction

A plastic nail wall reduction is performed only after an acute infection has resolved following either successful conservative care or partial or complete toenail avulsion. This technique is often used in younger patients. [1485] [1496] [1497] A wedge of tissue from the lateral toenail pulp, including skin and subcutaneous tissue, is excised from the medial or lateral border or from the tip of the toenail. The skin is approximated loosely with interrupted nylon sutures. Often this procedure reduces the prominent lateral toenail fold, removing the source of impingement of the lateral toenail edge (Fig. 25H-46).

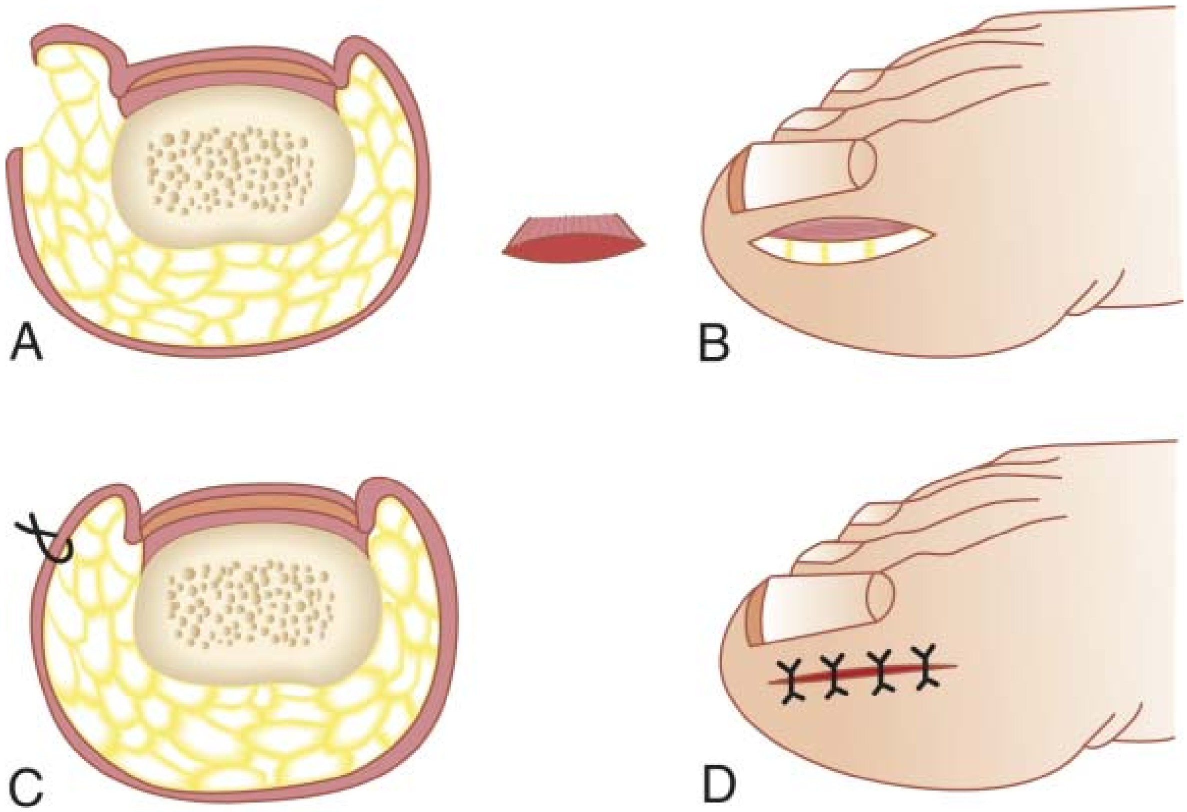

Figure 25H-46 Soft tissue plastic repair to decompress lateral nail fold. **A,** Cross section of plastic toenail repair. **B,** Lateral view shows elliptical skin incision and soft tissue excision. **C,** Cross section after closure of skin and soft tissue ellipse. **D,** Lateral view after closure of ellipse.

Partial Onychectomy (Winograd or Heifetz Procedure)

Partial onychectomy is performed only after an acute infection has resolved, usually following partial nail plate avulsion. [1486] [1498] [1499] A vertical incision is made along the edge of the toenail (usually where the previous nail plate edge was avulsed). The resection is carried distally to the terminal extent of the nail matrix (the Heifetz procedure) [156] or the terminal extent of the nail bed (the Winograd procedure) [168] and proximally to the proximal edge of the nail matrix. An oblique incision is made at the apex of the nail bed, and the proximal nail matrix and edge of the cuticle are removed. Care must be taken not to injure the extensor tendon insertion and not to violate the interphalangeal joint. The germinal matrix is characterized by a pearly white color and smooth texture. [155] The resection is carried into the nail fold laterally. The cortex of the distal phalanx is stripped of remaining matrix, and the skin is approximated with interrupted nylon sutures (Fig. 25H-47).

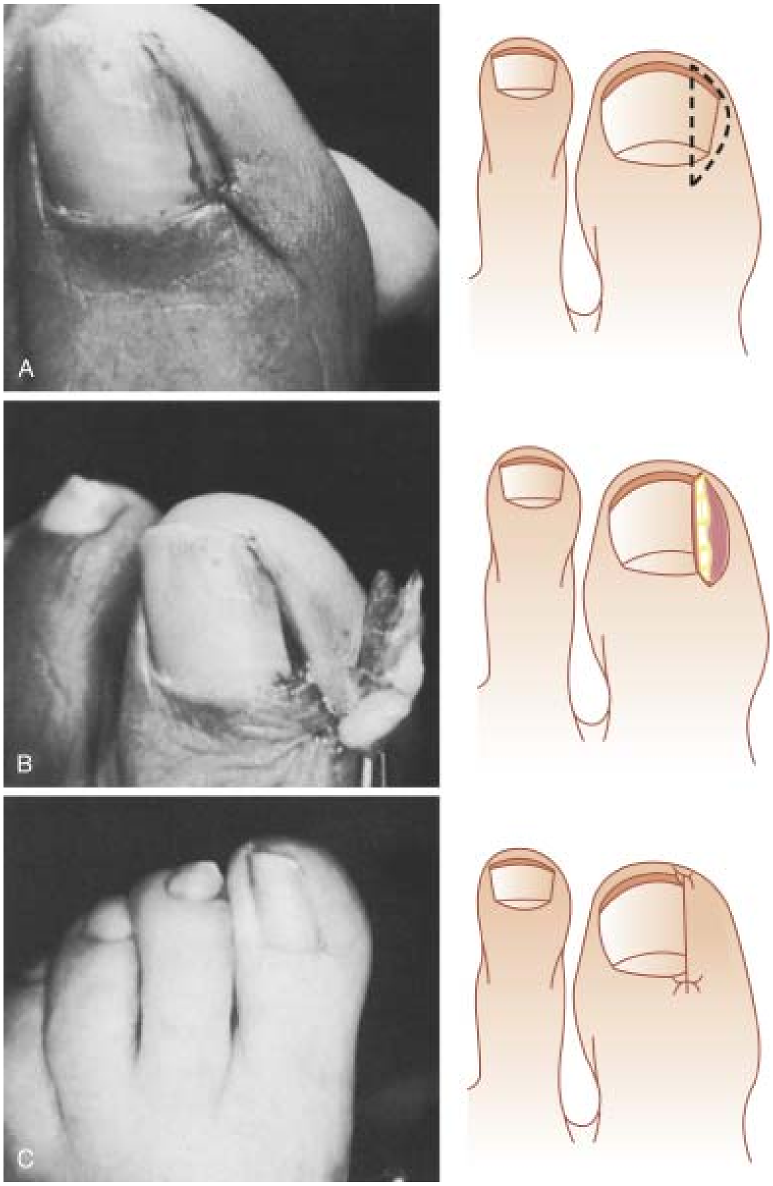

Figure 25H-47 Winograd procedure. **A,** Longitudinal incision of toenail matrix with oblique incision at apex. **B,** After excision of toenail matrix. **C,** Postoperative photograph shows bilateral Winograd procedure. *(From Mann RA, Coughlin MJ: Video Textbook of Foot and Ankle Surgery. St. Louis, Medical Video Production, 1991, p 63.)*

Complete Onychectomy (Zadik Procedure)

Occasionally, a patient desires a complete and permanent removal of a toenail. The procedure usually is performed after an initial toenail avulsion when previous infection has been present. [170] It is advantageous to delay surgery until the infection and inflammation have subsided completely. An oblique incision is made at the medial and lateral apices of the nail bed. The toenail is avulsed. The cuticle, eponychium, and proximal nail bed are excised completely, and the matrix is excised proximal to the cuticle and distally as far as the lunula. The resection includes each nail fold as well. The skin is approximated with interrupted nylon sutures loosely closing the proximal edge of the remaining nail bed (Fig. 25H-48). Excess tension on the suture line may lead to sloughing of the skin.

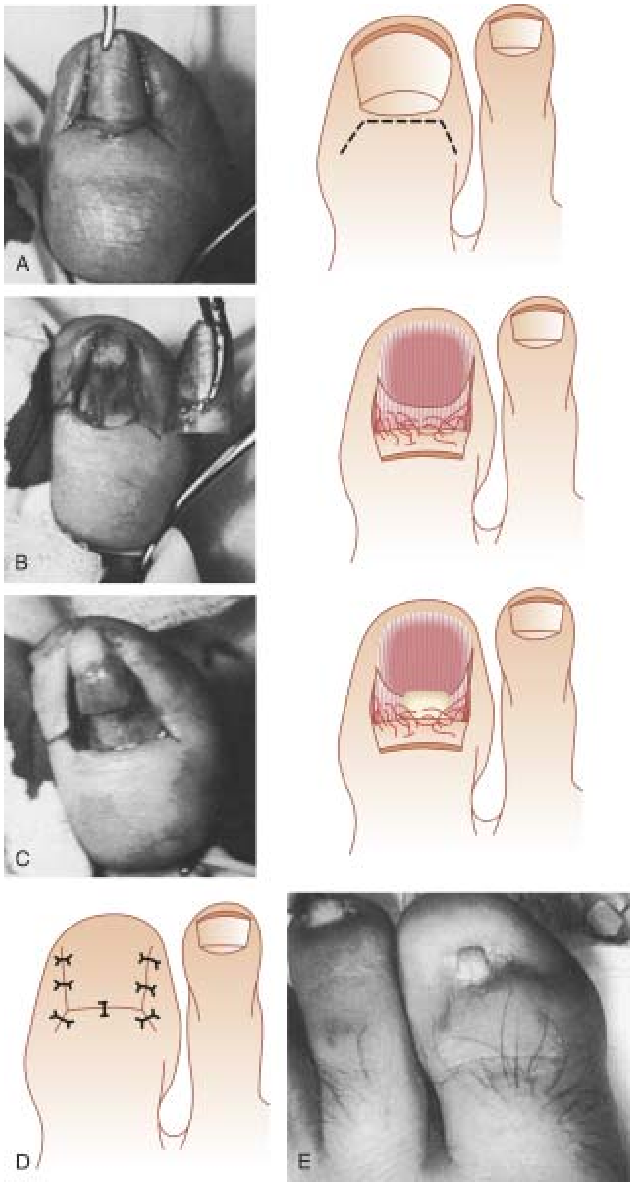

Figure 25H-48 Toenail ablation. **A,** Incisions are made at the apex of the lateral nail grooves, and the cuticle is excised. **B,** The toenail is avulsed. **C,** Toenail matrix and lateral nail fold are excised. **D,** Skin edges are loosely approximated. **E,** Occasionally, recurrence of toenail tissue may develop after an attempt at complete toenail ablation. *(From Mann RA, Coughlin MJ: Video Textbook of Foot and Ankle Surgery. St. Louis, Medical Video Production, 1991.)*

Syme Amputation (Thompson-Terwilliger Procedure)

For symptomatic recurrence or when the patient requests a more reliable ablation, Thompson and Terwilliger [171] described a Syme amputation of the distal phalanx. An elliptical incision is used to resect the entire nail bed, matrix, lateral and proximal nail folds, cuticle, and proximal border of skin. About half of the distal phalanx is removed, and the remaining edges of bone are beveled. Skin edges are approximated with interrupted nylon sutures (Fig. 25H-49).

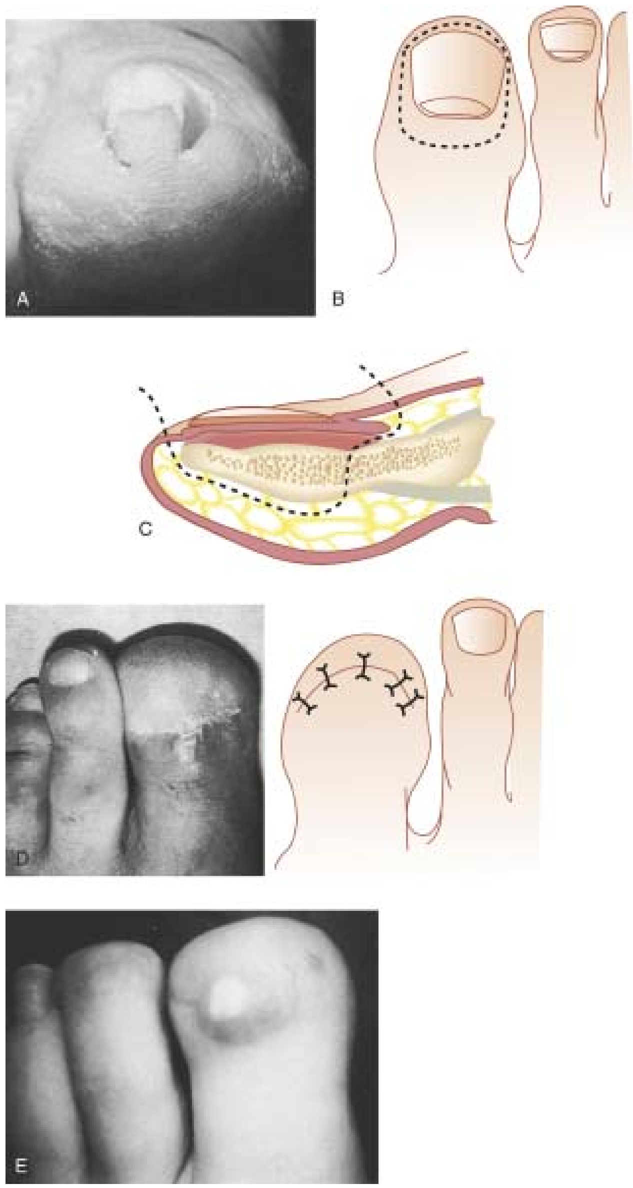

Figure 25H-49 **A,** Chronic incurved toenail. **B,** Proposed excision. **C,** Cross section shows area of resection. **D,** After Syme amputation of the great toe. **E,** Development of inclusion cyst with recurrence after attempted Syme amputation. *(**A** and **E,** From Mann RA, Coughlin MJ: Video Textbook of Foot and Ankle Surgery. St. Louis, Medical Video Production, 1991; **B** to **D,** © M. J. Coughlin. Used by permission.)*

Phenol and Alcohol Matrixectomy

A phenol and alcohol matrixectomy may be used rather than surgical resection. [1485] [1502] [1503] The advantage of this technique is that it can be performed in the presence of concurrent infection. The nail plate edge is avulsed as previously described. The toenail groove and nail matrix are curetted. A cotton-tipped applicator is used for the procedure, but most of the cotton is removed from the applicator so that it can fit along the area of the lateral nail fold where the toenail has been avulsed and into the matrix area below the eponychium. The cotton-tipped applicator is moistened with 88% fresh carbolic acid (phenol), and the excess is blotted on a gauze pad. The applicator is inserted into the nail groove and matrix area for 1 minute. The applicator is removed, and the area is flushed with alcohol. After this application, two subsequent 30-second phenol applications are performed. After each application, alcohol is used to flush the nail groove and matrix region. Any area of skin that should be protected is covered with petroleum jelly to prevent injury to the tissue (Fig. 25H-50).

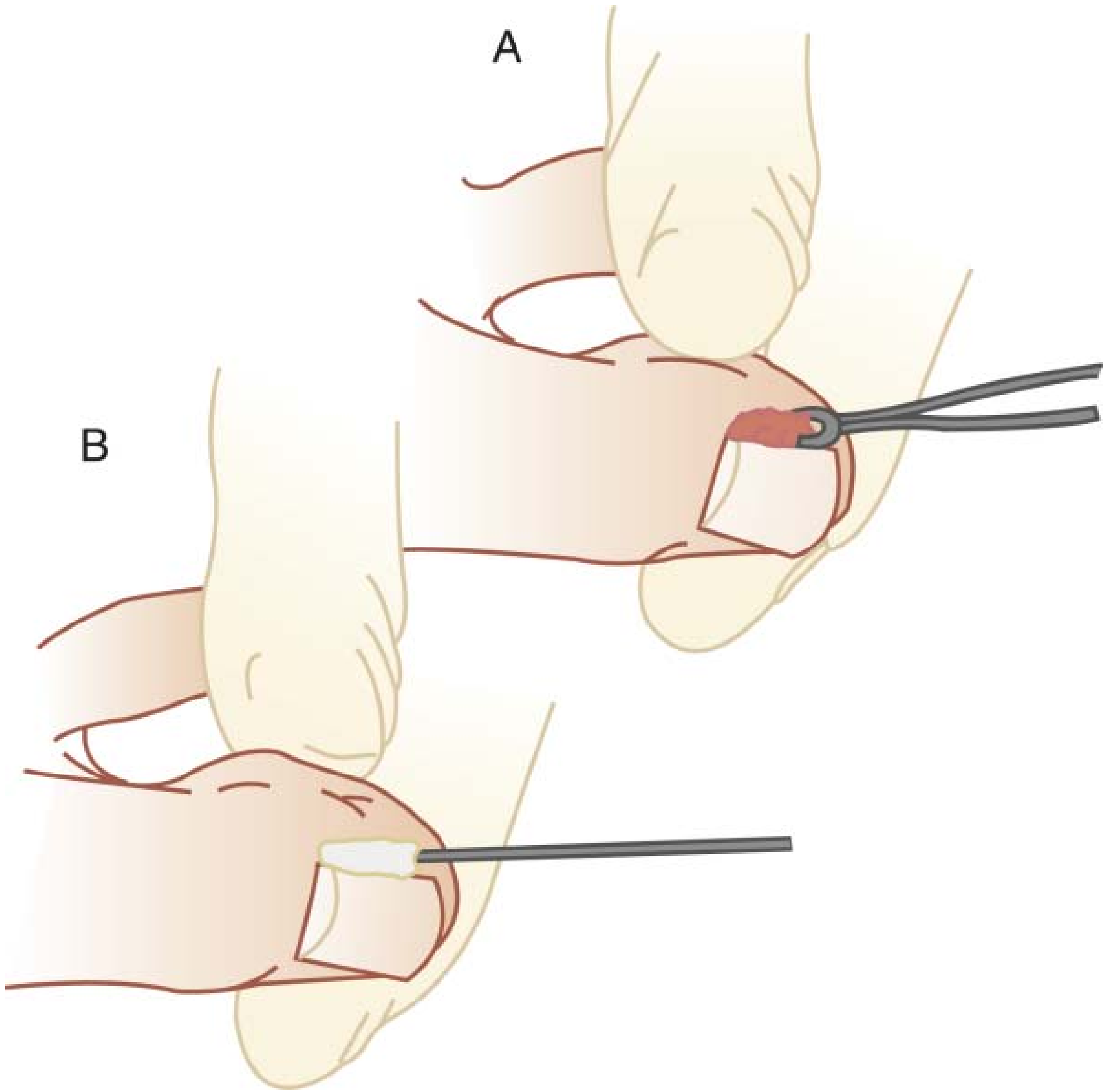

Figure 25H-50 Phenol ablation of toenail edge. **A,** Curettage of nail bed and matrix. **B,** Phenol application with cotton-tipped applicator along nail edge.

Weighing the Evidence

The most frequent complication after attempted toenail ablation is recurrence. After partial nail avulsion, the recurrence rate with subsequent infection is 47% to 77% when further definitive care is deferred. [1495] [1504] Murray [175] estimated that recurrent infection occurred in 64% of cases after the initial nail avulsion and in 86% of cases after a second toenail avulsion in the absence of definitive treatment. Although a toenail avulsion gives dramatic relief not only of an infection but also of symptoms, it has a poor long-term cure rate, and usually a second procedure is needed. Dixon [155] noted a much higher recurrence rate in patients in whom multiple avulsions of a single toenail have been performed. Lloyd-Davies and Brill [165] reported that within 6 months after nail avulsion, 31% of patients required further treatment. Palmer and Jones [176] reported a 70% recurrence rate after a nail plate avulsion. Plastic nail wall reduction has a reported recurrence rate of 25%. [174]

After partial onychectomy with either a Winograd or a Heifetz procedure, recurrence rates of 6% to 33% have been reported. [1498] [1504] [1506] [1507] [1508] Murray and Bedi [177] noted a 27% recurrence rate after an initial Winograd procedure and a 50% recurrence rate if a double Winograd procedure was performed. Gabriel and colleagues [179] reported a 1.7% recurrence rate with a 79% satisfaction rate following this procedure. Wadhams and coworkers [180] reported the development of 10 epidermal inclusion cysts after 147 partial matrixectomies (7%) at an average of 5.5

months after surgery.

Complete toenail ablation procedures are associated with a recurrence rate of 16% to 50%. [1506] [1507] [1511] All patients should be informed that minor recurrence of toenail tissue can emanate from the remaining toenail bed. Although this regrowth is not a significant problem, occasionally a repeat excision is necessary. The cause of this recurrent nail plate growth may be germinal cells within the nail bed.

Pettine and associates [178] found that the Heifetz procedure had a 90% rate of patient satisfaction and a recurrence rate of 6%. Nail excision with chemical matrixectomy had an 80% rate of patient satisfaction and a 20% recurrence rate. Fewer patients underwent complete nail ablation and a Syme procedure; for both techniques, a satisfaction rate of about 80% to 90% was reported. Shaath and associates [182] compared recurrence after complete ablation with the Zadik procedure with chemical ablation. They found a recurrence rate of 60.5% in the patients treated surgically compared with 15.6% after chemical ablation. Murray and Bedi [177] concluded that because of the recurrent nail plate regrowth after either a Winograd or partial or complete nail plate ablation a terminal Syme procedure was the procedure of choice.

AUTHOR'S PREFERRED METHOD

The goal in treatment of toenail infections is to control the infection initially. A partial toenail avulsion is preferable in the presence of acute or chronic infection. Once the infection has resolved, definitive surgery can be contemplated, but surgery can be deferred depending on the patient's desire to participate in competitive sports.

A plastic nail wall reduction is useful for younger patients with mild disease. Excision of the toenail edge and matrix (the Winograd procedure) is also preferable because of its high rate of predictability. Complete nail ablation is complicated by a high rate of recurrence as a result of the presence of microscopic nail matrix in the nail bed. This procedure is preferable for a chronic toenail abnormality or for recurrent toenail infections that have not been amenable to more conservative methods. A Syme amputation is associated with a low rate of recurrence but is a more radical procedure and probably is less cosmetically acceptable to patients. Syme amputation should be reserved for more chronic toenail problems in older patients.

Postoperative Prescription, Outcomes Measurement, and Potential Complications

Postoperative care after a toenail avulsion requires initial hemostasis, a compressive dressing, and efforts to reduce inflammation and infection. Usually, removal of the toenail edge allows rapid resolution of inflammation. Prophylactic antibiotics are used in more severe infections, but for the most part, antibiotics are not necessary. Soaks in tepid salt solution should be initiated within 24 to 48 hours after toenail avulsion.

Aftercare is important after a partial toenail avulsion. As the toenail regrows, the advancing edge is at risk for recurrent infection. A cotton wisp must be placed beneath the nail plate to elevate it as the edge grows distally. A digital block may be necessary to replace the initial packing. The patient is instructed in the packing procedure, and continues to perform the nail edge packing until the advancing nail edge has grown past the distal edge of the nail groove.

With more definitive procedures such as complete or partial toenail ablation or a Syme amputation, a compression dressing is applied and changed 24 hours postoperatively. Repeat dressings are changed intermittently until there is no further drainage. Then a bandage is used to protect the toe. Sutures are removed 2 to 3 weeks after surgery.

A sterile dressing is applied and changed daily after phenol and alcohol matrixectomy. The patient is instructed to soak the foot daily in a tepid salt solution. Daily soaking continues until the drainage has subsided, usually between 2 and 8 weeks.

Criteria for Return to Play

In athletes, a partial or complete toenail avulsion is used to resolve an infectious process quickly, allowing the patient to return to competitive athletics within a short time. A patient is allowed to return to athletic participation after the inflammation has subsided and drainage is minimal. Aggressive running, jumping, and cutting and contact sports are possible after the inflammation subsides.

Definitive surgery, such as partial or complete toenail ablation or a Syme amputation of the toe, should be deferred until adequate time for healing is available. These procedures are reserved best for the off-season or when an athlete can afford several weeks of down time. When definitive surgery is performed, the patient must be willing to avoid significant running activities until adequate healing has occurred (3 to 6 weeks) (Box 25H-31).

BOX 25H-31

CRITERIA FOR RETURN TO PLAY AFTER TREATMENT OF INGROWN TOENAIL

- Resolution of pain and inflammation after conservative care or partial or complete toenail avulsion
- Complete wound healing after definitive nail ablation procedures (usually 3-6 weeks)

SUBUNGUAL EXOSTOSIS

A subungual exostosis is a benign tumor of the distal phalanx most commonly occurring in the great toe. [1523] [1524] [1525] [1526] An osteochondroma has a similar clinical presentation; however, the two entities are characterized by differences in histology, [187] age of occurrence, [1523] [1525] [1528] [1529] and etiology. [1523] [1528] [1530] [1531] [1532] [1533]

Anatomy and Biomechanics

The histologic differentiation of the cartilage cap is the primary distinguishing factor between a subungual exostosis and a subungual osteochondroma. [187] A subungual exostosis is composed of a fibrocartilaginous cap with reactive fibrous growth and cartilage metaplasia. [1513] [1517] Jahss [194] suggested that the histology is characteristic of chronic irritation. A trabecular bone pattern typically underlies the fibrocartilaginous cap and connects with the distal phalanx. [183] In a subungual osteochondroma, hyaline cartilage composes the cartilaginous cap; however, a trabeculated bone pattern often underlies the cap.

Ippolito and colleagues [189] reported a high incidence of subungual exostosis in individuals participating in sports. In 21 patients with subungual exostosis, 12 were noted to be dancers, gymnasts, cyclists, or football players. Pressure of the toe box against the dorsal aspect of the nail plate often causes discomfort, [195] and athletic activity tends to exacerbate the symptoms. The association of trauma with the later development of subungual exostosis, as suggested by Miller-Breslow and Dorfman [183] and Bendel, [190] may indicate a high risk for this abnormality in athletes. Many patients describe a history of pain that is aggravated by walking or running. This pain is due to the pressure of an expanding lesion beneath the nail plate that is traumatized by extrinsic pressure from either the end or the medial aspect of the toe box.

Classification

A specific classification system for subungual exostoses has not been described. However, several key factors help distinguish a subungual exostosis and subungual osteochondroma (Table 25H-3). [1513] [1515] [1517] [1518] [1519] [1520] [1521] [1522] [1523] [1526]

TABLE 25H-3 -- Subungual Exostosis Versus Subungual Osteochondroma

	Age (yr)	Gender	Etiology	Histology	Radiographic Features
Subungual exostosis	20-40	Female	Trauma	Fibrocartilage cap	Trabeculated bone from dorsomedial tuft
Subungual osteochondroma	10-25	Male		Hyaline cartilage	Trabeculated bone from juxtaepiphyseal region

Evaluation

Clinical Presentation and History

The patient often gives a history of discomfort with excessive walking, jogging, or running. An athlete may give a history of traumatic injury to the toe with later development of pain (Box 25H-32).

BOX 25H-32

TYPICAL FINDINGS IN PATIENTS WITH SUBUNGUAL EXOSTOSIS

- Pain occurs with activities such as walking or running.
- Discomfort is elicited on examination with direct pressure on the dorsal nail.

- Lateral or oblique radiographs best show the lesion.
- The bony portion of the lesion may be smaller than the actual size due to the fibrocartilaginous cap.

Physical Examination

On physical examination, direct pressure on the dorsal aspect of the toenail causes discomfort. A subungual hemorrhage can occur and is often mistaken for a chronic subungual hematoma, when in fact the nail has been elevated by an underlying exostosis. [190] Pressure of an underlying subungual exostosis often separates the toenail from the toenail bed. [195] Occasionally, the mass will penetrate the nail plate and can be mistaken for a malignant tumor (see Box 25H-32). [186]

A subungual exostosis is frequently misdiagnosed or confused with other toenail abnormalities. [189] Elevation of the nail bed can resemble a subungual hematoma or chronic onychomycosis (Fig. 25H-51). Differential diagnosis includes subungual verruca, pyogenic granuloma, glomus tumor, melanotic whitlow, keratoacanthoma, carcinoma of the nail bed, subungual nevus, subungual melanoma, and myositis ossificans. [1513] [1514] [1518] [1519] [1523] [1527]

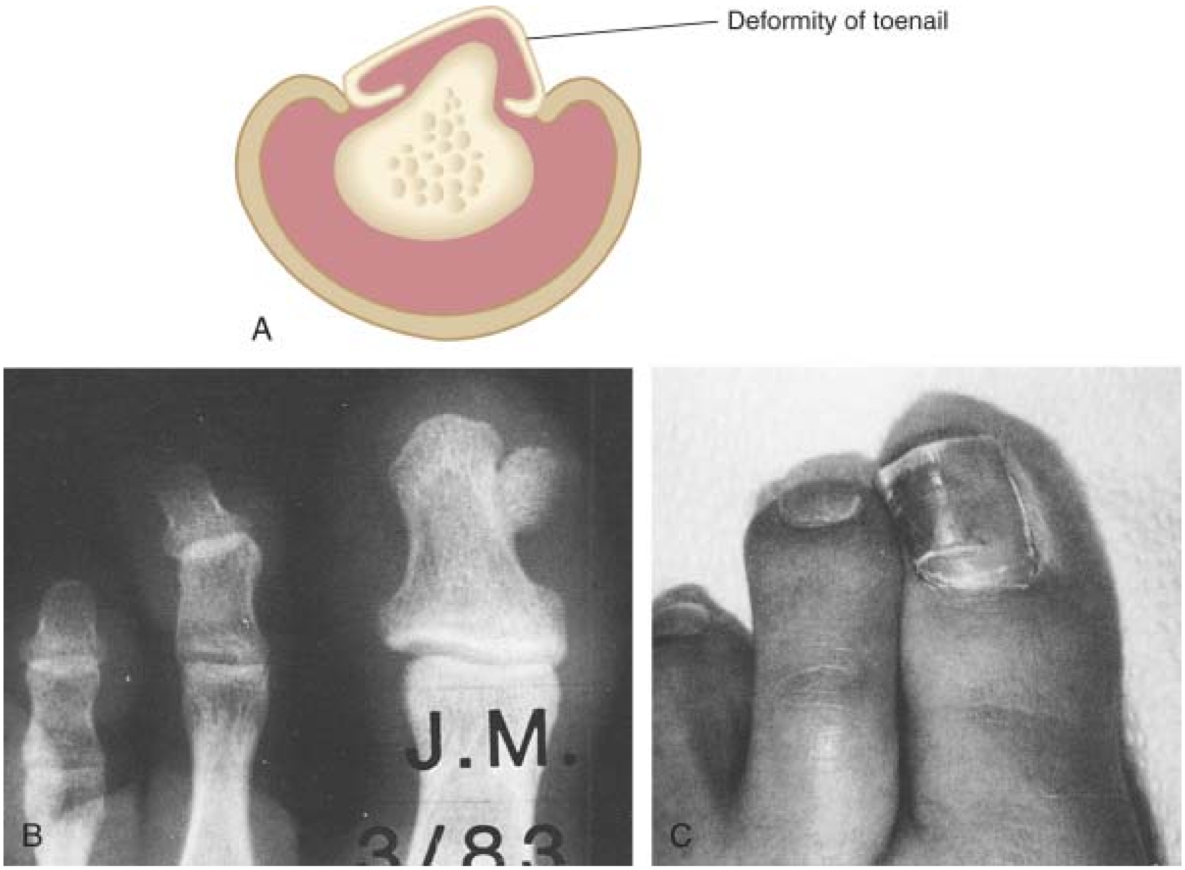

Figure 25H-51 **A,** Subungual exostosis may elevate the toenail and may be misdiagnosed as a chronic fungal infection. **B,** Anteroposterior radiograph shows subungual exostosis (same patient). More commonly, a lateral or an oblique radiograph may be used to show the exostosis. **C,** Often, the toenail is deformed. The exostosis often deviates in a medial direction. *(**B** and **C**, From Mann RA, Coughlin MJ: Video Textbook of Foot and Ankle Surgery. St. Louis, Medical Video Production, 1991.)*

Imaging

The key to the correct diagnosis is radiographic demonstration of the exostosis. [198] Lateral or oblique radiographs are most helpful when the anteroposterior radiograph fails to show the lesion. The exostosis arises from the dorsal medial aspect of the distal phalanx (see Fig. 25H-56). It is often oval in shape and irregular in density. [195] The trabecular pattern is usually contiguous with the distal phalanx. Often, if the cartilage cap is of significant size, radiographs show a bony lesion much smaller than the actual size of the growth (see Box 25H-32).

The major reason for delay in diagnosis of a subungual exostosis is the misdiagnosis of the condition as a toenail deformity or as onychomycosis. The presence of a painful deformed toenail in a younger athlete may be an indication of a subungual exostosis. Radiographic examination is the key to accurate diagnosis.

Treatment Options

Nonoperative

An asymptomatic subungual exostosis seen on radiographs does not require intervention. Once the exostosis becomes symptomatic, surgical treatment is preferred.

Operative

Surgical resection of a subungual exostosis is the most frequent treatment. A partial or complete toenail avulsion is performed depending on the size and location of the exostosis (Fig. 25H-52). A longitudinal incision is made in the nail bed, and the nail bed is reflected from the exostosis. Care should be taken to avoid damage to the toenail matrix. The exostosis is resected with an osteotome or bone cutter, and the base is curetted. The nail bed is relocated and sutured when possible. The wound is covered with a compression dressing to encourage hemostasis.

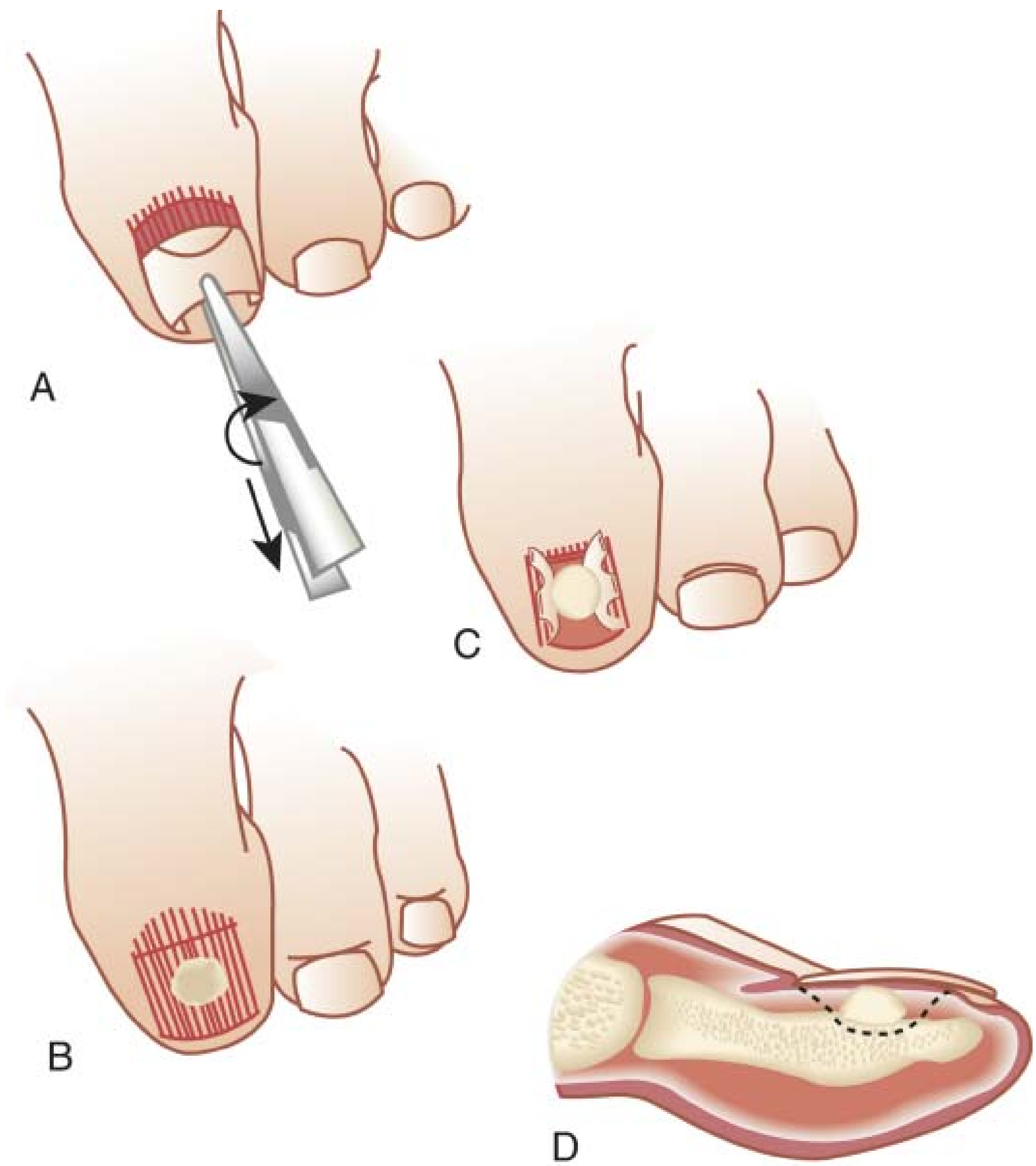

Figure 25H-52 Resection of subungual exostosis. **A,** The toenail is avulsed. **B** and **C,** The toenail matrix is incised longitudinally, then peeled off the exostosis to allow adequate exposure. **D,** The exostosis is resected at its base, and the base is curetted (see line of resection).

Weighing the Evidence

DePalma and colleagues [199] reported successful resection in 11 cases. Recurrence of the exostosis, although not a common occurrence, results if incomplete resection occurs [1518] [1519] or if a continuing source of irritation is present. Miller-Breslow and Dorfman [183] noted a 53% incidence of recurrence when subtotal excisional biopsy was performed. With wide local excision and curettage of the base, a 5% to 6% rate of recurrence can be expected. [192] Fickry and coworkers [200] reported a recurrence in 4 of 28 cases without evidence of malignant transformation.

Postoperative toenail deformity can occur if the surgical dissection damages the nail matrix. Ippolito and associates [189] reported a 66% rate of nail deformity or absence of nail growth after resection of a subungual exostosis. Although postoperative infection is a possibility with surgical resection of the exostosis, the infrequent occurrence of infection is most likely due to the fact that these lesions are not associated routinely with active infection or ingrown toenails.

AUTHOR'S PREFERRED METHOD

Partial or complete toenail avulsion followed by a wide surgical excision and curettage of the base is the preferred treatment. A patient should be informed that surgical excision may be complicated by recurrence or by postoperative toenail deformity caused by toenail matrix injury.

Postoperative Prescription, Outcomes Measurement, and Potential Complications

Weight-bearing in a postoperative shoe begins immediately after the procedure. The surgical dressing is changed at the first postoperative visit preferably 1 to 3 days after surgery. Dressing changes are continued until the wound is clean and dry, and the nail bed is protected until tenderness resolves. Sutures are removed 2 weeks after surgery. Athletic shoes are permitted once the nail bed is nontender and the postoperative wound is healed.

Criteria for Return to Play

Gradual return to sport occurs when the wound is healed and all pain, swelling, and drainage resolve. This usually occurs 4 to 6 weeks after surgery.

INTRACTABLE PLANTAR KERATOSES

An IPK is a localized callosity occurring on the plantar aspect of the foot. [201] An isolated keratotic lesion typically develops beneath a bony prominence as a direct result of increased pressure or friction. Establishing the correct diagnosis is essential to treatment. Care must be taken to distinguish an IPK from other lesions of the plantar skin (Box 25H-33 ; Figs. 25H-53 to 25H-55 [1195] [1200] [1205]). [1532] [1533]

BOX 25H-33

DIFFERENTIAL DIAGNOSIS OF INTRACTABLE PLANTAR KERATOSIS

- Verrucae plantaris, plantar wart (see Fig. 25H-53)
- Seed corn (small, well-circumscribed)
- Diffuse callous formation (see Fig. 25H-54)
- Discrete well-localized callous formation (see Fig. 25H-55)
- Epidermal cysts
- Blistering or ulceration due to vascular insufficiency

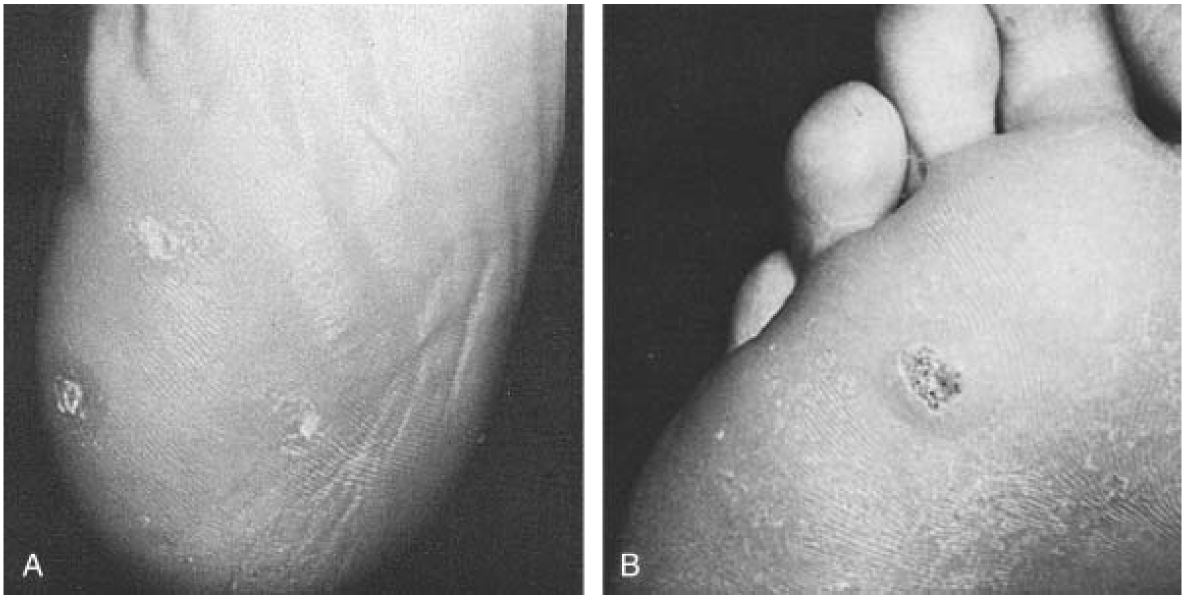

Figure 25H-53 **A,** Verruca plantaris, or wart. Warts usually are located in areas other than beneath the metatarsal head (e.g., heel region). **B,** Wart beneath the third metatarsal head. *(**A,** © M. J. Coughlin. Used by permission; **B,** from Mann RA, Coughlin MJ: Video Textbook of Foot and Ankle Surgery. St. Louis, Medical Video Production, 1991, p 86.)*

Figure 25H-54 Diffuse plantar keratosis. *(© M. J. Coughlin. Used by permission.)*

Figure 25H-55 A well-circumscribed intractable plantar keratosis resulting from a prominent fibular condyle. *(From Mann RA, Coughlin MJ: Video Textbook of Foot and Ankle Surgery. St. Louis, Medical Video Production, 1991, p 86.)*

Generalized callus can develop in the forefoot of an athlete as a result of increased pressure. It is normal for a moderate amount of callus to form. In contrast, an IPK is a well-localized keratotic lesion in an area of mechanical irritation such as friction or pressure.[201] Lesions may be either discrete or diffuse.

Anatomy and Biomechanics

Anatomic abnormalities often lead to callus formation. A plantarflexed metatarsal (Fig. 25H-56), [1534] [1535] an elongated metatarsal in relation to adjacent metatarsals, [1531] [1534] [1536] or a malunion of a metatarsal after fracture[204] can cause increased pressure beneath a metatarsal and subsequent callus formation. Positional deformities[204] (Box 25H-34 ; Fig. 25H-57) often lead to malalignment manifested by increased pressure beneath one or more metatarsal heads. Jahss[204] noted that minor physiologic variations in the athlete can lead to development of callosities as a result of the repeated stresses that are involved with particular sporting activities. The repetitive stresses of athletic activity such as running, jogging, or walking increase mechanical irritation to vulnerable areas, frequently leading to increased symptoms.

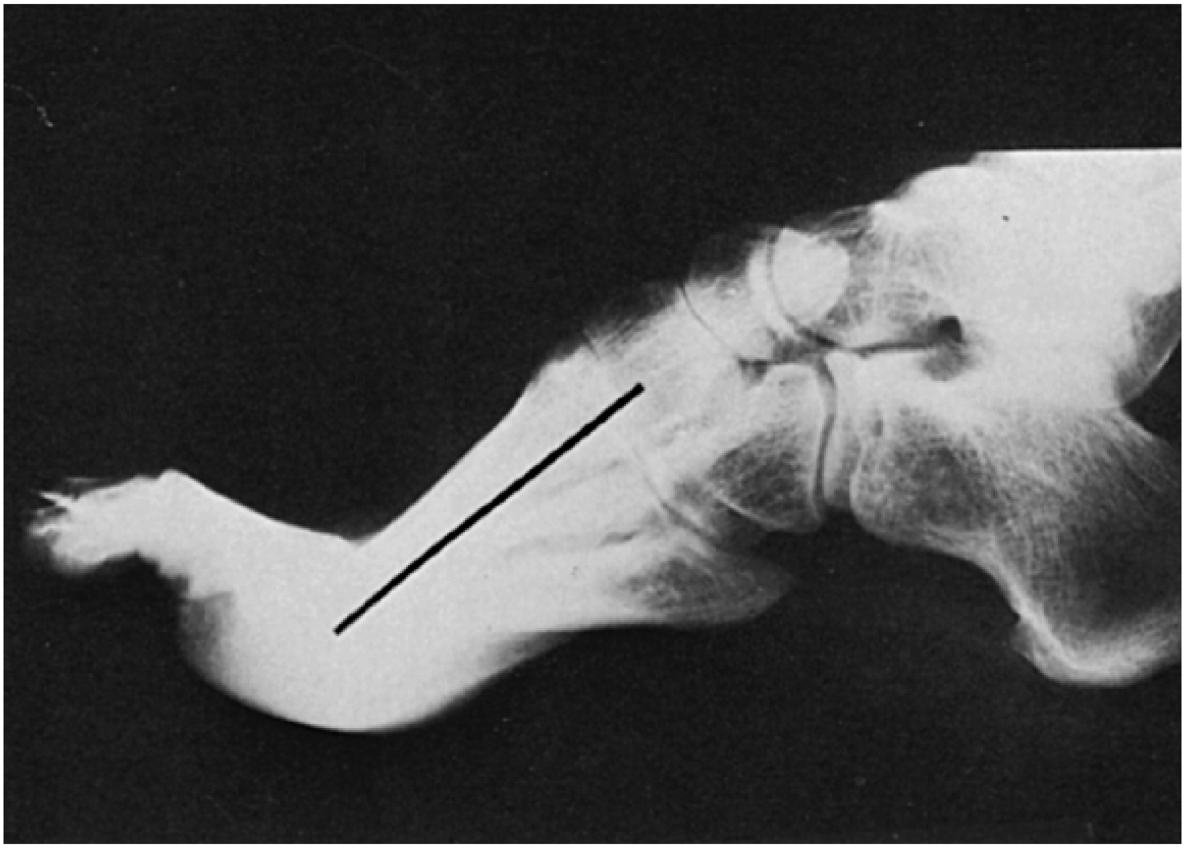

Figure 25H-56 Plantar flexed first metatarsal. *(© M. J. Coughlin. Used by permission.)*

BOX 25H-34

POSITIONAL DEFORMITIES ASSOCIATED WITH INTRACTABLE PLANTAR KERATOSIS FORMATION

- Ankle equinus
- Forefoot equinus
- Cavus midfoot coupled with a rigid forefoot (see Fig. 25H-62)
- Rotatory deformity of the midfoot
- Varus or valgus malalignment of the forefoot

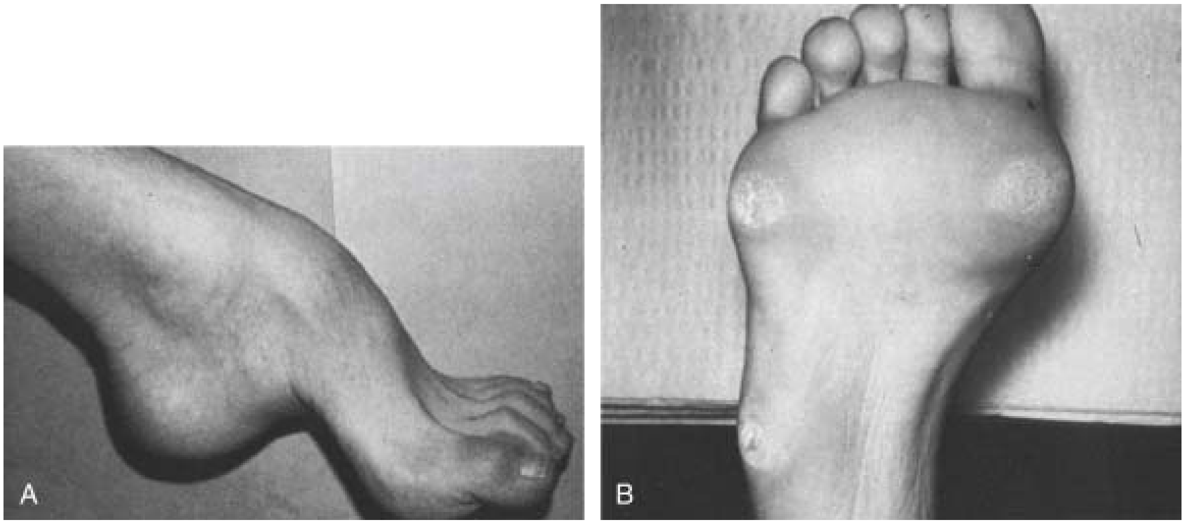

Figure 25H-57 **A** and **B,** A cavus deformity may lead to multiple areas of callus formation. *(© M. J. Coughlin. Used by permission.)*

The first, fourth, and fifth metatarsal-tarsal articulations allow considerable flexibility compared with the more rigid second and third metatarsal-tarsal articulations.[204] Reduced flexibility in the middle rays can increase susceptibility to pressure buildup beneath the metatarsal heads. The length of individual metatarsals is variable, but in some situations, an abnormally long second metatarsal (Fig. 25H-58) or second and third metatarsals subject a forefoot to increased weight-bearing, leading to symptoms of increased pressure. [1537] [1538] [1539]

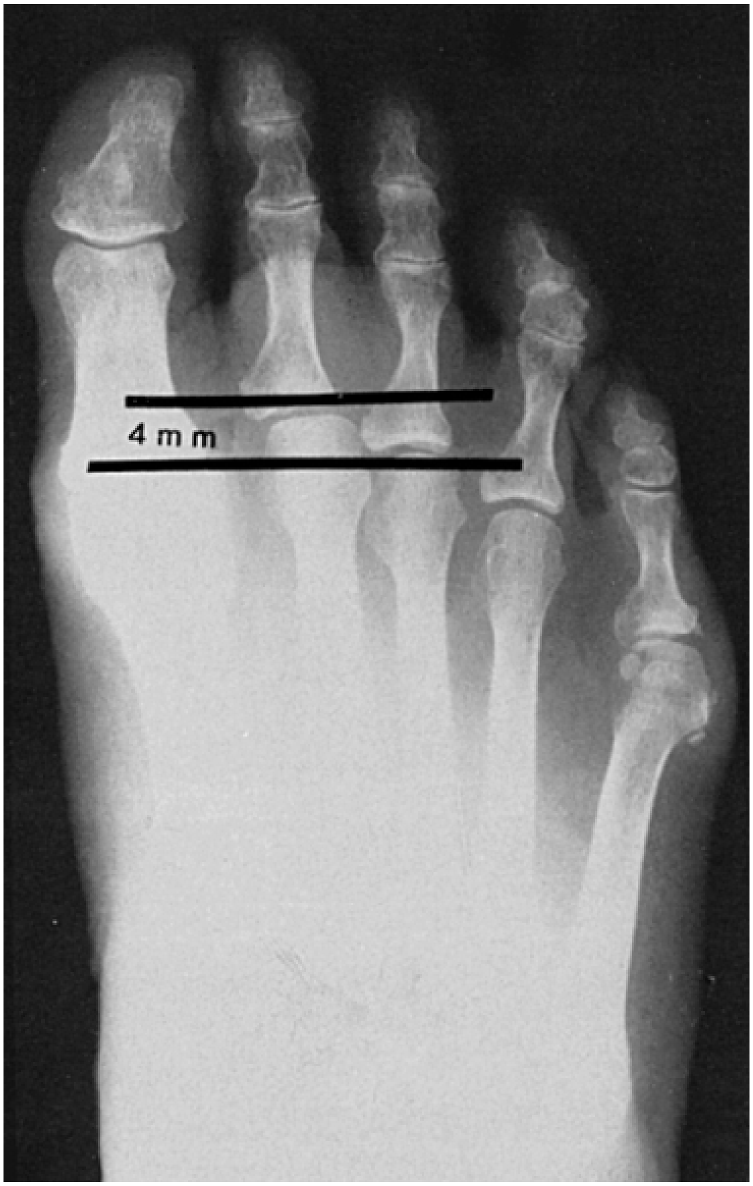

Figure 25H-58 A long second metatarsal may lead to increased callus formation. *(© M. J. Coughlin. Used by permission.)*

Midfoot flexibility and alignment can have a considerable effect on the distal metatarsals. A pes planus configuration presents a flexible forefoot that rarely is associated with plantar keratoses.[204] However, a cavus midfoot is characterized by decreased flexibility and is associated with a higher incidence of plantar keratoses. A rotary midfoot deformity (Fig. 25H-59) can result in varus or valgus malalignment. A varus abnormality often causes increased pressure on the fifth ray, whereas a valgus malalignment is characterized by increased pressure beneath the first ray. Forefoot equinus can occur without a significant hindfoot deformity, presenting as a rigid forefoot that is less resistant to pressure beneath the metatarsal heads.

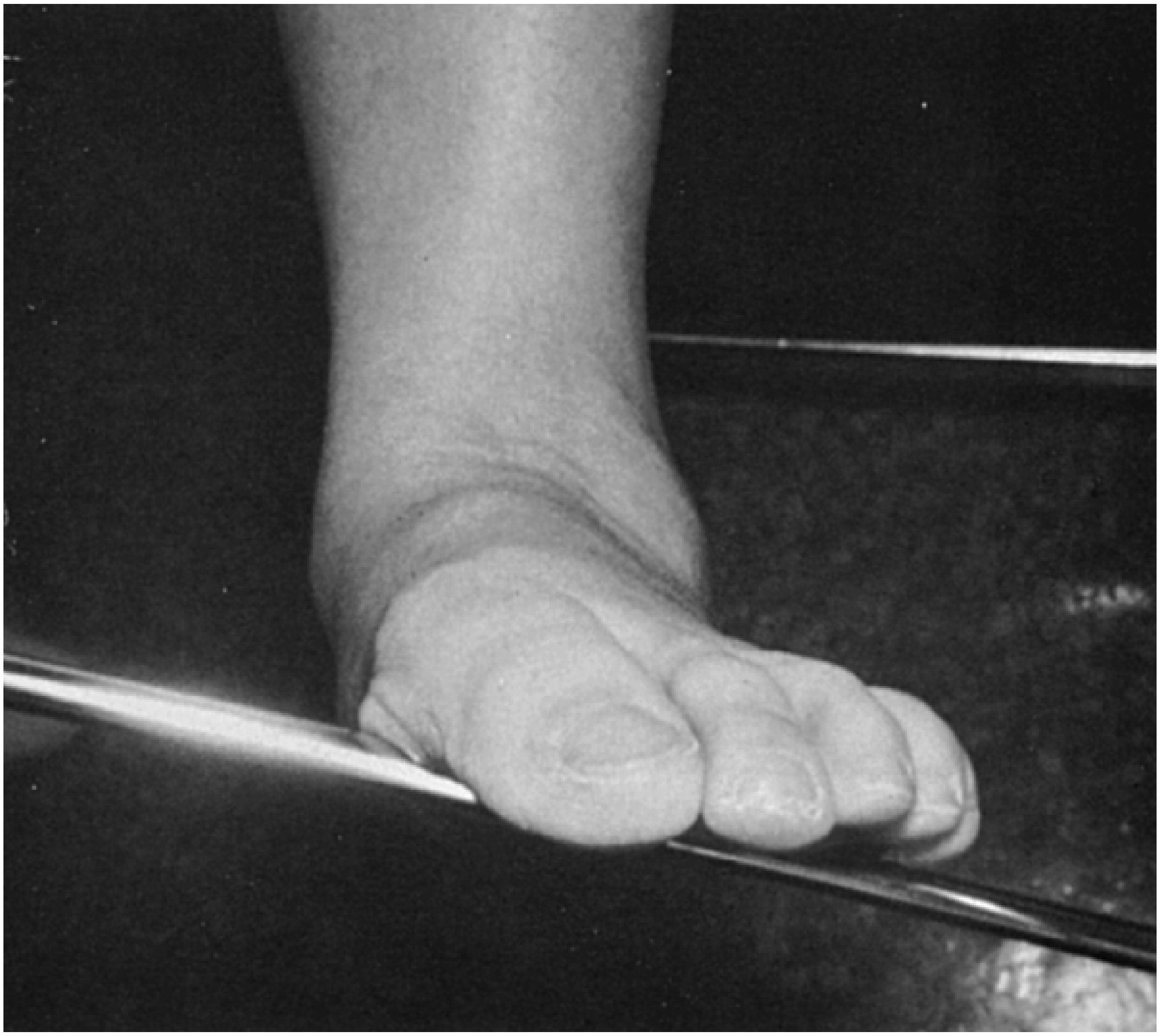

Figure 25H-59 A rotatory midfoot deformity may subject the lateral border of the foot to callus formation. A varus deformity of the forefoot with a fixed hindfoot deformity is shown. *(© M. J. Coughlin. Used by permission.)*

In differentiating a plantar wart from an IPK, there are significant differences not only in the cutaneous presentation but also in location. Typically, an IPK is located under the weight-bearing part of the forefoot beneath the metatarsal heads. [203] A wart is rarely located under a metatarsal head and is more likely to be seen under other areas on the plantar aspect of the foot. When an IPK is trimmed, a small, pearl-gray avascular core is noted. This keratotic core is a buildup of the plantar callosity that invaginates owing to localized pressure beneath the metatarsal head. A wart is a well-localized lesion with sharp margins. When shaved, it bleeds vigorously because of the end arterioles that are present in the lesion.

Classification

Keratotic lesions have not been specifically classified. Lesions are usually characterized as discrete or diffuse (Box 25H-35).

BOX 25H-35

Classification of Intractable Plantar Keratosis

- Small core (or seed corn) with keratotic buildup around the periphery
- Well-circumscribed lesion (<1 cm in size)

- Broad diffuse lesion (>1 cm in size)

Evaluation

Clinical Presentation and History

Clinical evaluation includes a thorough history of the aggravating and relieving factors related to the lesion. Athletes often report increased symptoms with specific activities or shoewear. Pain is usually directly under the callus (Box 25H-36).

BOX 25H-36

TYPICAL FINDINGS IN PATIENTS WITH INTRACTABLE PLANTAR KERATOSES

- Patients report pain directly under the callus.
- Patients may have hindfoot, midfoot, or forefoot deformity that leads to areas of increased pressure.
- Trimming of the callus reveals a well-circumscribed, keratotic lesion occurring directly under a bony prominence.
- Keratotic lesions are avascular, so significant bleeding with trimming may indicate the presence of a plantar wart.
- Radiographs may reveal a bony lesion adjacent to the callus.

Physical Examination

Physical examination first includes careful evaluation of the alignment of the foot when standing. Position of the hindfoot, the arch, and any great toe or lesser toe deformity should be noted. The plantar aspect of the foot should be carefully evaluated, noting the characteristics and location of any lesions. Trimming of the lesion usually reveals a well-circumscribed keratotic lesion. Keratotic lesions occur directly under a bony prominence, whereas plantar warts are less likely to occur underneath the metatarsal heads.[203] Warts are quite vascular, so bleeding will be encountered with trimming a wart, unlike keratotic lesions, which are avascular (see Box 25H-36).

Imaging

Evaluation includes weight-bearing views of the foot. Sesamoid views are included depending on location of the lesion. Careful attention is given to bony abnormalities that may be responsible for areas of increased pressure (see Box 25H-36).

Treatment Options

Nonoperative

The initial treatment of an IPK involves trimming the lesion to reduce the keratotic buildup. The keratotic center of a callus is delineated as it is shaved. This keratosis is a typical response to increased pressure; with time, the lesion invaginates slowly, and the keratosis increases in depth. As it deepens, it typically becomes more symptomatic. Frequently, it is not possible to trim a keratotic lesion completely in a single office visit; it may be necessary for a patient to return for subsequent visits to reduce the callus further.[201] As the keratotic core becomes more superficial, it typically becomes less symptomatic. A seed corn has a well-differentiated keratotic core, usually 1 to 2 mm in size, and responds well to trimming or curettage.[201] Trimming a callus also helps in differentiating it from a wart.

When the callus has been trimmed, a soft metatarsal pad (Fig. 25H-60) is placed proximal to the keratosis to redistribute the pressure more uniformly. Jahss[204] recommended relieving the pressure on areas of excess weight-bearing and increasing pressure on areas of too little weight-bearing. The use of a soft insole (Fig. 25H-61) can alleviate the pressure further in athletes.[210] Athletic shoewear should provide a wide toe box and a soft sole to lessen impact when running.

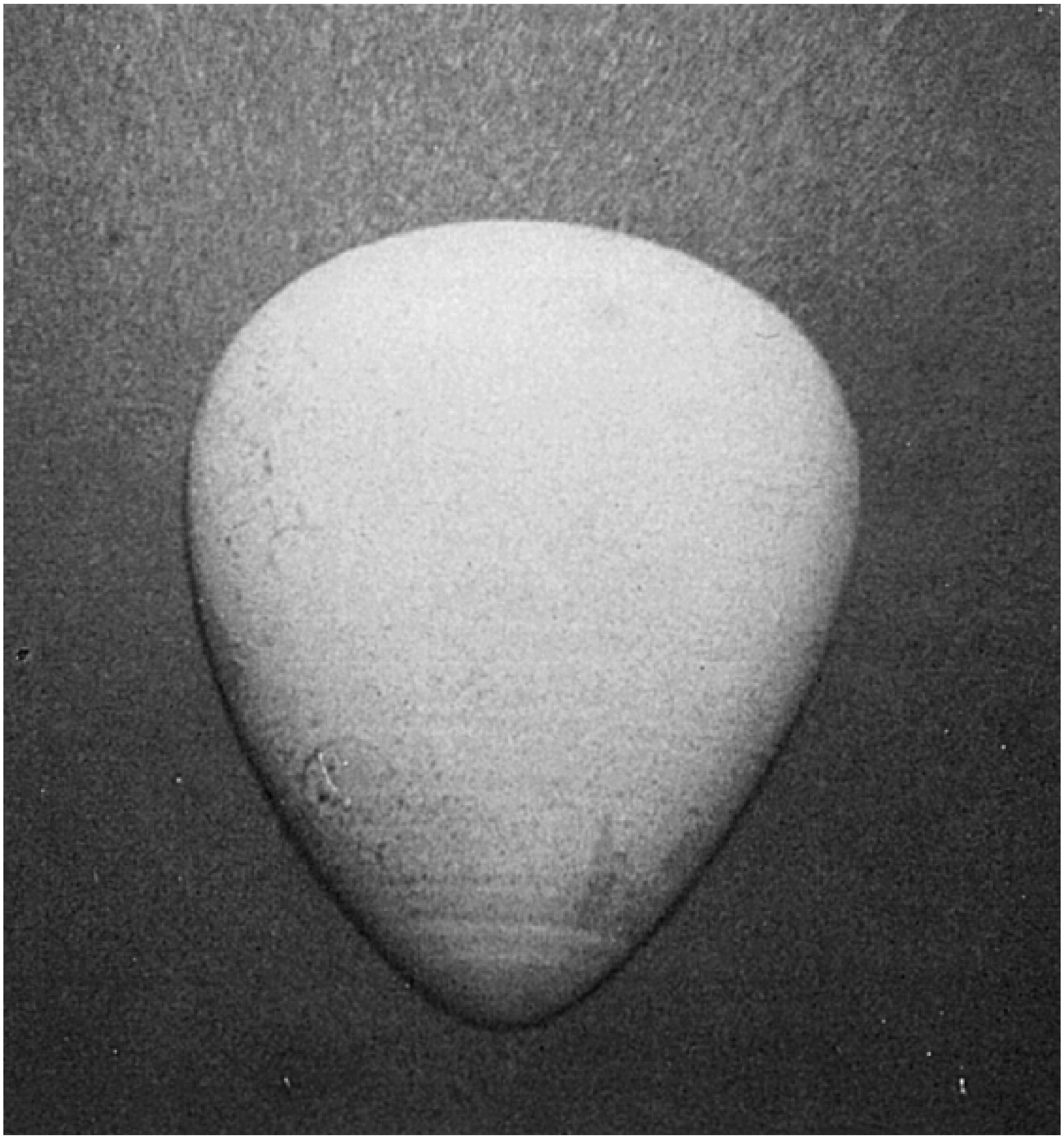

Figure 25H-60 A small metatarsal pad may be placed just proximal to the keratosis to redistribute the pressure. *(© M. J. Coughlin. Used by permission.)*

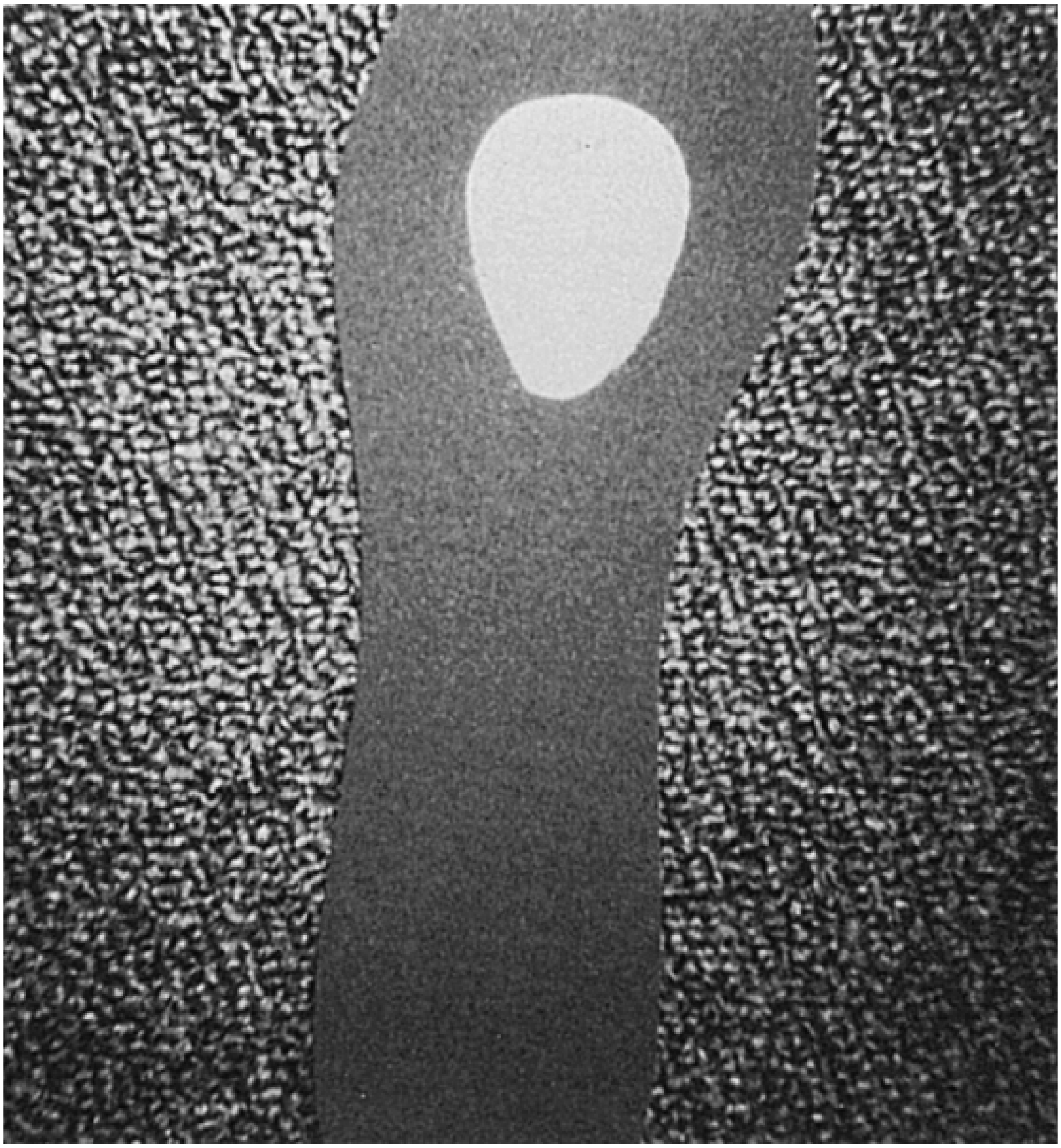

Figure 25H-61 A soft insole may be used to decrease the pressure in the forefoot region. *(© M. J. Coughlin. Used by permission.)*

When varus or valgus malalignment of the forefoot or hindfoot results in subsequent development of an IPK, appropriate orthotic devices are selected to compensate for the malalignment and to redistribute weight-bearing forces beneath the metatarsal head. A Plastizote orthosis of medium or high density can be fabricated to relieve pressure beneath the IPK and provide correction for a postural deformity.

If a keratotic lesion continues to be symptomatic and significantly impairs athletic function, surgical intervention is considered. Because of the lengthy postoperative recovery time, the possibility of restricted MTP motion, and the possibility of recurrence of a lesion or development of a transfer lesion, a rigorous trial of trimming, padding, and orthotic management should be carried out before surgery is performed.

Operative

IPKs beneath the second, third, and fourth metatarsal heads usually are classified as diffuse or discrete keratoses. Differentiating between these two lesions is important to institute proper treatment.

A large, diffuse IPK often is associated with a long or plantar flexed metatarsal. Although the second and third metatarsals are the most common sites of occurrence, [203] the other metatarsals are occasionally involved. Typically, these lesions are 1 to 2 cm in size and do not have an invaginated keratotic core. Multiple osteotomies have been described for treatment of metatarsalgia and plantar keratoses (Box 25H-37). [1531] [1532] [1533] [1541] [1542] [1543] [1544] Giannestras [203] described a step-cut proximal metatarsal osteotomy that is used to decrease the length of a symptomatic metatarsal. Mann and Mann [1531] [1532] suggested the use of a long oblique longitudinal osteotomy rather than a step-cut osteotomy to shorten the elongated metatarsal (Fig. 25H-62). Giannestras [203] reported development of transfer lesions in 10% of patients postoperatively. Mann [206] reported a 5% rate of transfer lesions. Delayed union occasionally occurs; however, with time, most osteotomies go on to successful healing.

BOX 25H-37

METATARSAL OSTEOTOMIES FOR INTRACTABLE PLANTAR KERATOSES

- Step cut osteotomy
- Long oblique metatarsal osteotomy
- Distal oblique (Helal) osteotomy
- Capital oblique (Weil) osteotomy
- Dorsal closing wedge osteotomy
- Vertical chevron osteotomy
- Segmental metatarsal osteotomy

Figure 25H-62 A, An oblique osteotomy of the proximal metatarsal may be used to achieve shortening. **B,** Preoperative radiograph of a patient with intractable plantar keratosis beneath the second metatarsal head. **C** and **D,** After longitudinal step-cut osteotomy with internal fixation, successful healing is shown (**C,** anteroposterior view; **D,** lateral view). **E,** Failure of fixation with fracture of Kirschner wire after osteotomy. *(**B** to **E,** © M. J. Coughlin. Used by permission.)*

Mann and Du Vries [215] proposed that a small, discrete, intractable plantar keratosis is caused by a prominent fibular condyle on the plantar aspect of the metatarsal head. A discrete callus can develop after a metatarsal head fracture with a plantar flexion deformity of the metatarsal and a hyperextended MTP joint, leading to buckling of a toe. Discrete lesions are also associated with an idiopathic plantar flexed metatarsal. DuVries [216] described a plantar condylectomy to correct this deformity. Coughlin recommends removal of 20% to 30% of the condyle through a dorsal incision. [217] This procedure was later modified by Mann and DuVries, [215] who performed an MTP arthroplasty, removing about 2 mm of the articular surface along with the plantar condylectomy (Fig. 25H-63). After MTP joint arthroplasty, MTP joint motion is diminished by 25% to 50%. Although stiffness does not always affect the function of a sedentary person, a competitive athlete typically requires more normal motion, and if so, an MTP arthroplasty is contraindicated.

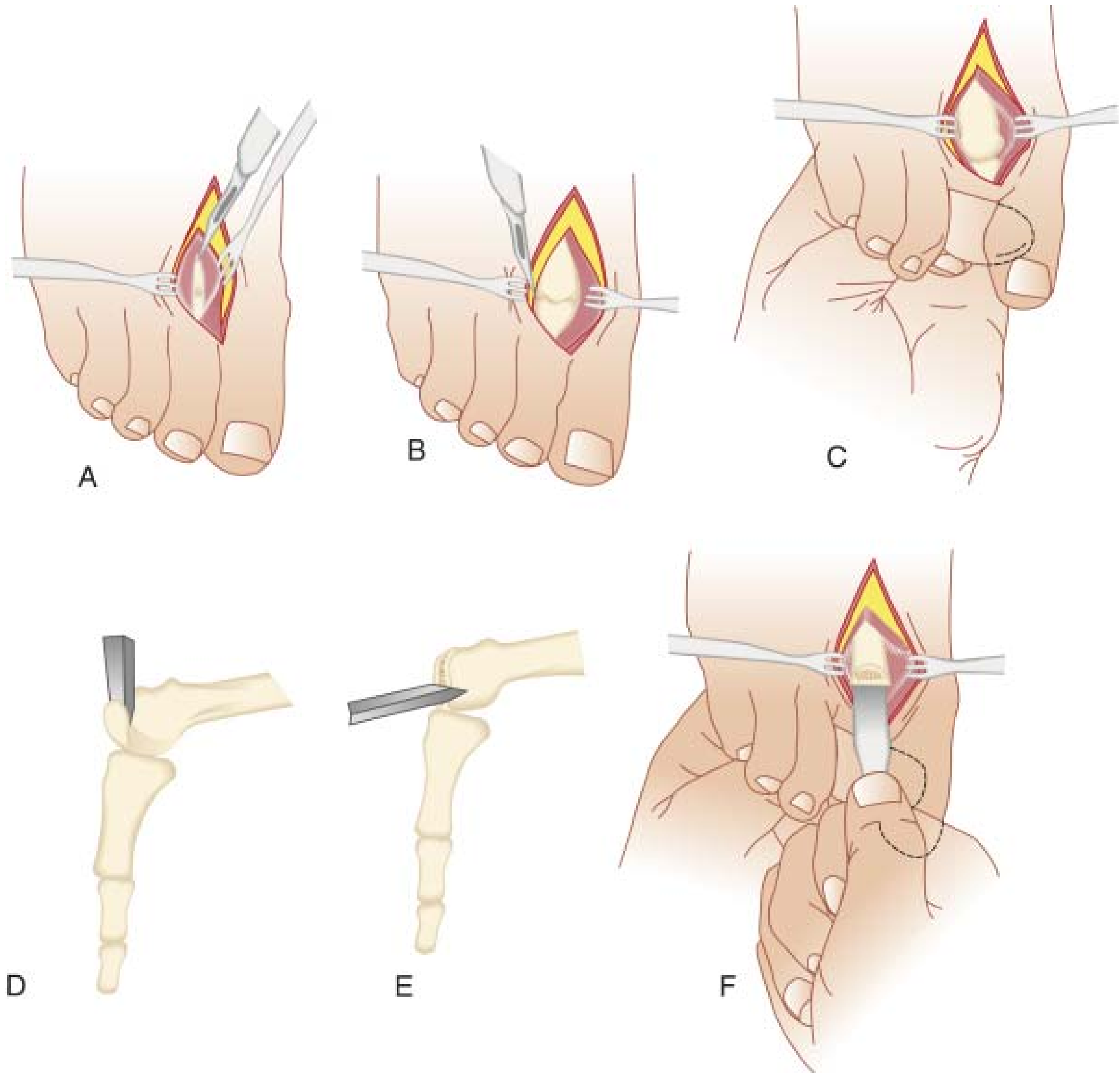

Figure 25H-63 **A,** A hockey-stick–shaped incision from the middle of the metatarsal shaft into the adjoining web space. The extensor tendon and the skin are retracted, and the capsule is excised longitudinally. **B,** The capsule is released on the medial and lateral aspects. **C,** The toe is plantar flexed, exposing the metatarsal head. **D,** Two millimeters of the distal metatarsal articular surface are resected. **E,** The plantar condyle is removed. **F,** The articular surfaces are smoothed to create a congruous surface. Often, there is a certain amount of restricted motion after this procedure.

Mann and DuVries evaluated 100 patients with discrete IPKs and noted a recurrence rate of 17.6% after MTP arthroplasty.[215] Of these, 5% recurred under the symptomatic metatarsal. A transfer lesion developed in 13% of cases. Despite these results, 93% of the patients were satisfied with their outcome. There was a 5% rate of complications, which included fracture of the metatarsal head, avascular necrosis, drift of the involved toe, and cock-up of the involved toe.

When a competitive athlete requires a more normal motion, a distal metatarsal osteotomy is considered. A vertical chevron procedure (Fig. 25H-64),[1535] [1544] [1548] a distal oblique osteotomy (Fig. 25H-65),[1542] [1543] [1549] [1550] [1551] [1552] or capital oblique osteotomy[223] (Fig. 25H-66) is commonly used. A distal oblique osteotomy allows dorsal displacement and shortening of the osteotomy. It is believed that elevation of about 3 mm is necessary for adequate decrease of pressure beneath the symptomatic metatarsal head.[224]

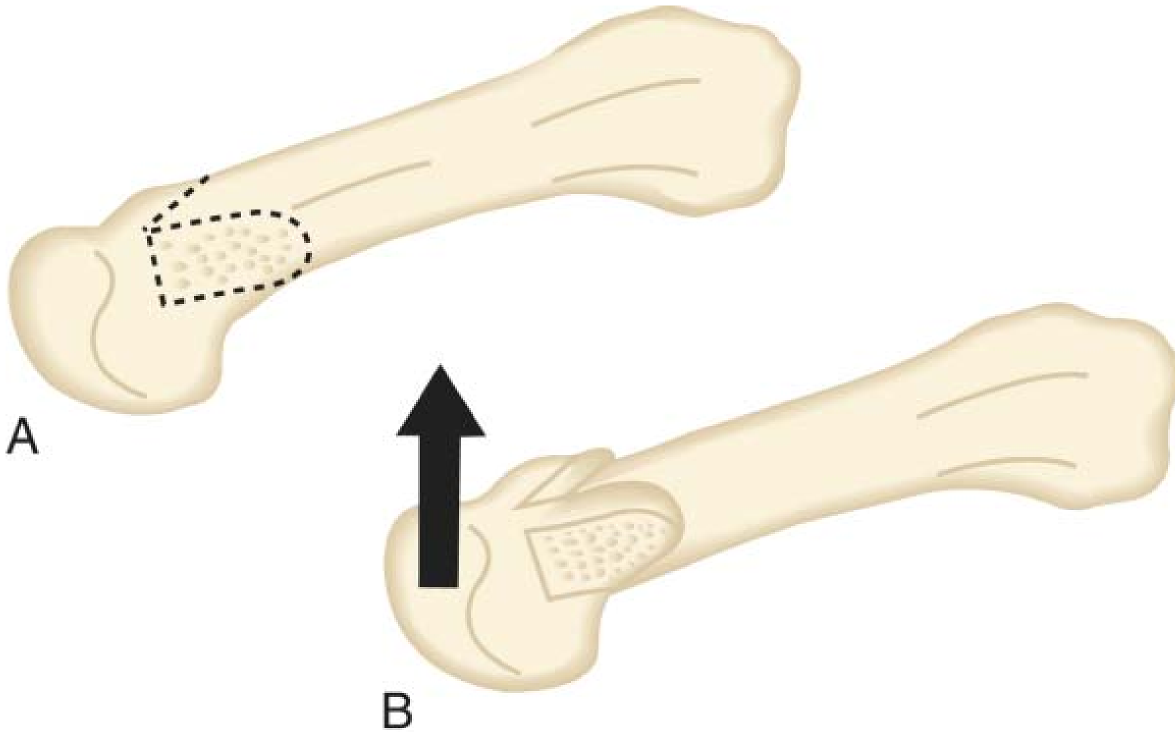

Figure 25H-64 Technique of vertical chevron osteotomy. **A,** Preoperatively. **B,** After osteotomy.

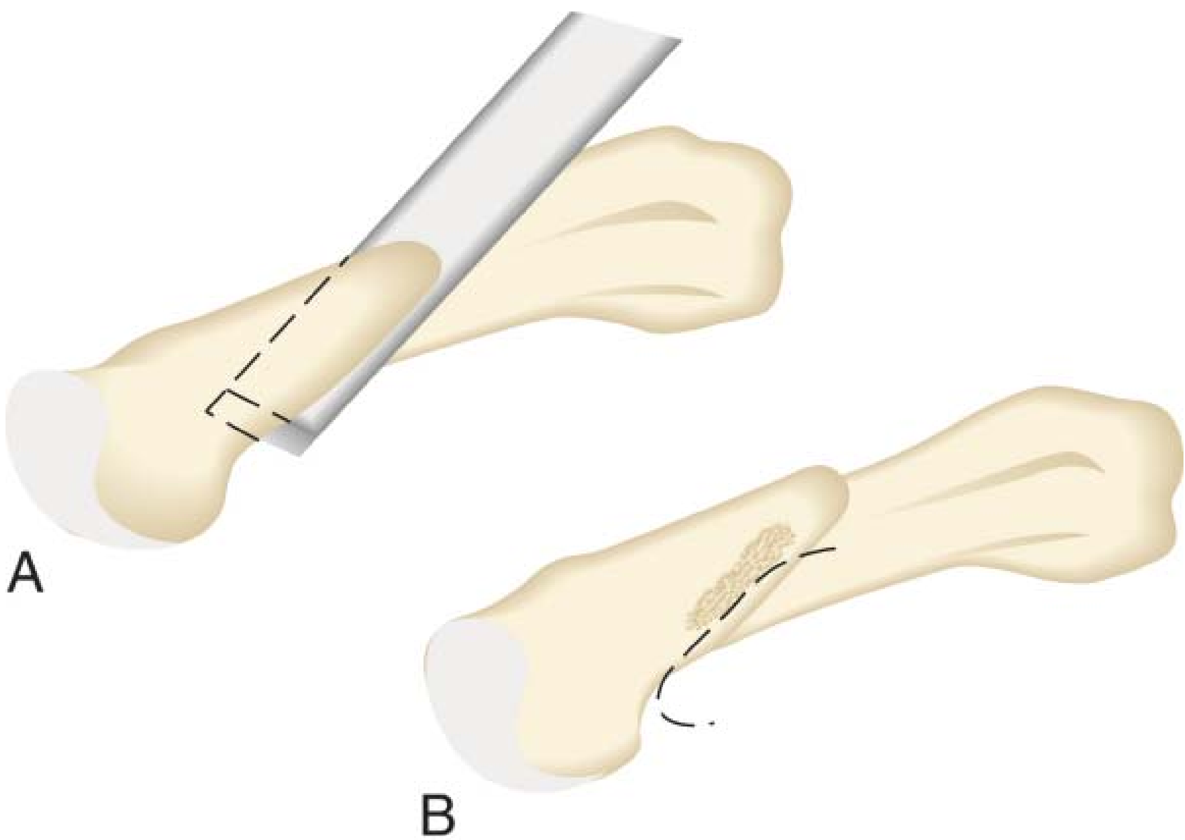

Figure 25H-65 Technique of oblique lesser metatarsal osteotomy showing dorsal displacement. **A,** Preoperatively. **B,** After osteotomy.

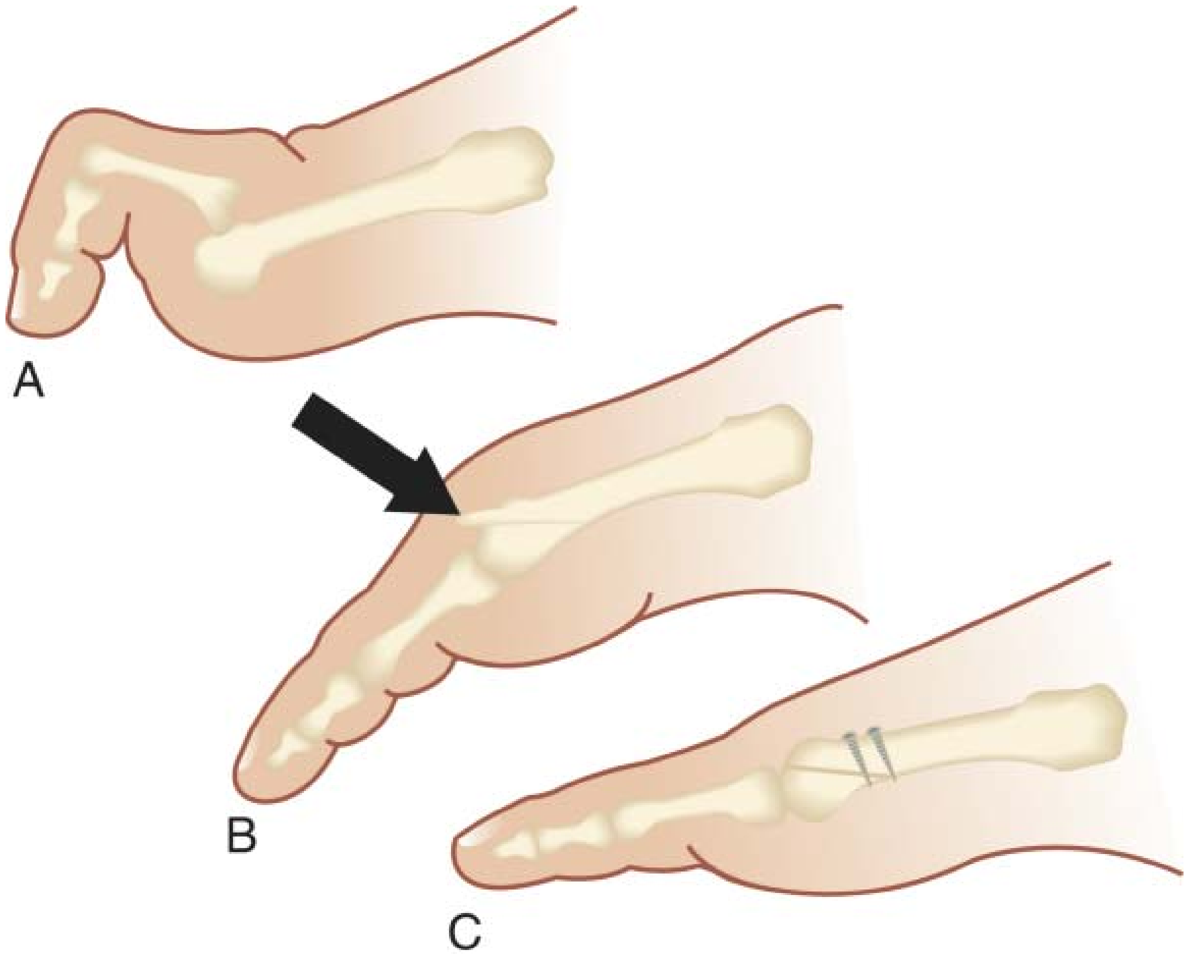

Figure 25H-66 **A,** Preoperative diagram shows dislocated lesser metatarsophalangeal joint. **B,** Technique of capital oblique osteotomy allows shortening but not plantar flexion. **C,** After internal fixation and resection of the dorsal flare, the lesser metatarsophalangeal joint has been reduced.

For discrete lesions under the tibial sesamoid, tibial sesamoid shaving has been advocated. A broad, diffuse callus under the first metatarsal is usually associated with a plantar flexed first metatarsal, whereas a discrete lesion is associated most commonly with a prominent sesamoid. When conservative treatment is unsuccessful, a dorsal-based closing wedge osteotomy of the proximal first metatarsal is used to correct the plantarflexed first ray. Sesamoid shaving or, more infrequently, sesamoid excision is considered for discrete lesions (see earlier section on sesamoid dysfunction).

Weighing the Evidence

Helal, [1542] [1543] in describing the oblique distal metatarsal osteotomy, noted that the purpose was to elevate the metatarsal head and to reduce length. Although in his initial report, Helal [212] did not note specific complications, in a retrospective review of 310 patients (508 feet), 84% were noted to have no pain, and an 8% recurrence of plantar callosities was noted. [213]

Winson and coworkers [222] reported on 94 patients (124 feet) who underwent a similar procedure. In their report, 53% of the patients had significant postoperative symptoms, including transfer lesions in 32%, nonunion in 13%, and an overall recurrence of an IPK in 50% of patients. In 66 of the 124 feet, major complaints were noted postoperatively. The authors stressed that in either a cavus or a rigid foot, a distal oblique sliding osteotomy was contraindicated. These results agreed with those of Giannestras, [203] indicating that the presence of a contracture at the MTP joint was a further contraindication to a metatarsal osteotomy.

Pedowitz, [221] in a report on 69 distal oblique osteotomies in 49 patients, reported good results in 83% of cases. There was a 27% incidence of either residual callosity or transfer lesion and two nonunions in his series. Pedowitz [221] stressed that this procedure was contraindicated in patients who had a fixed MTP joint deformity. Idusuyi and associates, [220] in a report on 20 patients (23 feet) who had single osteotomies of the second, third, or fourth metatarsals without internal fixation, noted a 20% reoperation rate for recurrent plantar callosities. Of these patients, 65% were limited with footwear choices or required a shoe insert. Of 23 feet, 13 (56%) were rated as poor or fair, and the authors had significant reservations about recommending this procedure.

Trnka and colleagues,[223] in a comparison of the results of an intra-articular capital oblique osteotomy (Weil type) and a distal oblique osteotomy (Helal type), reported a high level of satisfaction and a lower incidence of recurrent metatarsalgia in transfer lesions with the capital oblique osteotomy. No transfer lesions were noted with the capital oblique osteotomy, whereas 41 feet with a Helal-type osteotomy developed transfer lesions. Five malunions and three pseudarthroses occurred in the 15 cases that underwent the Helal-type osteotomy compared with no malunions or pseudarthroses in the Weil-type group. Trnka and colleagues[223] concluded that the Weil-type osteotomy was a satisfactory method for treating metatarsalgia, but because of the high complication rate, the Helal-type osteotomy was not an acceptable procedure.

Metatarsal head resection should be avoided because it tends to concentrate pressure beneath the remaining metatarsal heads.

Several authors have investigated the mechanism and magnitude of pressure relief after capital oblique osteotomy of the metatarsal. [1555] [1556] [1557] [1558] Grimes and Coughlin[225] recommended using a 2-mm saw blade for a capital oblique osteotomy in order to offset plantar displacement of the metatarsal head. Khalafi and colleagues,[226] Snyder and coworkers,[227] and Vandeputte and colleagues[228] found that overall plantar pressure decreased under the metatarsal head with capital oblique osteotomy. However, when compared with a vertical chevron osteotomy,[227] both procedures resulted in decreased pressure, but only the chevron osteotomy demonstrated a corresponding decrease in load.

Hatcher and colleagues[219] advocated multiple metatarsal osteotomies as a method of reducing the occurrence of transfer lesions and the recurrence of plantar keratoses. Mann[206] and others[222] favored an isolated osteotomy as a more reliable procedure. If two adjacent metatarsals are significantly longer, an osteotomy of these two metatarsals is considered.[201] Internal fixation allows the surgeon to alter either the length or the inclination of the metatarsal, then to stabilize it in the desired position. A higher rate of displacement, angulation, and shortening can occur if the metatarsal osteotomy is not internally fixed.[222] Rigid internal fixation also tends to reduce the chance of malunion or delayed union.

Although delayed union or nonunion is possible with the use of a proximal dorsal closing wedge osteotomy for an IPK, the location of this osteotomy in the metaphysis of the involved metatarsal usually allows fairly rapid healing. A transfer lesion can develop if an excess amount of bone is resected, and an IPK can recur if too little bone has been resected. Mann[206] reserved this osteotomy for recurrent lesions after failure of a longitudinal oblique osteotomy.

Occasionally, an IPK develops beneath a hallucal sesamoid. [1559] [1560] Mann and Wapner[229] reported on the results in 12 patients and noted that 58% had excellent results with no recurrence and 33% had good results with slight recurrence of a plantar callosity. Van Enoo and Cane[230] reported on 17 tibial sesamoid shavings with an average 4-year follow-up. In two of the feet, a mild recurrent callosity developed postoperatively.

AUTHOR'S PREFERRED METHOD

Localized trimming, instruction of the athlete in methods of reducing the callus by self-care, redistribution of pressure with metatarsal pads or orthotic devices, and use of proper footwear are the preferred methods of treatment. Surgery should be reserved for individuals who are limited significantly by an IPK and in whom other conservative methods have been unsuccessful. The significant risk for reduced range of motion, recurrence of plantar keratosis, and development of transfer lesions makes a strong case for the use of conservative care in competitive athletes.[231] Surgical intervention should be considered only after a lengthy period of conservative care.

A capital oblique osteotomy is useful in the setting of either a diffuse or discrete IPK. In patients with a small, discrete IPK secondary to a prominent fibular condyle, a capital oblique osteotomy (Weil type) is preferable either to an MTP joint arthroplasty (because of the possibility of restricted postoperative range of motion) or to a vertical chevron osteotomy. However, the metatarsal head must be raised or at the least not depressed, or symptoms can be made worse. When a contracture of the MTP joint has occurred, a plantar condylectomy or a distal metatarsal osteotomy should be avoided because the buckling of the toe at the MTP joint leads to plantar keratoses.[232]

Postoperative Prescription, Outcomes Measurement, and Potential Complications

A compressive dressing is placed at the time of surgery, and the patient is allowed to bear weight in a postoperative shoe. If necessary, a cast can be used for increased protection. Kirschner wires are removed usually 3 to 4 weeks after surgery. After a metatarsal osteotomy, adequate time must be allowed for healing. Premature athletic activity may lead to failure of fixation, displacement of an osteotomy, or nonunion. In general, proximal metatarsal osteotomies require 6 to 12 weeks for osseous union to occur. A vertical chevron osteotomy, if performed with a thin oscillating saw blade, heals in about 6 weeks. After an MTP joint arthroplasty and condylectomy, about 6 weeks is necessary for adequate healing to occur at the MTP joint.

Once pins are removed and adequate healing has occurred (4 to 6 weeks), gentle range of motion is initiated at the MTP joint to diminish postoperative stiffness. When radiographs demonstrate bony union, aggressive walking activity is initiated. Taping of the forefoot or the use of soft metatarsal pads helps to alleviate symptoms with athletic activity during surgical recovery. About 4 weeks after the initiation of walking, jogging is initiated, followed by running activities as pain permits.

Decreased range of motion is a significant risk after MTP arthroplasty as well as after distal metatarsal osteotomy. Recurrence of plantar keratoses or development of a transfer lesion occurs in 10% to 50% of patients after metatarsal osteotomy.

Criteria for Return to Play

Full return to athletic activity is expected when bony healing occurs. Usually between 6 and 12 weeks after surgery, aggressive walking is initiated. Walking is advanced to jogging after 4 weeks as pain and swelling allow. The athlete then increases running activities as tolerated, gradually returning to sport between 3 and 6 months postoperatively (Box 25H-38).

BOX 25H-38

CRITERIA FOR RETURN TO PLAY AFTER OSTEOTOMY FOR INTRACTABLE PLANTAR KERATOSIS

- Bony healing of osteotomies on radiographs and clinical examination
- Resolution of pain and swelling
- Full return to activities expected between 3 and 6 months

Special Populations

All athletes are at risk for plantar keratoses. The risk for developing an IPK is higher in athletes with mild positional deformity or a plantarflexed or elongated metatarsal. Risk may also be increased in athletes involved in repetitive running activities.

LESSER TOE ABNORMALITIES

The most common pathologic entities involving the lesser toes are hammer toes, mallet toes, and claw toes. [233] Other pathologic entities of the lesser toes include lateral fifth toe corns, interdigital corns, and bunionettes. The severity and frequency of occurrence of these deformities increases with the age of the patient, and they have been reported as more frequent in women. [1564] [1565] [1566] Although there appears to be a correlation between the use of high-fashion footwear and the development of lesser toe deformities, intrinsic predisposing factors, such as a wide forefoot, an abnormally long ray, inflammatory arthritis, isolated or repetitive trauma, or neuromuscular disease may predispose the lesser toes to deformity. [233] The development of callosities on the lateral aspect of the fifth toe or between the lesser toes often causes problems with walking or running. Keratoses in either of these areas frequently necessitate modification of athletic activities. Although the onset of these problems usually is insidious, [233] they can cause significant discomfort in older athletes, specifically athletes in their 40s through 60s.

Anatomy and Biomechanics

The position of the lesser toes relies on a balance between intrinsic and extrinsic muscle forces. On the dorsal aspect of the lesser toes, the extensor digitorum longus dorsiflexes the proximal phalanx by its insertion into the extensor expansion. [235] When the proximal phalanx is in a neutral position, the extensor digitorum longus extends the proximal interphalangeal joint. [1565] [1567] When the MTP joint is hyperextended, the dorsiflexion force at the interphalangeal joint decreases significantly. [1565] [1567] The flexor digitorum longus inserts into the distal phalanx on the plantar aspect of the toes and flexes the distal interphalangeal joint (Fig. 25H-67 **A**). The flexor digitorum brevis inserts into the middle phalanx and plantar flexes the proximal interphalangeal joint. Neither flexor has a significant influence on MTP joint plantar flexion because there is no insertion into the plantar aspect of the proximal phalanx. [237] The interossei and lumbricals pass plantar to the axis of motion of the MTP joint and dorsal to the axis of the interphalangeal joints. These muscles provide a plantar flexion force at the MTP joint and an extension force at the proximal interphalangeal and distal interphalangeal joints (see Fig. 25H-67 **B** and **C**). [1567] [1568]

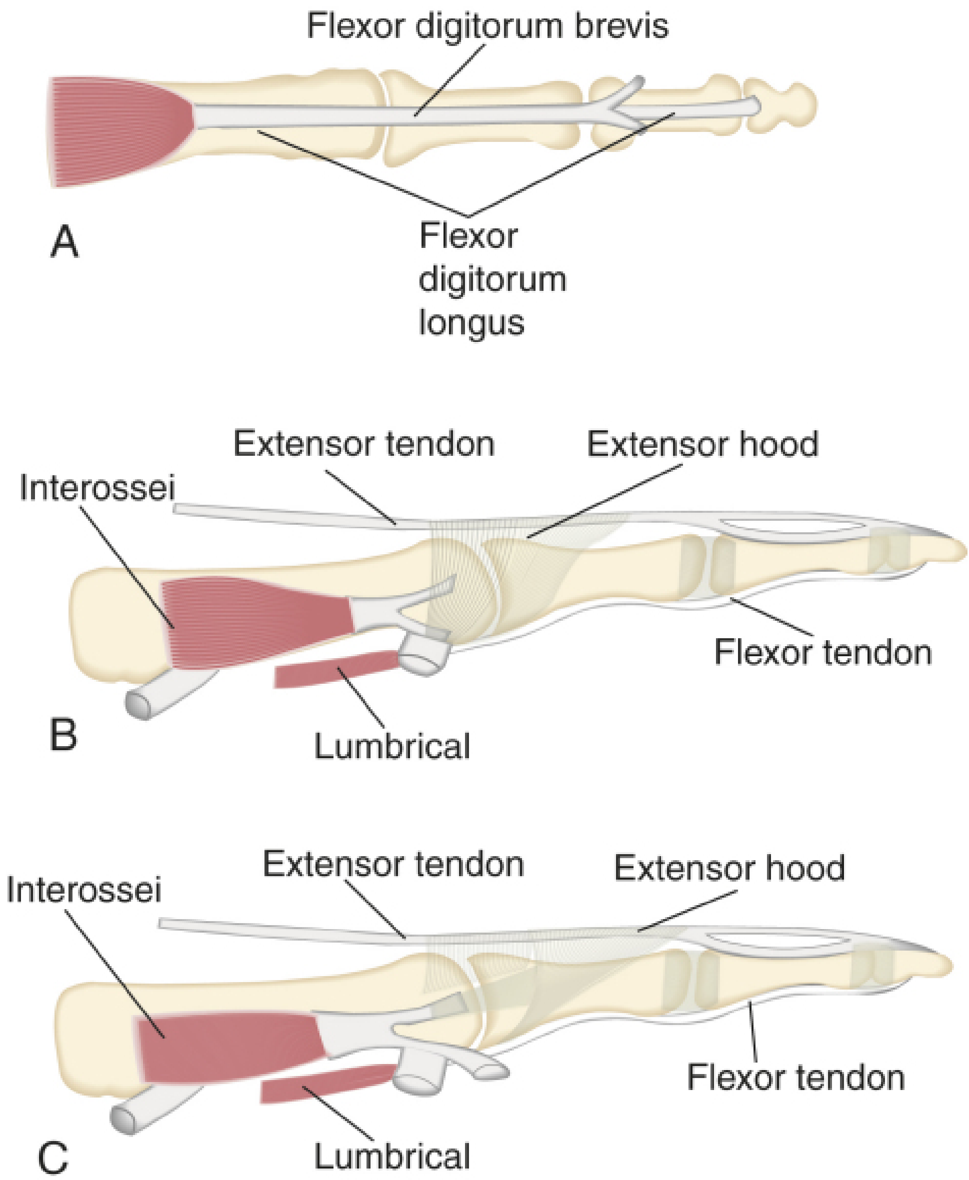

Figure 25H-67 **A,** Plantar view of lesser toe. The tendon of the flexor digitorum longus inserts onto the base of the distal phalanx. **B** and **C,** Side views show the insertion of the intrinsic muscles, which allows them to plantar flex the metatarsophalangeal joint and extend the interphalangeal joints.

The plantar aponeurosis and plantar capsule are static structures that provide a stabilizing force on the plantar aspect of the MTP joint. [1565] [1567] Aided by the dynamic power of the interossei and lumbricals, plantar flexion of the proximal phalanx occurs.

At the MTP joint, the long and short extensor muscles are opposed by the plantar aponeurosis, plantar capsule, and intrinsic musculature. The reactive force as the foot strikes the ground helps to achieve passive extension at the MTP joint; however, the long and short extensors are significantly stronger than the intrinsics. At the interphalangeal joints, the flexor digitorum longus and flexor digitorum brevis are opposed by the interossei and lumbricals. At both the MTP joint and the interphalangeal joints, a mismatch occurs (Fig. 25H-68) in which the more powerful extrinsic muscles overpower the weaker intrinsic muscles. [237] The position of the lesser toes is crucial in whether this mismatch leads to development of pathology. When the MTP joint is held in a neutral position, the extensor digitorum longus assists in extending the proximal interphalangeal joint and the flexor digitorum longus flexes the MTP joint. When the MTP joint is hyperextended, these functions are diminished. The long and short flexors come under increased tension, and the interossei and lumbricals are overpowered by the stronger extrinsic muscles. [1565] [1567] As a result, a chronic hyperextension deformity develops at the MTP joint and the flexion deformity at the interphalangeal joints increases.

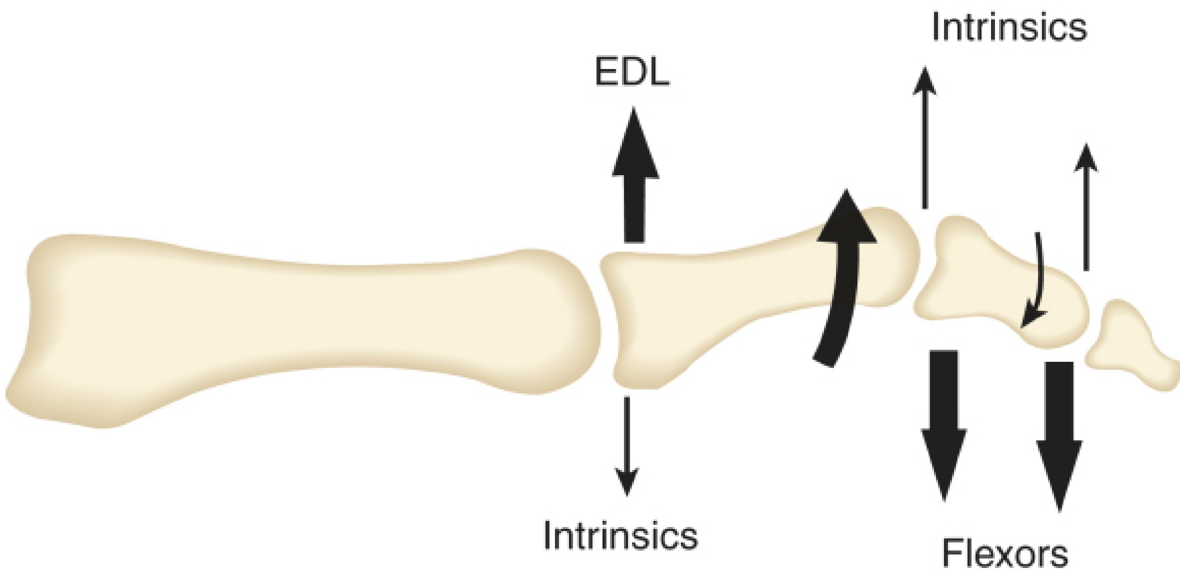

Figure 25H-68 At the various joints of the lesser toe, intrinsic and extrinsic muscles oppose each other. An obvious mismatch occurs between the larger intrinsic and the smaller intrinsic muscles, leaving two deformities. EDL, extensor digitorum longus.

Subluxation or dislocation of the MTP joint commonly occurs as a result of the imbalance between stabilizing plantar structures and intrinsic and extrinsic muscle forces. [1569] [1570] [1571] [1572] [1573] The plantar aponeurosis or plantar capsule becomes incompetent as a result of repetitive trauma, a constricting toe box, or excessive length of the metatarsal. [237] This diminishes the stabilizing force of the plantar structures over the MTP joint. [1565] [1567] [1574] Concurrent contractures of the dorsal capsule and extensor tendons add to the deformity. A hallux valgus deformity destabilizes the toe by exerting extrinsic pressure on the second toe. [237] With time, subluxation can progress to frank dislocation.

Although the most common deformity of the second MTP joint is dorsal dislocation, occasionally the second toe deviates medially. [1567] [1570] [1572] A gap or space occurs between the second and third toes (Fig. 25H-69). [1570] [1571] [1572] This deformity is often associated with a hallux valgus deformity. Medial deviation of the toe can also be seen after trauma, [240] as a result of degenerative or rheumatoid arthritis, [1567] [1570] nonspecific synovitis, [245] synovial cyst or ganglion formation, [240] or erosion of the fibular collateral ligament. [240]

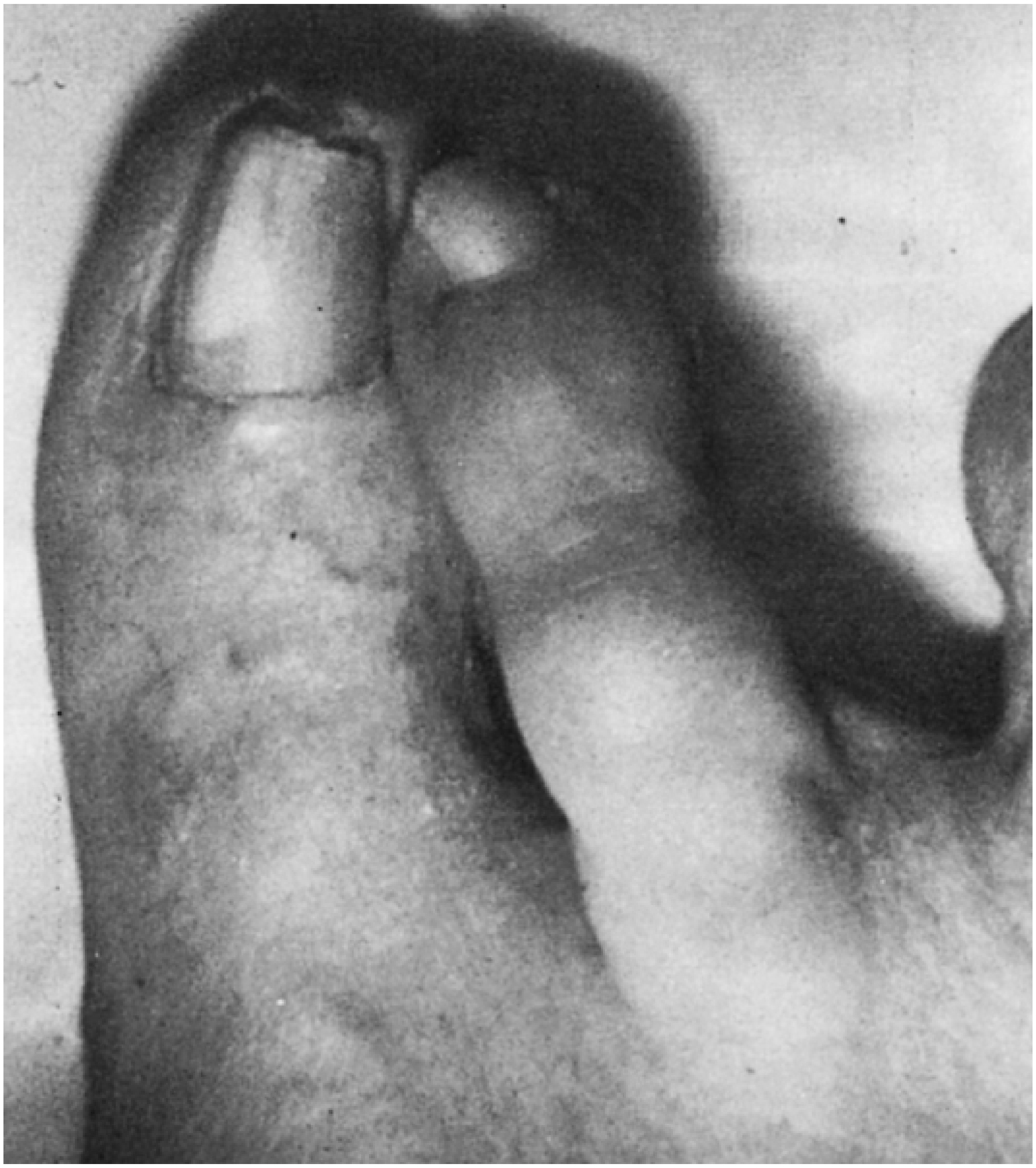

Figure 25H-69 Crossover second toe shows space between the second and third toes. *(© M. J. Coughlin. Used by permission.)*

A corn occurs when a keratosis develops over the lateral aspect of the fifth toe. Pressure from constricting footwear over the area of the lateral condyle of the interphalangeal joint can lead to the development of keratoses (Fig. 25H-70). [246] Occasionally, skin breakdown occurs, although callus formation is a more common abnormality.

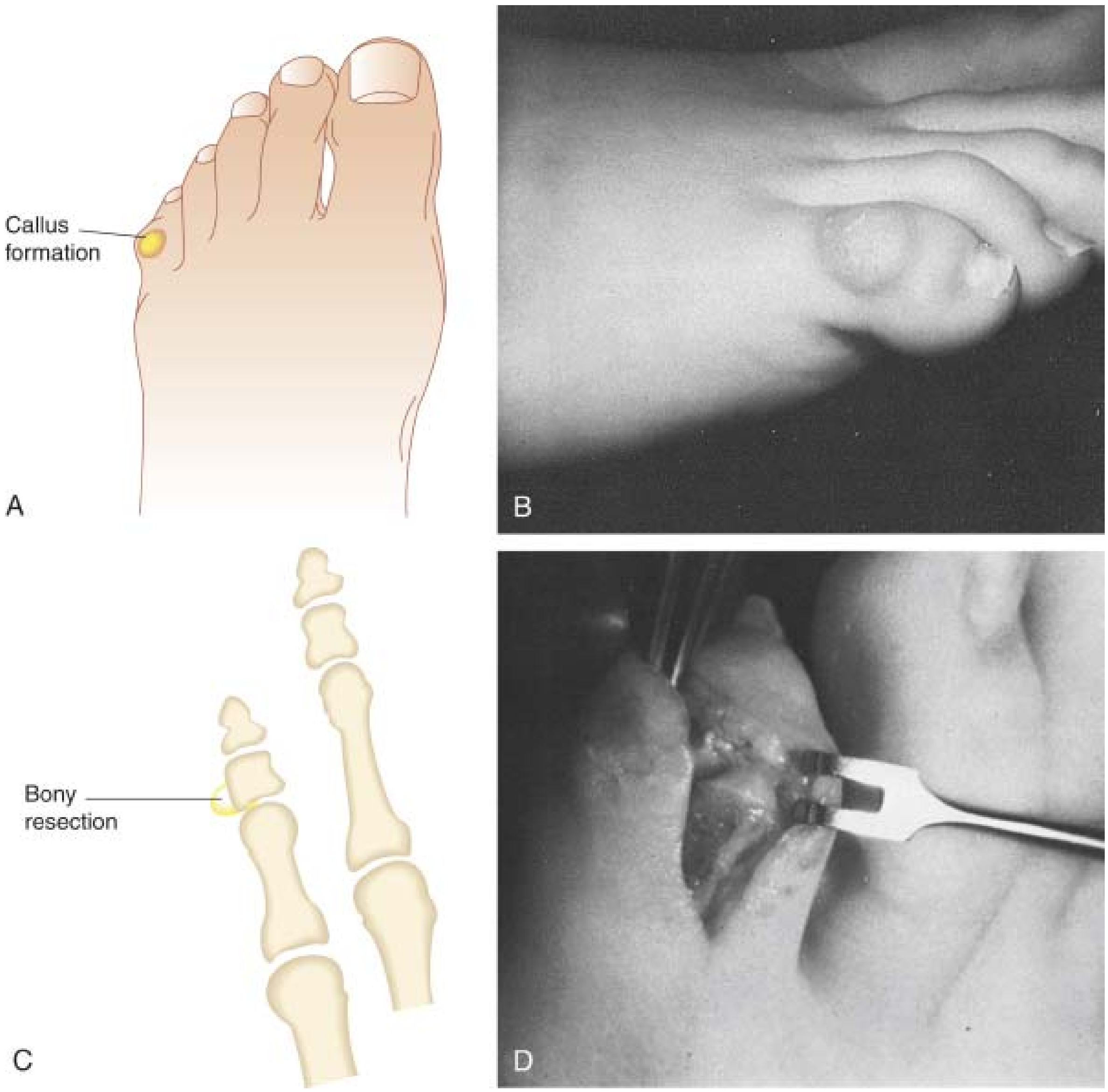

Figure 25H-70 **A** and **B,** Lateral fifth toe corn. **C** and **D,** Resection of lateral condyle. Capsular repair helps prevent postoperative deformity. *(**A** and **C,** From Mann RA, Coughlin MJ: Video Textbook of Foot and Ankle Surgery. St. Louis, Medical Video Production, 1991, p 50; **B** and **D,** © M. J. Coughlin. Used by permission.)*

An interdigital corn is a hypertrophic keratotic lesion that occurs between the lesser toes either along the shaft or in the web space as a result of pressure between two prominent areas on adjacent toes (Fig. 25H-71). [1563] [1576] This lesion often is mistaken for a fungal infection when there is maceration between the lesser toes. [1563] [1576]

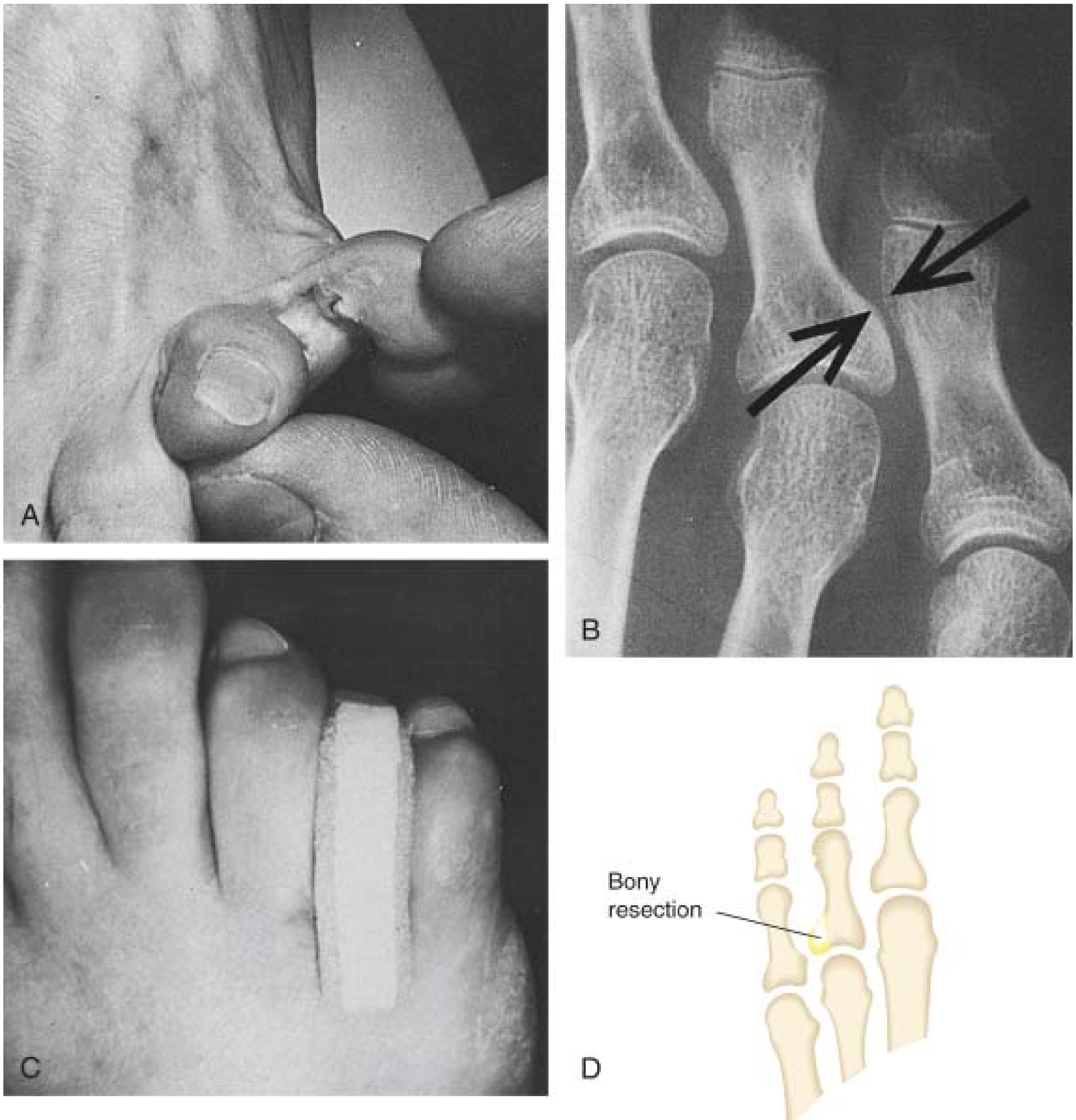

Figure 25H-71 A, Interdigital corn in web space. **B,** Radiograph shows area of bone impingement between distal aspect of proximal phalanx of fifth toe and proximal aspect of proximal phalanx of fourth toe. **C,** Padding may be placed in a symptomatic web space to relieve symptoms. **D,** Resection of condyle. *(**A-C,** From Mann RA, Coughlin MJ: Video Textbook of Foot and Ankle Surgery. St. Louis, Medical Video Production, 1991, p 51.)*

Classification

Lesser toe deformities include hammer toes, claw toes, mallet toes, and curly toes (Table 25H-4). These clinical entities are characterized by the location of the deformity (MTP, proximal interphalangeal, or distal interphalangeal joints) and the direction of the deformity (hyperextension or flexion). The deformity is further identified as passively correctable, flexible, or rigid.

TABLE 25H-4 -- Characteristics of Lesser Toe Deformities

	Metatarsophalangeal Joint	Proximal Interphalangeal Joint	Distal Interphalangeal Joint	
Hammer toe	Hyperextension	Flexion	None	Acquired deformity

	Metatarsophalangeal Joint	Proximal Interphalangeal Joint	Distal Interphalangeal Joint	
				Common in second toe Typically single toe involvement
Claw toe	Hyperextension	Flexion	± Flexion deformity	Multiple toe involvement
Mallet toe	None	None	Flexion	Trauma, impingement in shoes Second toe most commonly involved
Curly toe	None	Flexion	Flexion	Congenital

A hammer toe is a plantar flexion contracture of the proximal interphalangeal joint [1567] [1577] and frequently is associated with a hyperextension deformity of the MTP joint (Fig. 25H-72). [235] Hammer toes can occur in multiple toes, although most commonly they occur in the second toe. [247] A hammer toe is believed to be an acquired deformity. Neuromuscular disease, degenerative disk disease, and connective tissue disorders may be associated with hammer toe development [1564] [1567] ; however, the long-term use of fashionable footwear probably is the most common cause that leads to the development of progressive contractures of the lesser toes. [1564] [1565] [1578]

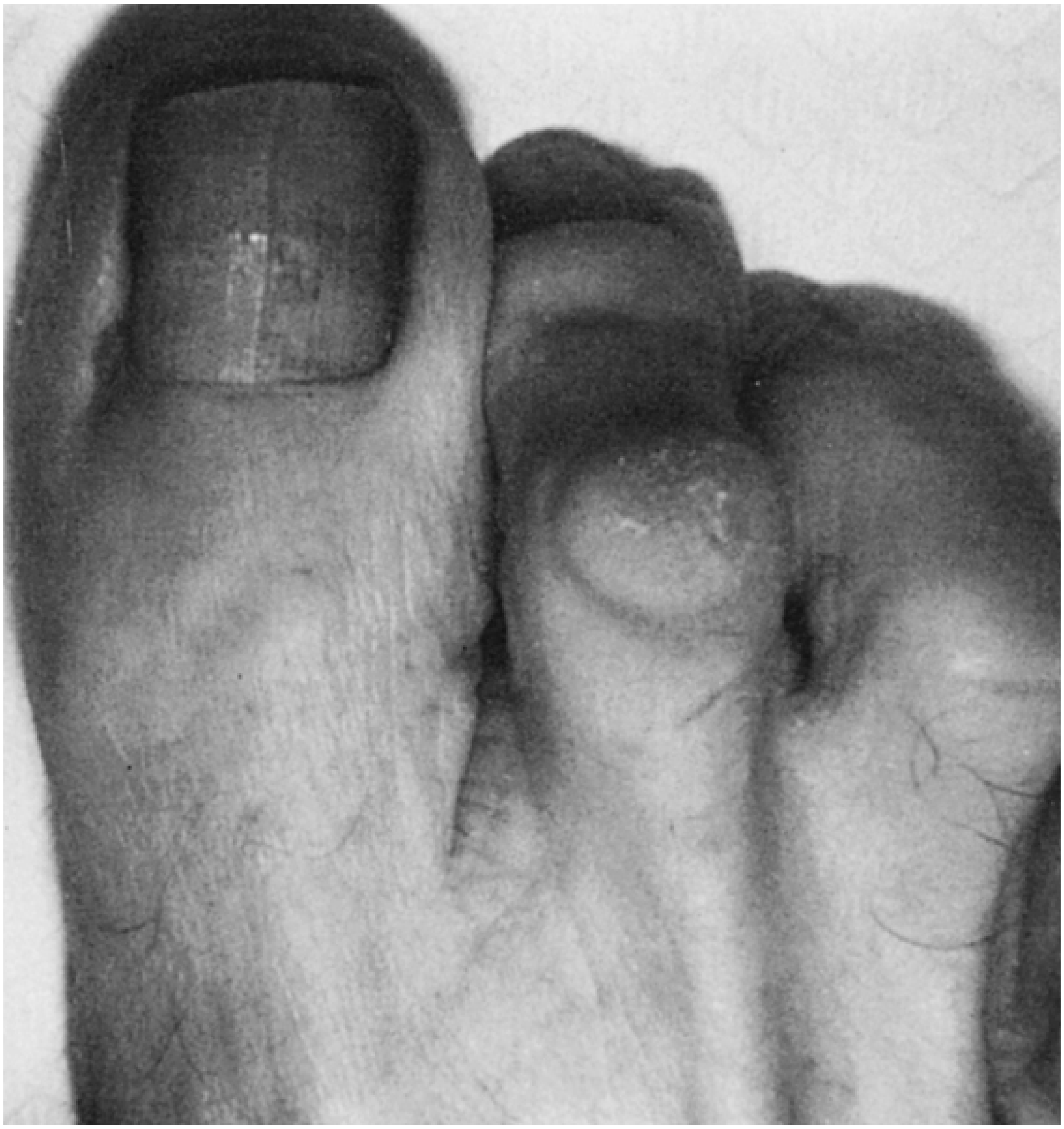

Figure 25H-72 Hammer toe deformity. *(© M. J. Coughlin. Used by permission.)*

A claw toe is characterized by dorsiflexion of the MTP joint and plantar flexion of the proximal interphalangeal joint. [1563] [1567] Multiple toes often are involved. Neurologic conditions, such as poliomyelitis, cerebral palsy, spinal cerebellar degeneration, muscular dystrophy, meningomyelocele, Friedreich's ataxia, diastematomyelia, and Charcot-Marie-Tooth syndrome, are common causes, but many of these deformities have no clear cause. [1563] [1567]

One distinction between a hammer toe and a claw toe is that with a claw toe many toes are involved, and there is always a hyperextension deformity of the MTP joint. [237] Both deformities typically have a fixed flexion deformity of the proximal interphalangeal joint. Sometimes there is no clear distinction, and often the treatments are similar.

A mallet toe is characterized by a contracture or deformity of the distal interphalangeal joint (Fig. 25H-73). [1567] [1579] The abnormality is believed to result from pressure of the toe box of a shoe against a long second toe. [234] In younger patients, contracture of the flexor digitorum longus is often the cause of this typically flexible deformity. In older patients, this is frequently a fixed contracture. [237]

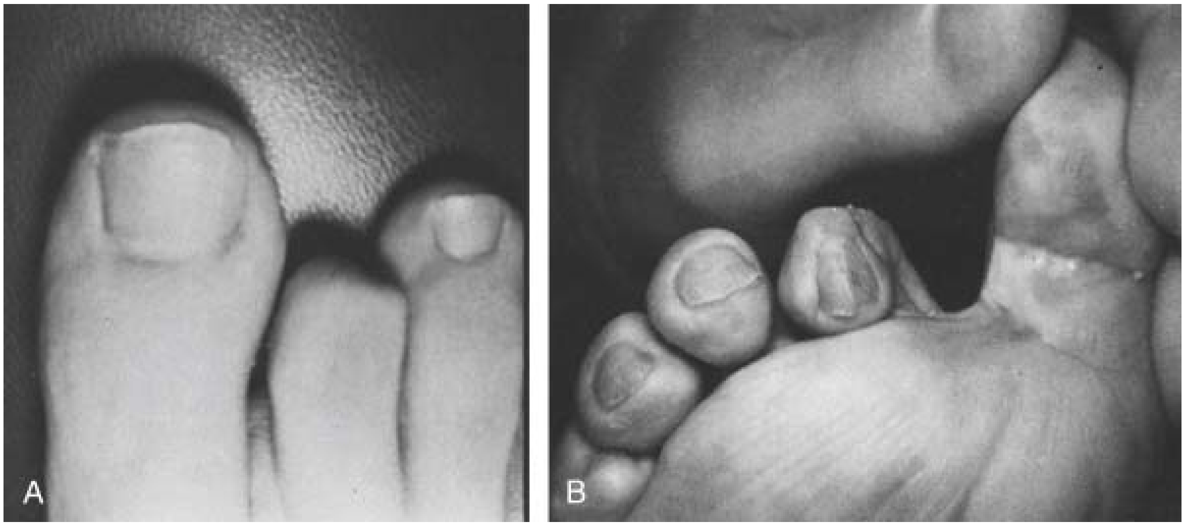

Figure 25H-73 **A** and **B,** Mallet toe deformity. *(From Mann RA, Coughlin MJ: Video Textbook of Foot and Ankle Surgery. St. Louis, Medical Video Production, 1991, p 48.)*

Evaluation

Clinical Presentation and History

The major complaint of the patient with a lesser toe deformity is discomfort related to pressure on the toe with shoewear or callus formation. In a hammer toe deformity, discomfort is located over the dorsal aspect of the proximal interphalangeal joint where a callosity develops as the toe buckles and strikes the top of the toe box (Fig. 25H-74). [248] On the plantar aspect of the foot, a callus develops beneath the metatarsal head secondary to subluxation or dislocation at the MTP joint.

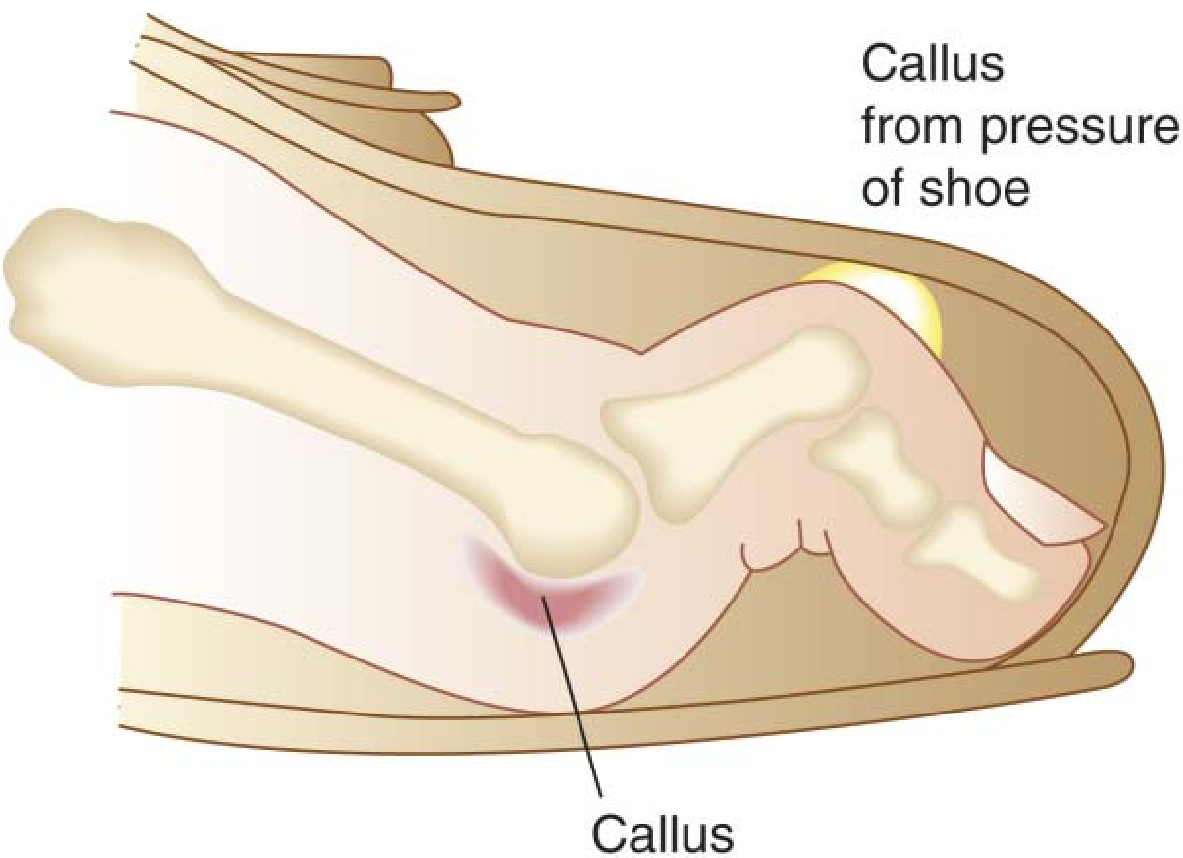

Figure 25H-74 Dorsal aspect of the proximal interphalangeal joint strikes the toe box, leading to callus formation. An intractable plantar keratosis may develop beneath the metatarsal head.

Claw toe deformities can present with the same pattern of pain and callus formation as a hammer toe. These clinical entities can be difficult to distinguish; however, claw toe deformities typically involve all of the lesser toes. Patients with a mallet toe develop a callus at the tip of the toe from repetitive injury as the toe strikes the ground. Occasionally a callus can develop over the dorsal aspect of the distal interphalangeal joint.

Instability, subluxation, or dislocation at the MTP joint occurs with a lesser toe deformity or in isolation. Patients frequently complain of pain caused by the development of a callus beneath the metatarsal head or pain on the plantar aspect of the MTP joint at the insertion of the plantar capsule. [237] With the development of a deviated second toe, a patient complains of vague pain in the second intermetatarsal space. [240] This condition is often difficult to distinguish from an interdigital neuroma except that typically paresthesias or neuritic symptoms are not present in the toes (Box 25H-39).

BOX 25H-39

TYPICAL FINDINGS ASSOCIATED WITH LESSER TOE DEFORMITIES

Hammer Toe

- Callus formation under metatarsal head and over proximal interphalangeal joint
- Metatarsophalangeal joint subluxation or dislocation
- Flexion deformity of adjacent toes (contracture of flexor digitorum longus)
- Adjacent toe deformity such as hallux valgus

Claw Toe

- Callus formation under metatarsal head and over proximal interphalangeal joint
- Metatarsophalangeal joint subluxation or dislocation
- Multiple toe involvement

Mallet Toe

- Callus formation over the distal interphalangeal joint or on the tip of the toe

Metatarsophalangeal Joint Instability

- Plantar capsule pain
- Pain with drawer testing of the joint
- Dorsal, medial, or lateral deviation of the toe
- Pain in the adjacent interspace

When a keratosis develops over the lateral aspect of the fifth toe, patients complain of pain with pressure from shoewear directly over the area of callus formation (see Fig. 25H-70 **A** and **B**). [246] Occasionally, skin breakdown occurs, although callus formation is a more common abnormality. In the case of an interdigital corn, the patient may complain of symptoms of recurrent infection, and in such cases a sinus tract is found extending into a mass of subcutaneous scar tissue. Occasionally, patients have been treated for a long time with antibiotics with no resolution of the problem.

Physical Examination

The patient with a lesser toe deformity should be examined in a standing as well as a sitting position. [1563] [1567] [1578]

Each joint is evaluated for deformity, and, when present, deformities are identified as either flexible or rigid. It is important to evaluate for subluxation or dislocation of the MTP joint, most commonly of the second toe. Subluxation and dislocation (Fig. 25H-75) occur in a dorsal medial or lateral plane. [1570] [1572] Pain can occur with ambulation or is elicited with palpation or manipulation of the MTP joint. A dorsal plantar drawer test (Fig. 25H-76) is administered by thrusting the toe in a dorsal plantar direction. With capsulitis or MTP joint instability, pain is elicited. When a patient complains of presumable MTP pain, but no deformity is present, eliciting pain with a drawer test assists in making the correct diagnosis. [236]

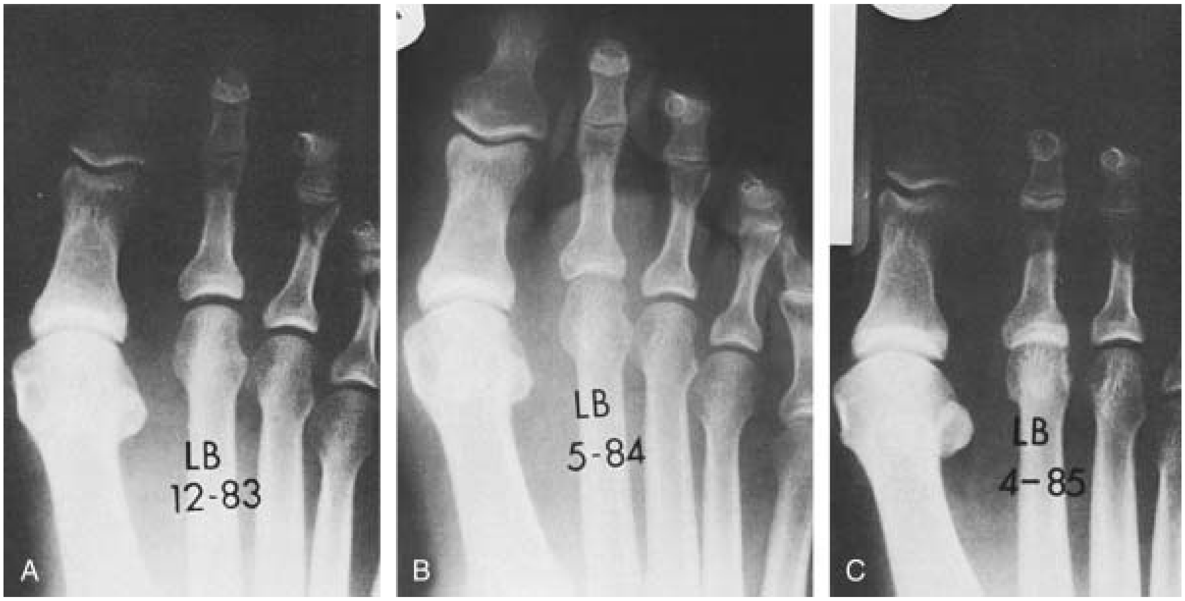

Figure 25H-75 A-C, Gradual subluxation of the metatarsophalangeal joint may occur. This tennis player developed subluxation and dislocation during a 5-year period. *(**A,** From Coughlin MJ: Lesser toe deformities. Orthopaedics 10:65, 1987; **B,** from Coughlin MJ: Lesser toe deformities. In Mann RA, Coughlin MJ [eds]: Surgery of the Foot and Ankle, 6th ed. St. Louis, Mosby, 1993.)*

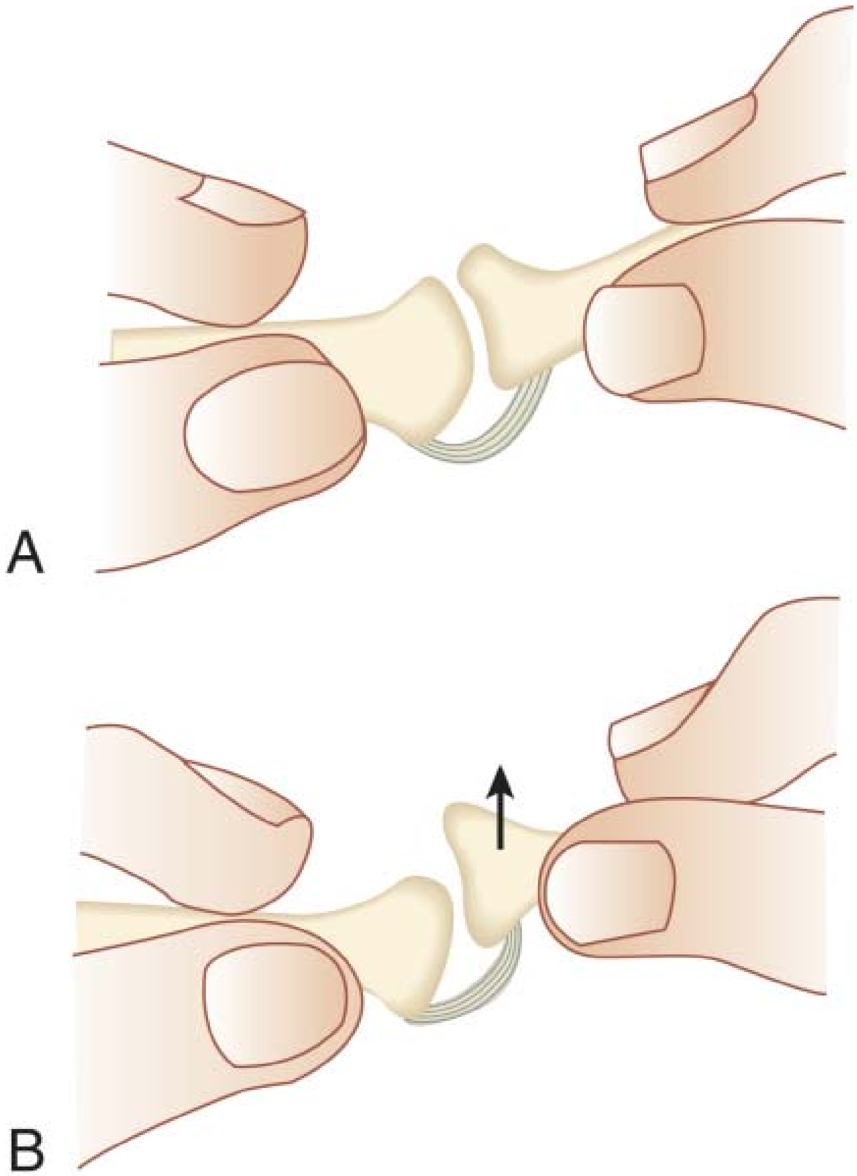

Figure 25H-76 Drawer test for metatarsophalangeal (MTP) joint instability. **A,** The toe is grasped between the thumb and the second finger. **B,** With a dorsal force, an attempt is made to subluxate the MTP joint. With instability of the MTP joint, pain is elicited with stress on the plantar structures. *(From Coughlin MJ, Mann RA: Lesser toe deformities. In Coughlin MJ, Mann RA [eds]: Surgery of the Foot and Ankle, 7th ed. St. Louis, Mosby, 1999, p 354.)*

In the evaluation of lesser toe deformities, it is important to note the amount of space present between adjacent toes. Lesser toes are inspected for deformity, callus formation, and interdigital corns. Occasionally, deformity diminishes the space available for a lesser toe correction. This diminished space requires correction of an asymptomatic adjacent toe (i.e., hallux valgus or an adjacent lesser toe deformity). [237]

With single hammer toe deformity, the adjacent toes are also evaluated in a standing position. Contracture of the flexor digitorum longus can result in a flexion deformity of an adjacent toe (Fig. 25H-77). In this case, in addition to carrying out a hammer toe repair, a flexor tenotomy is beneficial in the treatment of the hammer toe deformity. [237]

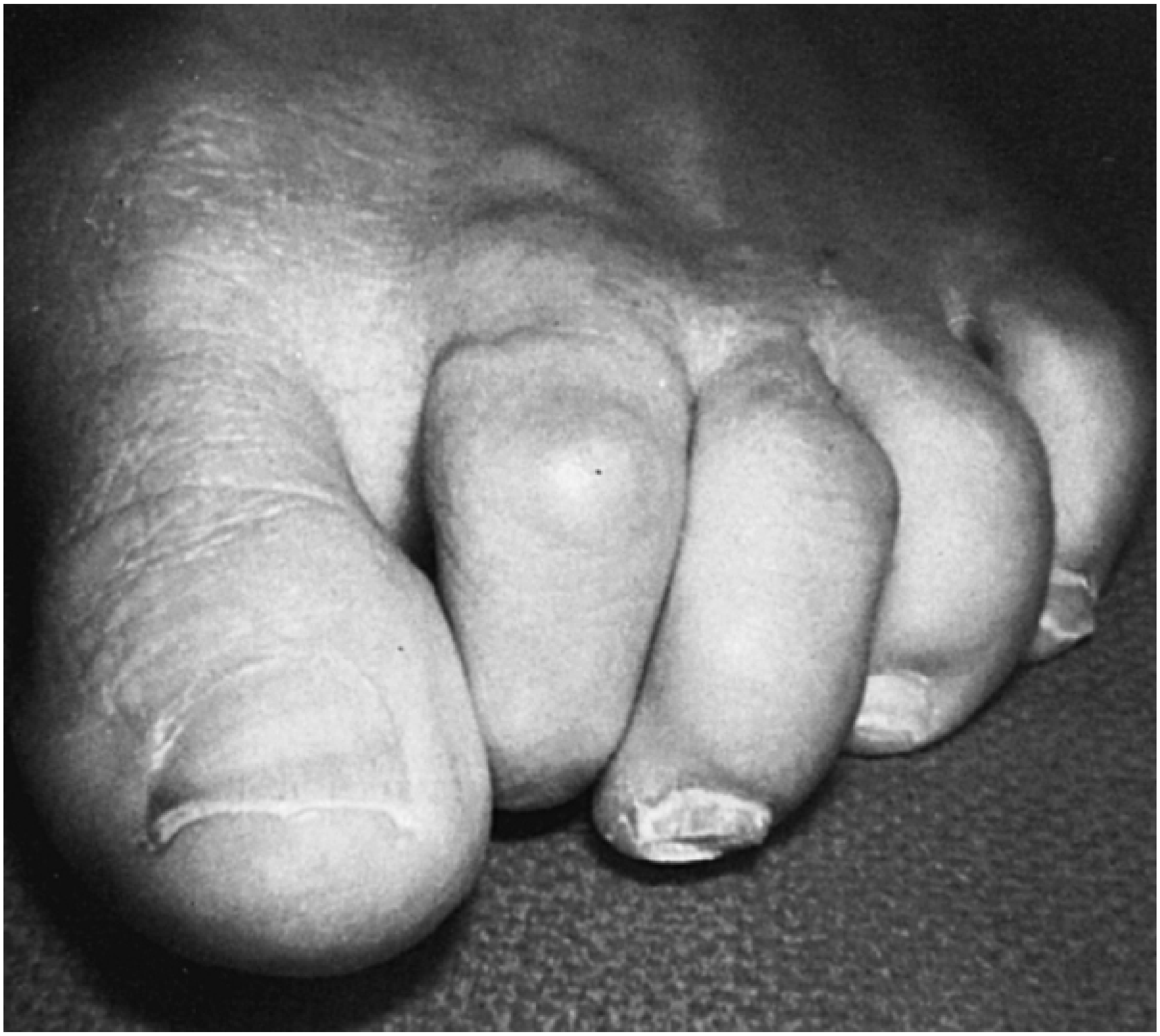

Figure 25H-77 Contracture of all the lesser toes may indicate a tightness of the flexor digitorum longus. *(© M. J. Coughlin. Used by permission.)*

On physical examination, deviation of the second toe either medially or dorsally is common. Pain is noted with ambulation and elicited with palpation of the second intermetatarsal space (see Box 25H-39). [240]

Imaging

Radiographs of the forefoot are important in analyzing lesser toe deformities. The presence of subluxation or dislocation at the MTP joint is best seen on an AP radiograph, whereas evaluation of a hammer toe is carried out best with a lateral radiograph (see Box 25H-39). Attention is given to bony abnormality or deformity underlying interdigital or lateral corns.

Treatment Options

Nonoperative

In the early stages of a lesser toe deformity, a roomy shoe with a low heel and an adequate toe box is appropriate for most flexible deformities. [1563] [1567] [1578] Range of motion exercise at each joint on a regular basis can help maintain flexibility of the toe. [237]

A deeper toe box eliminates dorsal pressure on the hammer toe or claw toe at the level of the proximal interphalangeal joint. [237] When a callus has developed, use of a soft insole or liner can decrease symptoms. [237] Foam or viscoelastic padding over a callosity or padding at the tip of a toe where calluses have occurred (Fig. 25H-78) often relieves the pressure. A toe cap or foam rubber pad can protect the end of the second toe in a mallet toe deformity. Padding the bony prominence over the lateral aspect of the fifth toe and shaving the keratotic lesion are recommended for symptomatic relief.[233] Padding placed in the interspace between the toes relieves discomfort of interdigital corns (see Fig. 25H-71 **C**). If an interdigital corn shows signs of chronic infection, the infection is treated by performing a culture and sensitivity studies and then instituting appropriate antibiotic therapy. When the acute infection has resolved, surgical treatment is considered to prevent recurrence.

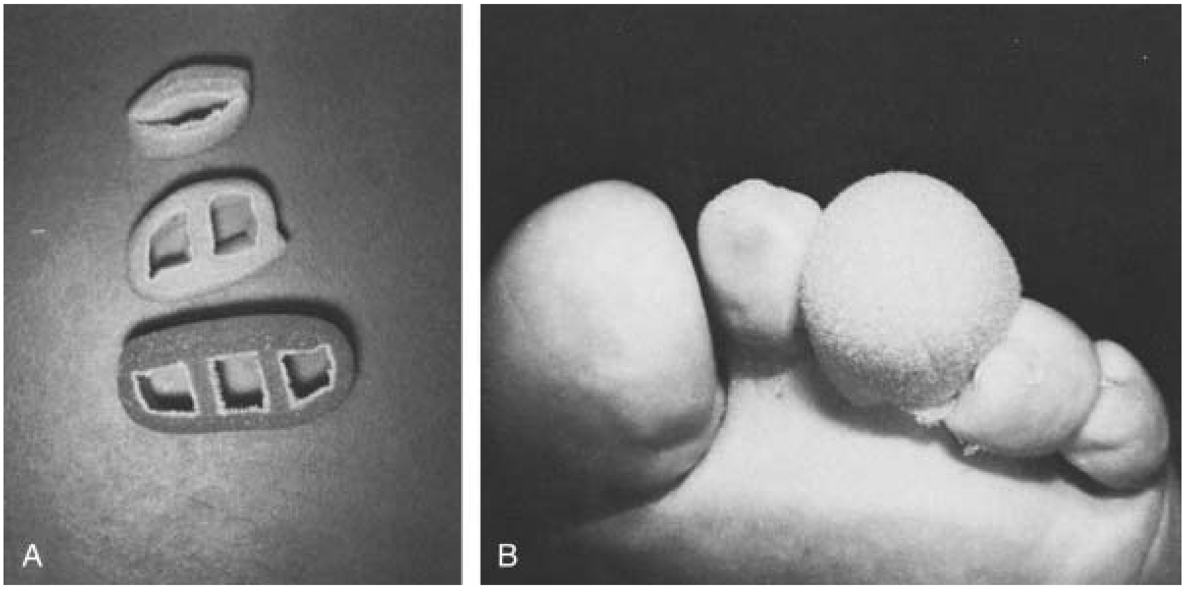

Figure 25H-78 **A,** Tube gauze may alleviate pain over a hammer toe. **B,** A toe cap pads a callus at the tip of the toe. *(**A,** From Coughlin MJ: Lesser toe deformities. In Mann RA, Coughlin MJ [eds]: Surgery of the Foot and Ankle, 6th ed. St. Louis, CV Mosby, 1993; **B,** from Mann RA, Coughlin MJ: Video Textbook of Foot and Ankle Surgery. St. Louis, Medical Video Production, 1991.)*

Metatarsal pads placed just proximal to the MTP joint alleviate plantar pressure and can decrease the hyperextension deformity at the MTP joint.[235] Taping the toe in a corrected position helps to stabilize the subluxated joint (Fig. 25H-79)[240]; with dislocation, however, taping rarely is successful in alleviating symptoms.[237] When a painful deformity does not respond to conservative care, surgical intervention is considered.

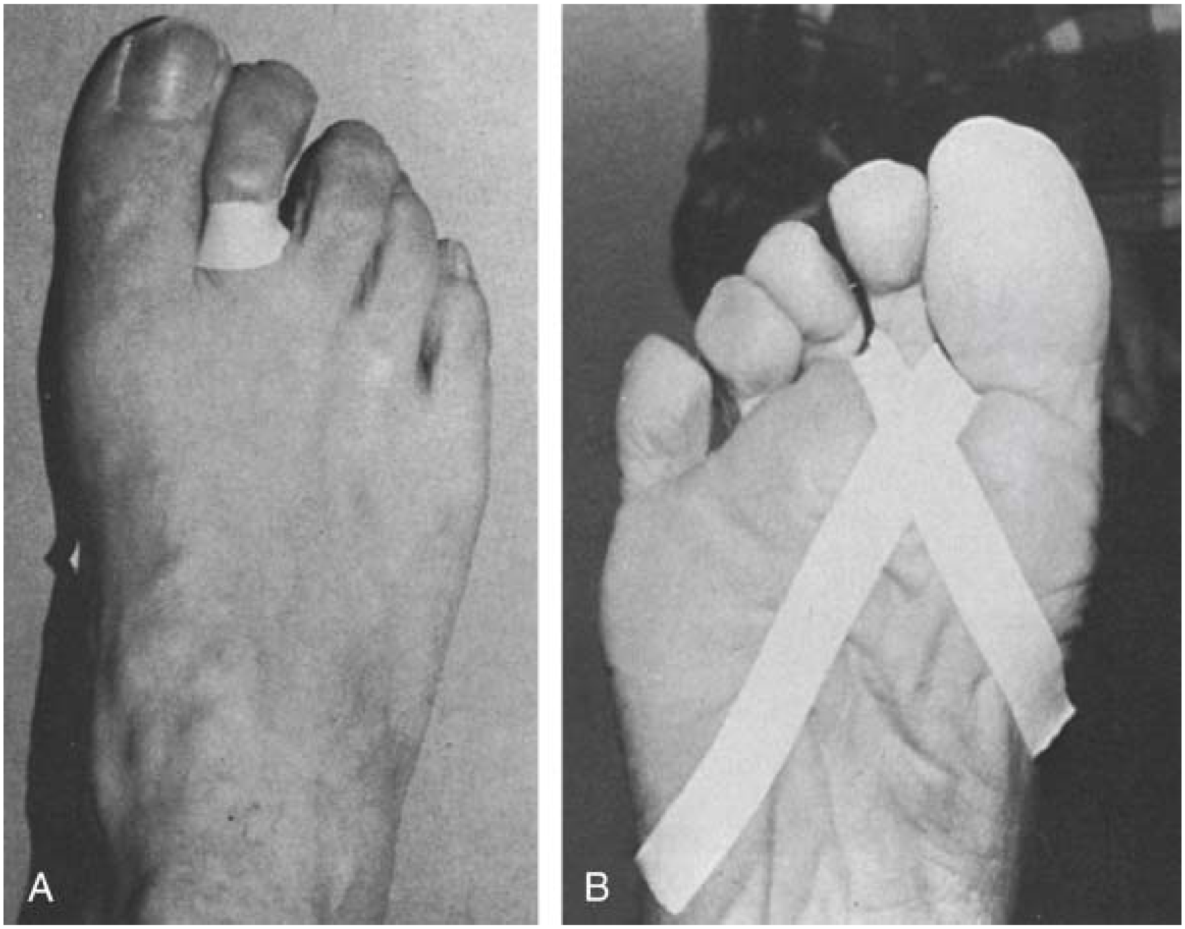

Figure 25H-79 **A** and **B,** A toe may be taped into a stable position. *(From Coughlin MJ: Crossover second toe deformity. Foot Ankle 8:29-39, 1987. © American Orthopaedic Foot and Ankle Society, 1987.)*

Operative

When a rigid deformity of the proximal (hammer toe or claw toe) (Fig. 25H-80) or distal (mallet toe) (Fig. 25H-81) interphalangeal joint is present, an interphalangeal joint arthroplasty is carried out (Box 25H-40 ; Figs. 25H-82 and 25H-83 [1340] [1345]). The technique is similar at either the proximal interphalangeal joint for a hammer toe or claw toe or the distal interphalangeal for a mallet toe. Whether an arthrodesis or an arthrofibrosis is obtained is not as important as the correction of the deformity and attaining stiffness at the interphalangeal joint. [244] With a fibrous proximal interphalangeal joint arthroplasty, about 15 degrees of motion is expected. [1567] [1578]

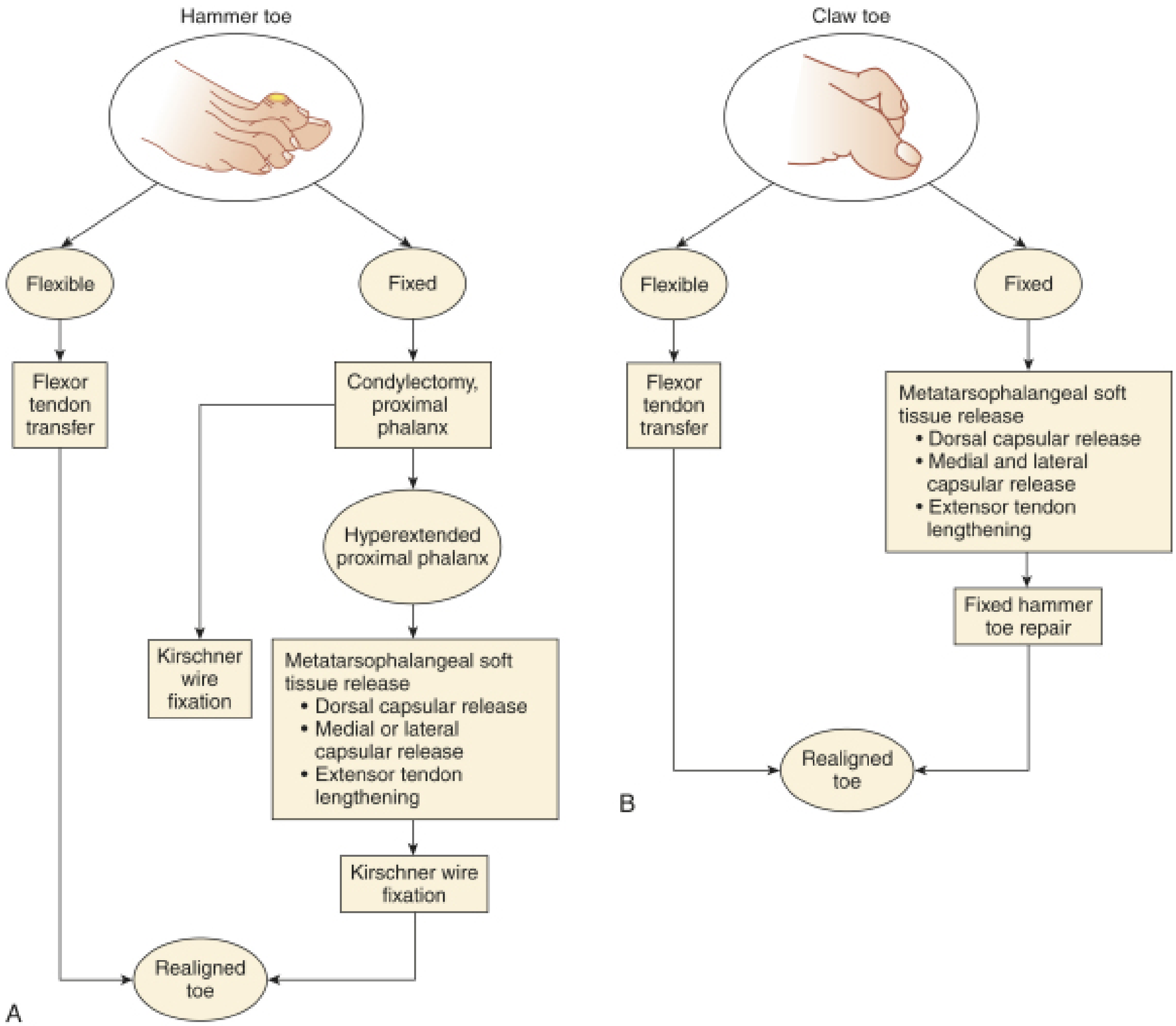

Figure 25H-80 **A,** Algorithm for treatment of hammer toe deformity. **B,** Algorithm for treatment of claw toe deformity. *(**A** and **B,** From Coughlin MJ, Mann RA: Lesser toe deformities. In Coughlin MJ, Mann RA [eds]: Surgery of the Foot and Ankle, 7th ed. St. Louis, Mosby, 1999, pp 331, 348.)*

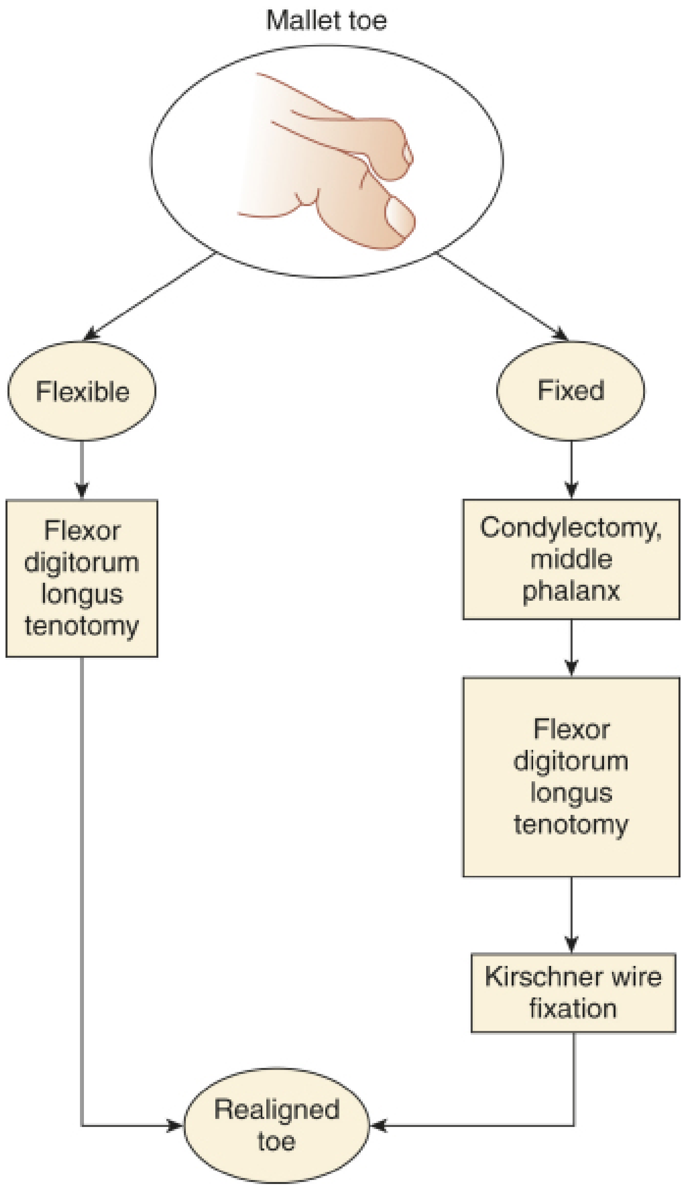

Figure 25H-81 Algorithm for mallet toe repair. *(From Coughlin MJ, Mann RA: Lesser toe deformities. In Coughlin MJ, Mann RA [eds]: Surgery of the Foot and Ankle, 7th ed. St. Louis, Mosby, 1999, p 343.)*

BOX 25H-40

INTERPHALANGEAL JOINT ARTHROPLASTY TECHNIQUE

- Center an elliptical skin incision over the dorsal aspect of the proximal (hammer toe and claw toe) or distal (mallet toe) interphalangeal joint.
- Excise the dorsal callus, the extensor tendon, and the joint capsule. [1563] [1567]
- Release the plantar capsule and the collateral ligaments carefully to deliver the head of the phalanx.
- Resect the condyles of the proximal (hammer toe and claw toe) or middle (mallet toe) phalanx (the amount of bone removed depends on the severity of the contracture).
- Perform a flexor tenotomy in a hammer toe repair if there is significant tightness of the long flexor tendon or an adjacent lesser toe appears to be contracted. [237] In a mallet toe deformity, the flexor digitorum longus is released through the same incision.

- Remove the articular cartilage of the base of the middle (hammer toe and claw toe) or distal (mallet toe) phalanx.
- Fix the toe with a 0.045 intramedullary Kirschner wire. [1567] [1578]
- Close the skin with vertical mattress sutures and apply a small compression dressing (see Figs. 25H-87 and 25H-88 [1365] [1370]).

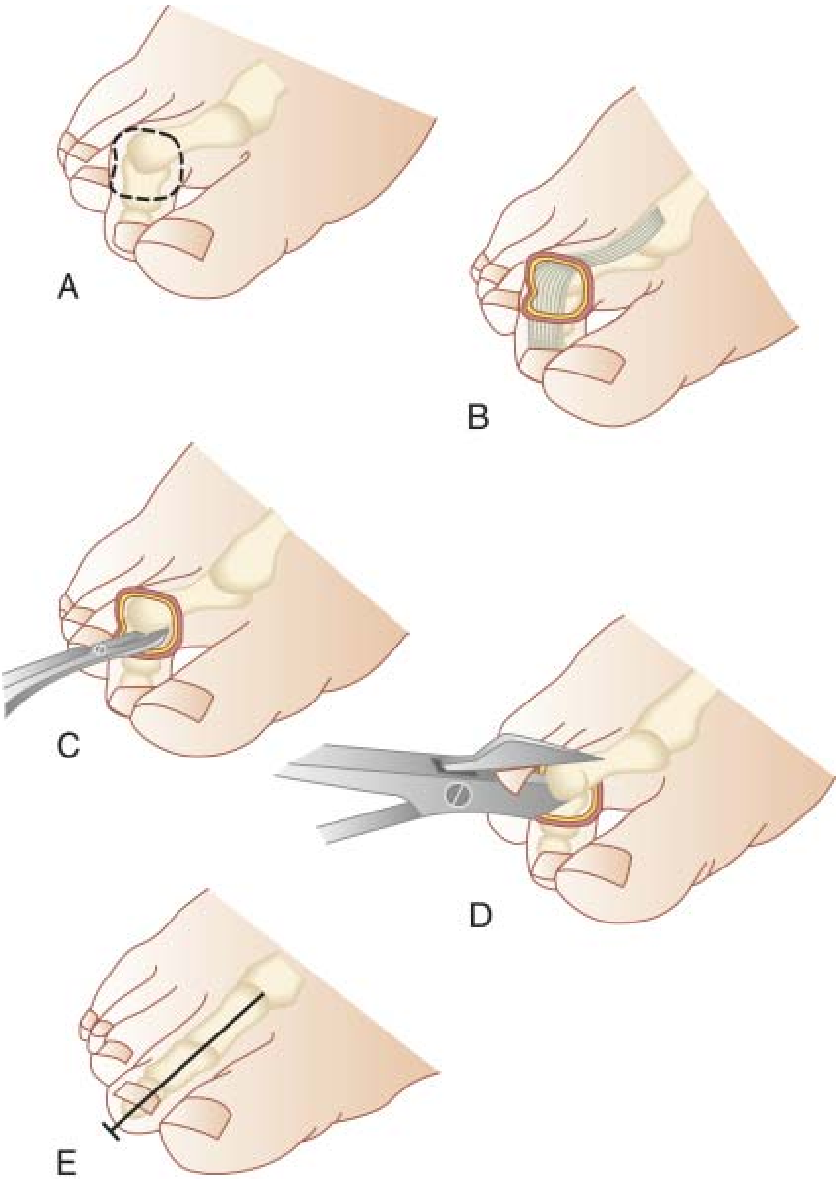

Figure 25H-82 **A,** Technique of fixed hammer toe repair. A dorsal elliptical incision excises skin, extensor tendon, and dorsal capsule. **B,** The collateral ligaments are released. **C,** The condyles of the proximal phalanx are delivered. **D,** The condyles are resected with a bone cutter. **E,** An intramedullary Kirschner wire is used for fixation.

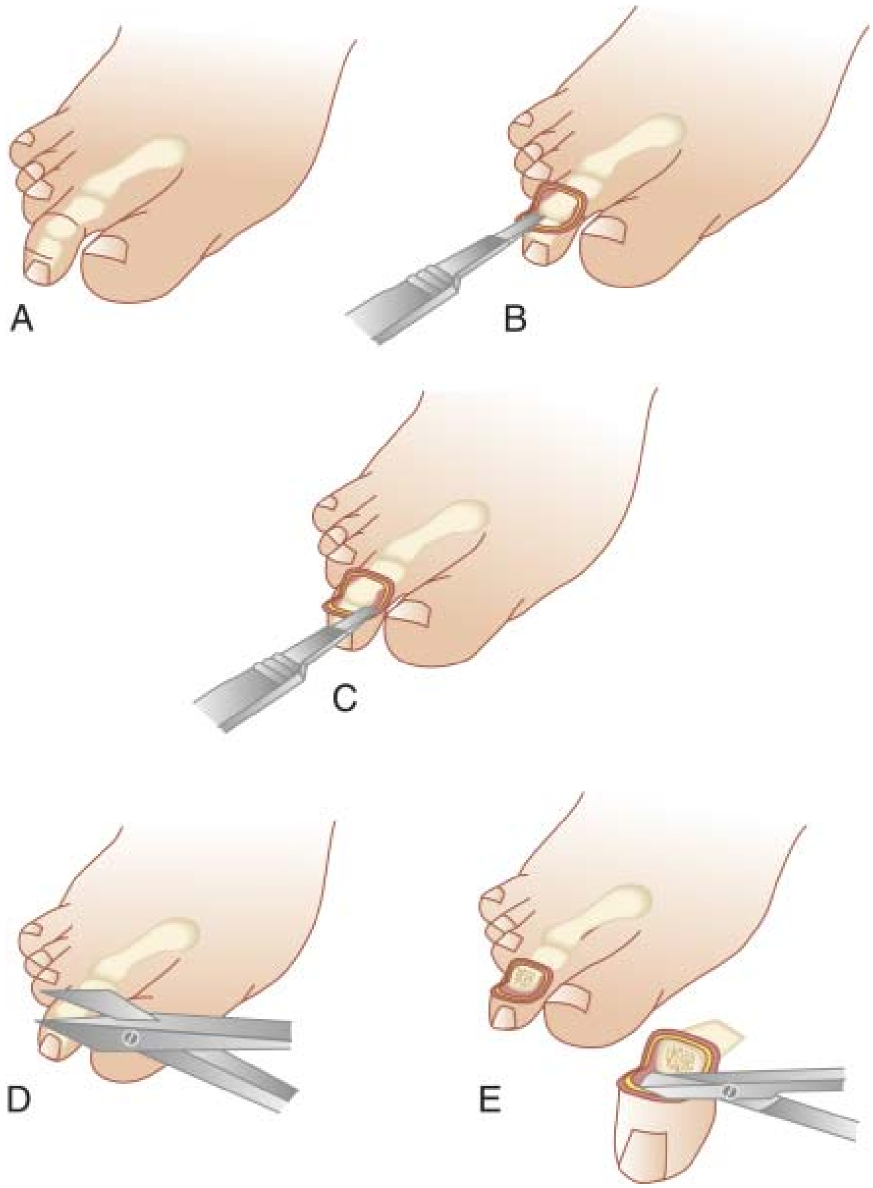

Figure 25H-83 Operative repair of mallet toe deformity. **A,** Elliptical skin incision. **B,** Extensor tendon and dorsal capsule are excised. **C,** The collateral ligaments are severed, exposing the condyles in the middle phalanx. **D,** The condyles of the middle phalanx are excised in the supracondylar region with a bone-cutting rongeur. **E,** The articular surface of the distal phalanx is excised. *(From Coughlin MJ, Mann RA: Lesser toe deformities. In Coughlin MJ, Mann RA [eds]: Surgery of the Foot and Ankle, 7th ed. St. Louis, Mosby, 1999, p 344.)*

When the deformity of the proximal interphalangeal joint in a hammer toe or claw toe is flexible, a flexor tendon transfer is performed (Box 25H-41 ; Fig. 25H-84). A Kirschner wire is placed from the tip of the toe in a proximal direction. Even when the wire fails to penetrate the metatarsal head, it still acts as a splint for the MTP joint. The Kirschner wire is used only to stabilize the repair. If the toe is not corrected completely, the deformity can recur when the wire is removed. Alternate fixation devices such as bioabsorbable pins have also been described. [250]

BOX 25H-41

FLEXIBLE HAMMER TOE CORRECTION TECHNIQUE

- Center a dorsal longitudinal incision over the proximal phalanx. [1567] [1599] [1600] [1601] [1602] [1609]
- Dissect superficial to the extensor tendon and adjacent to the extensor hood on each side of the toe. This dissection is superficial to the extensor hood and deep to the neurovascular bundle.

- Make a transverse incision at the plantar flexion crease of the metatarsophalangeal joint.
- Identify the tendon sheath of the flexor tendons and incise longitudinally. The tendon of the flexor digitorum longus is characterized by a median raphe and is the largest and deepest of the three tendons in the sheath (see Fig. 25H-89). [248]
- Apply tension to the tendon through this proximal plantar incision and percutaneously detach the tendon distally at the level of the distal interphalangeal joint.
- Pull the tendon into the proximal plantar incision and split longitudinally along the median raphe.
- Pass each half of the tendon ion either side of the proximal phalanx in a dorsal direction.
- With the ankle held in a neutral position and the toe held in about 20 degrees of plantar flexion, suture the tendon to the extensor expansion or to the other limb of the tendon.
- Tighten either limb until adequate alignment is achieved.
- Stabilize the repair with a 0.045 Kirschner wire (the toe should be realigned adequately before placement of the Kirschner wire).

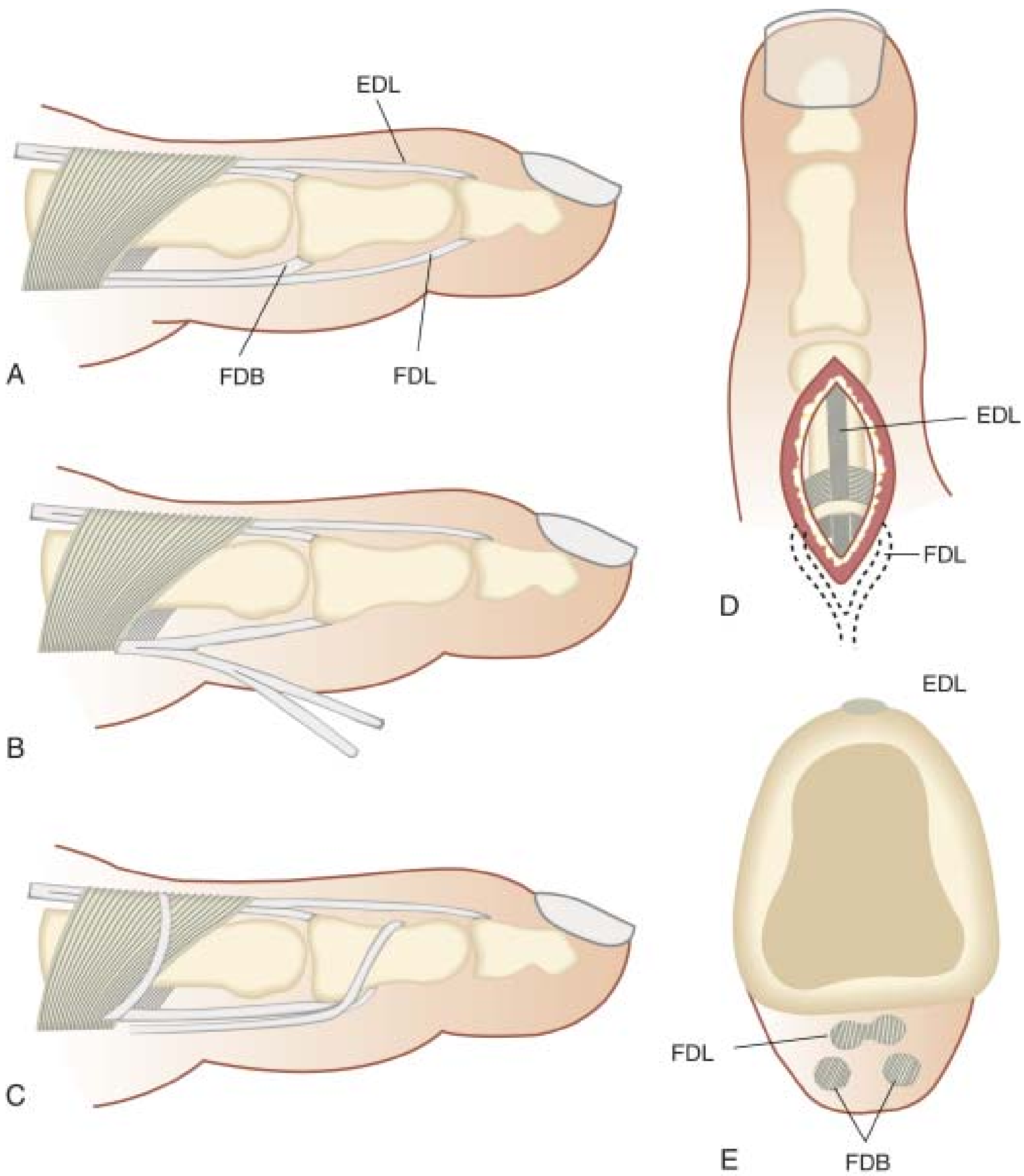

Figure 25H-84 Technique of flexor tendon transfer. **A,** Lateral view shows flexor digitorum longus (FDL), flexor digitorum brevis (FDB), and extensor digitorum longus (EDL). **B,** The flexor digitorum longus is detached through a distal puncture wound and is delivered through a transverse incision at the plantar metatarsophalangeal joint flexion crease. **C,** The tendon is split longitudinally, and each half is delivered on either side of the proximal phalanx and is sutured into either the extensor expansion or the corresponding limb of the flexor tendon. **D,** Dorsal view shows transferred flexor digitorum longus tendon. **E,** Cross-sectional view shows the characteristic position of the flexor digitorum longus tendon. It is deep to the flexor digitorum brevis and is characterized by a midline raphe.

An MTP joint abnormality is assessed in regard to the severity and rigidity of the deformity. Surgical correction is performed at this level to prevent recurrence of deformity. [233] A soft tissue release at the MTP joint (Box 25H-42) is used for mild (Fig. 25H-85 **A**) or moderate (see Fig. 25H-85 **B**) subluxation when the toe can be reduced easily. By achieving arthrofibrosis at the MTP joint, the stability of the second MTP joint is increased; however, this occurs at the expense of motion. With more severe instability and deformity (Fig. 25H- 86), further surgery is necessary to realign the second MTP joint. In this case, a complete soft tissue release of the MTP joint capsule and ligaments does not achieve a stable reduction. A flexor tendon transfer (Fig. 25H-87) can enhance the correction and achieve adequate MTP joint stability. Each limb of the flexor tendon can be sutured in place (see Box 25H-41), and the stability of the second toe is evaluated by dorsiflexion and plantar flexion of the ankle. When instability is still present or if the toe cannot be reduced easily, decompression of the MTP joint must be performed. [237] Several procedures, including partial proximal phalangectomy (Fig. 25H-88), metatarsal head arthroplasty (Fig. 25H-89), and shortening osteotomy of the metatarsal, have been described to decompress the MTP joint in more severe deformities. A partial proximal phalangectomy [251] (see Fig. 25H-88) frequently requires a syndactylization between the second and third toes to stabilize the floppy toe. Although a partial proximal phalangectomy decompresses the MTP joint, a more mechanically stable articulation is achieved by preserving the base of the proximal phalanx and decompressing the joint instead through a metatarsal osteotomy (Box 25H-43) or joint arthroplasty. [1567] [1578] [1582] [1583]

BOX 25H-42

METATARSOPHALANGEAL JOINT RELEASE TECHNIQUE

- Center a curvilinear incision over the metatarsophalangeal joint.
- Lengthen or release the extensor tendon. [240]
- Release the dorsal, medial, and lateral capsules and resect any hypertrophic synovium.
- Release or tighten additional structures if needed to reduce the metatarsophalangeal joint:
    - Release the first lumbrical if it is acting as a deforming force. [237]
    - Release plantar adhesions. [237]
    - Tighten elongated medial or lateral collateral ligaments to correct varus or valgus deformity.
- Reduce the subluxated metatarsophalangeal joint after the soft tissue release and test for stability by passive dorsiflexion and plantar flexion of the ankle joint.
- Stabilize the toe with an intramedullary 0.045 Kirschner wire, if the toe is stable and does not redislocate.

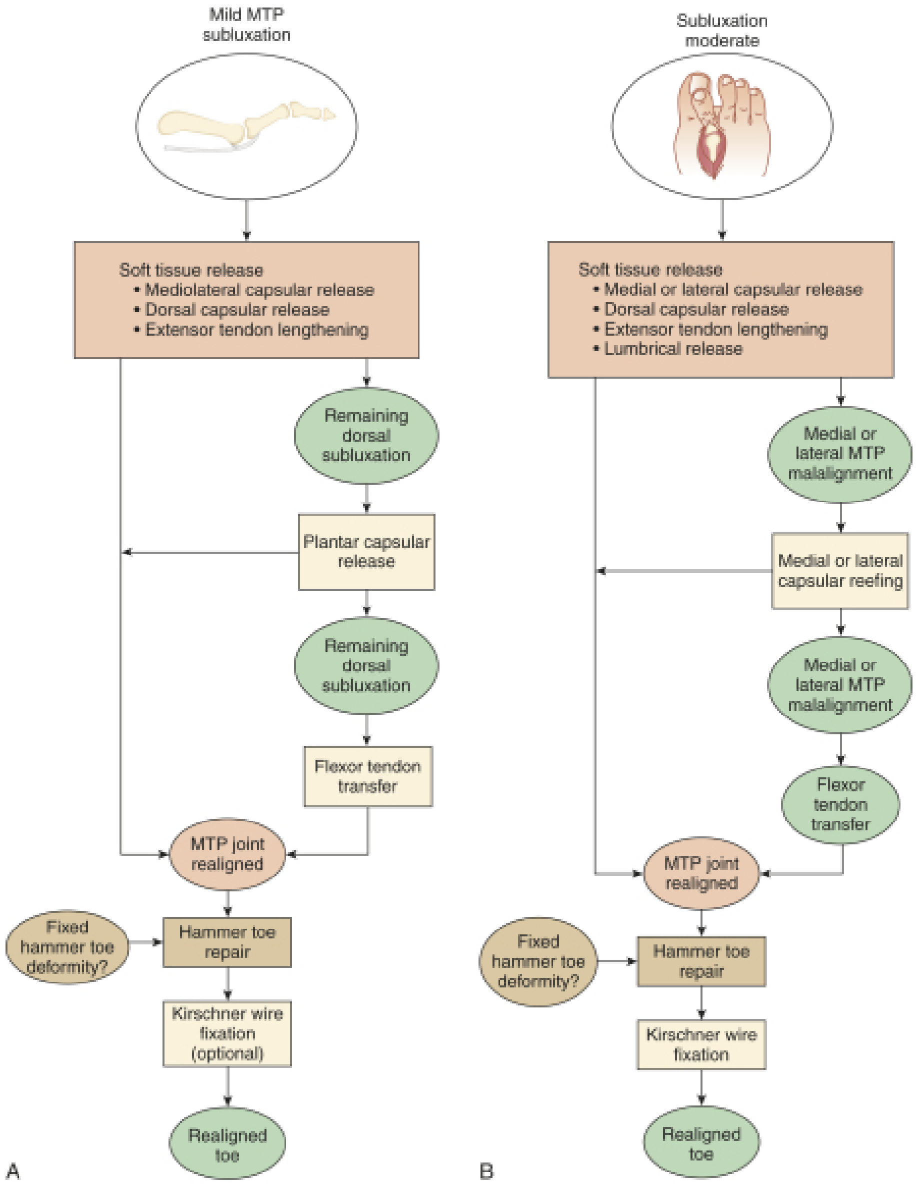

Figure 25H-85 A, Algorithm for treatment of mild subluxation of lesser toe metatarsophalangeal (MTP) joint. **B,** Algorithm for treatment of moderate MTP joint subluxation. *(From Coughlin MJ, Mann RA: Lesser toe deformities. In Coughlin MJ, Mann RA [eds]: Surgery of the Foot and Ankle, 7th ed. St. Louis, Mosby, 1999, pp 360, 362.)*

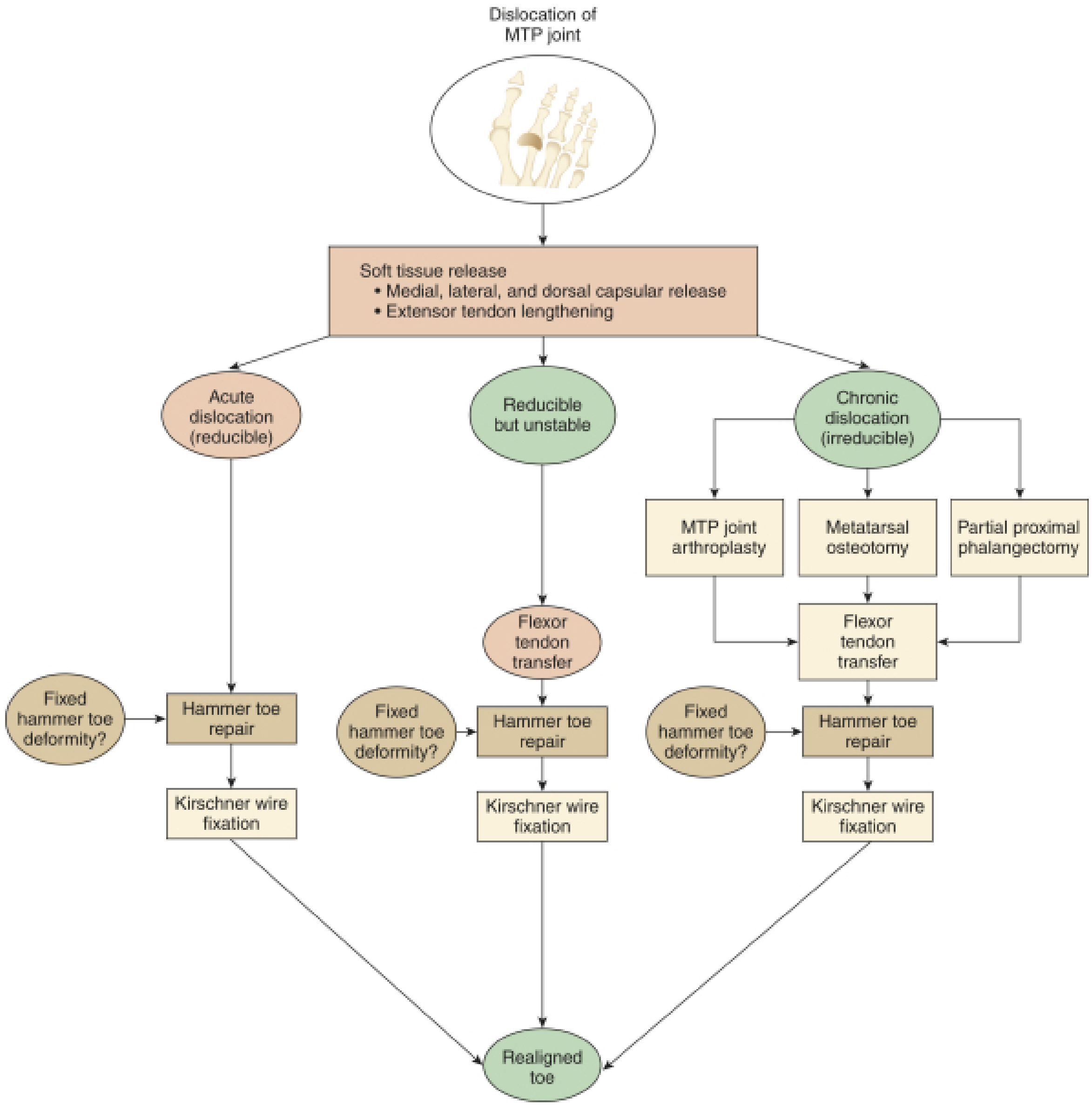

Figure 25H-86 Algorithm for treatment of subluxation/dislocation of metatarsophalangeal (MTP) joint. *(From Coughlin MJ, Mann RA: Lesser toe deformities. In Coughlin MJ, Mann RA [eds]: Surgery of the Foot and Ankle, 7th ed. St. Louis, Mosby, 1999, p 366.)*

Figure 25H-87 **A,** Preoperative photograph of crossover second toe deformity. **B,** Preoperative radiograph shows second toe deformity. **C,** Three years after soft tissue release and flexor tendon transfer. **D,** Radiograph 3 years after surgical repair. *(From Coughlin MJ: Cross over second toe deformity. Foot Ankle 8:29-39, 1987. © American Orthopaedic Foot and Ankle Society, 1987.)*

Figure 25H-88 A partial proximal phalangectomy may be used to decompress the second metatarsophalangeal joint. *(© M. J. Coughlin. Used by permission.)*

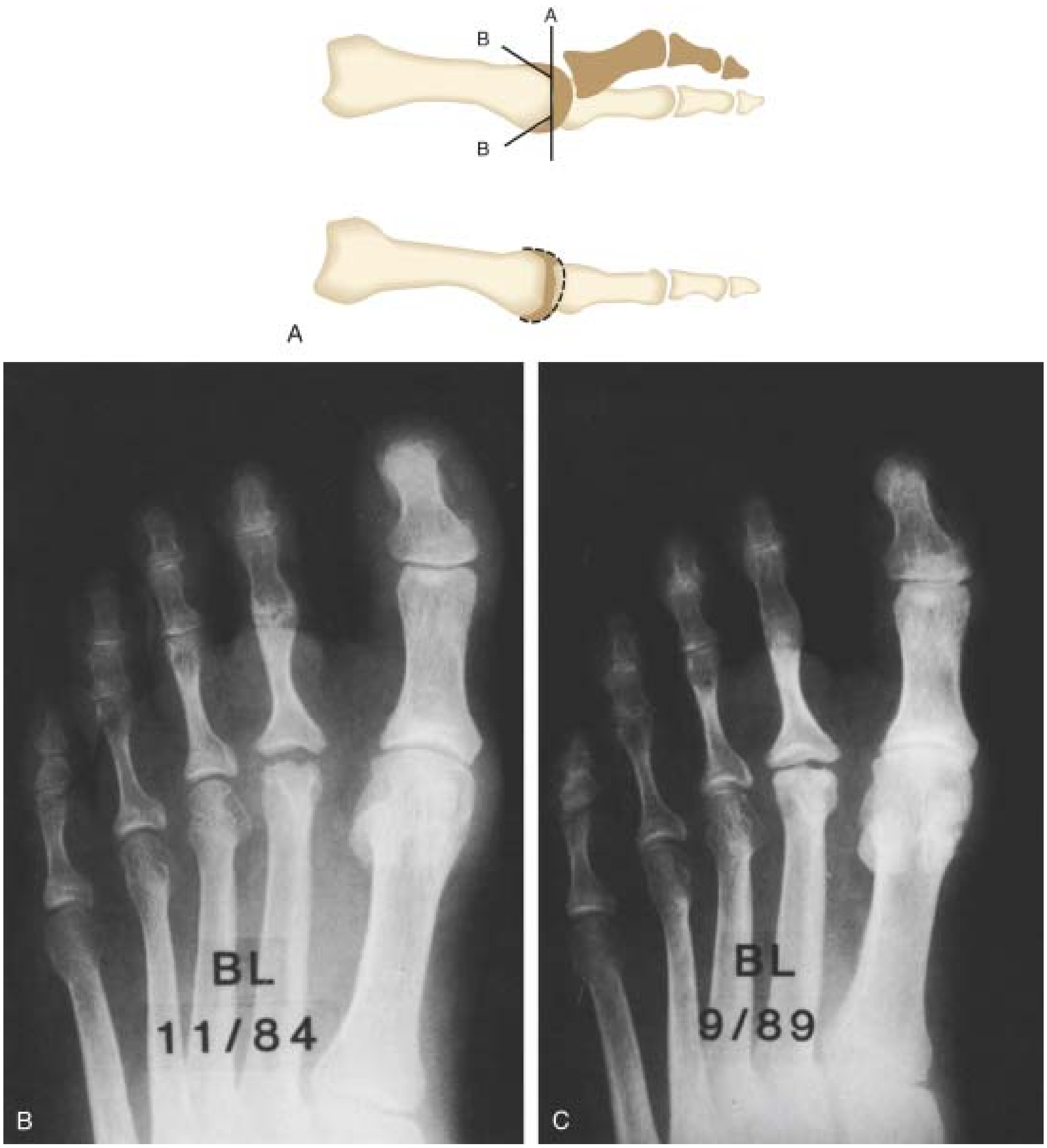

Figure 25H-89 A, A metatarsophalangeal arthroplasty requires removal of 2 to 3 mm of articular surface and beveling of the metatarsal head on the dorsal and plantar aspects. **B,** Radiograph after metatarsophalangeal arthroplasty. **C,** Five-year follow-up after metatarsophalangeal arthroplasty. Joint motion is reduced about 50%. *(**A,** © M. J. Coughlin. Used by permission; **B** and **C,** from Mann RA, Coughlin MJ [eds]: Surgery of the Foot and Ankle, 6th ed. St. Louis, Mosby, 1993, p 390.)*

BOX 25H-43

CAPITAL OBLIQUE OSTEOTOMY TECHNIQUE

- Center a 3-cm longitudinal incision over the lesser metatarsophalangeal joint.
- Release the dorsal, medial, and lateral capsule.
- Reduce the metatarsal head and plantar flex the phalanx to expose the metatarsal head.
- Perform a longitudinal oblique distal metatarsal osteotomy, holding the saw blade parallel to the plantar surface of the foot
- Penetrate the distal superior metatarsal articular surface 2 to 3 mm inferior to the dorsal metatarsal surface.

- Create the osteotomy in the plane parallel to the plantar surface of the foot and proceed in a proximal direction until the saw blade has penetrated the proximal metatarsal cortex.
- Translate the distal fragment proximally by the desired amount (2 to 6 mm).
- Stabilize the osteotomy with one or two minifragment lag screws. (Excessively long screw can cause plantar pain.)
- Resect the excess dorsal metatarsal surface that extends beyond the articular surface with a rongeur (see Fig. 25H-66).

Lateral fifth toe corns are treated with removal of a bony prominence at the interphalangeal joint. [1567] [1578] If only the lateral condyle is prominent, it is excised (see Fig. 25H-70 **C** and **D**). When the base of the middle phalanx is prominent, it is also shaved with a rongeur. When a contracture of the interphalangeal joint is present, resection of the condyles of the proximal phalanx achieves a more long-lasting repair. [233] A technique similar to that used for a fixed hammer toe deformity is employed (see Box 25H-40), stabilizing the toe with an intramedullary Kirschner wire. With recurrence of a lateral fifth toe corn after a condylectomy, a flexor tenotomy reduces the fifth toe contracture and diminishes symptoms.

Interdigital corns with an underlying exostosis are treated with removal of the exostosis (see Fig. 25H-71 **D**). [1567] [1578] [1584] The incision should be kept out of the web space because these wounds are slow to heal and can become macerated or secondarily infected. If possible, the capsule of the involved joint should be repaired. When prominent exostoses are adjacent to each other, both are resected. [246]

Often hindfoot or midfoot deformity is the cause of discomfort in patients with lesser toe deformity, especially claw toes. [237] It is important to address the major deformity when treating the patient.

Weighing the Evidence

Ohm and colleagues [255] reported on 25 patients (62 hammer toe repairs) in whom an interphalangeal fusion was performed. A 100% fusion rate was achieved. An equal number of corrections were performed in the second, third, and fourth toes. Although many authors advocate attempted proximal interphalangeal arthrodesis, [1586] [1587] [1588] adequate resection with realignment that achieves a stable alignment of the toe is considered a successful result. [1589] [1590] [1591] A fusion of the proximal interphalangeal joint or an arthrofibrosis succeeds by converting the flexor digitorum longus to a flexor of the entire digit.

Coughlin and associates [247] reported on 63 patients (118 toes) with a fixed hammer toe deformity. Involvement of the second toe was noted in 35%, the third toe in 21%, the fourth toe in 24%, and the fifth toe in 20%. After a resection arthroplasty technique with intramedullary Kirschner wire fixation, fusion of the proximal interphalangeal joint occurred in 81% of the involved toes. Subjective acceptable alignment was achieved in 86% of cases. Pain was relieved in 92%, and subjective satisfaction was noted by 84% of patients. Malalignment and numbness were the major factors reported to be associated with an unsuccessful result.

The rate of pseudarthrosis in some series approaches 50%, although a higher fusion rate has been achieved with peg-and-dowel type of technique. [1586] [1592] [1593] Lehman and Smith [262] reported a 50% patient satisfaction rate with this technique, however. Major reasons for postoperative dissatisfaction included angulation of the lesser toe and incomplete relief of pain. McConnell [1594] [1595] reported on a large series of patients treated with diaphysectomy. This technique was useful in treating a hammer toe deformity as well as shortening a substantially long lesser toe. The actual postoperative alignment of the lesser toe and complication rate were not reported in either of McConnell's series. Daly and Johnson [266] reported on a large series of hammer toes treated with partial proximal phalangectomy. Although 75% patient satisfaction was noted, 43% of patients noted moderate footwear restrictions, 27% reported residual pain, 28% noted cosmetic dissatisfaction, and 18% reported recurrent cock-up deformity. Cahill and Connor [267] reported on 78 patients (84 feet). They noted 50% poor results after partial proximal phalangectomy. Conklin and Smith [268] noted 29% postoperative dissatisfaction after this procedure.

Taylor, [1599] [1600] Pyper, [271] and others [1596] [1602] reported satisfactory results ranging from 51% to 89% after correction of a flexible hammer toe deformity. Thompson and Deland [273] reported excellent pain relief; however, only 54% of patients examined were noted to have complete correction of deformity. Kuwada [274] and Barbari and Brevig [275] reported greater than 90% satisfaction in patients after flexor tendon transfer. Boyer and DeOrio [276] used a modification of the flexor tendon transfer with 72% satisfaction.

Trnka and colleagues [253] reported on 25 osteotomies of the second, third, and fourth metatarsals. Of 15 patients, 12 were satisfied with their results. Reasons for dissatisfaction included pain associated with a prominent plantar screw.

Three patients developed an asymptomatic callus beneath the involved lesser metatarsal head. All osteotomies healed. All 25 dislocated MTP joints were relocated successfully with an average metatarsal shortening of 4.4 mm. No cases of avascular necrosis were described, and the authors concluded that this technique enabled an accurate metatarsal shortening because the distal metatarsal fragment could be positioned exactly and stabilized with internal fixation.

Coughlin[240] reported on the surgical correction of 15 toes (11 patients) with a variety of methods depending on the severity of the deformity. Extensor tendon lengthening or tenotomy, MTP joint release, flexor tendon transfer, Kirschner wire fixation, and occasionally metatarsal articular resurfacing were used in the treatment of these acute and chronic MTP joint subluxations. Satisfactory results were reported in 93%. Of these patients, 79% were women with an average age of 60 years. In a follow-up study, Coughlin[242] reported on a younger group of patients (9 patients [11 toes]) with an average age of 50 years. A positive result on drawer testing was noted in all cases and was pathognomonic of early second MTP joint instability. Using a similar soft tissue realignment technique, good and excellent results were reported in 71%.

After a mallet toe repair, generally satisfactory results have been reported. Coughlin[249] obtained successful fusion in 72% of cases; 86% of patients were satisfied with their results. Of patients with a fibrous union, 75% were satisfied, although slightly less so than patients with a successful distal interphalangeal arthrodesis. Pain relief was noted by 97% and correction of deformity by 91% of patients. Although not performed in all cases, a flexor tenotomy was associated with a slightly higher rate of satisfaction and maintenance of correction.

Barbari and Brevig[275] performed a flexor tendon transfer for correction of a flexible claw toe deformity and noted satisfactory results in 90% of cases, although one third of patients reported postoperative metatarsalgia. Postoperatively, 12 patients noted no interphalangeal joint motion, 15 had reduced motion, and 12 reported the same amount of motion at the interphalangeal joint. Continuing complaints were noted by patients who had a fixed claw toe deformity that would have been treated more appropriately with resection arthroplasty. With refinement of this procedure and the use of a flexor digitorum longus transfer, Thompson and Deland[273] reported improved results, although complete realignment of MTP subluxation was achieved in only 54%.

After a simple condylectomy to repair a lateral fifth toe corn, in about 2% of patients, a keratotic lesion persists despite resection of the bony prominence.[237] In this situation, soft tissue trimming with a scalpel usually results in reduction of the keratotic lesion to a minimally symptomatic state. An excision of an excessive portion of the middle phalanx should be avoided because this can destabilize the fifth toe.

Zeringue and Harkless[277] reported their results after the treatment of 30 patients with an interdigital corn excision. Of the affected feet, 94% were noted to have rotation of the fifth toe. These patients were treated with a proximal interphalangeal joint arthroplasty of the fifth toe combined with lateral-based condylectomy of the fourth toe proximal phalanx. A high level of satisfaction was found at an average follow-up of 3 years. Recurrence of a soft corn is the most common postoperative complication. When recurrence develops, a more extensive resection is considered. A complete condylectomy may be necessary in cases of recurrence in the presence of a severe deformity.[246]

AUTHOR'S PREFERRED METHOD

Deformities of the lesser toes can cause significant morbidity in athletes. Pressure from ill-fitting footwear leads to blistering or callus formation over bony prominences. Padding of areas at risk, coupled with the use of well-fitting shoes, alleviates many symptoms pertaining to the lesser toes. Because most surgery of the lesser toes involves some form of joint arthroplasty, reduced range of motion of the involved joint is a common postoperative occurrence. An athlete should be counseled that although surgery typically reduces discomfort significantly, decreased range of motion can impair athletic activity or performance. It usually is preferable to perform surgery in athletes when the athlete already has modified activity because of discomfort and conservative treatment has been unsuccessful.

A fixed hammer toe or claw toe deformity is corrected by performing a condylectomy of the proximal phalanx. A flexor tenotomy is used when contractures of adjacent toes are present. Release of contracted dorsal medial and dorsal lateral capsular structures is performed when a hyperextension deformity of the MTP joint is present. If severe subluxation or dislocation has occurred, decompression of the MTP joint often is necessary. A distal metatarsal shortening osteotomy is preferable because it affords more stability to the MTP joint and preserves the articular cartilage. A flexor tendon transfer often is necessary to give a stabilizing plantar flexion force to the proximal phalanx. Care must be taken not to perform excessive surgery on the second toe that leads to vascular compromise.

A flexible hammer toe or claw toe is repaired by performing a flexor tendon transfer. During the preoperative physical examination, it is important to determine that the toe is passively correctable. If a flexor tendon transfer is performed on a toe with a fixed deformity, a less than complete repair results.

A flexible mallet toe in a younger person is corrected with a flexor tenotomy. When a fixed contracture develops, a condylectomy of the middle phalanx, coupled with a flexor tenotomy, allows realignment of the toe.

A lateral fifth toe corn is treated by removing the prominent exostosis of the lateral condyle of the proximal phalanx. A complete capsular repair is important to realign the toe. When a more severe deformity is present or when a flexion deformity is present at the interphalangeal joint, a condylectomy of the proximal phalanx is performed, and Kirschner wire stabilization is used. An interdigital corn repair is addressed by removing either one or both of the corresponding exostoses. [246] Through a dorsal approach, the exostosis is identified and resected. If possible, a capsular repair is performed.

The most important aspect of surgery on the lesser toes is the need to repair all components of the deformity so that adequate alignment is achieved. Care must be taken to protect the vascular supply to the lesser toes. Sometimes an intramedullary Kirschner wire must be removed when postoperative vascular compromise occurs. It is better to perform a two-stage lesser toe repair than to risk severe ischemia or necrosis of a lesser toe postoperatively.

Postoperative Prescription, Outcomes Measurement, and Potential Complications

When surgical intervention is used to repair hammer toes, mallet toes, claw toes, and hard and soft corns, postoperative management should concentrate on the goals of maintaining physical fitness, while protecting the repaired lesser toes until adequate healing has occurred. The patient is allowed to ambulate in a postoperative shoe immediately. While wound healing is occurring, upper extremity activities and exercising on a stationary bicycle can be used to maintain fitness. Sutures are removed 2 to 3 weeks after surgery. Pins are removed 4 weeks after surgery. After pin removal, the involved lesser toe is taped to maintain adequate alignment for another 6 weeks. In the case of a flexible hammer toe repair or release of the MTP joint, the toe is taped in slight plantar flexion (see Fig. 25H-79), or, in cases of medial deviation or a crossover second toe, the toe is taped in a lateral direction. If correction is performed at the fifth toe interphalangeal joint, the fifth toe is taped to the fourth toe after the pin is removed. It is important during the 6-week period of taping after pin removal to protect the toe from stress that increases the risk for recurrence of the deformity. Walking activities are allowed about 6 weeks after surgery; the involved toe is protected in a shoe with a stiff sole and a roomy toe box. Running should be avoided until 9 to 12 weeks after surgery to allow adequate healing to occur.

Lesser toe deformity correction frequently results in postoperative stiffness at either the interphalangeal joint or the MTP joint (Box 25H-44). Passive manipulation helps to alleviate these symptoms. Patients should be counseled that an interphalangeal joint arthroplasty will leave residual stiffness. After a tendon transfer, the ability to curl the toes is sacrificed. When correction of a flexible deformity is planned, a patient should be counseled preoperatively that dynamic function of the lesser toe is absent after flexor tendon transfer. Although this is not a cause of disability, a patient must be counseled about the tradeoff of function of the flexor digitorum longus for stability or realignment of the lesser toe. After distal metatarsal osteotomy, patients experience limitation of plantar flexion at the MTP joint as well as reduced range of motion.

BOX 25H-44

EXPECTATIONS AFTER CORRECTION OF LESSER TOE DEFORMITY

- Swelling frequently lasting 6 months or more
- Stiffness at the metatarsophalangeal or interphalangeal joints
- Decreased function after flexor to extensor transfer
- “Molding” of the toe

After an operative procedure on any of the lesser toes, the most frequent postoperative finding is swelling. Although swelling often persists for 1 to 6 months, it invariably subsides with time. [1579] [1585] [1592] Postoperatively, the toe often assumes the shape of adjacent toes, which is known as *molding*. Molding of a lesser toe because of intrinsic pressure from an adjacent toe is a common occurrence. It is often unavoidable, [233] and it is best to counsel the patient preoperatively that this molding may occur. Usually, the main anxiety of the patient is the cosmetic appearance of the toes. Preoperative counseling alerts the patient to this possibility.

Recurrence of deformity occasionally is noted (Box 25H-45). Adequate decompression of the deformity at the joint involved is necessary to prevent recurrence. Excessive resection can lead to an unstable or floppy toe, and inadequate resection can lead to recurrence. If the toe is not corrected completely at the time of surgery, an intramedullary Kirschner wire should not be used to achieve correction only for stabilization. In this situation, when the Kirschner wire has been removed, the deformity often recurs. In the face of recurrence of a hammer toe deformity, a flexor tenotomy can aid in correction. Occasionally, recurrence of a mallet toe deformity is noted because the flexor digitorum longus tendon is not released. Postoperative malalignment is also a source of patient dissatisfaction.[247] Arthrodesis or arthrofibrosis gives an inherent stability to the digit and helps to resist the deforming forces of lesser toes. Complications associated with Kirschner wire fixation are uncommon, but migration or breakage or pin tract infection occasionally occur.[278] If patients develop pain at the site of the pseudarthrosis, an injection of corticosteroid usually gives lasting relief.

BOX 25H-45

COMPLICATIONS ASSOCIATED WITH CORRECTION OF LESSER TOE DEFORMITY

- Recurrent deformity
- Postoperative malalignment
- Vascular compromise
- Localized numbness
- Kirschner wire complications: migration, breakage, infection

The most significant complication after lesser toe surgery is vascular compromise. If extensive surgical correction is required, it is preferable to do a two-stage repair rather than to incur a vascular injury to a lesser toe. If the circulatory status of the toe is impaired, constrictive dressings are corrected first. Occasionally, a Kirschner wire must be removed to improve circulation. When the Kirschner wire fixation is removed, the surgical correction is maintained with postoperative dressings.

Occasionally, a Kirschner wire fatigues and breaks. Usually, this break occurs proximal to the metatarsal articular surface, and if the proximal portion of the pin is retained completely within the metatarsal, it does not need to be removed. If the pin compromises joint function, it can be removed through an arthrotomy of the involved joint.

Injury to an adjacent digital nerve results in an area of numbness, which is associated with patient dissatisfaction. Preoperative toenail deformity typically does not resolve after correction of a mallet toe, and the patient should be counseled appropriately.

Criteria for Return to Play

Walking is initiated 6 to 8 weeks after surgery in a stiff-soled shoe with a roomy toe box. Taping of the toe begins at 4 weeks when pins are removed and continues for a total of 6 weeks. Occasionally, swelling prohibits appropriate shoewear, and initiation of walking is delayed. The athlete progresses to running at 9 to 12 weeks postoperatively. Osteotomies of the metatarsal should be healed both clinically and radiographically before advancing to running activities. Athletic activities are advanced after 12 weeks as pain and swelling allow. Full return to sport is expected 4 to 6 months after surgery (Box 25H-46).

BOX 25H-46

CRITERIA FOR RETURN TO PLAY AFTER CORRECTION OF LESSER TOE DEFORMITY

- Clinical and radiographic evidence of bony healing after metatarsal osteotomy
- Decrease in swelling that allows a stiff sole shoe with a roomy toe box
- Return to sport expected 4 to 6 months after surgery

BUNIONETTES

A bunionette, or tailor's bunion, is characterized by a prominence of the lateral eminence of the fifth metatarsal head. This deformity originally was described by Davies,[280] who observed that pressure over the lateral aspect of the fifth metatarsal head could lead to chronic irritation of the overlying bursa. Sitting in a cross-legged position gave rise to the term *tailor's bunion*. Pressure from constricting footwear leads to the development of thickened keratoses over the lateral [1611] [1612] [1613] or plantar lateral [284] aspect of the metatarsal head. Typically, the fifth toe deviates in a medial direction at the MTP joint, whereas the fifth metatarsal head deviates in a lateral direction with respect to the fourth metatarsal.

Anatomy and Biomechanics

Kelikian [285] stated that a bunionette could be considered analogous to the medial eminence of the first metatarsal in hallux valgus. The cause and anatomic variations that are present with a bunionette appear to be much more complex, however, than those originally described by Kelikian [285] and Davies. [280] Several anatomic factors have been attributed to the development or presence of a bunionette deformity (Box 25H-47).

BOX 25H-47

ANATOMIC FACTORS CONTRIBUTING TO BUNIONETTE DEFORMITY

- Enlarged or prominent fifth metatarsal head (width > 13 mm)
- Hypertrophy of the lateral condyle of the fifth metatarsal head
- Increased 4,5 intermetatarsal angle (>8 degrees)
- Fifth metatarsal lateral bowing
- Pes planus

An enlarged fifth metatarsal head can lead to a bunionette deformity (Fig. 25H-90 **A**). [1611] [1616] [1617] [1618] Although hypertrophy of the lateral condyle of the fifth metatarsal head occurs, [1611] [1618] Throckmorton and Bradlee [287] and later Fallat and Buckholz [286] reported that with pronation of the forefoot, the lateral plantar tubercle of the fifth metatarsal head rotates to a more lateral position, creating the radiographic impression of fifth metatarsal head enlargement. These investigators noted an average increase of the 4-5 intermetatarsal angle of 3 degrees with pes planus. Whether there is true hypertrophy of the fifth metatarsal head or a prominence of the fifth metatarsal head owing to pronation of the foot, the lateral condyle of the metatarsal head may become symptomatic without divergence of the fifth metatarsal.

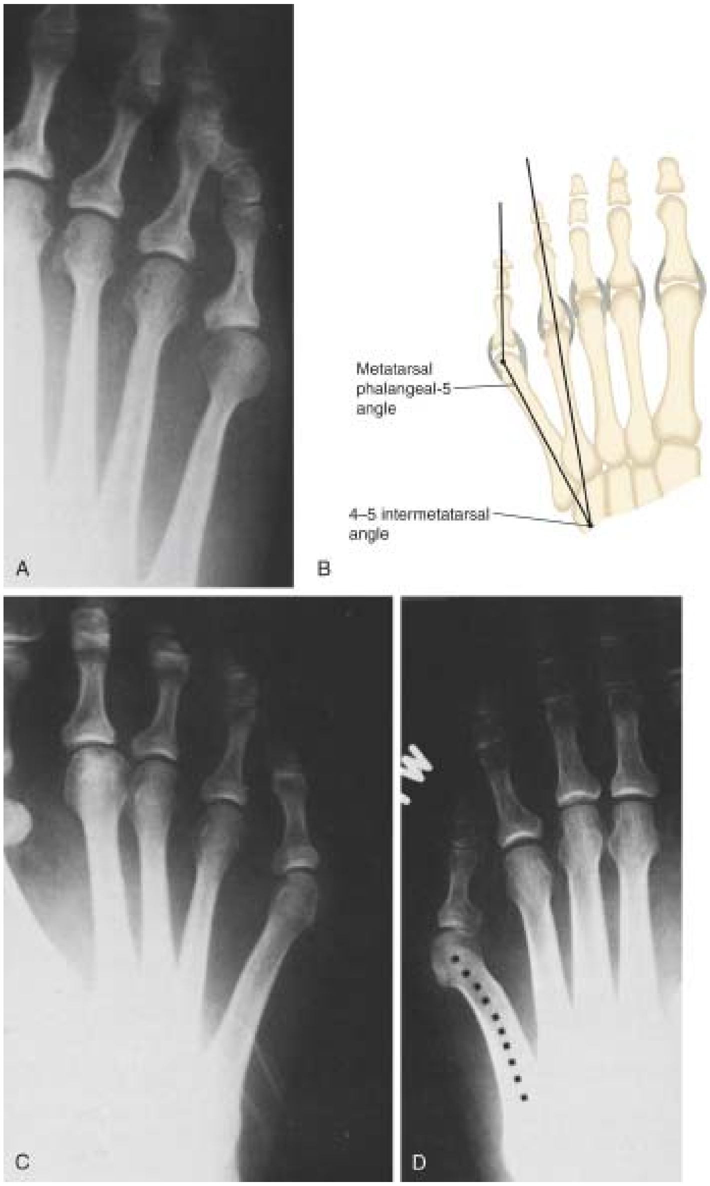

Figure 25H-90 **A,** Enlarged metatarsal head. **B,** The 4-5 intermetatarsal angle and metatarsophalangeal 5 angle. **C,** Increased 4-5 intermetatarsal angle. **D,** Lateral angulation of the distal fifth metatarsal. *(**A, C,** and **D,** © M. J. Coughlin. Used by permission.)*

The angular measurements that define a bunionette deformity are the 4-5 intermetatarsal angle and the MTP-5 angle (see Fig. 25H-90B). The MTP-5 angle is the magnitude of medial deviation of the fifth toe in relation to the fifth metatarsal shaft. The 4-5 intermetatarsal angle calculates the divergence of the fourth and fifth metatarsals and is measured by the intersection of lines bisecting the base and neck of the fourth and fifth metatarsals.[289] A 4-5 intermetatarsal angle of greater than 8 degrees is considered abnormal (see Fig. 25H-90 **C**). [1618] [1619] [1620] [1621]

Lateral bowing of the fifth metatarsal diaphysis results in prominence of the lateral metatarsal condyle (see Fig. 25H-90 **D**). [1611] [1616] [1621] [1622] [1623] [1624] Nestor and colleagues,[293] in reporting on the anatomic variations in patients with symptomatic bunionettes, found that an increased 4-5 intermetatarsal angle frequently was associated with a bunionette deformity, whereas fifth metatarsal bowing and an enlarged fifth metatarsal head were seen much less frequently.

In general, a bunionette deformity is a static deformity that is exacerbated by excess pressure of footwear against a prominent fifth metatarsal head. Anatomic variations and repetitive activity can lead to symptoms in the athletic population. A thickened or inflamed bursa with an associated hyperkeratotic lesion overlying the deformity is seen in athletes participating in repetitive activities such as running.

Classification

Three types of deformity have been described (Box 25H-48). [286] Coughlin [295] further reported the frequency of occurrence of each type of deformity. Coughlin noted a type 1 deformity in 27% of cases, type II in 23% of cases, and type III in 50% of cases.

BOX 25H-48

CLASSIFICATION OF BUNIONETTE DEFORMITIES
Type I: Enlargement of the metatarsal head or lateral exostosis
Type II: Abnormal lateral bend of the fifth metatarsal with a normal 4,5 intermetatarsal angle (23%)
Type III: Increased 4,5 intermetatarsal angle (>8 degrees [50%])

Evaluation

Clinical Presentation and History

The major subjective complaints of an athlete are pain and irritation caused by friction between the underlying bony abnormality and restricting footwear. Patients complain of swelling, pain with shoes, and callus formation over the deformity (Box 25H-49).

BOX 25H-49

TYPICAL FINDINGS IN BUNIONETTE DEFORMITIES

- Pain and irritation over the lateral aspect of the fifth metatarsal head
- Plantar or lateral keratosis formation
- Elevated 4,5 intermetatarsal angle or metatarsophalangeal 5 angle

Physical Examination

On clinical evaluation, the examiner inspects the lateral eminence and lateral border of the foot for an inflamed bursa, [1614] [1617] [1626] a plantar keratosis, [1614] [1627] [1628] a lateral keratosis, [299] or a combined plantar lateral keratosis. [1612] [1629] Diebold and Bejjani [299] noted that two thirds of the patients in their series had significant pes planus. Diebold and Bejjani [299] also noted that one third of the patients had a plantar lesion, and half had a lateral keratotic lesion. Force plate studies or imprints that evaluate the pressure concentration on the plantar aspect of the foot are helpful in the analysis of a plantar keratosis (see Box 25H-49).

A bunionette can develop in combination with a hallux valgus deformity. An increased 1-2 intermetatarsal angle combined with an increased 4-5 intermetatarsal angle results in a wide or splay foot abnormality. [1610] [1614] [1620] [1630] [1631]

Imaging

Radiographic evaluation includes standing anteroposterior and lateral radiographs. The 4-5 intermetatarsal angle and the MTP-5 angle are measured on standing anteroposterior radiographs (see Box 25H-49).

Treatment Options

Nonoperative Treatment

Most bunionette deformities respond well to nonoperative measures. Constricting footwear is often a significant cause of symptoms in the athlete. Pain, swelling, and chronic irritation over the lateral bursa of the fifth metatarsal head are reduced significantly by the use of properly fitted shoes. [1610] [1613] [1615] [1624] [1626] [1632] Padding of the prominent metatarsal head [1613] [1624] and shaving of the hypertrophic callus will relieve symptoms. An orthotic device can control pronation and secondarily can reduce discomfort over the prominent fifth metatarsal head (Box 25H-50).

BOX 25H-50

TREATMENT OPTIONS FOR BUNIONETTE

Nonoperative

- Shoe modifications
- Callus shaving
- Orthotics

Operative

- Lateral condylectomy
- Metatarsal head resection
- Distal metatarsal osteotomy
- Diaphyseal osteotomy
- Proximal fifth metatarsal osteotomy
- Fifth ray resection

Operative Treatment

Surgical treatment of a bunionette is indicated for deformities that continue to cause symptoms despite appropriate nonoperative measures. Numerous operative techniques have been proposed for surgical correction of a symptomatic bunionette deformity (see Box 25H-50).

Lateral condylectomy is considered when an isolated enlargement of the fifth metatarsal head of lateral condyle occurs (Box 25H-51 ; Figs. 25H-91 and 25H-92 [1385] [1390]). Failure of lateral condylectomy as a treatment of the bunionette deformity has led to the development of more extensive resection procedures. Excision of the fifth metatarsal head,[303] resection of the distal half of the metatarsal,[298] and fifth ray resection[304] all have been used to treat a bunionette deformity but are not appropriate in the initial treatment of the symptomatic athlete. These procedures are used as salvage procedures for infection, ulceration, or severe deformity. Less radical operative procedures that preserve foot or toe function are preferred in athletes.

BOX 25H-51

LATERAL CONDYLECTOMY

- Center a longitudinal skin incision over the lateral condyle of the fifth metatarsal.
- Protect the dorsal cutaneous nerve of the fifth toe.
- Create an inverted L-type capsular incision by detaching the dorsal and proximal capsular attachments, allowing exposure of the fifth metatarsal head (see Fig. 25H-91).
- Distract the fifth metatarsophalangeal (MTP) joint and release the medial capsule.
- Resect the lateral eminence with an osteotome or sagittal saw (see Fig. 25H-92 A).
- Close the MTP capsule by suturing it to the dorsal periosteum and to the abductor digiti quinti proximally (see Fig. 25H-92 B).
- If necessary, place a suture through a drill hole in the fifth metatarsal metaphysic dorsal and lateral to ensure a stable capsular closure and prevent recurrence or lateral subluxation of the MTP joint.

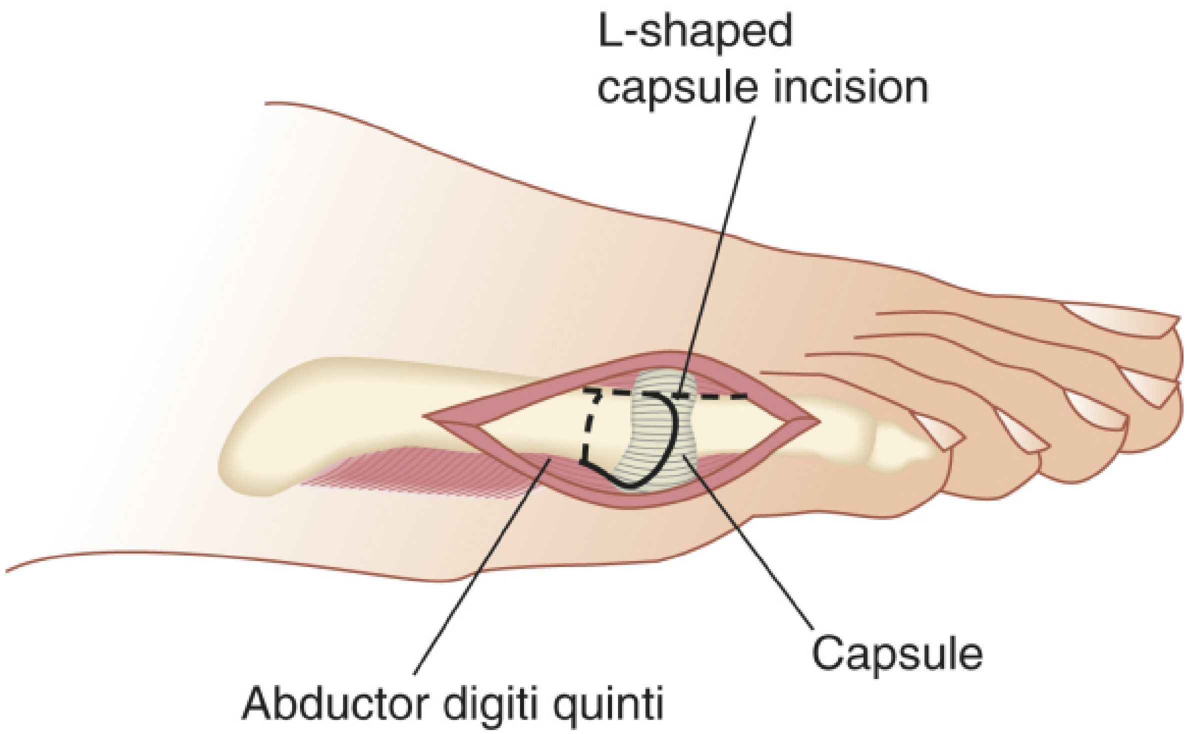

Figure 25H-91 An L-shaped capsular incision is used to expose the fifth metatarsal head.

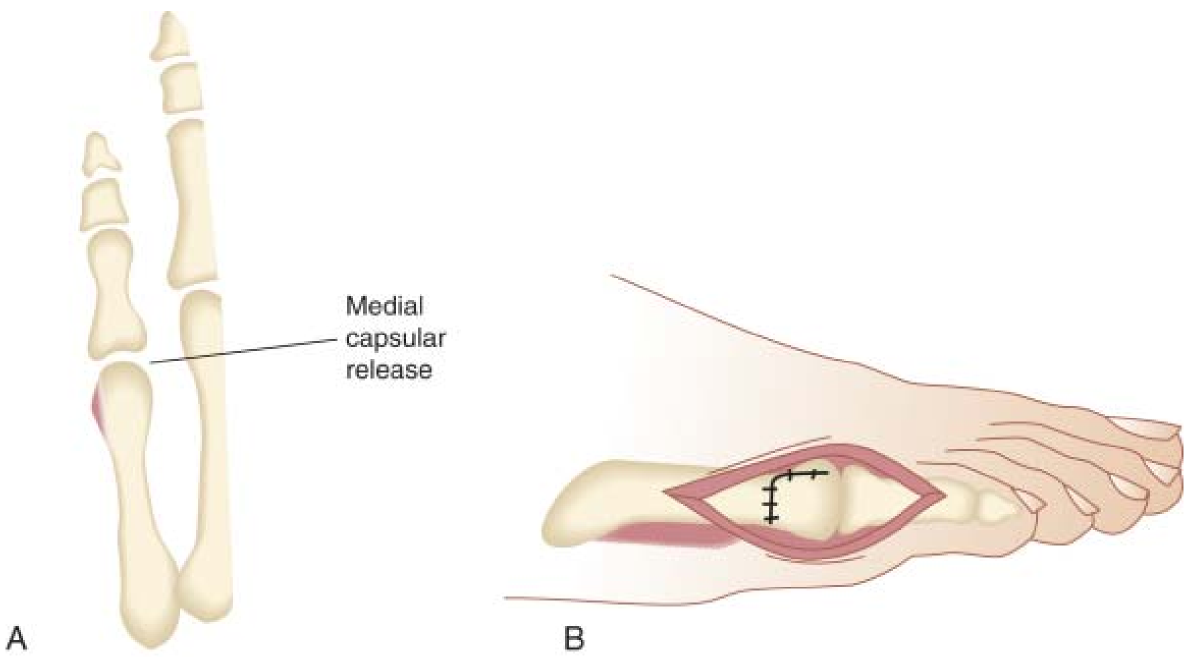

Figure 25H-92 **A,** The lateral eminence is resected, and a medial capsulotomy is performed. **B,** Lateral capsular reefing may be reinforced by a drill hole through the lateral metaphysis of the fifth metatarsal.

Many different distal fifth metatarsal osteotomy techniques have been described for treating the symptomatic bunionette (Box 25H-52). Hohmann[305] originally described a transverse osteotomy of the metatarsal neck, although the lack of stability of this procedure increases the risk for a transfer lesion or a malunion. Kaplan and associates[282] used a distal closing wedge osteotomy internally fixed with 2-mm Kirschner wire. These authors[282] suggested the use of internal fixation because they believed that a distal osteotomy is unstable and can rotate postoperatively with loss of correction.

BOX 25H-52

DISTAL METATARSAL OSTEOTOMY TYPES

- Distal chevron osteotomy
- Distal oblique osteotomy
- Crescentic osteotomy
- Transverse distal osteotomy
- Distal closing wedge osteotomy

Most frequently, a distal oblique, crescentic, or chevron osteotomy is considered. A distal oblique osteotomy [1630] [1631] [1636] is performed from distal lateral to proximal medial (Box 25H-53 ; Figs. 25H-93 to 25H-95 [1395] [1400] [1405]). Haber and Kraft[284] used a distal crescentic osteotomy. These authors did not use internal fixation and reported delayed healing and excessive callus formation at the osteotomy site. An alternative procedure is resection of the prominent lateral condyle in combination with a distal chevron osteotomy. Throckmorton and Bradlee[287] performed a transverse chevron-type osteotomy without fixation, relying on this stable osteotomy shape to hold the postoperative position (Box 25H-54 ; Fig. 25H-96). Boyer and DeOrio[307] describe fixation of the chevron osteotomy with a bioabsorbable pin. Recently, several minimally invasive techniques have been reported in the literature with good results. [1638] [1639]

BOX 25H-53

DISTAL OBLIQUE OSTEOTOMY

- Make a midlateral longitudinal incision over the lateral eminence.
- Release the proximal and dorsal capsule using an L-type capsular incision (see Fig. 25H-91)
- Release the abductor digiti quinti and resect the lateral eminence with an osteotome or sagittal saw (see Fig. 25H-93 A)
- Create the oblique osteotomy of the metaphyseal neck using either a saw or osteotome (see Fig. 25H-93 A). The osteotomy is oriented in a proximal lateral to distal medial direction.
- Displace the distal fragment medially on the metatarsal and impact the bone on the proximal fragment (see Fig. 25H-93 B and C). (The osteotomy can be fixed with a Kirschner wire [see Fig. 25H-94B]).

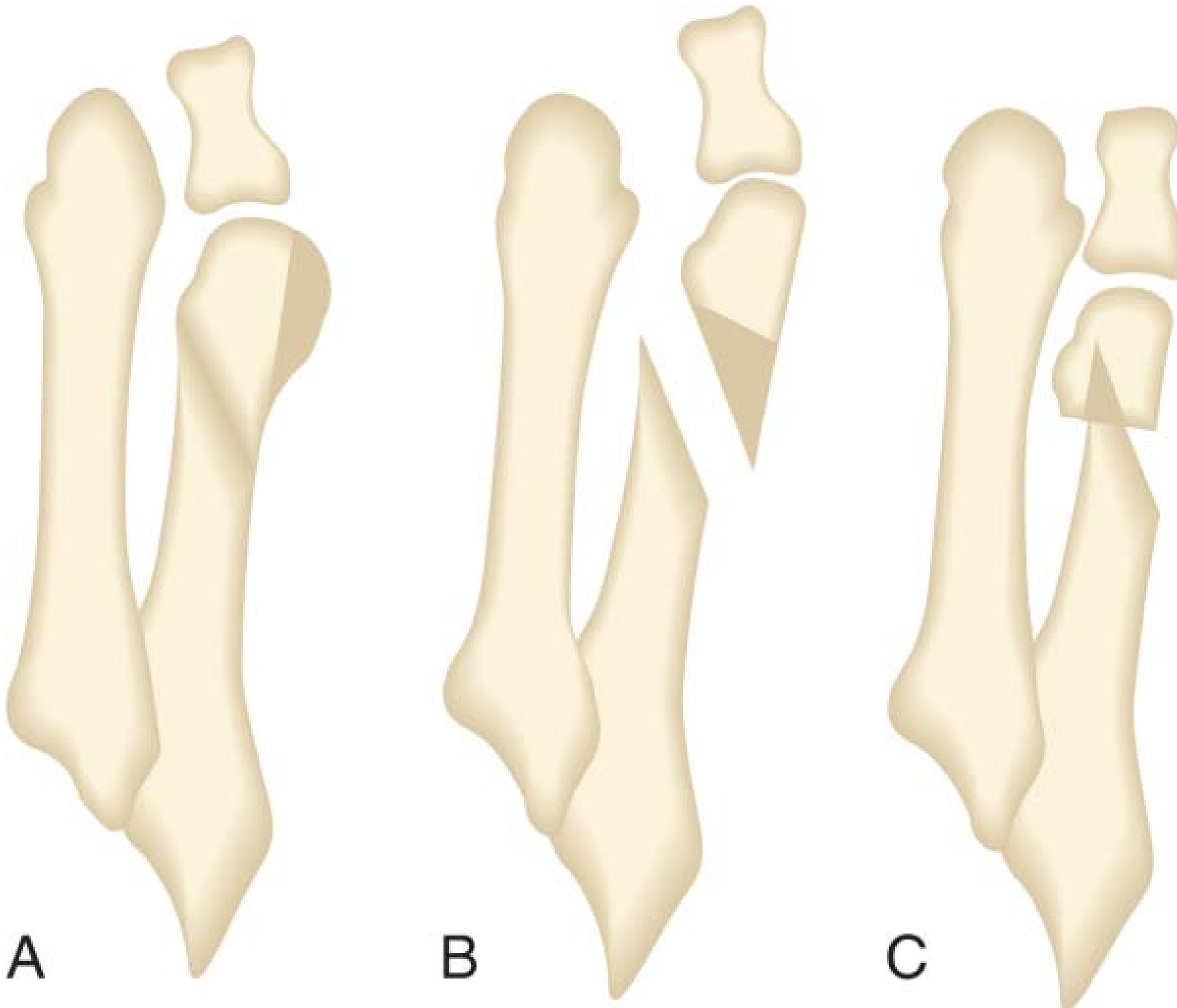

Figure 25H-93 A, Distal oblique osteotomy coupled with lateral eminence resection. **B,** Resection of fifth metatarsal metaphysis with distal oblique osteotomy. **C,** Impaction of osteotomy site.

Figure 25H-94 **A,** Preoperative radiograph shows severe bunionette deformity. **B,** After distal oblique osteotomy with internal fixation. *(Courtesy of H. Zollinger-Kies, Zurich, Switzerland.)*

Figure 25H-95 **A** and **B,** Preoperative and postoperative radiographs of distal oblique osteotomy. *(**A** and **B,** © M. J. Coughlin. Used by permission.)*

BOX 25H-54

DISTAL CHEVRON OSTEOTOMY

- Make a midlateral longitudinal incision over the lateral eminence.
- Release the proximal and dorsal capsule using an L-type capsular incision (see Fig. 25H-91).
- Avoid soft tissue stripping to avoid vascular insult to the distal metatarsal fragment.
- Remove about 2 mm of the lateral eminence with an osteotome or a sagittal saw.
- Mark the apex of the osteotomy with a drill hole in the midportion of the metatarsal.
- Create a horizontal chevron osteotomy with a sagittal saw. The osteotomy is based proximally with an angle of 60 degrees (see Fig. 25H-96 A) and oriented in a lateral-to-medial direction.
- Displace the distal fragment about 2 to 3 mm in a medial direction and impacted onto the proximal phalanx (see Fig. 25H-96 B).
- Use Kirschner wire fixation when necessary.
- Remove any remaining prominent bone in the metaphyseal region of the fifth metatarsal with a sagittal saw.
- Reef the lateral capsule to the abductor digiti quinti or the dorsal periosteum of the fifth metatarsal. If necessary, reattach the capsule through drill holes on the dorsal aspect of the metaphysis.

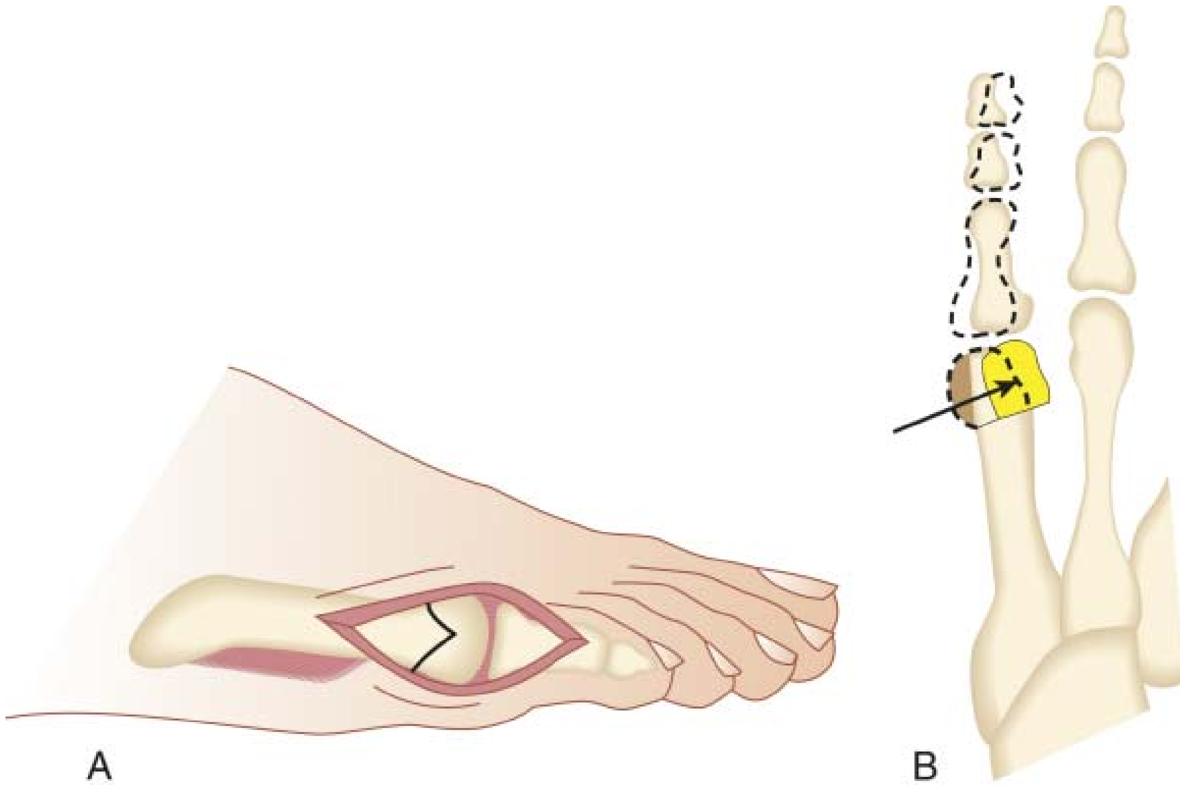

Figure 25H-96 **A,** Lateral view of a fifth metatarsal chevron osteotomy. **B,** Anteroposterior view after chevron osteotomy.

The indications for a diaphyseal fifth metatarsal osteotomy are a bunionette deformity associated with either an increased 4-5 intermetatarsal angle or lateral bowing of the distal metatarsal. MTP joint realignment with a lateral eminence resection is performed simultaneously if necessary (Box 25H-55 ; Fig. 25H-97). A diaphyseal metatarsal osteotomy has been used to correct a bunionette deformity. [1625] [1640] Voutey [288] carried out a transverse osteotomy in the diaphysis but described problems with rotation, angulation, and pseudarthrosis. Yancey [292] used a double transverse closing wedge osteotomy in the diaphyseal region to correct a bunionette deformity characterized by lateral angulation of the fifth metatarsal. Gerbert and colleagues [311] recommended use of a biplane osteotomy for a combined plantar lateral keratotic lesion to displace the distal fragment in a medial direction. Mann [294] and Coughlin [1625] [1640] used an oblique diaphyseal fifth metatarsal osteotomy to treat diffuse keratotic lesions on either the plantar or plantar lateral aspects of the fifth metatarsal. The oblique orientation of the metatarsal osteotomy permits a dorsal medial translation of the metatarsal as the distal fragment is rotated. Internal fixation was recommended with either a small fragment screw or wire loop or a Kirschner wire. Mann [294] did not realign the fifth MTP joint with this procedure, and no results were reported, although one case of nonunion was noted. Coughlin [310] modified Mann's oblique diaphyseal osteotomy by performing a fifth MTP joint realignment and lateral eminence resection (see Box 25H-55). The fifth MTP medial capsular structures are not released because release might impair the circulation to the fifth metatarsal head. If a combination plantar lateral keratosis is present, the oblique osteotomy is oriented in a cephalad direction to create an elevating effect on the distal fragment, then the fragment is rotated (Fig. 25H-98).

BOX 25H-55

OBLIQUE DIAPHYSEAL OSTEOTOMY

- Make a midlateral longitudinal incision is from the base of the fifth metatarsal to the middle of the proximal phalanx (see Fig. 25H-97 A).
- Carry the dissection down to the fifth metatarsal shaft.
- Protect the dorsal cutaneous nerve and retract the abductor digiti quinti in a plantar direction to expose the diaphysis of the fifth metatarsal.
- Create an L-type capsular incision (see Fig. 25H-91) to expose the lateral eminence.

- Remove the lateral eminence with a sagittal saw or osteotome.
- Perform the resection in a line parallel with the metatarsal shaft.
- Make a direct horizontal osteotomy in a dorsal proximal–to–plantar distal plane (see Fig. 25H-97 B).
- Drill the fixation holes before final displacement of the osteotomy site.
- Drill a gliding hole in the dorsal distal fragment and create a tapped fixation hole in the proximal plantar fragment.
- Complete the osteotomy and rotate the distal fragment medially (see Fig. 25H-97 C).
- Fix the osteotomy with either a small fragment compression screw or two minifragment compression screws.
- Repair the fifth MTP joint capsule and bring the fifth toe into proper alignment (see Fig. 25H-97 D and E).
- Approximate the abductor digiti quinti and the MTP capsule. If necessary, reattach the capsule through drill holes on the dorsal aspect of the metaphysis.

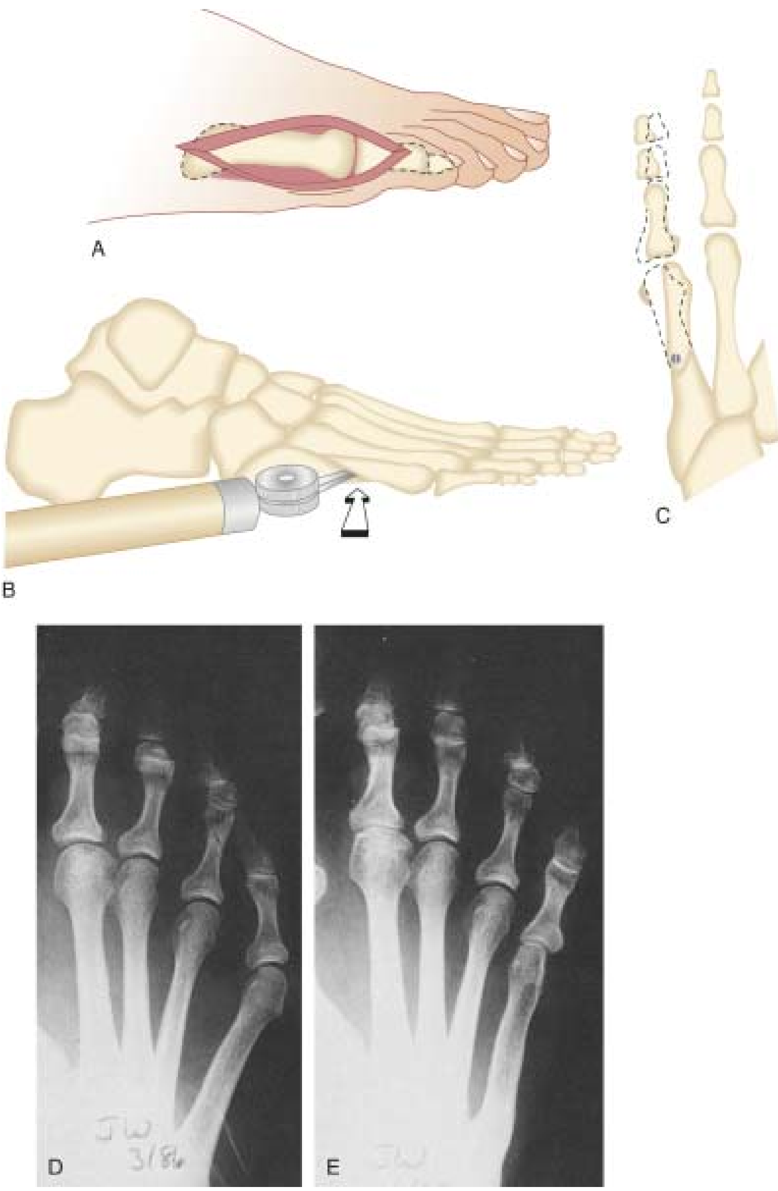

Figure 25H-97 **A,** Incision for a fifth metatarsal diaphyseal osteotomy. **B,** The horizontal osteotomy is performed from a proximal-dorsal to a plantar distal site. **C,** The osteotomy is rotated. **D,** Preoperative radiograph. **E,** Postoperative radiograph after oblique osteotomy. *(**D and E,** © M. J. Coughlin. Used by permission.)*

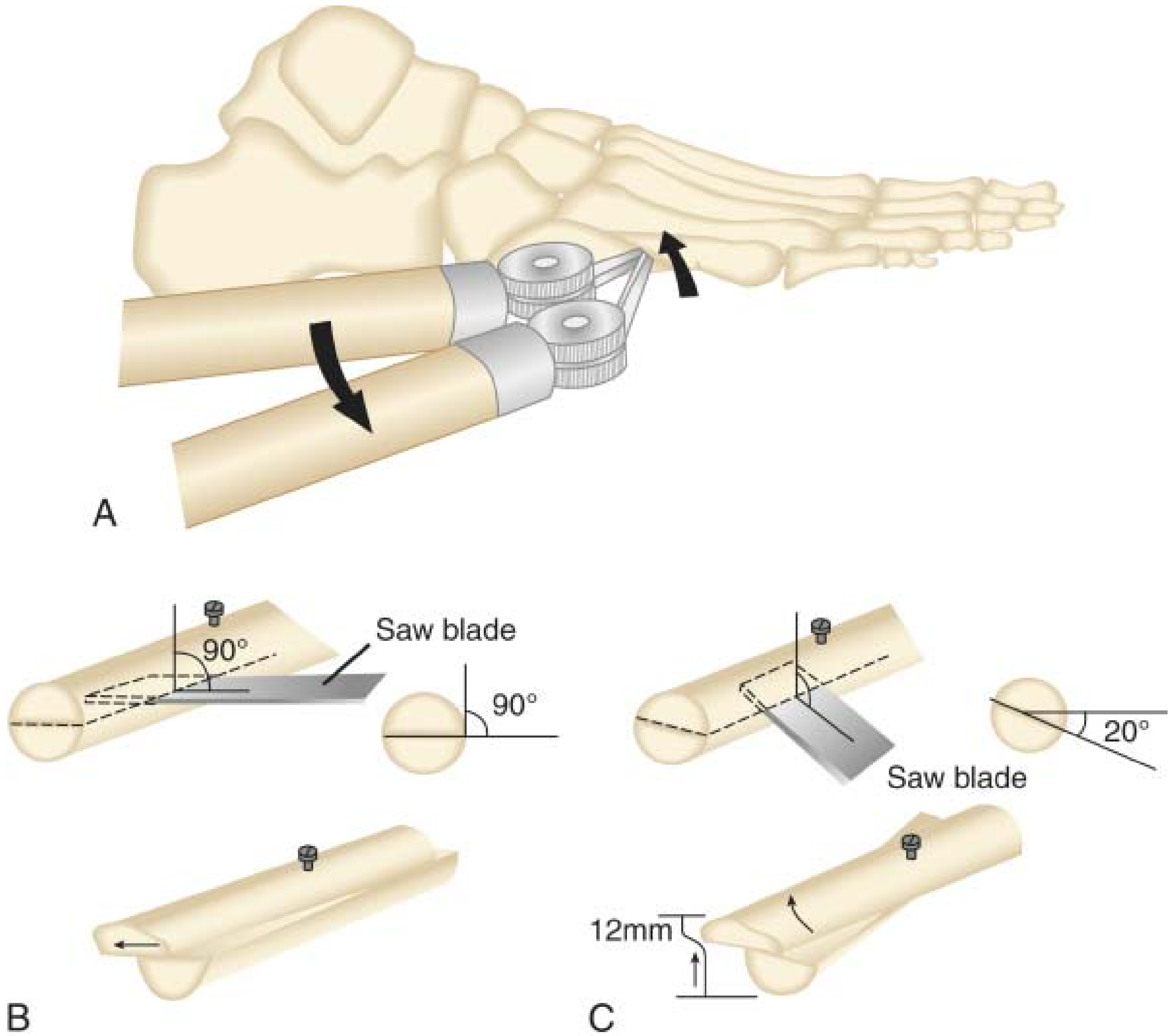

Figure 25H-98 **A,** To achieve elevation of the fifth metatarsal head, the saw is oriented in a cephalad direction, and the osteotomy site is rotated. **B,** Schema illustrating effect of horizontal osteotomy. With the saw blade oriented in a lateral-to-medial direction, the osteotomy site is rotated and does not elevate the distal metatarsal. **C,** Schema illustrating effect of oblique osteotomy. With the saw blade oriented in a medial-to-lateral but also superior direction, as the osteotomy site is rotated, the distal fragment is elevated. *(**B** and **C,** Adapted from Lutter L: Atlas of Adult Foot and Ankle Surgery. St. Louis, Mosby, 1997, pp 110-111.)*

Proximal osteotomies are associated with a higher incidence of nonunion secondary to potential injury to the blood supply to the fifth metatarsal. [1629] [1642]

Weighing the Evidence

Kitaoka and Holiday [313] reported on 21 feet that had undergone a lateral condylar resection. These authors [313] concluded that a minimal correction was achieved with the procedure, although it did relieve symptoms. As Kelikian [285] noted, "at best a lateral condylectomy is a temporizing measure like simple exostectomy on the medial side of the foot; in time, deformity will recur." The only indication for a lateral condylectomy is an enlarged lateral condyle. Symptomatic relief often follows a condylectomy alone; however, a distal metatarsal osteotomy achieves greater correction of the deformity.

Kitaoka and Leventen [314] reported an average of 5 degrees of correction of the 4-5 intermetatarsal angle and a diminished forefoot width of 4 mm with 87% patient satisfaction after distal oblique osteotomy. Sponsel, [301] who advocated an oblique distal osteotomy, noted an 11% delayed union rate, and Keating and coworkers [300] reported 75% of patients to have transfer lesions with a 12% recurrence rate. Pontious and colleagues [315] reported a much higher rate of success in oblique osteotomies that were internally fixed (see Fig. 25H-94).

Throckmorton and Bradlee [287] and others [1613] [1646] [1647] reported high levels of good and excellent results with the chevron osteotomy. Kitaoka and associates [317] reported the 4-5 intermetatarsal angle was reduced an average of 2.6 degrees, and the MTP-5 angle was reduced an average of 8 degrees with a chevron osteotomy. Moran and Claridge [318] stressed that there was a low margin of error with this osteotomy and that there was a high risk for either recurrence or overcorrection. As a result, these authors encouraged the use of Kirschner wire stabilization of the osteotomy site.

Coughlin [295] reported on 30 feet that had undergone a midshaft diaphyseal metatarsal osteotomy. All went on to successful union. The average 4-5 intermetatarsal angle was reduced 10 degrees, and the MTP-5 angle was reduced 16 degrees. No transfer lesions developed, and a 93% patient satisfaction rate was reported. The average foot width was reduced 6 mm. Midshaft osteotomies do not appear to have an increased nonunion rate. Vienne and colleagues [319] reported good or excellent results in 97% of their series of patients with diaphyseal osteotomies for bunionette correction. In this study, the 4-5 intermetatarsal angle was reduced from an average of 10 degrees preoperatively to 1 degree postoperatively. Shereff and associates [312] noted that more proximally positioned osteotomies are at increased risk for delayed healing as a result of interruption of the interosseous and extraosseous blood supply to the proximal fifth metatarsal (Fig. 25H-99).

Figure 25H-99 A, Preoperative radiograph shows moderate bunionette deformity. **B,** After proximal fifth metatarsal osteotomy. **C,** Symptomatic nonunion after proximal osteotomy. This osteotomy took about 12 months to heal and prevented the patient from participating in high school athletics for that season. *(From Mann RA, Coughlin MJ [eds]: Surgery of the Foot and Ankle, 6th ed. St. Louis, Mosby, 1993.)*

Salvage procedures are less desirable in the athletic population. Although McKeever [298] reported a high level of success in his series of 60 cases of metatarsal head resection, no criteria for postoperative evaluation were included. Kitaoka and Holiday [320] reported on a small series and noted that 82% had fair or poor results, including the complications of severe shortening, transfer metatarsalgia, stiffness, and continuing symptoms. Dorris and Mandel [321] reported malalignment of the fifth toe in 59% of patients, and Addante and colleagues [322] reported malalignment and wound problems after metatarsal head resection and silicone implant arthroplasty.

Author's Preferred Method

Conservative management of a symptomatic bunionette includes the use of padding, shaving of keratotic lesions, and roomy footwear. In many cases, an athlete can continue sports activities with the use of orthotic devices or pads. Development of chronic bursal thickening, blistering, and symptomatic keratoses leads to operative treatment in certain patients. Because of the risk for transfer lesions, recurrence of deformity and malunion, delayed union, or nonunion of osteotomy sites, surgical intervention should be delayed until a patient experiences significant difficulty in sports activities.

Surgical versatility in the treatment of a bunionette deformity is important. Attention to the underlying pathology helps to determine whether a condylectomy with a distal soft tissue repair, a distal metatarsal osteotomy, or a diaphyseal biplane osteotomy offers the best treatment for the symptomatic bunionette deformity in the athlete. Analysis of the physical findings including examination of the plantar aspect of the foot for the presence of keratotic lesions helps to differentiate the type of bunionette present and the appropriate treatment. Evaluation of radiographs is necessary to analyze the nature of the deformity.

When an enlarged fifth metatarsal head or medial eminence is present (with or without a pronated foot or fifth ray), lateral condylectomy with MTP joint realignment or distal metatarsal osteotomy is the treatment of choice. The presence of a pure lateral keratotic lesion makes a chevron osteotomy preferable because of the stability of this osteotomy. Kirschner wire fixation often helps to stabilize the osteotomy site. When a plantar lateral keratotic lesion is present (with or without an increased 4-5 intermetatarsal angle or lateral deviation), a distal oblique osteotomy as described by Kitaoka and Leventen [314] or a diaphyseal biplane osteotomy is considered. When there is an abnormally wide 4-5 intermetatarsal angle or when lateral deviation of the distal fifth metatarsal is present, a diaphyseal biplane osteotomy affords an excellent means of correction.

Although there is some disagreement about the need for internal fixation after a fifth metatarsal osteotomy, [315] the development of delayed union, malunion, nonunion, or transfer lesions in patients in whom floating osteotomies have been performed indicates a need for internal fixation.

Postoperative Prescription, Outcomes Measurement, and Potential Complications

Although recovery from bunionette surgery usually is relatively rapid, conservative methods often are used either to alleviate symptoms or to help postpone surgery until the off-season. Postoperatively, the foot is wrapped in a soft gauze and tape dressing, and the patient ambulates in a postoperative shoe. Sutures are removed 2 weeks after surgery. Pins are removed at 6 weeks, and the toe is taped in the proper alignment for 4 more weeks. A below-knee cast can be used if the surgeon is concerned about fixation or the reliability of the patient. Weight-bearing in a postoperative shoe usually is carried out by having the patient bear more weight on the inner aspect of the foot at first. By 3 weeks, a plantigrade stance and gait pattern are encouraged.

Recurrence of deformity is the most common complication after lateral condylectomy (Fig. 25H-100). Occasionally, fifth MTP joint realignment is complicated by joint subluxation or dislocation (Fig. 25H-101). An adequate capsular joint repair helps to avoid this complication. Any dissection in this area can result in injury to the lateral cutaneous nerve to the fifth toe (a branch of the sural nerve), leading to numbness or formation of neuroma.

Figure 25H-100 A, Radiograph shows type 1 bunionette deformity with a large metatarsal head. **B,** After lateral condylectomy. **C,** Recurrence 3 years after lateral condylectomy.

Figure 25H-101 Dislocated metatarsophalangeal joint after simple condylectomy.

Dorsal angulation with development of an IPK or transfer lesion beneath the fourth metatarsal head is a reported complication of distal metatarsal osteotomy. Delayed union or nonunion has been observed as well. The use of internal fixation reduces the incidence of malunion and nonunion after fifth metatarsal osteotomies. An oblique diaphyseal osteotomy can also be complicated by malunion, delayed union, nonunion, or transfer metatarsalgia.

Criteria for Return to Play

Return to athletic activities is expected earlier after a lateral condylectomy or MTP joint realignment than after a fifth metatarsal osteotomy. After a lateral condylectomy, usually aggressive walking can be initiated 4 weeks after surgery, with running after 6 weeks. After a distal osteotomy, aggressive walking is initiated at 8 weeks, and if no complications are encountered, jogging and running can be started progressively between 10 and 12 weeks postoperatively. Diaphyseal osteotomies require slightly longer healing time. In these cases, aggressive walking is initiated between 8 and 10 weeks and progressive running after 12 weeks. If there is clinical concern of incomplete healing of the osteotomy, progression of activity should be delayed. Roomy footwear with an adequate toe box is more comfortable during initiation of athletic activities.

CRITICAL POINTS

- Most conditions of the forefoot are successfully treated nonoperatively in athletes.
- When athletes with forefoot deformity (hallux valgus or lesser toe deformity) have pain associated with shoewear, modification of shoes by stretching constricting areas or relieving pressure areas can relieve the athlete's symptoms completely.
- Choosing appropriate orthotics or shoes for an athlete depends on careful examination and recognition of subtle malalignment that may be contributing to symptoms.
- When choosing operative treatment, the surgical procedure(s) must address all anatomic abnormalities present.
- Athletes should expect up to 3 to 6 months until full return to sport after most forefoot surgeries, especially when deformity correction through osteotomy is required.

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SECTION I Osteochondroses and Related Problems of the Foot and Ankle

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OVERVIEW OF OSTEOCHONDROSIS

The term *osteochondrosis* has been applied to more than 50 eponymic entities to describe a variety of conditions characterized by abnormal endochondral ossification of physeal growth. *Osteochondrosis* is the singular term and is used to describe a noninfectious disease process involving the growth or ossification centers in children that begins as a degeneration or osteonecrosis followed by regeneration or recalcification. Because normal endochondral ossification does not always present a uniform radiographic pattern, the differentiation of osteochondrosis from normal growth often is difficult.

The etiology of osteochondrosis is complex and has been described as traumatic, constitutional, idiopathic, and hereditary. Most authors now believe that multiple factors are responsible for these changes. For example, excessive physical demands during athletic activity may incite osteochondral changes in growing bone made vulnerable by constitutional factors. Once the process has begun, repetitive trauma or pressure may prolong recovery or contribute to deformity. All osteochondroses heal, but treatment may be required to relieve pain or prevent residual deformity, especially in osteochondroses around the foot and ankle in athletes.

The osteochondroses have been classified according to etiology, anatomic location, and type of growth center, but none has had much practical application. In a clinically oriented classification, Siffert [1] divided the osteochondroses into three basic groups: articular, nonarticular, and physeal (Table 25I-1). Mintz and colleagues [2] described a classification based on the magnetic resonance imaging (MRI) appearance of the lesion (Table 25I-2). Osteochondroses of specific locations also have been further classified, and these specialized classification systems are discussed with the specific osteochondroses.

TABLE 25I-1 -- Clinical Classification of Osteochondrosis

Type	Involvement (Example)	Characteristics	Treatment
Articular	Primary: articular and epiphyseal cartilage and subadjacent endochondral ossification (Freiberg disease) Secondary: articular and epiphyseal cartilage as consequence of osteonecrosis of adjacent bone (Legg-Calvé-Perthes disease, Köhler disease)	Degenerative arthritis, pain, limitation of motion	Minimize epiphyseal deformity Encourage joint congruity
Nonarticular	Tendon attachment (Osgood-Schlatter disease) Ligament attachment (vertebral ring, epicondyle) Impact sites (Sever disease, Iselin disease)	Local pain with activity, local tenderness, adolescents, self-limited	Individualized Allow rapid, safe return to activity while minimizing sequelae
Physeal	Long bones (tibia vara) Vertebrae (Scheuermann disease)		

Modified from Siffert RS: Classification of the osteochondroses. Clin Orthop 158:10-18, 1981

TABLE 25I-2 -- Magnetic Resonance Imaging Classification of Osteochondrosis

Grade	Magnetic Resonance Imaging Appearance

Grade	Magnetic Resonance Imaging Appearance
0	Normal cartilage
1	Abnormal signal but intact
2	Fibrillation or fissures not extending to bone
3	Flap present or bone exposed
4	Loose undisplaced fragment
5	Displaced fragment

Data from Mintz DN, Tashjian GS, Connell DA, et al: Osteochondral lesions of the talus: A new magnetic resonance grading system with arthroscopic correlation. Arthroscopy 19:353-359, 2003

According to the Siffert classification, osteochondroses involving the foot and ankle generally are articular or nonarticular. Articular osteochondroses, such as Freiberg and Köhler diseases, may result in degenerative arthritis, pain, and limitation of motion, and treatment should be aimed at minimizing epiphyseal deformity and maximizing joint congruity. Nonarticular osteochondroses, such as Sever disease of the calcaneus and Iselin disease of the base of the fifth metatarsal, cause local pain with activity and local tenderness, usually occur in adolescents, and are self-limited conditions. Treatment of any osteochondritic condition must be individualized to allow the athlete a rapid, safe return to activity and to minimize sequelae of the condition.

OSTEOCHONDROSES OF THE FOOT AND ANKLE

Although a number of osteochondritic conditions in the foot and ankle have been described, most are rare and usually asymptomatic. The most common osteochondroses in the foot and ankle that cause symptoms and require treatment affect the talus, calcaneus, navicular, cuneiforms, metatarsals, and sesamoids (Fig. 25I-1). Because an osteochondrosis may have its onset in childhood or adolescence and not become evident until adulthood, it is difficult to divide these conditions into clear-cut adult and pediatric categories.

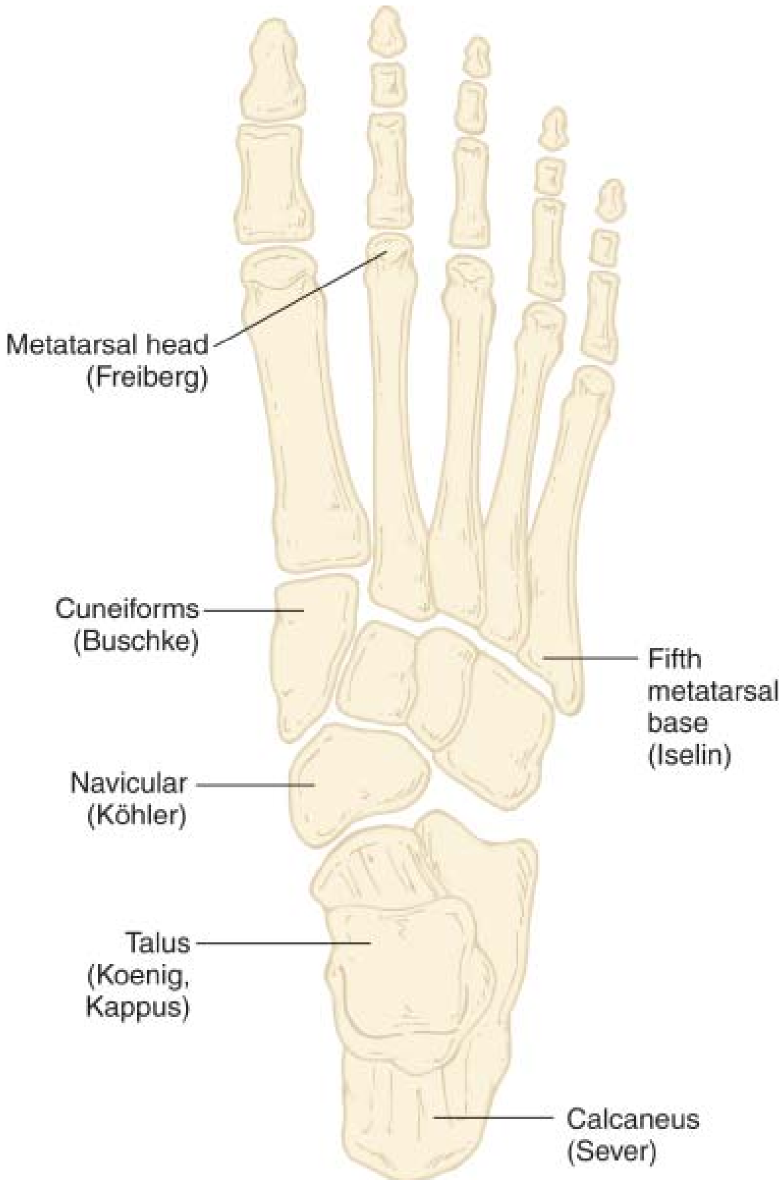


Figure 25I-1 Common sites of osteochondrosis in the foot. The specific form of disease is mentioned in parentheses.

Osteochondral Lesions of the Talus

Konig, [3] in 1888, first used the term *osteochondritis dissecans* to describe loose bodies in the knee joint, theorizing that they were caused by spontaneous necrosis of bone. In 1922, Kappis [4] noted the similarity of lesions of the ankle to those in the knee and referred to osteochondritis dissecans of the ankle. In 1932, Rendu [5] suggested that osteochondritic lesions represented traumatic intra-articular fractures, and the primarily traumatic etiology of osteochondral lesions of the talus (OLT) has been supported by numerous authors. [1659] [1660] [1661] [1662] [1663] [1664] [1665]

Relevant Anatomy and Biomechanics

The talus is a uniquely shaped bone divided into three anatomic regions: dome, neck, and head. The talar dome articulates with the tibia and fibula on its superior, medial, and lateral surfaces to form the ankle joint. Inferiorly, it articulates with the posterior facet of the calcaneus and, along with the inferior surfaces of the head and neck of the talus, forms the subtalar joint. It plays a key role in ankle motion and in supporting the axial load during weight-bearing. Because it lacks muscular or tendinous insertions, indirect perfusion of the talar dome is limited; injury to the artery of the tarsal canal, a branch of the posterior tibial artery, disrupts the main intraosseous blood supply to the central two thirds of the talar dome. In addition, about 60% of the talar dome is covered with hyaline cartilage, further reducing its vascular supply and reparative capacity. [13]

Boyd and Knight [14] showed that the tibiotalar articulation is subjected to more load per unit area than any other joint in the body, and Millington and coworkers [15] demonstrated that more force than previously thought is placed at the talar shoulders where osteochondral lesions typically occur. Several authors [1669] [1670] [1671] [1672] [1673] have hypothesized that even minimal talar displacement can result in medial stress concentration in the tibiotalar joint and lead to cartilage damage.

Berndt and Harty [21] proposed two possible mechanisms for osteochondral fractures of the talus: (1) compressive injury to a dorsiflexed and inverted ankle (direct tibiotalar impact) that crushes the subchondral bone of the lateral talar dome, with or without overlying cartilage damage; and (2) inversion and external rotation forces on a plantar flexed ankle that can produce osteochondral injuries to the medial surface of the talus. O'Farrell and Costello [22] also described a combination of inversion and plantar flexion as a mechanism of injury for medial talar lesions. Other mechanisms have been described, including impaction of the talar dome against the lateral malleolus (causing lateral dome lesions) or against the posterior tibial lip (causing medial defects). [1674] [1676] [1677]

Ankle sprains have been identified as the most common injuries leading to OLT. [1678] [1679] [1680] [1681] In ankle sprains that involve a plantar flexed foot forcibly supinating and injuring the anterior talofibular ligament, the plantar flexed foot and talus subluxate or dislocate, and the posteromedial corner of the talus strikes the tibial plafond, causing a chondral bruise, chondral cracks, shearing off of the corner of the talus with intact chondral attachments, or a complete fracture of the corner of the talus.

Lateral lesions usually are located anterior or central on the talar dome. Also, they are shallower and more wafer shaped than medial lesions, possibly because of a more tangential force vector that results in shearing-type forces. Medial lesions are central and posterior as well as deeper and cup shaped because the combination of inversion, plantar flexion, and external rotation forces causing the posteromedial talar dome to impact the tibial articular surface has a relatively more perpendicular force vector. [1674] [1682]

Classification

The classification of Berndt and Harty, based on radiographic findings, remains a useful system for describing osteochondral lesions of the talus. However, more recent classification systems reflect advances in technology, such as computed tomography (CT), [30] MRI, [31] and ankle arthroscopy, [1685] [1686] (Table 25I-3) and may contribute to a more accurate prognosis.

TABLE 25I-3 -- Classification of Osteochondral Lesions of the Talus

Radiographic Classification [*]	
Stage	**Radiographic Finding**
I	Small area of compression of subchondral bone
IIa	Partially detached osteochondral fragment
IIb	Subchondral cyst on magnetic resonance imaging [38]
III	Completely detached osteochondral fragment remaining in the crater
IV	Displaced osteochondral fragment
V	Radiolucent lesions on computed tomography scan [43]
Computed Tomography Classification [†]	
Stage	**Computed Tomography Appearance**
I	Cystic lesion of talar dome with intact roof
IIa	Cystic lesion with communication to talar dome surface
IIb	Open articular surface lesion with overlying nondisplaced fragment
III	Nondisplaced lesion with lucency
IV	Displaced osteochondral fragment
Magnetic Resonance Imaging and Arthroscopy Classification [‡]	
Cartilage	

Grade	Magnetic Resonance and Arthroscopy Appearance
A	Viable and intact
B	Breached and nonviable
Bone	
Stage	**Magnetic Resonance and Arthroscopy Appearance**
1	Subchondral compression or bone bruise that appears as high signal on T2-weighted images
2	Subchondral cysts not seen acutely, develop from stage 1 lesions
3	Partially separated or detached fragments in situ
4	Displaced fragments
Arthroscopic Classification [§]	
Grade	**Arthroscopic Appearance**
I	Intact, firm, shiny articular cartilage
II	Intact but soft articular cartilage
III	Frayed articular cartilage
Arthroscopic Classification [¶]	
Grade	**Arthroscopic Finding**
A	Articular cartilage is smooth and intact but may be soft or ballottable
B	Articular cartilage has a rough surface
C	Articular cartilage has fibrillations or fissures
D	Articular cartilage with a flap or exposed bone
E	Loose, nondisplaced osteochondral fragment
F	Displaced osteochondral fragment

* Data from Berndt AL, Harty M: Transchondral fracture of the talus. J Bone Joint Surg Am 41:988-1029, 1959.

† Data from Ferkel RD, Sgaglione NA: Arthroscopic treatment of osteochondral lesions of the talus: Long-term results. Orthop Trans 17:1011, 1993.

‡ Data from Taranow WS, Bisignani GA, Towers JD, et al: Retrograde drilling of osteochondral fragments of the talar dome. Foot Ankle Int 20:474-480, 1999

§ Data from Pritsch M, Horoshovski H, Farine I: Arthroscopic treatment of osteochondral lesions of the talus. J Bone Joint Surg Am 68:862-865, 1986.

¶ Data from Cheng MS, Ferkel RD, Applegate GR: Osteochondral lesions of the talus: A radiologic and surgical comparison. In Ferkel RD, Whipple TL, Burst SE (eds): Arthroscopic Surgery: The Foot and Ankle. Philadelphia, Lippincott-Raven, 1996.

Raikin and colleagues [34] divided the talar dome articular surface into nine zones in a grid configuration: zone 1 was the most anterior and medial, zone 3 was anterior and lateral, zone 7 was the most posterior and medial, and zone 9 was the most posterior and lateral (Fig. 25I-2). From MRI examinations of 424 ankles with reported osteochondral talar lesions, they recorded the frequency of involvement and size of lesion for each zone. The medial talar dome was more frequently involved (62%) than the lateral talar dome (34%); in the anteroposterior direction, the midtalar dome was much more frequently involved (80%) than the anterior (6%) or posterior (14) thirds; zone 4 (medial and mid) was most frequently involved (53%), with zone 6 second (26%). Lesions in the medial third of the talar dome were significantly larger and deeper than those in the lateral talar dome.

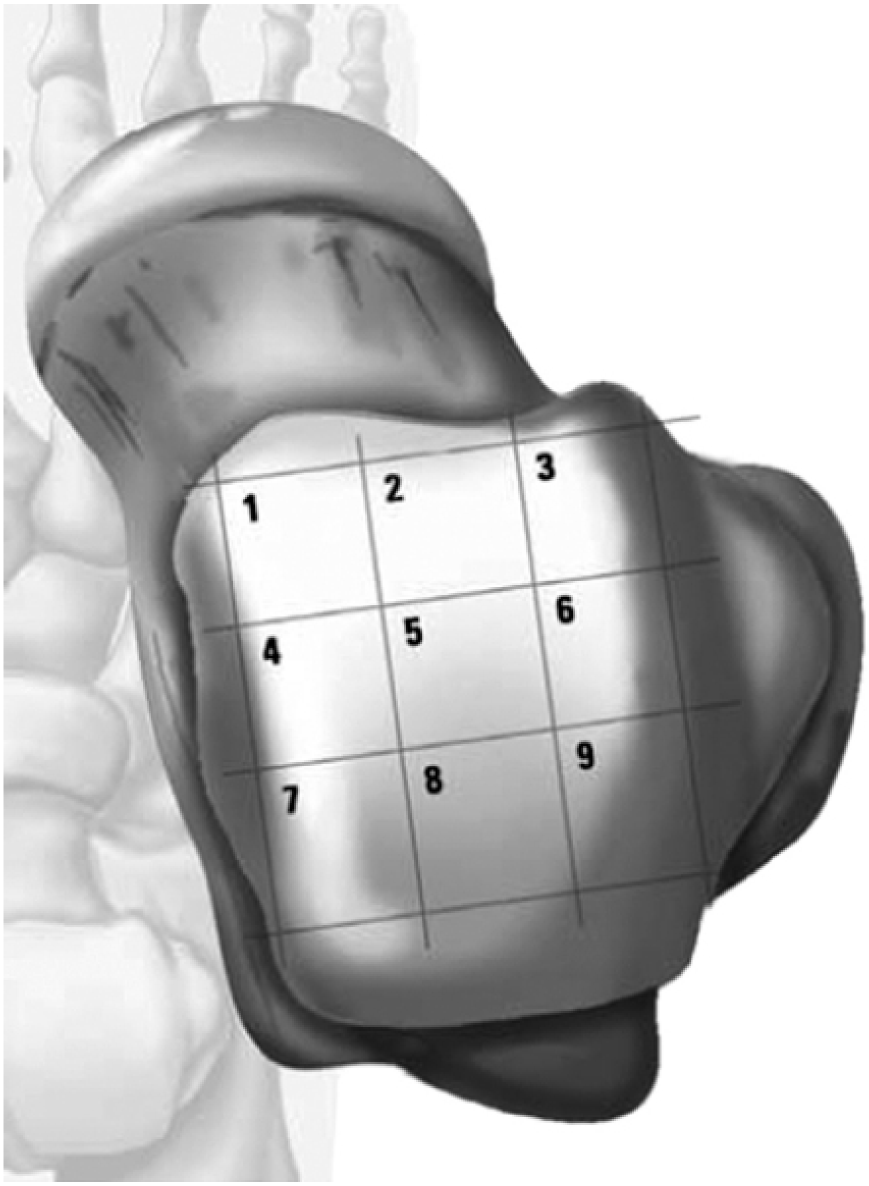


Figure 25I-2 Anatomic nine-zone grid scheme on the talar dome: nine equal surface area zones, with zones 1, 4, and 7 positioned on the medial talus and zones 1, 2, and 3 positioned anteriorly. *(From Raikin SM, Elias I, Zoga AC, et al: Osteochondral lesions of the talus: Localization and morphologic data from 424 patients using a novel anatomical grid scheme. Foot Ankle Int 28:154-161, 2007.)*

Evaluation

Clinical Presentation and History

In patients with acute injuries, the ankle and foot usually are swollen and painful, which can limit the specificity of physical examination. Patients with chronic injuries generally complain of mechanical symptoms, such as locking or giving way, or a feeling of instability of the ankle joint, in addition to pain and persistent swelling. Pain may occur only with certain ankle movements during sport or strenuous activity.

An OLT should be considered in any patient who presents with a history of a "persistent ankle sprain." Eighty percent of patients with traumatic OLT have a history of a seemingly benign ankle sprain. Taga and associates [35] found cartilage lesions in 89% of acutely injured ankles and in 95% of ankles with chronic injuries. They concluded that the longer the time from initial injury, the more severe the associated cartilage lesions. They suggested that these cartilage lesions are the primary cause of persistent pain in patients with chronic ankle instability. Studies have found cartilage damage in up to 66% of ankles with lateral ligament injuries and 98% of ankles with deltoid ligament injuries. [1689] [1690] No correlation has been found between the amount of cartilage damage and the severity of lateral ligament injury.[36] In contrast, Komenda and Ferkel[26] found chondral injuries in only 25% of 55 unstable ankles.

Physical Examination and Testing

The ankle and foot, especially, should be palpated to identify locations of tenderness; the medial and lateral corners of the talar dome should be palpated with the ankle maximally plantar flexed. A careful neurovascular examination is essential. Range of motion in the involved foot and ankle should be compared with that of the contralateral extremity. Stability of the ankle should be evaluated with an anterior drawer test with the ankle plantar flexed and dorsiflexed and with inversion and eversion stress testing. Other soft tissue or bony causes should be ruled out (Box 25I-1).

BOX 25I-1

DIFFERENTIAL DIAGNOSIS OF OSTEOCHONDRAL LESIONS OF THE TALUS

- Syndesmosis injury
- Ankle soft tissue impingement lesions
- Complex regional pain syndrome type I
- Degenerative arthrosis
- Occult talar fractures
- Lateral ankle instability
- Tarsal coalition
- Peroneal tendon subluxation or tendinitis
- Subtalar dysfunction

Imaging

Oblique and plantar flexed radiographic views that avoid tibial overlap generally show the lesion more clearly than plain films. If radiographs are suggestive but not diagnostic of OLT, technetium-99m bone scanning can help identify localized bony pathology. If an OLT is suggested by either radiograph or bone scan, CT or MRI, or both, can provide more definitive information. Axial and sagittal CT cuts can help determine the location of the lesion (anterior, medial, or posterior) as well as its depth and size (Fig. 25I-3). MRI is useful for both preoperative evaluation and postoperative follow-up. Anderson and colleagues[38] demonstrated that low signal intensity in T1-weighted images is an early and definitive sign of even stage I lesions. A high signal rim between the osteochondral fragment and the talar bed is considered indicative of instability of the fragment, with the presence of joint fluid or fibrous granulation tissue as a result of the mobility of these fragments. It has been noted that the diameter of the lesion measured on MRI was significantly larger than indicated on radiographs,[39] an important factor in preoperative planning. We recommend MRI evaluation to detect changes that provide information about detachment and viability of the fragment and help make the decision to preserve or to excise the fragment (Fig. 25I-4). MRI also may allow more appropriate treatment because it delineates the lesion more accurately than either radiography or CT.[40] OLTs that have a high signal rim on T2-weighted images are most likely unstable.[41] In a study of 22 ankles with OLT, Higashiyama and associates[42] found that the low and high signal rims present before surgery disappeared in 100% and 77% of ankles, respectively. A decrease in or disappearance of the signal rim correlated well with clinical results: no patient in whom the signal rim persisted had a good result. It has been suggested that helical CT, MRI, and diagnostic arthroscopy are significantly better than history, physical examination, and standard radiography for detecting or excluding OLT. Diagnostic arthroscopy does not perform better than helical CT and MRI.[40] In general, arthroscopy should not be used as the initial method for diagnosing OLT.

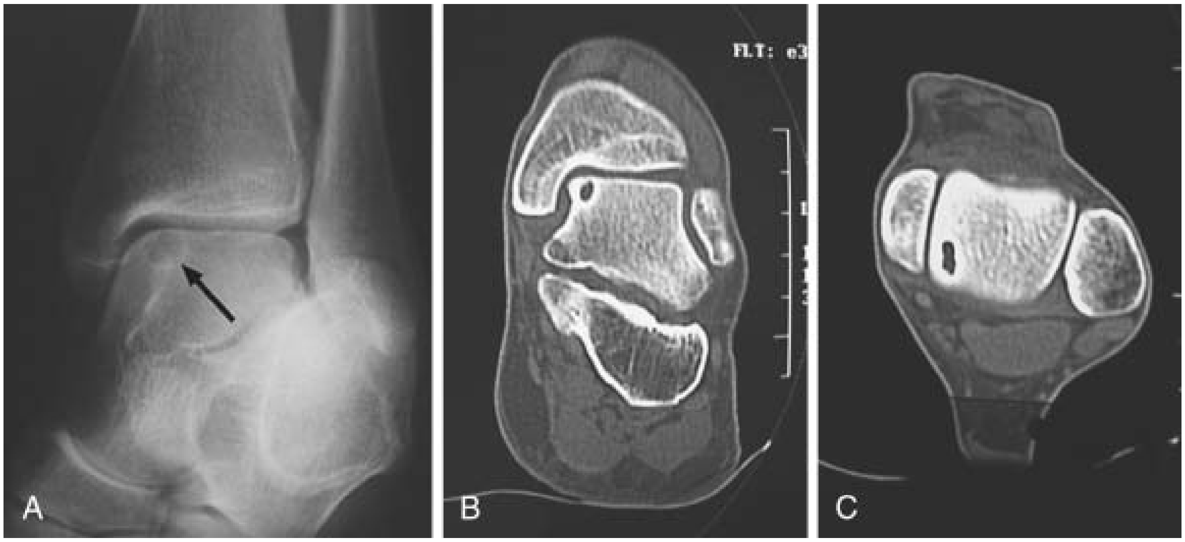


Figure 25I-3 A, Posteromedial osteochondral lesion of the talus (arrow). B, Coronal plane CT image. C, Axial plane CT image. *(From Richardson DR: Ankle injuries. In Canale ST, Beaty JH [eds]): Campbell's Operative Orthopaedics, 11th ed. Philadelphia, Elsevier, 2008.)*

Figure 25I-4 Magnetic resonance imaging appearance of stage IV osteochondral lesion of the talus.

Treatment Options

Nonoperative

Nonoperative treatment may be attempted for CT stage I or II lesions and for stage III lesions in skeletally immature patients. Nonoperative treatment of acute OLT generally involves an initial period of non–weight-bearing with cast immobilization, followed by progressive weight-bearing and mobilization to full ambulation by 12 to 16 weeks. The recommended duration of nonoperative treatment is varied, with some authors recommending 6 months [7] and others [6] up to 12 months before operative treatment is chosen. Based on the results of nonoperative treatment of 35 chronic cystic OLT, Shearer and colleagues [43] concluded that (1) nonoperative management of chronic cystic OLT is a viable option with little or no risk for developing significant osteoarthritis; (2) most lesions remain radiographically stable; (3) there is poor correlation between changes in lesion size and clinical outcome, although the few patients with lesions that decrease significantly in size tend to do well, and those with lesions that increase significantly in size tend to do poorly; (4) the development of mild radiographic changes of osteoarthritis does not correlate with clinical outcome; (5) the general course of chronic cystic OLT is benign, with more than half of patients improving to good or excellent results with nonoperative management; (6) lateral lesions tend to do better than medial ones; and (7) adult-onset lesions tend to do better than juvenile-onset lesions. Alexander and Lichtman [44] suggested that a delay in treatment does not affect outcome; however, more recent studies have questioned this. [1698] [1699] [1700] Lesions presenting more than 1 year after injury or the onset of symptoms may have a poorer prognosis. [45] Also, radiographic results are improved when the interval between injury and operative treatment is reduced. [1698] [1700] This indicates that early diagnosis and treatment are advisable.

Operative

Options for open or arthroscopic treatment of OLT generally are based on one of three specific goals: (1) stimulating the bone marrow by débridement or drilling, with or without loose body removal; (2) securing the lesion to the talar dome so that it will heal in place; or (3) stimulating the development of hyaline cartilage. Techniques include excision, drilling, and curettage, alone or in combination; internal fixation with screws, Kirschner wires, or bioabsorbable devices; cancellous bone grafting; osteochondral autograft or allograft procedures; and autologous chondrocyte implantation or transplantation.

Lavage, Débridement, and Excision

Small, chronic, symptomatic lesions may benefit from arthroscopic lavage and débridement by removing catabolic cytokines and loose bodies from the ankle, which can be the source of mechanical symptoms. However, adding curettage and drilling has been associated with better results. [1665] [1701]

Curettage, Drilling, and Microfracture

Marrow-inducing reparative techniques, such as abrasion, drilling, and microfracture, aim to stimulate chondroprogenitor cells within the underlying marrow. These stem cells populate the fibrin clot in the talar defect and produce a fibrocartilaginous matrix composed of chondroblasts, chondrocytes, fibrocytes, and an unorganized matrix that protects the surface from excessive loading. The disadvantage of these reparative techniques is the weaker mechanical properties of the fibrocartilage matrix, which lacks the normal biomechanical and viscoelastic characteristics of normal tissue. Arthroscopic results appear superior to open procedures. [48]

Internal Fixation

Fixation devices include permanent or bioabsorbable low-profile pins, nails, or headless screws. Acute OLTs do markedly better after fixation than do chronic lesions. Lesions need to be larger than 8 mm to allow secure internal fixation.

Restoration of Articular Hyaline Cartilage

Restorative techniques usually are recommended for defects larger than 2 cm [2]. These techniques can include autologous chondrocyte implantation (ACI), matrix or membrane ACI (MACI), collagen-covered autologous chondrocyte implantation (CACI), arthroscopic allograft or autograft (AAP) with platelet-rich plasma (PRP) implantation, osteochondral autograft, osteochondral autologous transfer system (OATS) and mosaicplasty, fresh osteochondral graft, stem cell–mediated implants, and scaffolds.

Osteochondral autografting procedures such as OATS and mosaicplasty require the harvest of grafts from a donor site, such as the lateral supracondylar ridge or intercondylar notch of the femur, for insertion into the OLT. These techniques generally are used for moderate to large grade III or IV lesions. Concerns about donor site morbidity have prompted graft harvest from sites other than the distal femur, including the anterior talar dome, and the use of allografts.

Fresh-frozen allografts (mega-OATS procedure) have been used for large osteochondral lesions in the knee, but seldom in the talus. Gross and colleagues [49] listed as their indication for this procedure a lesion at least 1 cm in diameter and 5 mm deep that could not be internally fixed. Chondrocyte viability is a primary concern, and it is essential that the graft be harvested within 24 hours of the donor's death and be stored at 4° C for less than 5 days. A benefit of using an ipsilateral talar allograft is the ability to harvest from a similar area as the defect and thus have a closely matched graft.

Autologous Chondrocyte Implantation or Transplantation

ACI involves harvesting 200 to 300 mg of autologous chondrocytes from the distal femur, growing the cells in vitro for 2 to 5 weeks, then implanting them into the defect. An autologous periosteal flap is harvested and sewn over the implanted cells and sealed with fibrin glue. A “sandwich” procedure has been described for lesions with concomitant bone loss. [50] The bony defect is grafted and covered with a periosteal patch with its cambium side facing the cartilage. A second periosteal patch with its cambium side facing the bone is sewn over the first patch to create a space for the cells. ACI is best suited for large and well-contained stage III or IV defects, large lesions with extensive subchondral cystic changes, and lesions in which previous operative treatment has failed. According to Ferkel and Hommen, [51] the ideal patient for ACI is between 15 and 55 years old and has no malalignment, degenerative joint disease, or instability of the joint. The procedure is contraindicated in bipolar (kissing) lesions that involve both the tibia and the talus. [1704] [1705] Because of concerns about donor-site morbidity after harvest from the distal femur, [1706] [1707] other donor sites have been suggested. Giannini and associates [55] used detached osteochondral fragments as a source of cells.

Weighing the Evidence

Most of the literature about OLT consists of case series (level IV evidence) or case reports (level V evidence). For some of the newer techniques, numbers are too small and follow-up too short to make definitive recommendations.

Shearer and colleagues [43] reviewed the results of nonsurgical management of 35 OLTs and concluded that nonsurgical management of chronic cystic (stage 5) OLT is a viable option with little or no risk for development of osteoarthritis. Their clinical results were good or excellent in 54%, fair in 17%, and poor in 29%. A meta-analysis of 14 studies with a total of 201 patients showed only a 45% success rate of nonsurgical treatment of grades I and II and medial grade II OLTs, and nonoperative treatment of chronic lesions had a success rate of 56%. [48] The highest success rate was obtained with excision, curettage, and drilling (85%), followed by excision and curettage (78%) and curettage alone (38%). Shelton and Pedowitz [56] reported just 25% satisfactory results after nonoperative treatment of grade II and III lesions.

Gobbi and coworkers, [57] in a randomized trial comparing chondroplasty, microfracture, and OATS in 33 patients, found no significant differences in clinical results among the three methods. However, each treatment group contained a small number of patients, and three different surgeons were involved in the surgeries.

Individual studies of various treatment methods have reported good results.

Lavage, Débridement, and Excision

In the meta-analysis by Verhagen and coworkers, [48] excision alone had an overall success rate of 38%, with a range of 30% to 100% in individual studies. Excision and curettage had a success rate of 76% (range, 53% to 100%). Arthroscopic procedures had a higher success rate (84%) than did open procedures (63%). Savva and associates [58] described repeat arthroscopic débridement in 12 of 215 patients who had arthroscopic treatment of OLT; at an average 6-year follow-up, results were good in all 12, and 8 had returned to their preinjury levels of sports.

Curettage and Drilling or Microfracture, Bone Grafting

Good to excellent results after drilling have been reported in 28% to 93% of patients. Ferkel and colleagues [51] reported 72% excellent or good results in 64 patients, Taranow and coworkers [59] reported an 81% success rate in 16 patients with retrograde drilling, and Becher and Thermann [60] reported 83% excellent and good results and 17% satisfactory results in 30 patients at an average follow-up of 2 years after microfracture. To determine whether the presence of a subchondral cyst affected the results of arthroscopic microfracture or abrasion arthroplasty, Han and colleagues [61] compared the results in 20 defects with cysts to those in 18 defects without cysts and found no differences in the clinical results. They concluded that small cystic lesions can be successfully treated by arthroscopic microfracture or abrasion arthroplasty.

Autogenous Cancellous Bone Grafting

Saxena and Eakin [62] compared the results of microfracture procedures in 26 patients to those after bone grafting in 20 patients. Overall, 96% of patients had excellent or good results, and there was no difference between the groups in the percentages of those who returned to sports. Bone grafting, however, required a longer time to return to activity than did microfracture in high-demand patients, but the two groups had similar postoperative American Orthopaedic Foot and Ankle Society (AOFAS) scores. Regardless of treatment type, patients with anterolateral lesions had the fastest returns to activity and the highest AOFAS scores. Draper and Fallat [63] compared the results of 14 patients treated with bone grafting with those of 17 patients treated with curettage and drilling. At almost 5-year follow-up, those with bone grafting had better range of motion and less pain. Kolker and colleagues, [64] however, reported that 6 of 13 patients required further surgery after open antegrade autologous bone grafting and concluded that autologous bone grafting alone should not be used as primary treatment for patients with symptomatic advanced OLT and deficient or absent overlying cartilage.

Osteochondral Autografts (Osteochondral Autologous Transfer System, Mosaicplasty)

Scranton and associates [65] reported 90% good to excellent results in 50 patients with type V OLT at an average 3-year follow-up after osteochondral autograft transplantation using a single, arthroscopically harvested graft from the distal femur. Thirty-two of their 50 patients (64%) had at least one previous operation that failed to relieve symptoms. Hangody and collegues [66] described good to excellent results in 34 of 36 patients 2 to 7 years after mosaicplasty. Kreuz and coworkers [67] used mosaicplasty procedures for the treatment of 35 OLTs after failure of arthroscopic excision, curettage, and drilling. The osteochondral graft was harvested from the ipsilateral talar facet, and a malleolar or tibial wedge osteotomy was used to access central or posterior lesions. Although there were no nonunions of the osteotomies, patients with small osteochondral lesions accessible through an anterior approach without additional osteotomy had the best results.

Osteochondral Allografts

Although several studies have reported good results with this technique in the knee, there are few reports of its use in the ankle. Gross and associates [49] reported that six of nine allografts remained in situ with a mean survival rate of 11 years; three patients required arthrodesis because of graft resorption and fragmentation. Kim and colleagues [68] used tibiotalar osteochondral shell autografts in seven patients; at 10-year average follow-up, only four had excellent or good results. Complications included graft fragmentation, poor graft fit, graft subluxation, and nonunion.

Autogenous Chondrocyte Implantation or Transplantation

Koulalis and colleagues [69] reported excellent to good results at 17 months' follow-up in all 8 of their patients treated with ACI, and Whittaker and associates [70] described ACI in 10 patients, 9 of whom were "pleased" or "extremely pleased" with their results at 4-year follow-up; however, 1 year after surgery, Lysholm knee scores had returned to preoperative levels in only 3 patients, suggesting donor-site morbidity in the other 7. Baums and coworkers [52] reported 12 patients with ACI of the talus for defects that averaged 2.3 cm[2]. At about 5 years' follow-up, 7 had excellent results, 4 had good results, and 1 had a satisfactory result. The AOFAS mean score improved from 43.5 before surgery to 85.5 after surgery. Patients who had been involved in competitive sports were able to return to their full activity levels.

AUTHOR'S PREFERRED METHOD

For a stage I or II OLT, non–weight-bearing in a cast or boot is first tried for 6 to 10 weeks, depending on the size of the lesion. If this fails to relieve symptoms, arthroscopic excision, curettage, and microfracture or drilling is done. In a skeletally immature patient with a stage III OLT, a trial of conservative treatment is warranted before surgical treatment. For stage III or IV OLT in skeletally mature patients, arthroscopic microfracture or drilling is the first choice and has obtained good results in about 90% of our patients (Fig. 25I-5). The use of a noninvasive ankle distractor (Fig. 25I-6) will help with visualization of posterior lesions. If this option fails to relieve symptoms, an osteochondral autograft (lesion < 1.5 cm[2]) (Table 25I-4) or allograft (lesion >1.5 cm[2]) is used.

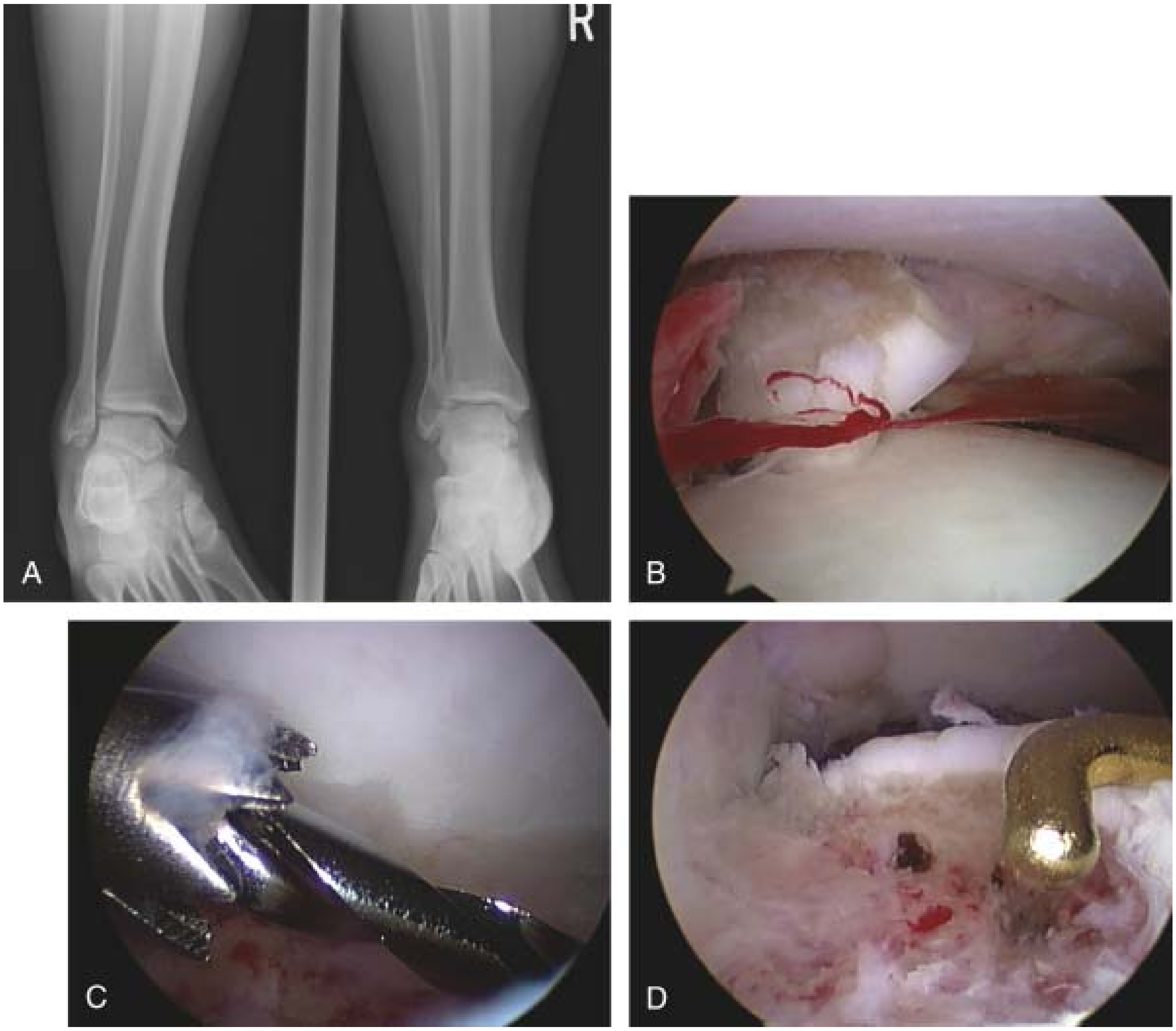


Figure 25I-5 **A,** Stage IV osteochondral lesion of the talus. **B,** Arthroscopic view of displaced osteochondral fragment. **C,** Arthroscopic excision and drilling. **D,** Note vascular channels created in defect.

Figure 25I-6 Noninvasive ankle distraction for ankle arthroscopy. *(From Richardson DR: Ankle injuries. In Canale ST, Beaty JH [eds]: Campbell's Operative Orthopaedics, 11th ed. Philadelphia, Elsevier, 2008.)*

TABLE 25I-4 -- Authors' Preferred Treatment of OLT

MRI Stage	Conservative Treatment	Primary Operative Treatment	After Failed Primary Procedure
I or II	Cast or boot: non–weight-bearing for 6-10 weeks depending on size; ankle brace for 3 months	Arthroscopic excision, curettage, microfracture/drilling	Damaged surface < 1.5 cm[2], osteochondral autograft transport; > 1.5 cm[2], osteochondral allograft
III	Cast or boot: Acute injury (< 10 weeks) skeletally immature: non-weight-bearing for 6-10 weeksSkeletally mature or chronic injury: proceed to operative treatment	Minimally damaged surface: arthroscopic transtalar drilling Damaged surface: microfracture/drilling	As above

MRI Stage	Conservative Treatment	Primary Operative Treatment	After Failed Primary Procedure
IV	No role for conservative treatment	Minimally damaged surface: If < 1 cm^2, arthroscopic transtalar drilling; If > 1 cm^2, internal fixation Damaged surface: microfracture or drilling	As above
V	Cast or boot: weight bearing as tolerated for 6-10 weeks; ankle brace for 6 months	Minimally damaged surface: arthroscopic transtalar drilling + bone graft Damaged surface: microfracture or drilling	Cyst < 1.5 cm: as above + bone graft Cyst > 1.5 cm: bulk allograft

Technique of Arthroscopic Drilling, Excision, or Pinning of OLT

- View anterolateral lesions through an anteromedial portal, with instrumentation for drilling, excision, or pinning inserted through an anterolateral portal, changing portals as necessary for optimal viewing and fixation.
- Posteromedial lesions can be more difficult to view and treat. With noninvasive distraction and use of a small, 2.7-mm scope in the anterolateral portal, most posteromedial lesions can be treated through anteromedial and posterolateral portals.
- Use a small, curved curet or curved microfracture awl to make perforations in the subchondral bone.
- If needed, make a small bony trough on the anteromedial tibia to improve access to posterior lesions.
- If the lesion still is not accessible, use a guide to place a Kirschner wire through the medial malleolus for drilling of the lesion (Fig. 25I-7).
- A malleolar osteotomy may be required for pinning of larger lesions.
- Other helpful instruments are an open-end curet, a small 2.7-mm full-radius resector, and a small 2.7-mm bur.

Technique of Osteochondral Autograft or Allograft Transplantation

- With the patient under general anesthesia, prepare the affected lower extremity from the ankle to the knee. Examine the ankle arthroscopically to further delineate the chondral lesion.
- Harvesters are made for lesions 5 to 11 mm (larger sizes also are available).
- Approach lateral lesions through an anterior sagittal incision and perform a medial malleolar osteotomy for medial lesions. Rarely, a lateral malleolar osteotomy will be needed to access posterolateral lesions.
- Use a commercially available recipient sizer and harvester to create a recipient hole for the donor osteochondral plug. Extract the plug to a depth of 10 mm (Fig. 25I-8 **A** and **B**). Place the harvester perpendicular for dome lesions (see Fig. 25I-8 **C**) and at 45 degrees for talar shoulder lesions.
- Drill multiple holes into the subchondral bone of the recipient hole (see Fig. 25I-8 **D**).
- Obtain a graft from the ipsilateral knee, arthroscopically from the medial femoral condyle, or from the lateral femoral condyle through a small incision (see Fig. 25I-8 **E** and **F**). For talar shoulder lesions, obtain a graft from the lateral trochlea.
- Use the specially designed donor harvester to obtain osteochondral grafts that measure 5 to 11 mm in diameter and 10 to 12 mm in depth (slightly deeper than the recipient hole).
- Insert the cylindrical grafts carefully into the recipient hole using the designed extruder or collared pin through the donor harvester (see Fig. 25I-8 **G** and **H**).

- Do not remove the OATS harvester before completion of full graft extrusion. Do not allow the harvester to deviate from the insertion angle. Either of these may cause fracture of the donor core.
- Use the sizer-tamp to gently tamp the core flush with the surrounding cartilage.
- Test range of motion of the ankle to ensure that the graft is well seated and secured.
- Close the incision and secure the osteotomy in the usual fashion (see Fig. 25I-8 **I**). Place one drain in the knee and apply a compressive dressing to the ankle. Apply a posterior splint with strips.

For very large lesions, allografts can be harvested from an ipsilateral donor talus (see Fig. 25I-8 J).

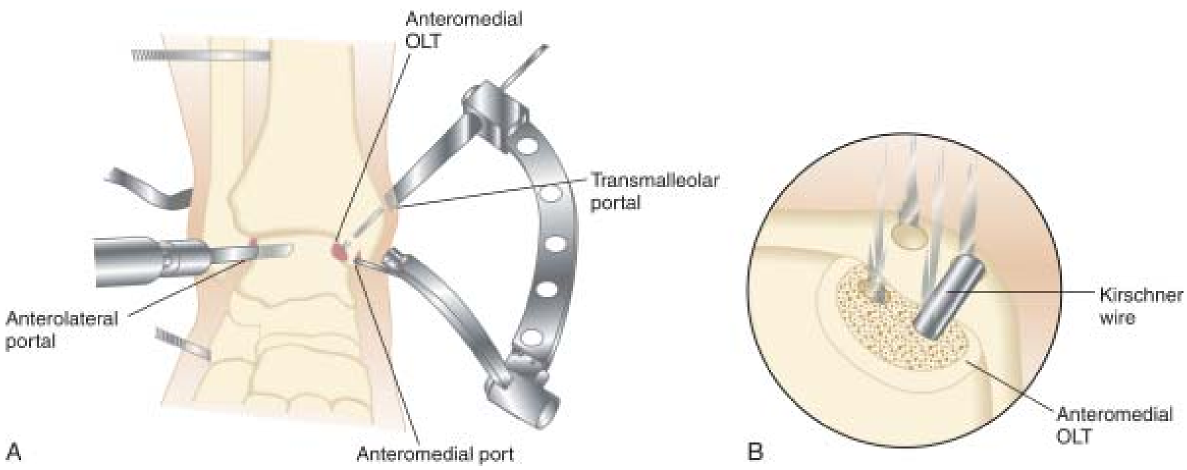
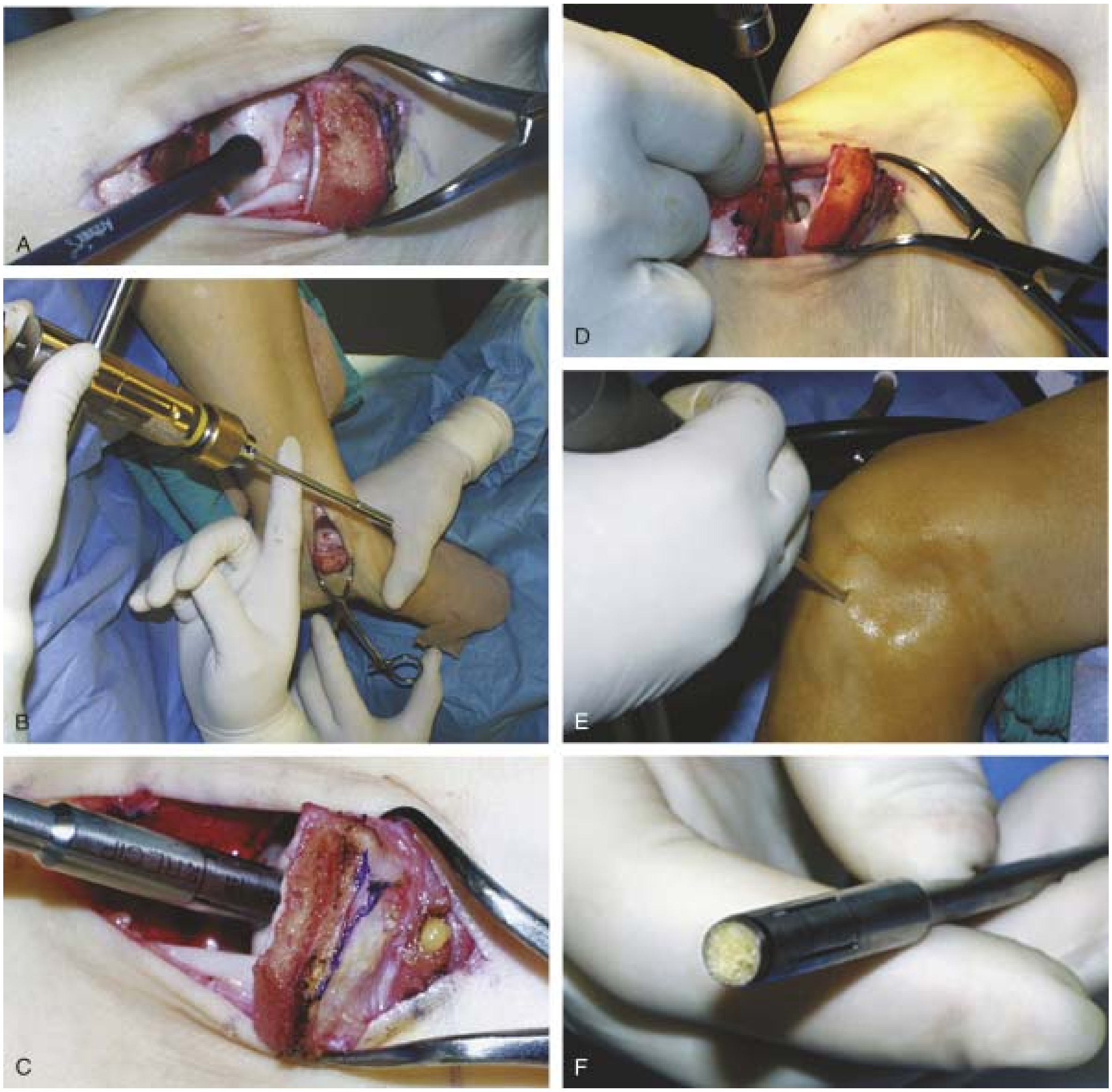


Figure 25I-7 A, Transmalleolar drilling of osteochondral lesion using a guide. The scope is in the anterolateral portal, and inflow is through the posterolateral portal. **B,** Holes are drilled through the medial malleolus into the talus down to areas of bleeding bone. *(From Ferkel RD: Arthroscopy of ankle and foot. In Mann RA, Coughlin MJ [eds]: Surgery of the Foot and Ankle, 8th ed. Philadelphia, Elsevier, 2006.)*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B
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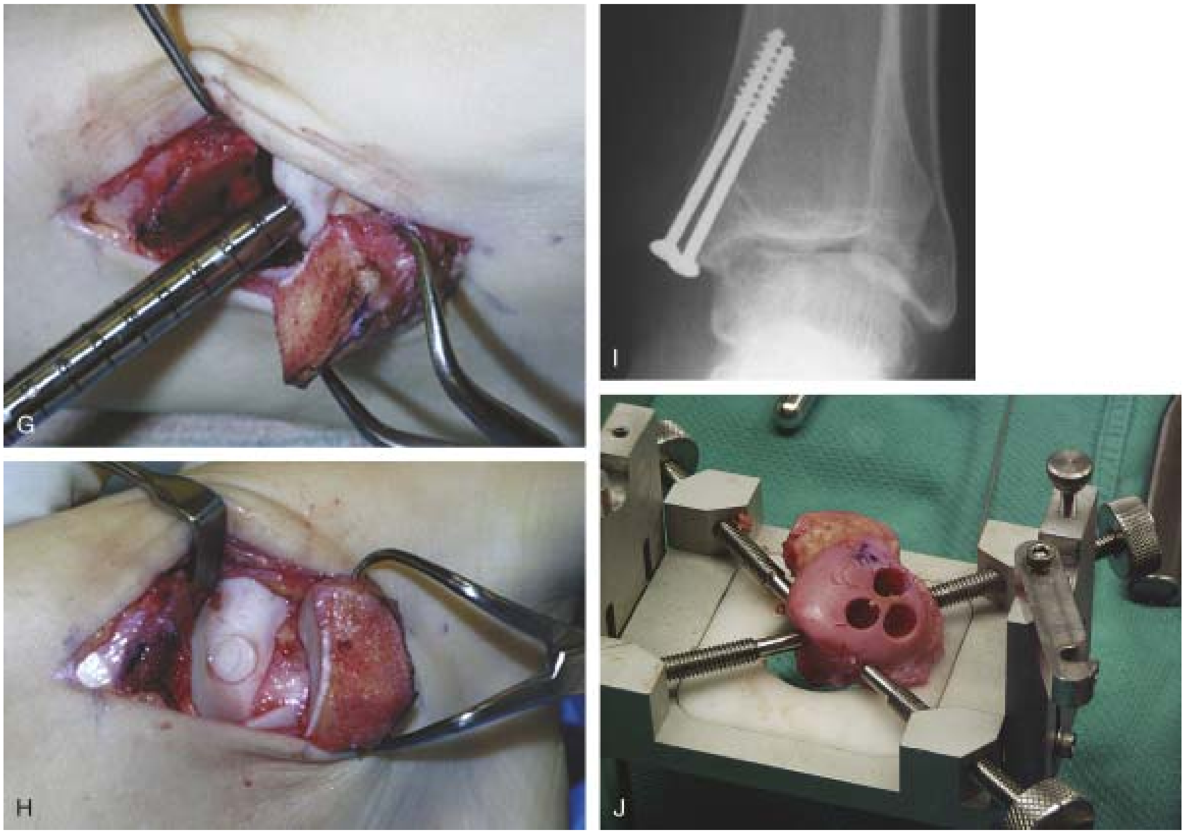

Figure 25I-8 Osteochondral autograft and allograft transplantation. **A,** Trial sizer for harvester. **B,** Recipient harvester. **C,** Plug 10 to 12 mm deep is removed from recipient site. **D,** Multiple holes are drilled at the base of the lesion. **E,** Autograft is obtained from the femoral condyle with a donor harvester (for talar shoulder lesions, graft is obtained from corner of trochlea). **F,** Donor graft in harvester. **G** and **H,** Graft is placed in recipient hole. **I,** Malleolar osteotomy is secured with two partially threaded cancellous screws (holes are predrilled before osteotomy). **J,** For large defects, allografts can be taken from donor talus. *(From Richardson DR: Ankle injuries. In Canale ST, Beaty JH [eds]: Campbell's Operative Orthopaedics, 11th ed. Philadelphia, Elsevier, 2008; courtesy of Dr. Robert Anderson, Charlotte, NC.)*

Postoperative Prescription, Outcomes Measurement, and Potential Complications

Postoperative Prescription

After arthroscopic excision, curettage, and drilling, the patient is non–weight-bearing in a boot for 4 weeks, then progresses to weight-bearing in the boot in physical therapy. Active motion is begun at 12 days after surgery. After internal fixation or OATS, the patient is non–weight-bearing in a cast for 8 weeks, then progresses to weight-bearing in a boot for 4 weeks in physical therapy. A brace is then worn during a gradual return to activities as symptoms dictate.

Outcomes Measures

Clinical outcomes measures include pain relief, ankle stability, and ankle motion.

Potential Complications

The most common complication is continued pain. Repeat MRI evaluation or second-look arthroscopy (Fig. 25I-9) is reasonable if pain persists after 4 months. We have had several patients with continued pain after osteochondral grafting of an OLT; all had complete or significant relief of symptoms after arthroscopic débridement of the graft. Other potential complications are wound infection and neural injury, most often to the superficial peroneal nerve.

Figure 25I-9 Second-look arthroscopy 15 months after osteochondral autologous transfer system (OATS) procedure shows good incorporation of the osseous portion of the graft, but incomplete incorporation of the cartilage. Repeat arthroscopy was done because of impingement symptoms, which resolved after surgery.

Criteria for Return to Play

- Minimal or no symptoms, minimal swelling
- Participating in physical therapy out of boot
- After internal fixation, OATS, or cartilage replacement, a brace must be worn while participating in sports for 6 months after the procedure.

Special Populations

Skeletally Immature Patients

The literature concerning the treatment of OLT in skeletally immature patients is scarce, and treatment recommendations usually are based on the Berndt and Harty classification: nonoperative treatment for stages I and II and medial stage III lesions and operative treatment for lateral stage III and stage IV lesions. Higuera and associates [71] reported excellent or good results in 18 of 19 patients, and Kumai and colleagues [45] reported good results in 10 of 11 with nonoperative treatment. In the series of Letts and associates, [72] 13 of 24 children initially treated nonoperatively required operative treatment. More recently, Perumal and associates [73] concluded that few OLTs in skeletally immature patients heal with 6 months of nonoperative treatment. In their 31 patients (mean age 12 years), after 6 months of nonoperative treatment only 5 (16%) had complete clinical and radiographic healing, 24 (77%) had persistent lesions on radiographs, and 2 had severe pain. Of the 13 who subsequently had operative treatment, 11 healed clinically and radiographically within 12 months; the other 2 had persistent lesions on radiographs but no clinical symptoms. In their compilation of the literature reporting operative treatment of OLT in 48 children, Letts and colleagues [72] found excellent or good results in 34 (71%), fair results in 12 (25%), and poor results in 2 (4%). In the only comparison of outcomes of surgery for OLT in adults and adolescents, Bruns and Rosenbach [74] found that adolescents had better long-term outcomes than adults, regardless of the severity of the lesion.

High-Level Athletes

Although a number of treatment methods have been shown to be successful in treating OLT in young, active patients, the ability to return to high-level athletic activity has not been well-documented. Saxena and Eakin, [62] in a series of 44 athletic patients with 46 OLTs, treated 26 with microfracture and 20 with autogenous bone grafting. Results were excellent or good in 44 (96%) of the 46 lesions, and the average time to return to sports activity was 17 weeks. The return to activity was significantly longer in the bone graft group (20 weeks) than in the microfracture group (15 weeks); return to sports was faster after arthroscopic treatment (16 weeks) than after arthrotomy (17.5 weeks), but there was no difference in postoperative AOFAS scores. Patients with anterolateral lesions had the fastest return to sports and the highest AOFAS scores.

Osteochondral Lesions of the Distal Tibia

Osteochondral lesions of the distal tibia are much less common than those of the talus, and there is little information in the literature about their etiology, natural history, or treatment. It appears that they, like talar lesions, are primarily caused by trauma. “Mirror image” or “kissing” lesions of the talus and distal tibia have been described. [75] In one of the largest series of osteochondral lesions of the distal tibia, [76] 11 of 17 patients recalled an inversion injury to the ankle. Symptoms may include pain, stiffness, swelling, locking, and instability. Radiographs usually are not helpful, but MRI and CT can identify the lesion (Fig. 25I-10). Treatment is similar to that of osteochondral lesions of the talus: débridement and curettage of the lesion, abrasion of the defect to subchondral bone, and drilling or microfracture of the subchondral bone. Mologne and Ferkel [76] reported excellent or good results in 14 of 17 patients an average of 44 months after débridement, curettage, abrasion, and drilling or microfracture. If this is unsuccessful, a retrograde osteochondral autograft or allograft transfer procedure can be done [1730] [1731] ; instrumentation has been developed to make this easier.

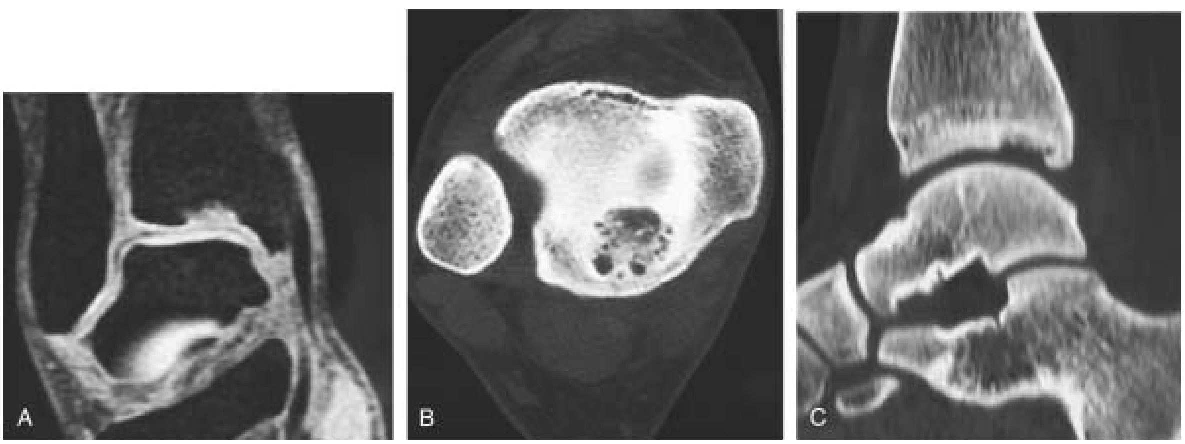

Figure 25I-10 Osteochondral lesion of the distal tibia in a female collegiate basketball player. **A,** Coronal fat-suppressed magnetic resonance imaging. **B,** Axial computed tomography (CT) shows posterior-central lesion and small subchondral cysts. **C,** Sagittal CT shows depth of the defect. *(From Mologne TS, Ferkel RD: Arthroscopic treatment of osteochondral lesions of the distal tibia. Foot Ankle Int 28:865-872, 2007.)*

OTHER LESIONS THAT CAN MIMIC ANKLE SPRAINS

Injuries of the ligaments around the ankle joint are common in athletic individuals, accounting for the highest percentage of injuries in epidemiologic studies of sports injuries, [1732] [1733] [1734] [1735] [1736] regardless of the sport, level of participation, or age or sex of the participants. Usually, these injuries are diagnosed promptly and treated appropriately; however, continued ankle pain or instability should raise suspicion of some other entity. Conditions that can be misdiagnosed as ankle sprains include fractures, neoplasms, impingement syndromes, and coalitions of the tarsal bones. Delayed or incorrect diagnosis can result in prolonged disability.

Fractures of the Talus and Calcaneus

Fractures of the talar and calcaneal processes can be mistaken for ankle sprains (Table 25I-5). Misdiagnosis or delayed diagnosis of these injuries can lead to nonunion, which can cause ankle pain that limits athletic activity. Missed lateral talar process fractures were found in about 1% of 1500 patients initially diagnosed with lateral ankle sprains [84] ;

in a series of 25 anterior calcaneal process fractures, 7 (28%) were initially diagnosed as anterior talofibular ligament sprains.[85] In 20 patients with avulsion fractures of the posterior talus that were all initially diagnosed as ankle sprains, the average number of physician visits before correct diagnosis was about 6, and 1 patient was seen 17 times.[86] Clark and coworkers[87] reviewed ankle radiographs of 1153 patients with acute ankle trauma and determined that an ankle effusion of 13 mm or more had a positive predictive value of 82% for occult fracture.

TABLE 25I-5 -- Talar and Calcaneal Process Fractures That Can Mimic Ankle Sprains

Fracture	Mechanism	Physical examination	Radiograph
Lateral talar process	Inversion + dorsiflexion	Point tenderness anterior and inferior to lateral malleolus (over lateral process); pain with plantar flexion, dorsiflexion, subtalar joint motion	Mortise view Lateral view
Posterior talar process (lateral tubercle)	Hyper–plantar flexion (compression fracture) or inversion (avulsion fracture)	Tenderness to deep palpation anterior to Achilles tendon, over posterolateral talus; plantar flexion may produce pain; swelling in posterolateral ankle	Lateral view
Posterior talar process (medial tubercle)	Dorsiflexion, pronation	Tenderness to deep palpation between medial malleolus and Achilles tendon; swelling posterior to medial malleolus and anterior to Achilles tendon	Oblique view (foot in 40 degrees of external rotation)
Anterior calcaneal process	Inversion + plantar flexion (avulsion fracture); dorsiflexion (compression fracture)	Point tenderness over calcaneocuboid joint (about 1 cm inferior and 3 to 4 cm anterior to lateral malleolus)	Lateral view Lateral oblique view

Relevant Anatomy and Biomechanics

The lateral talar process is an osseous protuberance that articulates superolaterally with the fibula and helps to stabilize the ankle mortise. Inferomedially, it articulates with the calcaneus to form the lateral portion of the subtalar joint. The posterior process of the talus is made up of the lateral and medial tubercles. The lateral tubercle is the larger of the two and serves as the attachment of the posterior talocalcaneal and posterior talofibular ligaments. The posterior third of the deltoid ligament attaches on the medial tubercle. The undersurface of both tubercles forms the posterior fourth of the subtalar joint. An accessory bone, the os trigonum, often is present posterior to the lateral tubercle and can be confused with a fracture of the lateral tubercle. Fractures of the lateral tubercle of the posterior process can be caused by hyper–plantar flexion or inversion, whereas those of the medial tubercle usually are caused by dorsiflexion and pronation injuries because the medial tubercle is avulsed by the deltoid ligament.

Evaluation and Classification

Most fractures of the talar and calcaneal processes result in pain, tenderness, or swelling in specific locations (see Table 25I-5) that help distinguish them from ankle sprains and from each other.

Patients with fractures of the anterior calcaneal process usually report a sudden twist of the ankle with immediate pain on the outer aspect of the midportion of the foot and discomfort with weight-bearing. Pain and tenderness are located in the region of the sinus tarsi, with maximal tenderness 2 cm anterior and 1 cm inferior to the anterior talofibular ligament, which helps distinguish this lesion from a lateral ankle sprain. Avulsion fractures of the anterior calcaneal process are best seen on a lateral oblique projection, whereas compression fractures are best seen on a lateral view of the hindfoot. Anterior calcaneal process fractures often are associated with other ankle pathology, including tarsal coalitions, ankle sprains, and bifurcate ligament abnormalities. In a review of 1479 foot and ankle MRI studies, Petrover and associates[88] found 15 fractures of the anterior process (1%), only 2 of which had no associated abnormality. Fractures of the anterior calcaneal process generally are classified according to displacement and involvement of the calcaneocuboid joint (Table 25I-6).[85]

TABLE 25I-6 -- Classification of Calcaneal Anterior and Lateral Talar Process Fractures

Classification of Calcaneal Anterior Process Fractures	
Type I	Nondisplaced tip avulsion

Type II	Displaced avulsion fracture not involving the calcaneocuboid articulation
Type III	Displaced, larger fragments involving the calcaneocuboid joint
Classification of Lateral Talar Process Fractures	
Type A	Small, minimally displaced, extra-articular avulsion
Type B	Medium-sized fracture involving only the talocalcaneal articular surface
Type C	Larger fracture involving both talocalcaneal and talofibular articulations

Fractures of the posterior process of the talus most often involve the lateral tubercle (Fig. 25I-11). Lateral tubercle factures can be caused by hyper–plantar flexion (compression) or inversion (avulsion) and have been associated with football and rugby kicking, which places the ankle in a forced plantar flexed position. Pain may be exacerbated by plantar flexion or, because of the proximity of the flexor hallucis longus tendon, by dorsiflexion of the hallux. In almost 50% of ankles, the os trigonum (fused or separate) is just posterior to the lateral tubercle of the posterior talar process and may be mistaken for a fracture. Differentiation of a fracture of the lateral tubercle from a nonunited secondary ossification center is best made on a lateral radiograph. An acute fracture is suggested by a rough, irregular cortical surface along the line of separation, whereas a normal os trigonum has a smooth and rounded cortical surface. These fractures also can be classified according to size, displacement, and joint involvement (see Table 25I-6).

Figure 25I-11 Lateral radiograph shows fracture of the lateral tubercle of the posterior talar process (*arrow*). *(From Judd DB, Kim DH: Foot fractures frequently misdiagnosed as ankle sprains. Am Fam Physician 66:785-794, 2002.)*

Fractures of the medial tubercle generally are caused by dorsiflexion-pronation injuries that cause avulsion of the medial tubercle by the deltoid ligament. They usually result in a tender, firm mass posterior to the medial malleolus, with no ankle instability or limitation of motion. These fractures are difficult to see on routine radiographs, and CT or MRI may be necessary to confirm the diagnosis. Ebraheim and colleagues, [89] in a cadaver study, determined that the 30-degree external rotation view of the ankle is most likely to show this injury. Fractures of the entire posterior process (both tubercles) have been described but are rare.

Fractures of the lateral talar process, the "snowboarder's fracture," are relatively infrequent but should be suspected in patients who complain of lateral ankle pain after an inversion or dorsiflexion injury. From 33% to 41% of these injuries are missed on initial examination. [1737] [1743] [1744] [1745] Point tenderness over the lateral talar process should prompt CT evaluation. Von Knoch and associates [93] found that 14 of 16 displaced or unstable lateral process fractures were associated with severe concomitant hindfoot injuries. A morphologic classification of these fractures has been described [94] : type I, chip fracture; type II, large fragment; type III, comminuted.

Treatment

Nonoperative treatment generally is sufficient for acute process and tubercle fractures with small (<1 cm), minimally displaced (<2 mm) fragments. [95] Nonoperative treatment also may be appropriate for larger fragments that are nondisplaced or minimally displaced. [96] Usually 6 weeks of immobilization in a non–weight-bearing cast is followed by transition to a removable walking boot and progressive weight-bearing with crutches. Operative treatment should be strongly considered for large (>1 cm), displaced (>2 mm) fragments with significant articular involvement. [1743] [1744] Valderrabano and colleagues [97] developed a treatment algorithm based on the fracture type (McCrory-Bladin [94] classification) (Fig. 25I-12). Operative treatment may consist of open reduction and internal fixation of large fragments, primary excision of severely comminuted fractures, or delayed excision of chronic nonunions.

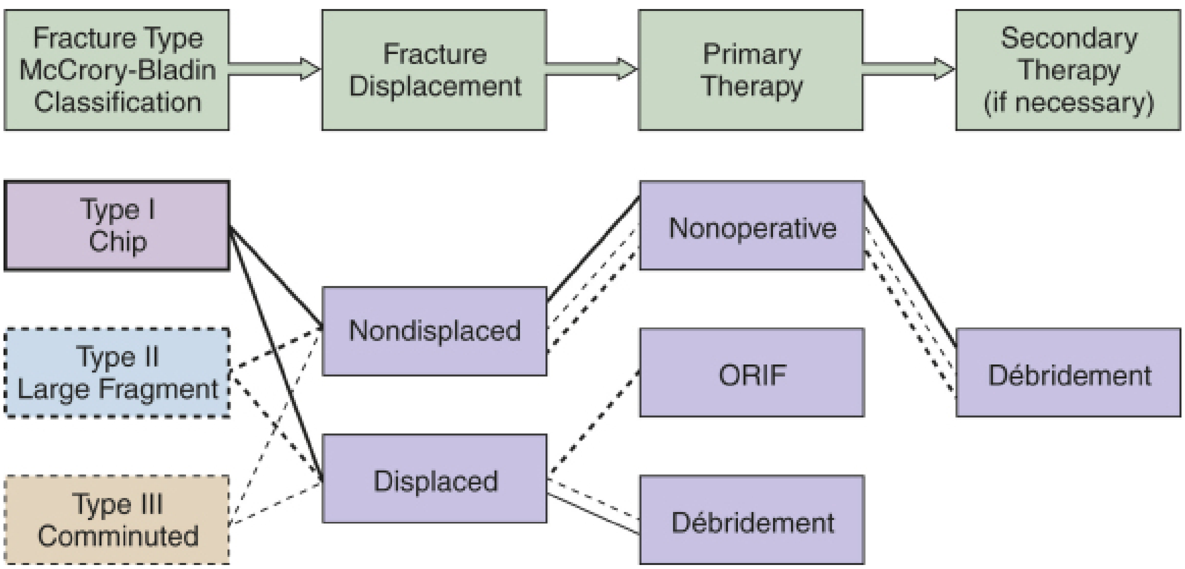

Figure 25I-12 Treatment algorithm for fractures of the lateral talar process. *(Redrawn from Valderrabano V, Perren T, Ryf C, et al: Snowboarder's talus fracture: Treatment outcome of 20 cases after 3.5 years. Am J Sports Med 33:871-880, 2005.)*

Weighing the Evidence

Because of the relatively infrequent occurrence of these process and tubercle fractures, there is little evidence in the literature on which to base treatment recommendations. Most studies include only a small number of patients or are isolated case reports. In one of the larger series of anterior calcaneal process fractures (25 fractures), all 18 treated nonoperatively had good results, whereas 5 of 7 treated with excision had good results. [85] The worst outcomes were in patients with the longest delays in diagnosis and treatment.

Medial talar process fractures generally are reported to do well with nonoperative treatment, if diagnosed and treated promptly. [1751] [1752] Although Giuffrida and colleagues [100] reported that five of six medial process fractures required arthrodesis after nonoperative treatment, the fractures in their series were all associated with high-energy medial subtalar dislocations.

Lateral talar process fractures appear to do less well with nonoperative treatment. In a series of 23 lateral talar process fractures in snowboarders,[93] 7 minimally displaced fractures were treated nonoperatively, and 16 displaced or unstable fractures were treated with open reduction and internal fixation. Fifteen of the patients (10 treated operatively and 5 treated nonoperatively) regained their preinjury levels of athletic activity. The 6 operatively treated patients who failed to regain preinjury participation levels all had severe associated injuries of the ankle or hindfoot. At 3.5-year follow-up of 20 lateral talar process fractures,[97] the AOFAS scores were higher in the 14 treated with open reduction and internal fixation (ORIF) (97 points) than in the 6 treated nonoperatively (85 points); all 14 patients treated with ORIF regained their preinjury levels of sport, whereas only 2 of the 6 treated nonoperatively did so.

Author's Preferred Method

Most fractures of the calcaneus and talus that mimic ankle sprains heal with cast immobilization, and this is our preferred treatment unless displacement or comminution is severe or the fracture fragment is large. ORIF is indicated for noncomminuted, displaced fractures because large, displaced, articular fragments, if unreduced, have a high risk for nonunion. For displaced intra-articular process and tubercle fractures, especially those of the lateral talar process, that are too comminuted for internal fixation, primary excision allows early mobilization without the risk for painful nonunion.

Postoperative Prescription, Outcomes Measurements, and Potential Complications

A short leg walking cast is applied over a compression dressing after ORIF and worn for 3 weeks, after which the dressing is removed, and a weight-bearing cast or brace is worn for another 3 weeks. After primary excision, immobilization usually consists of 2 to 3 weeks in a weight-bearing cast or removable boot. Full weight-bearing is allowed when radiographic healing of the fracture is evident. Physical therapy is instituted for muscular strengthening, proprioception, balance, and sport-specific functions.

The primary clinical outcome measures are pain relief, joint motion, and return to activity. The AOFAS hindfoot score can be used for functional evaluation. Radiographs or CT scans evaluate healing.

The most frequent complications after process and tubercle fractures of the hindfoot are chronic pain and the development of arthritis. Symptomatic nonunion is likely if these fractures are not diagnosed and treated promptly. Late excision of the un-united fragment can improve symptoms, [1749] [1754] [1755] [1756] but results appear to deteriorate the longer the interval between nonunion and excision. [1738] [1754]

Criteria for Return to Play

Return to play depends on both the fracture and the athlete. Motivated athletes generally can return to sports when fracture healing is documented and normal strength and motion have been regained. A range of motion equal to the uninjured ankle and 85% of contralateral strength should be obtained before returning to sport.

Impingement Syndromes

The term *ankle impingement* encompasses a wide variety of soft tissue and bony conditions around the ankle that can cause chronic pain and decreased motion and limit athletic activity. Various forms of mechanical impingement can be caused by synovial proliferation, bone spur formation, or ligamentous scarring and hypertrophy. Impingement generally is described as anterolateral, anterior, or posterior. Combined anterior and posterior impingement also has been described.[104]

Evaluation

Anterior impingement probably is the most common of the impingement syndromes and occurs most often in ballet dancers and football, basketball, and soccer players. The first symptom is pain that begins as a vague discomfort and becomes sharper and more localized to the front of the ankle and usually is exacerbated by cutting or pivoting maneuvers. Physical examination finds tenderness between the anterior tibial tendon and the medial malleolus, which is exacerbated with dorsiflexion and relieved with plantar flexion. With the ankle plantar flexed, exostoses can be palpated on the superior surface of the talar neck. Imaging studies show a beak-like prominence at the anterior rim of the tibial plafond, usually associated with a corresponding area of the opposed margin of the talus proximal to the talar neck, within the anterior ankle joint capsule (Fig. 25I-13). These osteophytes can impinge on each other, and soft tissues can become entrapped between them. Anterior impingement has been classified into four grades to indicate severity (Fig.

25I-14).[105] Two distinctive impingement lesions in the anterior talus have been described: a localized “divot” that accepts the growing tibial spur during dorsiflexion, prevents the formation of an osteophyte on the anterior talar neck, and allows unimpeded dorsiflexion[106]; and a “tram track” lesion, formed when a prominent osteophyte carves a longitudinal trough in the articular surface of the talar dome.[107]

Figure 25I-13 Anterior impingement syndrome. **A,** Magnetic resonance image shows osteophyte on distal tibia. **B,** Radiograph after excision of osteophyte. *(From Richardson DR: Ankle injuries. In Canale ST, Beaty JH [eds]: Campbell’s Operative Orthopaedics, 11th ed. Philadelphia, Elsevier, 2008.)*

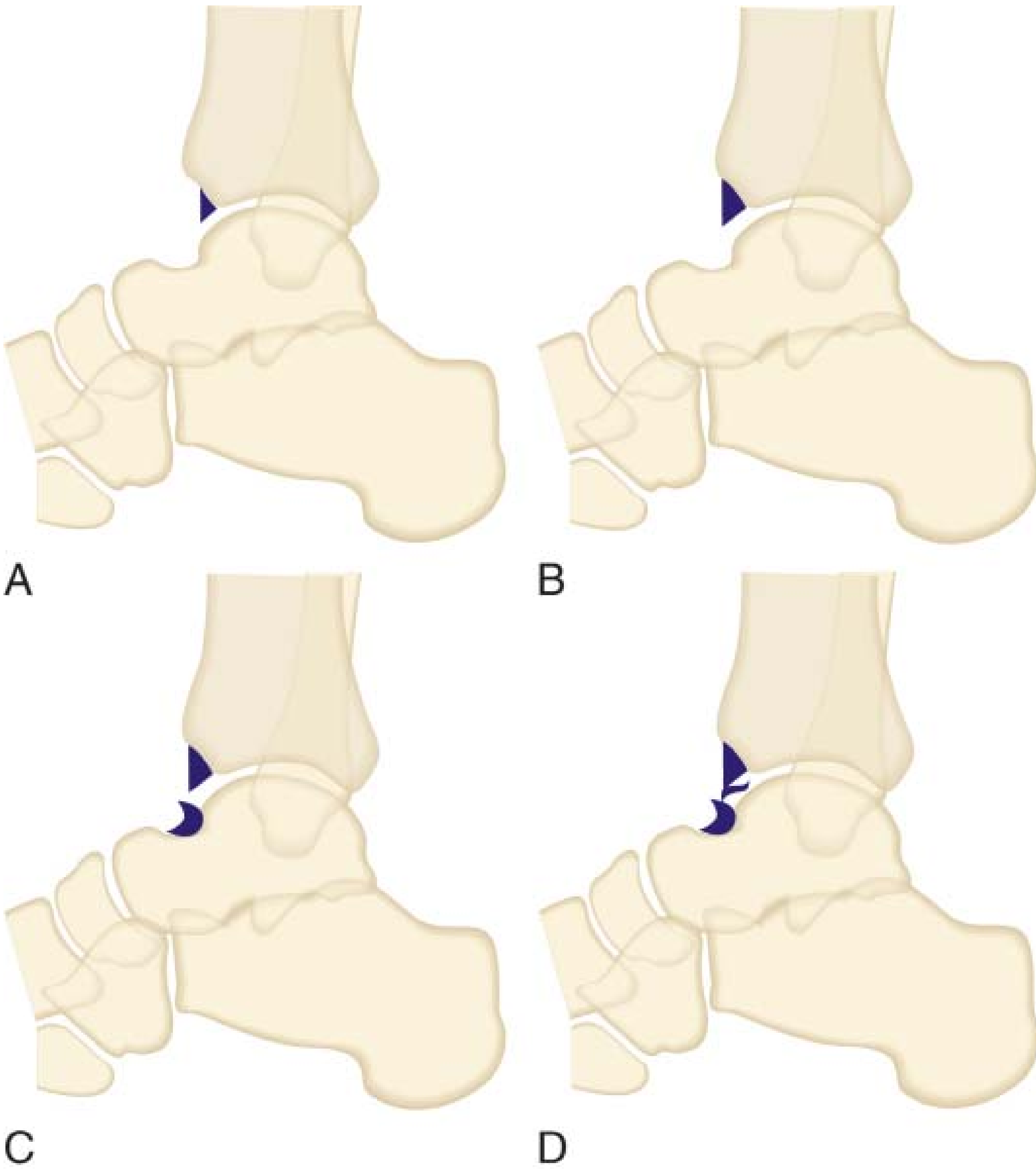

Figure 25I-14 Classification of anterior impingement. **A,** Grade I: synovial impingement, spur ≤3 mm. **B,** Grade II: osteochondral reaction exostosis, spur >3 mm. **C,** Grade III: severe exostosis with or without fragmentation, secondary spur on talus. **D,** Grade IV: pantalocrural osteoarthrotic destruction. *(Redrawn from Ferkel RD, Scranton PE: Current concepts review: Arthroscopy of the ankle and foot. J Bone Joint Surg Am 75:1233-1242, 1993.)*

Anterolateral impingement is believed to be caused by relatively minor inversion injuries of the ankle and to occur after about 3% of ankle sprains. [108] Tearing of the anterolateral soft tissues and ligaments, without substantial associated mechanical instability, followed by repeated microtrauma can result in hypertrophy of the synovial tissue and fibrosis in the anterolateral ankle gutter, causing pain and mechanical impingement. Several studies [1762] [1763] [1764] [1765] [1766] have suggested that a contributing factor may be hypertrophy of an accessory fascicle of the anterior tibiofibular ligament (Bassett ligament) (Fig. 25I-15). Most patients complain of chronic vague pain over the anterolateral ankle, exacerbated by cutting or pivoting maneuvers, and physical examination identifies tenderness along the lateral gutter and the anterior tibiofibular ligament. Maximal dorsiflexion and deep palpation to the anterolateral corner of the ankle joint reproduces the pain. The “charger” stance, with the patient bearing weight, flexing the knee, and keeping the heel flat on the ground (Fig. 25I-16), also produces pain in this corner of the ankle. [114] Impaired proprioception also has been identified in patients with anterolateral impingement.

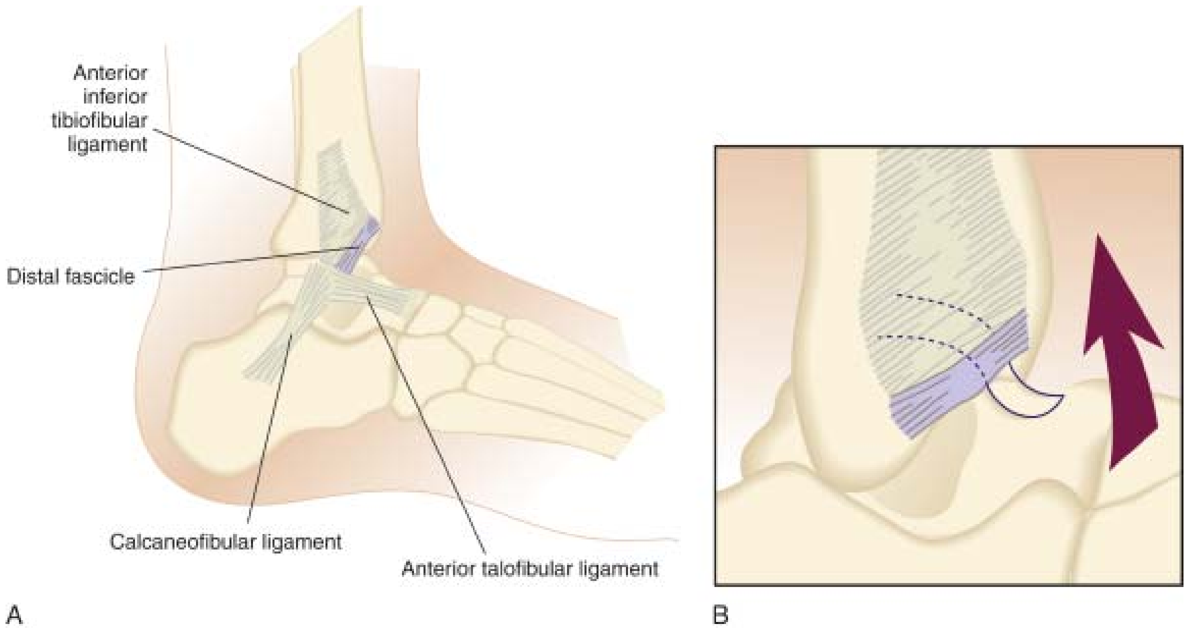

Figure 25I-15 A, Distal fascicle of the anterior inferior tibiofibular ligament is parallel and distal to the anterior tibiofibular ligament proper and is separated from it by a fibrofatty septum. **B,** With dorsiflexion of the ankle, the distal fascicle may impinge on the anterolateral aspect of the talus. *(From Bassett FH 3rd, Gates HS 3rd, Billys JB, et al: Talar impingement by the anteroinferior tibiofibular ligament. J Bone Joint Surg Am 72:55-59, 1990.)*

Figure 25I-16 "Charger" stance causes pain in the anterolateral corner of the ankle in patients with anterolateral impingement. *(From Hyer CF, Buchanan MM, Philbin TM, et al: Ankle arthroscopy. In ElAttrache NS, Harner CD, Mirzayan R, Sekiya JK [eds]: Surgical Techniques in Sports Medicine. Philadelphia, Lippincott Williams & Wilkins, 2007.)*

Anteromedial impingement is an uncommon cause of chronic ankle pain that also is associated with an inversion mechanism that injures the lateral and medial ankle ligaments. A meniscoid lesion or a thickened anterior tibiotalar portion of the deltoid ligament may impinge on the anteromedial corner of the talus during dorsiflexion of the ankle, resulting in osteophyte formation or a chondral lesion, or both.

Posterior impingement has been described as os trigonum syndrome, talar compression syndrome, and posterior block of the ankle and has been extensively described in ballet dancers as well as in gymnasts, soccer players, and down-hill runners. [1763] [1764] [1768] [1769] The mechanism of injury has been compared with a nutcracker because the posterior talus and surrounding soft tissues are compressed between the tibia and the calcaneus during plantar flexion of the foot. [117] Bony causes of posterior impingement include the os trigonum (an accessory ossicle of the lateral talar tubercle that persists in as many as 7% of adults), the Stieda process (an elongated lateral talar tubercle), a prominent posterior calcaneal process, and loose bodies (Table 25I-7). Soft tissue factors can include synovitis of the flexor hallucis longus tendon sheath, the posterior synovial recess of the subtalar and tibiotalar joints, and the posterior intermalleolar ligament. Pain is worse with forced plantar flexion and with push-off maneuvers.

TABLE 25I-7 -- Etiology of Posterior Ankle Impingement Syndrome

Pathology	Example
Trigonal process	Fracture (acute or chronic)
Synchondrosis injury	
True compression	
Flexor hallucis longus dysfunction	Tenosynovitis
Tibiotalar pathology	Posterior capsuloligamentous injury
Osteochondritis	
Fracture	
Subtalar pathology	Osteochondritis
Arthritis	
Other	Calcified inflammatory tissue
Prominent calcaneus posterior process	
Combined	Flexor hallucis longus tenosynovitis and synchondrosis injury

From Maquirriain J: Posterior ankle impingement syndrome. J Am Acad Orthop Surg 13:365-371, 2005.

Posteromedial impingement occurs after severe ankle inversion injury that damages the deep posterior fibers of the medial deltoid ligament. Chronic inflammation and hypertrophy of the ligament result in fibrotic scar tissue that can be trapped between the medial wall of the talus and the posterior margin of the medial malleolus.

Treatment Options: Weighing the Evidence

Initial treatment of any impingement lesion should be nonoperative, including restriction of activity, orthoses, nonsteroidal anti-inflammatory drugs (NSAIDs), and possibly cortisone injection. If nonoperative methods are unsuccessful at relieving pain and mechanical symptoms, arthroscopic débridement of soft tissue lesions or excision of bony lesions, or both, is successful in most patients. In two of the larger series of anterolateral lesions, [1771] [1772] including 64 lesions, 58 (91%) had excellent or good results, 4 had fair results, and 2 had poor results after arthroscopic treatment. At a mean follow-up of 6 years after arthroscopy, 40 (70%) of 57 patients with anterior lesions had resumed sports, 21 of them playing soccer. [120] Results were good in all patients who had no preoperative osteoarthritis (OA), in 73% of those with grade I OA, and in only 29% of ankles with grade II OA. Posterior arthroscopy also can be used for excision of an os trigonum, tenolysis, loose body removal, spur excision, and débridement of OLT. [121] Two studies each reported that 14 of 15 patients returned to their preinjury levels of sports after posterior arthroscopy. [1774] [1775] Henderson and La Valette [104] described anterior arthroscopic and open posterior treatment for combined anterior and posterior impingement in 62 patients; 47 (81%) had excellent or good outcomes, 9 (15.5%) had fair outcomes, and 2 (3.5%) had poor outcomes.

AUTHOR'S PREFERRED METHOD

If nonoperative measures do not relieve symptoms, arthroscopy is indicated for excision, débridement, decompression, or synovectomy. Large exostoses may require open arthrotomy for excision.

Arthroscopic treatment of ankle impingement begins with a careful inspection of all structures that may be contributing factors (Table 25I-8). Standard anteromedial and anterolateral ankle portals are used (Fig. 25I-17).

TABLE 25I-8 -- Ankle Impingement Syndromes: Arthroscopic Examination

Medial Portal	Lateral Portal
Medial gutter	Lateral gutter
Medial malleolus	Lateral malleolus
Deep fibers of deltoid ligament	Anteroinferior talofibular ligament
Anterior joint line, anterior tibia	Anterior joint line, anterior tibia

Medial Portal	Lateral Portal
Talar dome	Talar dome
Tibiofibular joint, ligaments	Medial malleolus
Lateral gutter	Medial gutter

From Hyer CF, Buchanan MM, Philbin TM, et al: Ankle arthroscopy. In ElAttrache NS, Harner CD, Mirzayan R, Sekiya JK (eds): Surgical Techniques in Sports Medicine. Philadelphia, Lippincott Williams & Wilkins, 2007.

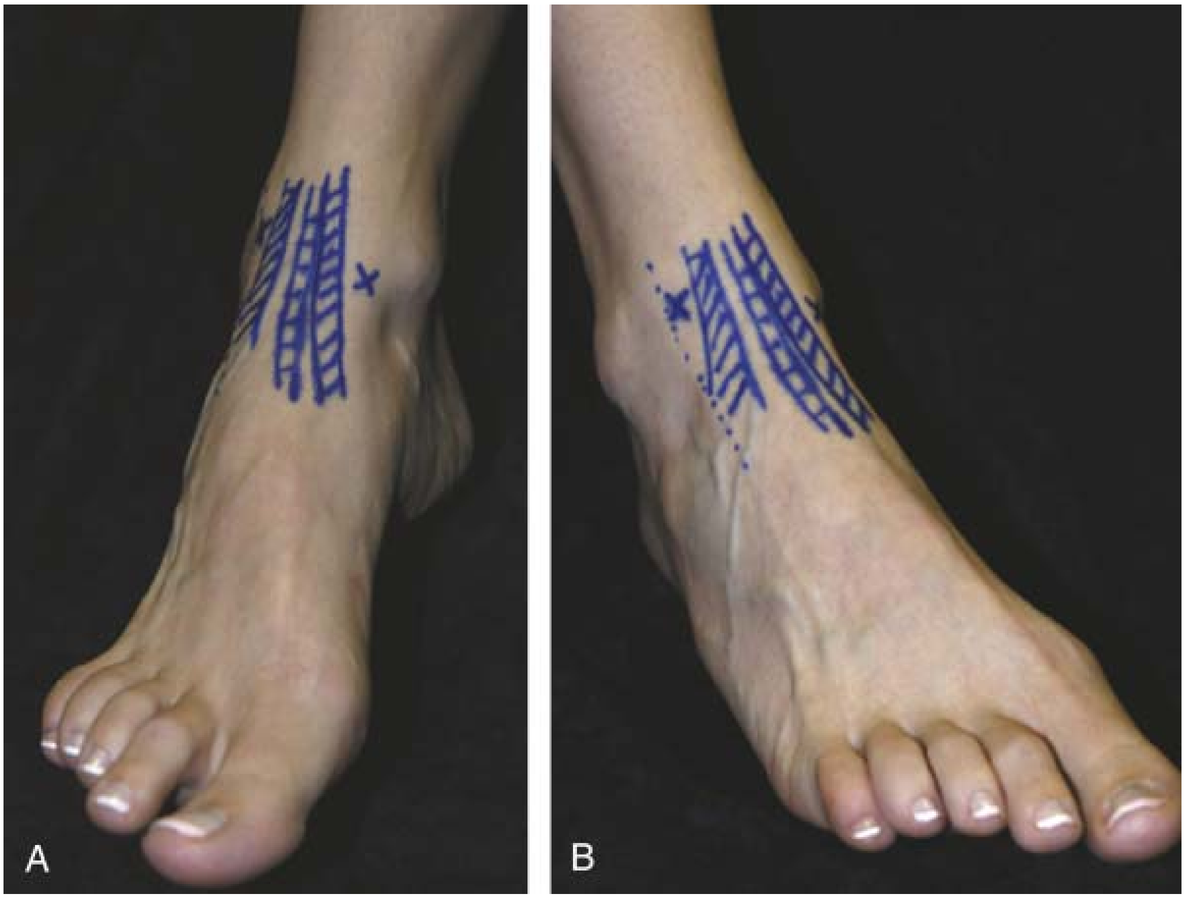

Figure 25I-17 Ankle arthroscopy portals. **A,** Anteromedial portal site. **B,** Anterolateral portal site. *(From Phillips BB: Arthroscopy of the lower extremity. In Canale ST, Beaty JH [eds]: Campbell's Operative Orthopaedics, 11th ed. Philadelphia, Elsevier, 2008.)*

Technique of Arthroscopic Treatment of Ankle Impingement

- With the patient under general anesthesia, apply and inflate a thigh tourniquet.
- Insert a needle just medial to the anterior tibial tendon and distend the ankle joint with 15 to 20 mL of saline.
- Make a small longitudinal incision to allow insertion of a 2.7-mm or 4.0-mm, 30-degree angle arthroscope through an anteromedial portal just medial to the anterior tibial tendon. Take care to pass the arthroscope across the anterior aspect of the joint and not across the dome of the talus.
- Make a separate anterolateral portal just lateral to the peroneus tertius tendon to allow inflow and outflow of saline. Be aware of the superficial peroneal nerve in this area. Instruments and the arthroscope can be switched to either portal as necessary.

- Fully examine the ankle with the use of a noninvasive ankle distraction device as necessary (see Fig. 25I-6). Distraction may need to be removed to identify and gain access to large anterior osteophytes, especially on the talus, because distraction may cause the anterior capsule to tighten.
- Use a pressure irrigation system with a 3.5-mm full-radius resector to clear the anterior synovium and define the anterior tibial and superior talar bony spurs.
- Use a 3-mm bur to remove the spurs, resecting them back to the level of normal cartilage.
- Smooth off the tibial surface with a 3.5-mm full-radius resector.
- Carry out a similar procedure on the superior neck of the talus.
- Again, examine the whole ankle by passing the arthroscope gently over the dome of the talus. This can be accomplished with the use of manual distraction in mid–plantar flexion or with a commercially available noninvasive ankle distraction device.
- After irrigation, place 20 mL of 0.25% bupivacaine into the joint, suture the incision, and apply a compression dressing.

If synovitis is found, typically along the anterior joint line and in the medial and lateral ankle gutters, an arthroscopic resector is used to remove the hypertrophic synovitis. Débridement begins in the medial gutter, then the anterior joint line, and finally the lateral gutter. Switching portals is necessary for complete débridement. Hypertrophy of the anterior tibiofibular ligament, which appears as a whitish, thick, scar-like lesion, is resected and débrided.

For anterior bony impingement, the anterior ankle capsule is carefully reflected and elevated superiorly off the anterior tibia and inferiorly off the talar neck. An arthroscopic bur is used to remove the exostoses on the tibia and talus. It is important to make sure the bur is facing bone to avoid damage to the soft tissues, especially the anterior neurovascular bundle. A trough can be made with a 3-mm bur about 1 mm proximal and parallel to the anterior edge of the tibia. This trough is taken down to subchondral bone to the level of surrounding normal cartilage, and an arthroscopic bone biter is used to remove the bony spur. This allows more control of the bur with less risk for damage to the articular surface than may occur with an arthroscopic shaver. [123] If the exostoses are large, open excision may be preferable through an anteromedial incision and arthrotomy. Tibial osteophytes typically are located laterally and the talar osteophytes medially. They can be easily removed with an osteotome.

Posterior synovectomy can be done arthroscopically through a posterolateral portal (Fig. 25I-18). For excision of posterior bony impingement, we usually prefer an open, posterolateral approach. The interval between the Achilles tendon and peroneal tendons is developed, with care to protect the sural nerve. The os trigonum is removed with subperiosteal dissection. The lateral tubercle of the posterior process of the talus (Stieda process) is removed with a curved osteotome or rongeur to ensure that the posterior talar surface is flush with the posterior tibial surface. If decompression of the flexor hallucis longus is indicated, a posteromedial approach can be developed along the tarsal tunnel

Figure 25I-18 Posterior portal site for ankle arthroscopy. *(From Phillips BB: Arthroscopy of the lower extremity. In Canale ST, Beaty JH [eds]: Campbell's Operative Orthopaedics, 11th ed. Philadelphia, Elsevier, 2008.)*

Postoperative Prescription, Potential Complications, and Criteria for Return to Play

After ankle arthroscopy, weight-bearing to tolerance, with or without crutches, usually is allowed the first week after surgery. Then a progressive program of strengthening, range of motion, and functional agility is begun. Generally, return to competitive sports is possible by about 6 weeks after surgery when the patient is pain free and range of motion and strength are comparable to the unaffected ankle. The most frequent complication of ankle arthroscopy is temporary numbness or tenderness at a portal site caused by local nerve damage. Osteophytes may recur, especially in athletes who have recurrent supination trauma or repeated forceful dorsiflexion of the ankle, but these are not always symptomatic. [120]

Tarsal Coalition

Particularly in children and adolescents, there is an association between frequent ankle sprains and tarsal coalitions, including fibrous (syndesmosis), cartilaginous (synchondrosis), and bony (synostosis) coalitions. Although the frequency of tarsal coalition has long been estimated at 1%, more recent information indicates a frequency of 11%, possibly because of more frequent identification by MRI. [124] Patients may complain of mild deep pain and limited range of motion, usually after a traumatic ankle sprain that "just never seems to get better." Often symptoms are relieved by rest and aggravated by prolonged or heavy activity, suggestive of ankle ligament injury. [125] Radiographs or CT scans can confirm the diagnosis (Fig. 25I-19). If nonoperative measures, such as bracing or casting and NSAIDs, fail to relieve symptoms, open or arthroscopic excision of the coalition is indicated. [126] After coalition excision, a removable boot usually is worn for about 6 weeks, and strengthening and flexibility exercises are done.

Figure 25I-19 CT shows medial facet tarsal coalition of talus and calcaneus.

Neoplasms

Neoplasms such as osteoid osteoma, eosinophilic granuloma (Fig. 25I-20), and pigmented villonodular synovitis (Fig. 25I-21) can mimic ankle sprain. Neoplasms of the ankle can mimic a number of ankle conditions, including chronic sprains, anterior impingement, stress fracture, osteomyelitis, and osteonecrosis. Appropriate treatment of these lesions often is delayed because of misdiagnosis; one study reported a 2.5-year delay in the diagnosis of osteoid osteomas in five patients, [127] and another reported a delay of more than 3 years in one patient. [128] A typical feature of osteoid osteoma is pain that worsens at night and is relieved by aspirin, but this may not be present in all patients with osteoid osteomas. Treatment of osteoid osteomas usually is en bloc excision or curettage of the nidus. Successful arthroscopic treatment of osteoid osteomas of the ankle has been described, [1781] [1782] [1783] [1784] which allows an earlier return to activity (2 to 3 months after surgery) than open procedures.

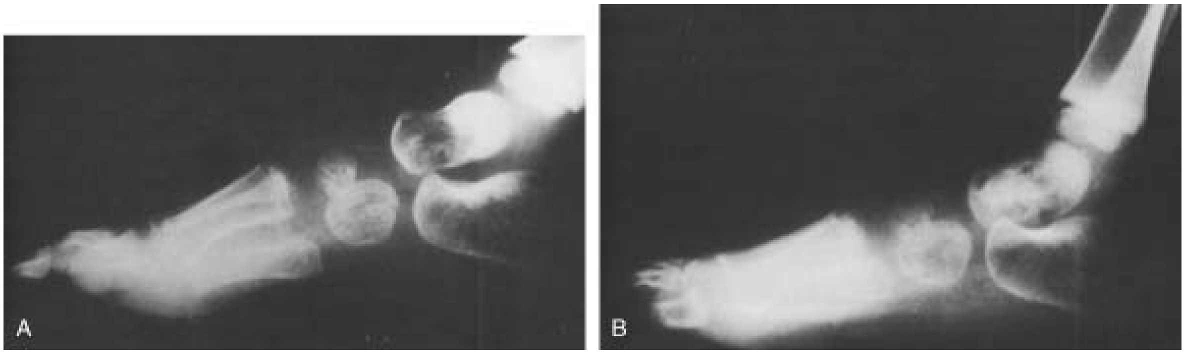

Figure 25I-20 Eosinophilic granuloma may cause symptoms of ankle sprain. **A,** Lesion in head and neck of talus. **B,** After excision and bone grafting.

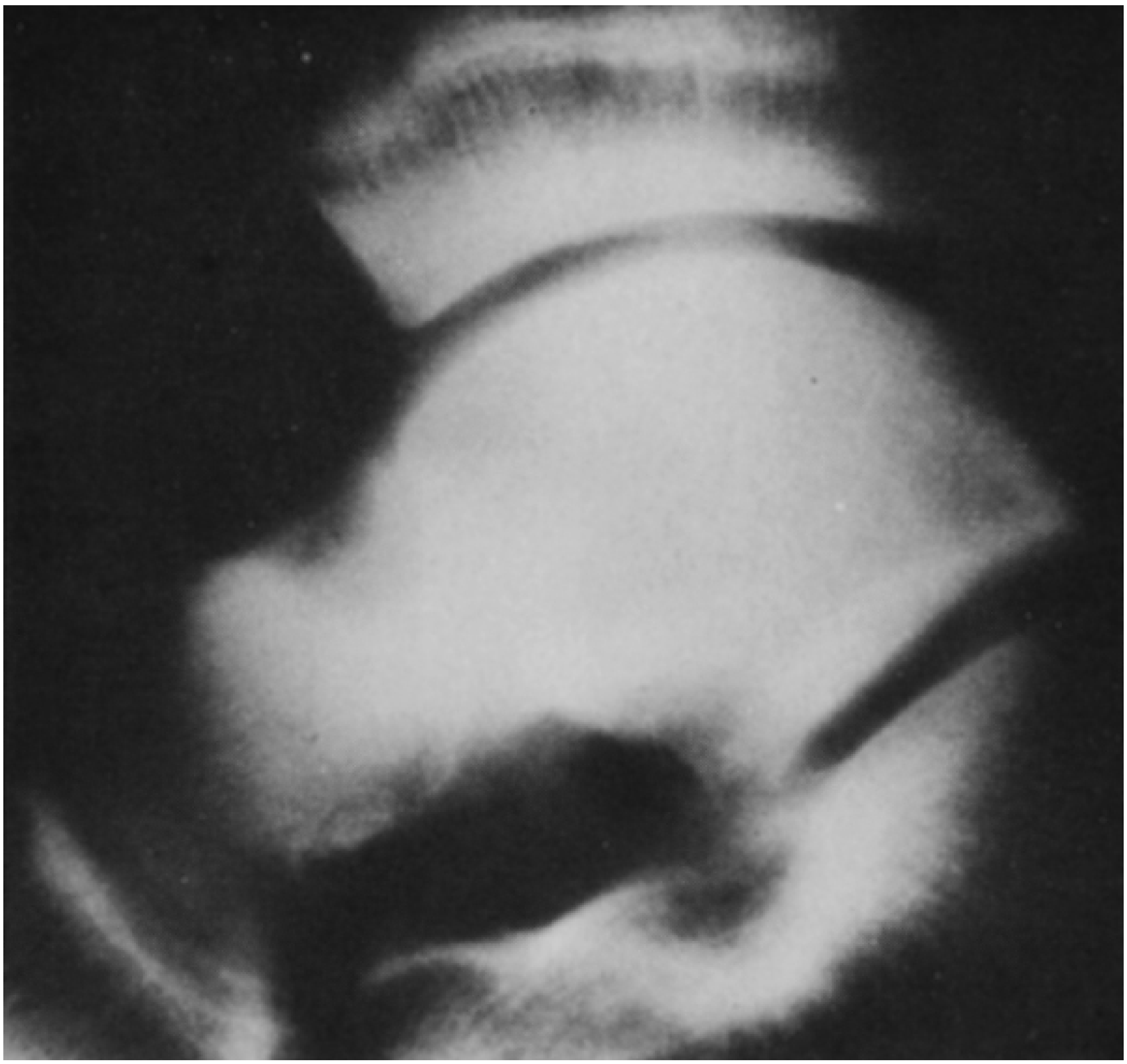

Figure 25I-21 Pigmented villonodular synovitis eroding into superior portion of the talar neck caused symptoms of ankle sprain in young athlete.

OTHER APOPHYSITIS, OSTEOCHONDRITIS, AND DEVELOPMENTAL ANOMALIES OF THE FOOT THAT CAN

CAUSE DISABILITY IN ATHLETES

Calcaneal Apophysitis (Sever Disease)

First described by J. W. Sever [132] in 1912, calcaneal apophysitis (Sever disease) is a common cause of heel pain, especially during running, that most often occurs in children who are engaged in activities such as basketball and soccer. The calcaneal apophysis appears as an independent center of ossification in boys aged 9 to10 years and ossifies around the age of 17 years; in girls, this occurs earlier. The average age at presentation is 11 years in boys and 8 years in girls. Sever disease is 2 to 3 times more common in boys, and 60% of patients have bilateral involvement. Aptly described by Sever as an “inflammation of the calcaneal apophysis resulting in the clinical symptoms of pain at the posterior heel, mild swelling, and difficulty with walking,” the condition often causes a child to walk with an antalgic gait. The heel pad and posterior aspect of the heel are tender, but swelling usually is minimal. Pain is located at the most distal portion of the heel pad and along the posterior aspect of the heel up to the most distal portion of the Achilles tendon. The heel cord itself is not especially tight. Ogden and associates [133] suggested that this entity should be called *Sever injury* rather than *Sever disease* because MRI evidence indicated that the true pathogenesis is a stress microfracture related to chronic repetitive microtrauma. The stress microfractures were found in the metaphysis of the body of the calcaneus adjacent to, but not directly involving, the apophysis.

Radiographs are not essential to the diagnosis but can rule out other conditions, such as fracture, infection, or neoplasm. They may show a sclerotic and fragmented calcaneal apophysitis (Fig. 25I-22), but the radiographic appearance usually is normal.

Figure 25I-22 Increased sclerosis and fragmentation of calcaneal apophysis (Sever disease).

The necessity of curtailing the activity that incites pain is controversial. Because they consider this condition a chronic, repetitive injury to metaphyseal bone, some have suggested that temporary discontinuation of the activity is essential. Most patients are able to return to normal sports activity after 2 months of rest; in only the most severe cases is immobilization necessary. [1787] [1788] Conversely, Weiner and associates [136] reviewed the records of 227 patients with Sever disease and concluded that activity restriction is unnecessary; they recommended an in-shoe orthosis, no limits on physical activity, with 4 to 6 weeks of casting for persistent symptoms.

Osteochondrosis of the Tarsal Navicular (Köhler Disease)

First described by Köhler [137] in 1908, osteochondrosis of the tarsal navicular is a relatively uncommon cause of pain and limp in children. Like Sever disease, boys are more commonly affected than girls; however, the age of onset is younger, 5 years in boys and 4 years in girls. Thirty-three percent of patients have bilateral involvement. Tenderness in the area of the tarsal navicular is the most consistent physical finding. The radiographic findings are variable degrees of sclerosis, flattening, and irregular rarefaction of the navicular (Alka-Seltzer-on-end appearance) (Fig. 25I-23). Loss of the trabecular pattern and fragmentation also are common. Other bones of the foot usually are normal. Köhler disease is self-limiting, and the final outcome is not affected by treatment. Short-leg walking cast immobilization for 6 to 8 weeks has been reported to result in faster resolution of symptoms than conservative treatment. [138]

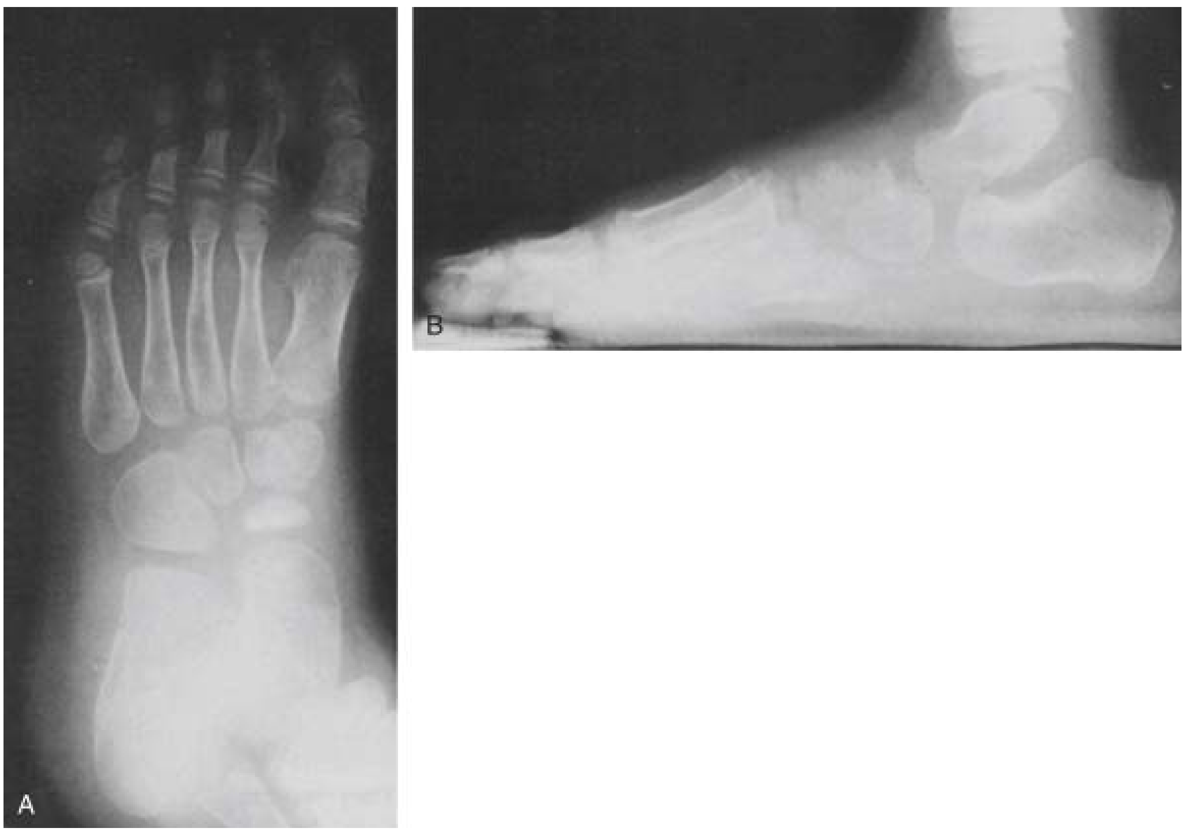

Figure 25I-23 **A** and **B,** Increased sclerosis and narrowing (Alka-Seltzer-on-end appearance) of the navicular (Köhler disease) in a young athlete with complaints of pain in the medial midfoot area.

Accessory Navicular

The accessory navicular is a separate ossification center for the tuberosity of the navicular that is present in about 14% of all feet, but most of these are asymptomatic and eventually fuse with the navicular. [1792] [1793] In some individuals, however, the accessory navicular can be a source of medial foot pain. The accessory navicular may exist as a separate ossicle within the posterior tibial tendon (type I), may form a synchondrosis with the navicular (type II), or may fuse with the navicular and create a cornuate navicular (type III) (Fig. 25I-24). Often the posterior tibial tendon inserts onto the accessory navicular instead of onto the tuberosity of the navicular, which has been suggested to alter the pull of the tendon and cause a flatfoot deformity. Symptoms are most common when the accessory navicular forms a synchondrosis with the navicular (type II). Symptoms usually begin in adolescence and are made worse by activity and weight-bearing. In adults, the onset of pain usually is acute after an eversion injury or other foot trauma. Pain is localized over the prominence of the accessory navicular and at the medial arch of the foot. A painful bursa may be present over the navicular prominence. Standard anteroposterior and lateral views of the foot (Fig. 25I-25), along with a 45-degree eversion oblique view, usually are sufficient for diagnosis, but bone scanning and MRI can be helpful if the diagnosis is in question.[141] Initial treatment of a symptomatic accessory navicular is nonoperative and directed at relieving pressure at the painful medial prominence and reducing inflammation. The classic operative treatment for an accessory navicular that does not respond to nonoperative measures is removal of the ossicle (Kidner procedure[142]), which generally gives good results.[1796] [1797] The benefits of transposition of the posterior tibial tendon to the plantar aspect of the navicular are questionable.[1793] [1798] [1799] [1800] Percutaneous drilling of the accessory navicular to induce or accelerate fusion was reported to obtain excellent or good results in 30 of 31 feet.[148] Percutaneous placement of one or two 3.5-mm or 4.0-mm cannulated screws across the synchondrosis has been recommended in athletes 14 to 30 years of age who have symptoms consistent with synchondrotic stress fracture.[149] Malicky and associates[150] described open fusion between the ossicle and navicular bone: after excision of the synchondrosis and the adjacent subchondral bone, one or two 2.7-mm or 3.5-mm lag screws are inserted to “arthodese” the bones together. They recommended this procedure in adults with large, painful accessory navicular bones because it leaves the posterior tibial tendon attachment intact and provides a more reliable healing surface.

AUTHORS' PREFERRED METHOD

Initial treatment is nonoperative and can include physical therapy aimed at controlling coexisting tendinitis and strengthening the posterior tibial tendon, molded orthoses, shoe modifications, and cast immobilization. If symptoms persist, we prefer simple excision of the accessory navicular. It is important to excise enough of the prominence to create a surface that is flush with the medial border of the midfoot.

Technique of Excision of Accessory Navicular

- Beginning 1 to 1.5 cm inferior and distal to the tip of the medial malleolus, curve the skin incision slightly dorsalward, peaking at the medial prominence of the accessory navicular and sloping distally to the base of the first metatarsal (Fig. 25I-26 **A**).
- Ligate the plantar communicating branches of the saphenous system and identify the posterior tibial tendon as it approaches the accessory navicular (see Fig. 25I-26 **B**).
- Identify the dorsal and plantar margins of the tendon 2 cm proximal to the accessory navicular, and expose the tendon distally, ending at the bone. This exposes the entire tendon without disturbing the part extending plantarward toward its multiple insertions.
- Use sharp dissection to shell the accessory navicular from the posterior tibial tendon (see Fig. 25I-26 **C**).

Return to Sport

After operative treatment, a short-leg cast is worn for 6 weeks. Range of motion exercises are begun after cast removal, and return to competition usually can be accomplished within 8 to 10 weeks.

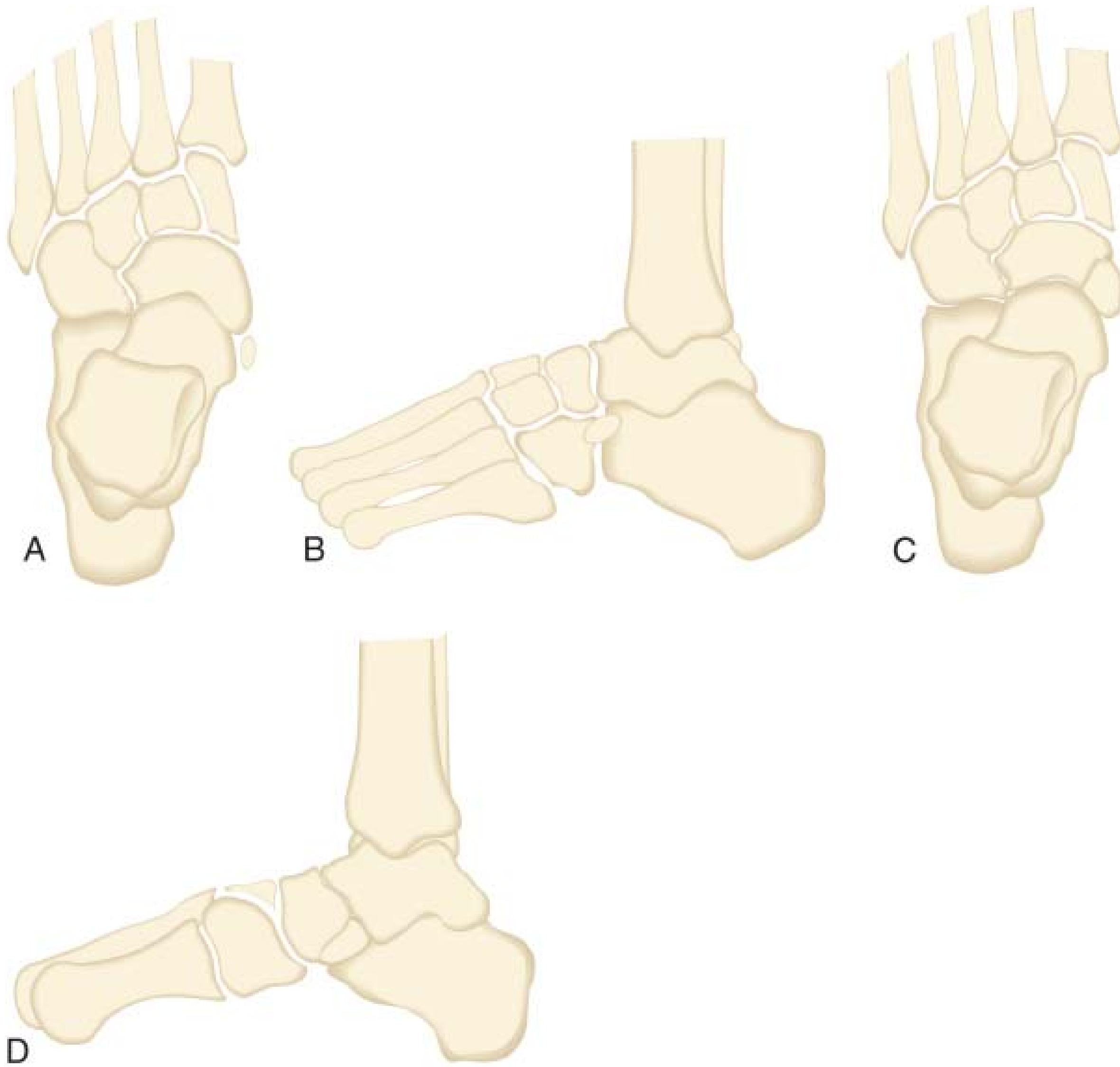

Figure 25I-24 Accessory navicular. **A** and **B,** Type I. **C** and **D,** Type II. *(Redrawn from MacNicol MJ, Voutsinas S: Surgical treatment of the symptomatic accessory navicular. J Bone Joint Surg Br 66:218-226, 1984.)*

Figure 25I-25 **A** and **B,** Bilateral accessory navicular. Note “opening up” of metatarsal cuboid-cuneiform articulation, suggesting flattening of the longitudinal arch. **C** and **D,** Note sag at talonavicular joint in left foot (**C**) compared with right foot (**D**). *(From Murphy GA: Pes planus. In Canale ST, Beaty JH [eds]: Campbell’s Operative Orthopaedics, 11th ed. Philadelphia, Elsevier, 2008.)*

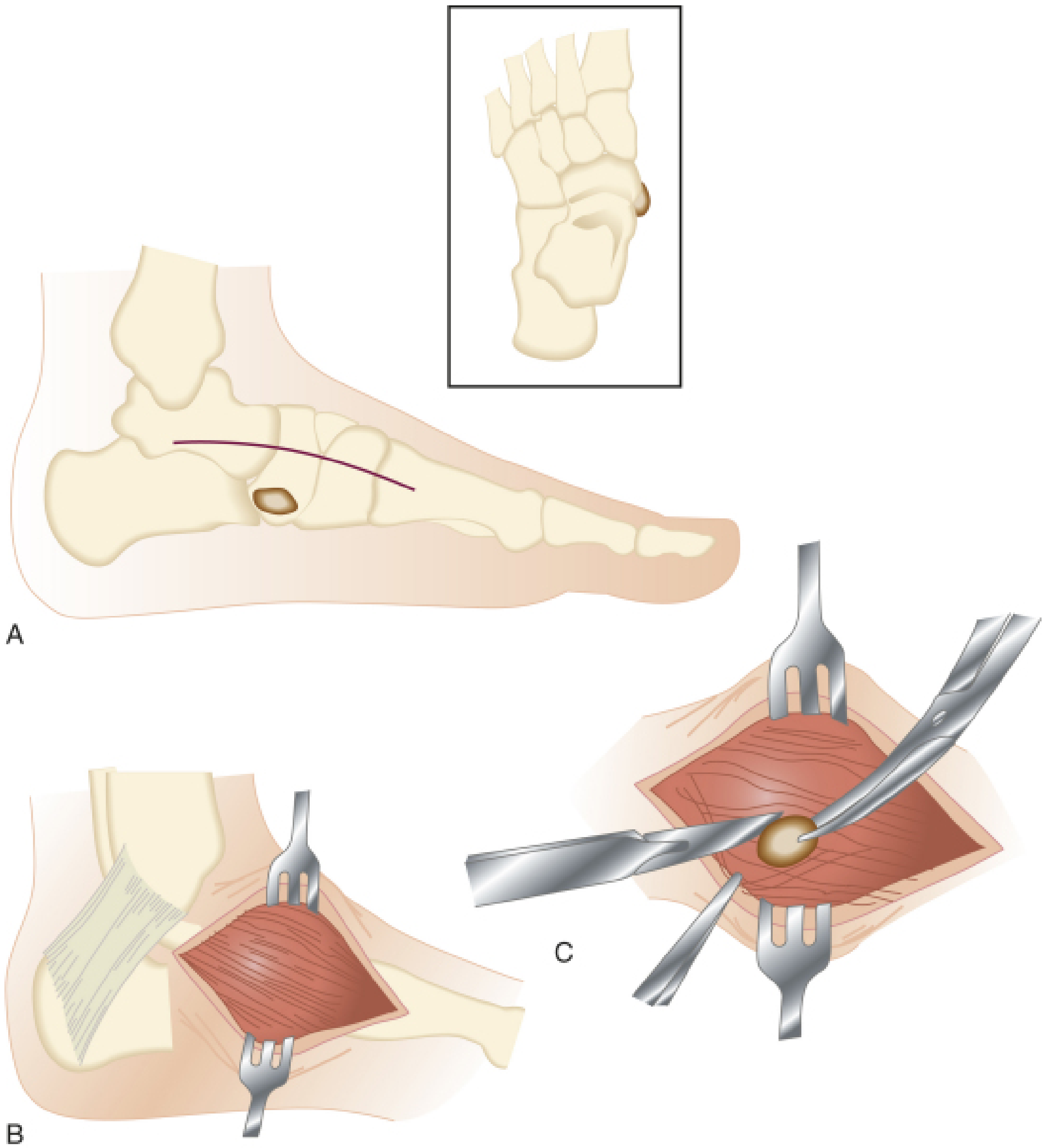

Figure 25I-26 Excision of accessory navicular. **A,** Incision. **B,** Exposure of the posterior tibial tendon. **C,** Removal of accessory navicular. *(From Murphy GA: Pes planus. In Canale ST, Beaty JH [eds]: Campbell's Operative Orthopaedics, 11th ed. Philadelphia, Elsevier, 2008.)*

Osteochondrosis of the Cuneiform

Osteochondrosis of the cuneiform (Buschke disease) is rare, with few reports in the literature. Repetitive trauma with insult to the developing osseous tissue is believed to be responsible for this osteochondrosis, especially in the pronated foot in which continuous pressure is placed on the medial side of the foot and the medial cuneiform. Of the 19 patients described in the English literature, 18 were boys; 12 were 5 or 6 years old, and the youngest was 2 years old. [1804] [1805] Pain and limping usually are present, but the lesion can be asymptomatic. Pain occasionally can be elicited by gentle pressure over the medial cuneiform, and mild swelling rarely is present. Radiographs usually show a sclerotic, fragmented medial cuneiform (Fig. 25I-27). Osteochondrosis of the medial cuneiform has been associated with other tarsal bone lesions in most patients and is almost always bilateral. Treatment is nonoperative and includes control of the foot pronation and reduction of pain with rest and medial arch supports.

Figure 25I-27 Increased sclerosis and fragmentation of the first cuneiform (Buschke disease) in a child who complained of pain in the medial midfoot area.

Osteochondrosis of the Metatarsal Head (Freiberg Disease)

First described by Freiberg [153] in 1914, osteochondrosis or osteonecrosis of the metatarsal head is most common in adolescent athletes who perform on their toes in either sprinting or jumping activities. It is more common in girls than in boys, making it the only osteochondrosis with a female predilection. The second, followed by the third, metatarsal head is most commonly involved. Fewer than 10% of patients have bilateral involvement. The pathogenesis is unknown, but Gauthier and Elbaz [154] found that patients with longer second metatarsals and excessive plantar pressure under the second metatarsal head had no significantly higher risk. These findings would appear to call into question mechanical stress being the sole or even the primary cause of Freiberg disease. The primary complaint often is a vague forefoot pain that is worsened by activity and weight-bearing and relieved with rest. The pain usually is worse at extremes of motion, with pain under and around the involved metatarsophalangeal joint. Palpation may identify swelling and slightly increased temperature. The initial process of pain and synovitis is followed by radiographic findings of sclerosis, resorption of the subchondral plate, fracture, collapse, and fragmentation. Secondary degenerative changes and remodeling then occur in the flattened metatarsal head (Fig. 25I-28). Several staging systems have been developed to correlate physical and radiographic findings with treatment (Fig. 25I-29 and Table 25I-9), but their reliability is still unproved.

Figure 25I-28 Freiberg infarction. **A** and **B,** Note flattening of second metatarsal head. **C,** Note marrow edema of second metatarsal.

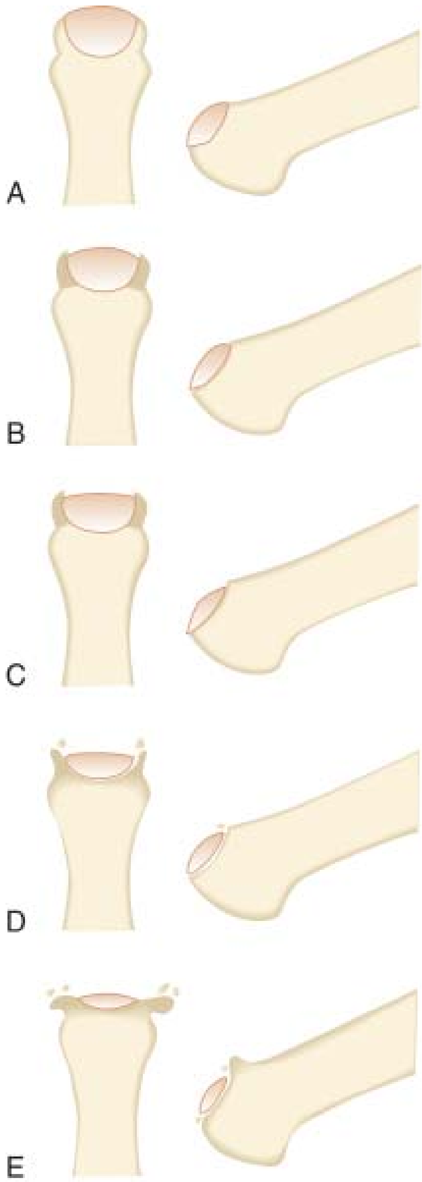

Figure 25I-29 Levels of progression of Freiberg disease. **A,** Early fracture of subchondral epiphysis. **B,** Early collapse of dorsal central portion of metatarsal with flattening of articular surface. **C,** Further flattening of metatarsal head with continued collapse of central portion of articular surface with medial and lateral projections; plantar articular cartilage remains intact. **D,** Loose bodies form from fractures of lateral projections and separation of central articular fragment. **E,** End-stage degenerative arthrosis with marked flattening of the metatarsal head and joint space narrowing. *(Redrawn from Katcherian DA: Treatment of Freiberg's disease. Orthop Clin North Am 25:69-81, 1994.)*

TABLE 25I-9 -- Staging and Classification Systems for Freiberg Disease

Katcherian	Smillie	Gauthier/Elbaz	Thompson/Hamilton	Description	Radiographs
Level A	Stage I	Stage 0 + 1	Type 1	Earliest form; fissure in epiphysis	Radiographs normal; bone scan may detect
Level B	Stage II	Stage 2	Type 2	Progression of subchondral fracture with bone resorption; collapse of dorsal central portion of metatarsal head and alteration of articular surface	Radiographs may show slight widening of joint, sclerosis of epiphysis, collapse and flattening of articular surface

Katcherian	Smillie	Gauthier/Elbaz	Thompson/Hamilton	Description	Radiographs
Level C	Stage III	Stage 2	Type 2	Further deformation and collapse of central portion of head	Progressive flattening of metatarsal head with osteolysis and collapse; zone of rarefaction around sclerotic bone as healing and revascularization take place; premature physeal closure may be present
Level D	Stage IV	Stage 3	Type 3	Fracture and separation of central portion and peripheral projections of involved metatarsal head resulting in loose bodies	Fragmentation of epiphysis, early joint narrowing, multiple loose bodies
Level E	Stage V	Stage 4	Type 3	Advanced arthrosis secondary to progressive flattening and deformity of metatarsal head	Joint space narrowing, hypertrophy of metatarsal head, irregularity of base of proximal phalanx with osteophyte formation

Modified from Katcherian DA: Treatment of Freiberg's disease. Orthop Clin North Am 25:69-81, 1994.

MT, metatarsal.

Treatment Options

Initial treatment is nonoperative, including metatarsal relief pads, restriction of running and jumping activities, and occasionally a short-leg walking cast worn for 6 to 12 weeks until acute symptoms are resolved. A number of operative procedures have been described for persistent symptoms, including débridement, synovectomy, drilling, osteotomy, interpositional arthroplasty, and joint replacement. Dorsal wedge or dorsiflexion osteotomy (Fig. 25I-30) has been used successfully for all stages of the disease, [1807] [1808] [1809] [1810] although the range of motion of the MTP joint is decreased. More recent innovations include the use of absorbable pins for fixation of the osteotomy, which obviates the need for a second operation for implant removal; arthroscopic techniques for synovectomy and drilling [1811] [1812] ; and osteochondral plug transplantation. [1811] [1813] Carro and coworkers [160] recommended an age-based approach to the treatment of Freiberg disease, beginning with arthroscopic synovectomy and débridement, followed by open or arthroscopic osteochondral transplantation in late adolescence and adulthood, and an arthroscopic Keller procedure (with or without interpositional arthroplasty) for more severe involvement in late adulthood. However, in our experience, resection of the base of the proximal phalanx or the metatarsal head should be avoided to avoid transfer metatarsalgia or hallux valgus caused by instability of the second digit.

AUTHORS' PREFERRED METHOD

Initial treatment is nonoperative, including restriction of sports activities and short-term cast-with-toe-plate immobilization. After acute symptoms resolve, metatarsal pads inserted proximal to the MTP joint are used during running and sports. Operative treatment is indicated for persistent symptoms. For adolescents and young adults, joint débridement and removal of loose bodies usually are sufficient, and return to sports generally is possible in 6 to 8 weeks. More extensive surgery (dorsiflexion or metatarsal shortening osteotomy) is reserved for older patients with late-stage involvement.

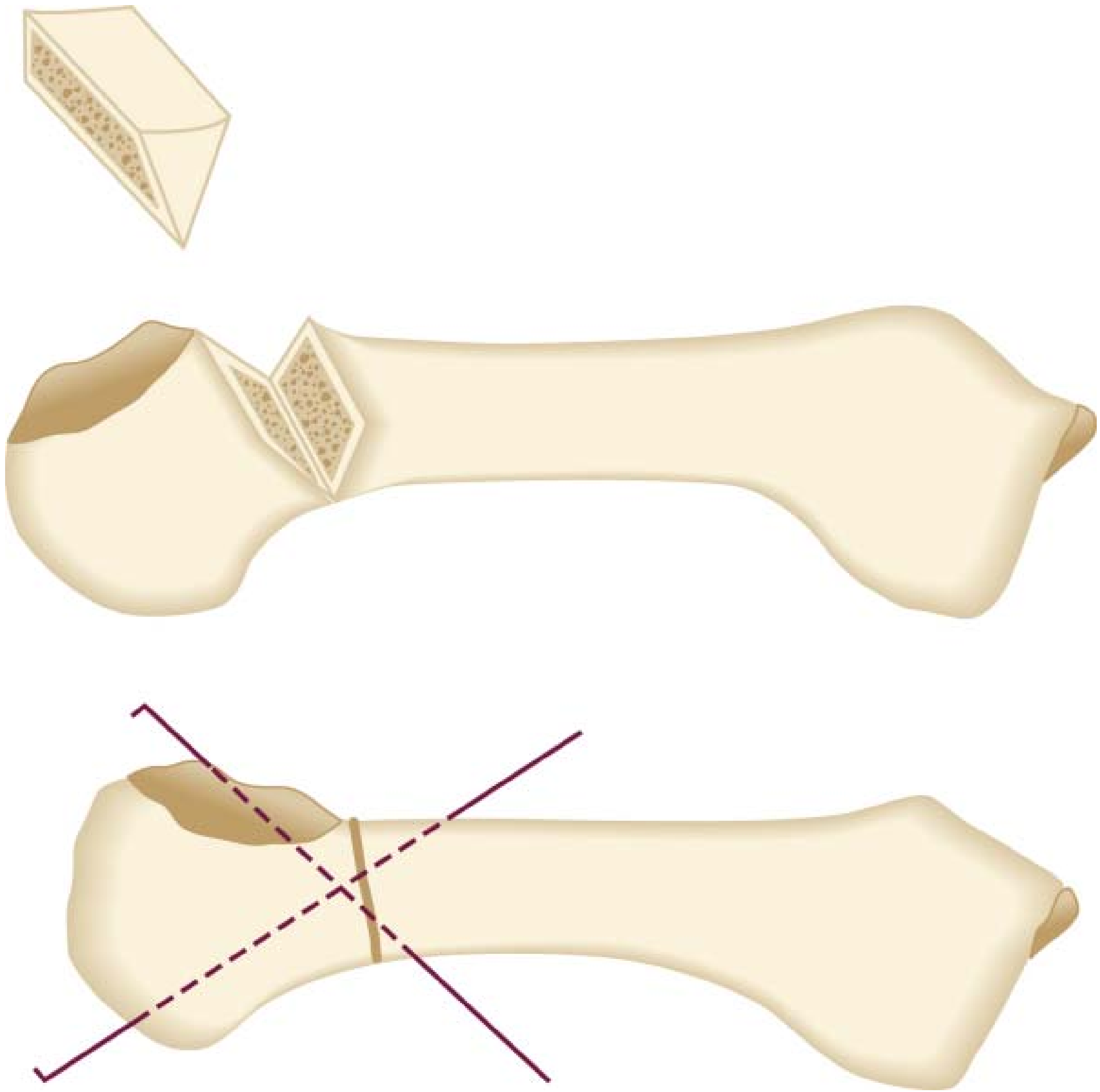

Figure 25I-30 Dorsal closing wedge osteotomy and cross-pinning for Freiberg infarction. *(Redrawn from Chao KH, Lee CH, Lin LC: Surgery for symptomatic Freiberg's disease: Extraarticular dorsal closing wedge osteotomy in 13 patients followed for 2 to 4 years. Acta Orthop Scand 70:483-486, 1999.)*

Iselin Disease

Iselin disease is a traction apophysitis of the base of the fifth metatarsal that occurs in late childhood or early adolescence at the time of the appearance of the proximal apophysis of the tuberosity of the fifth metatarsal. [161] This secondary ossification center is a small, shell-shaped fleck of bone oriented slightly obliquely with respect to the metatarsal shaft and located on the lateral plantar aspect of the tuberosity (Fig. 25I-31). It usually is not visible on anteroposterior or lateral radiographs but can be seen on an oblique view. Symptoms include tenderness over a prominent proximal fifth metatarsal and pain over the lateral aspect of the foot with weight-bearing. Participation in sports that require running, jumping, and cutting, which cause inversion stresses on the forefoot, is a common factor. [1815] [1816] The affected area over the tuberosity is larger than that on the noninvolved side, with soft tissue edema and local erythema. The area is tender to palpation at the insertion of the peroneus brevis, and resisted eversion and extreme plantar flexion and dorsiflexion of the foot elicit pain. Oblique radiographs show enlargement of the apophysis and often fragmentation of this secondary ossification center (Fig. 25I-32) and widening of the cartilaginous-osseous junction. Technetium-99m bone scanning shows increased uptake over the apophysis. Failure of the apophysis to fuse with the metatarsal (Fig. 25I-33) can cause symptoms into adulthood. Os vesalianum, a sesamoid in the peroneus brevis (Fig.

25I-34), must be distinguished from Iselin disease, and the nonunited apophysis should not be mistaken for a fracture.

Authors' Preferred Method

Treatment of Iselin disease is always nonoperative and includes limitation of activity, ice, and NSAIDs. If symptoms persist or are severe, a brief period of cast immobilization can be helpful. After resolution of acute symptoms, stretching exercises and range of motion exercises for the ankle and subtalar joints on a wobble board are initiated. Return to competition is allowed when discomfort is tolerable after activity. Plantar arch strapping or an arch support system placed in the shoe to elevate and relieve stress on the fifth metatarsal may allow athletic activity without pain.

Figure 25I-31 Fragmentation of secondary ossification center (Iselin disease) in a young athlete who complained of pain and swelling over the base of the fifth metatarsal.

Figure 25I-32 Enlargement and fragment of epiphysis in Iselin disease. *(From Canale ST: Osteochondrosis or epiphysitis and other miscellaneous affections. In Canale ST, Beaty JH [eds]: Campbell's Operative Orthopaedics, 11th ed. Philadelphia, Elsevier, 2008.)*

Figure 25I-33 Nonunion of fifth metatarsal as a result of Iselin disease. *(From Canale ST: Osteochondrosis or epiphysitis and other miscellaneous affections. In Canale ST, Beaty JH [eds]: Campbell's Operative Orthopaedics, 11th ed. Philadelphia, Elsevier, 2008.)*

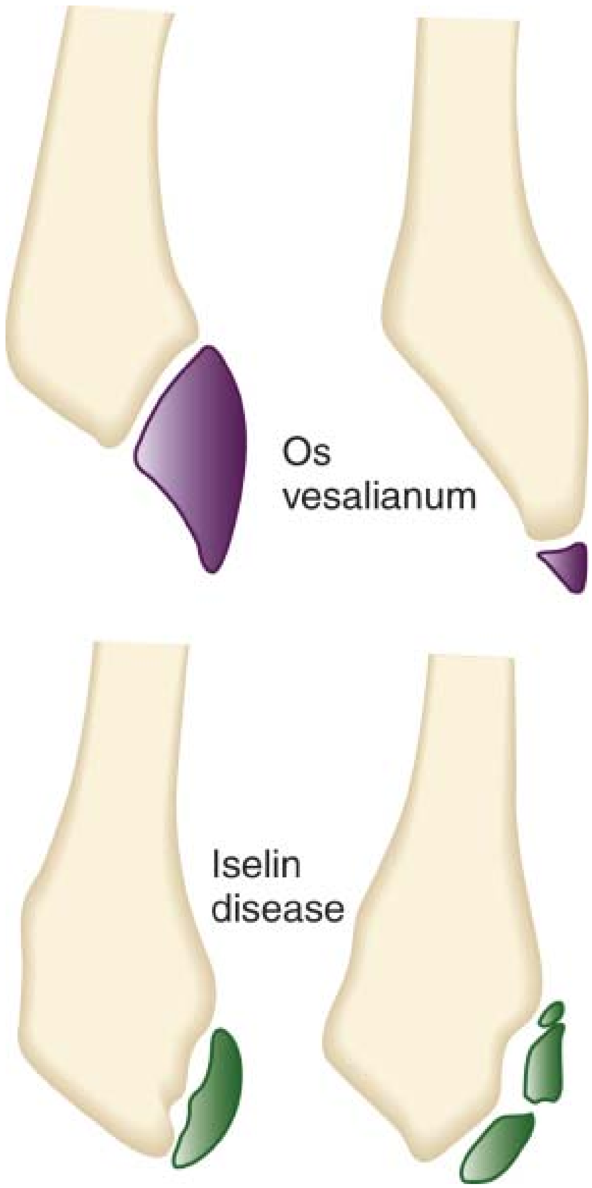

Figure 25I-34 Os vesalianum must be distinguished from Iselin disease. *(From Canale ST: Osteochondrosis or epiphysitis and other miscellaneous affections. In Canale ST, Beaty JH [eds]: Campbell's Operative Orthopaedics, 11th ed. Philadelphia, Elsevier, 2008.)*

Osteochondritis of the Sesamoids

Although small, seemingly insignificant bones of the foot, the sesamoids can cause disabling pain in an athlete. Galen, in 180 AD, named these small bones the sesamoids because of their resemblance to flat, oval sesame seeds. During the seventh or eighth week of embryonic development, both sesamoid bones of the hallux appear as islands of undifferentiated connective tissue between the first metatarsal heads. The fibular (lateral) sesamoid is present slightly sooner than the tibial (medial) sesamoid. Ossification begins at about 8 or 9 years of age in girls and 10 or 11 years of age in boys. There may be two or more centers of ossification, with the tibial sesamoid being bipartite at skeletal maturity in about 10% to 15% of the population; the fibular sesamoid rarely is bipartite. [1817] [1818] [1819] The two hallucal sesamoids are embedded in the tendons of the short flexor of the hallux and are held together by the intersesamoid ligament and the plantar plate, which inserts on the base of the proximal phalanx of the hallux (Fig. 25I-35). The sesamoids function to absorb weight-bearing pressure, reduce friction, and protect tendons. They are important to the dynamic function of the hallux and act as a fulcrum to increase the mechanical force of the flexor hallucis brevis tendon. The sesamoid complex normally transmits as much as 50% of body weight and during push-off may transmit loads of more than 300% of body weight. [167]

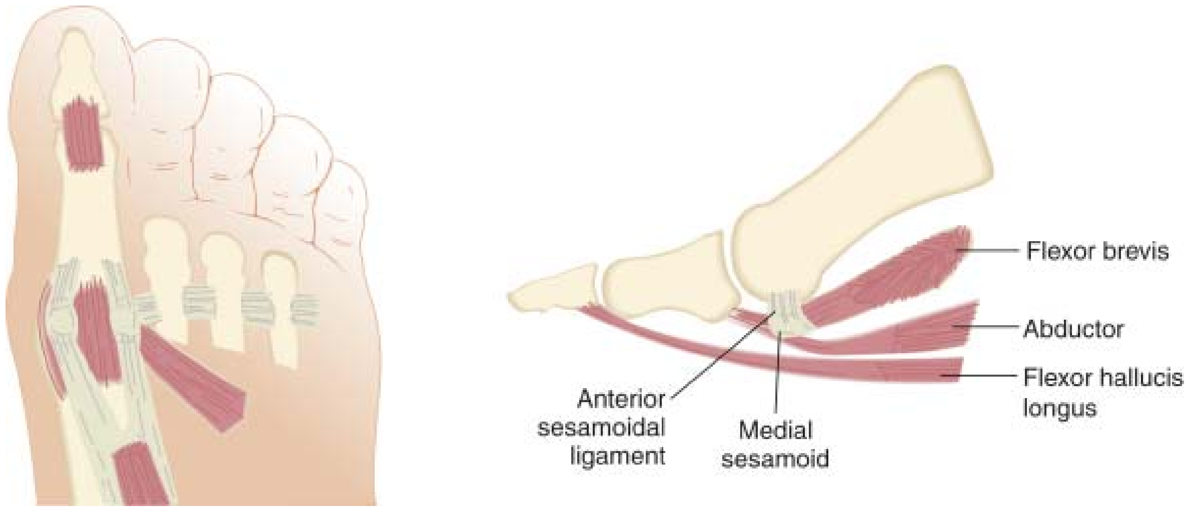

Figure 25I-35 Anatomy of the sesamoids. *(Redrawn from Leventen EO: Sesamoid disorders and treatment: An update. Clin Orthop 269:236-240, 1991.)*

Osteochondritis of the hallucal sesamoids is most common in young women but has been reported in nearly all age and gender groups from adolescence through adulthood. [164] Osteochondritis may occur as a primary pathologic entity or may be a late stage of repetitive stress injury involving osteonecrosis (Fig. 25I-36). Trauma is believed to the most frequent cause, but osteonecrosis with subsequent regeneration and excessive calcification may be present. The diagnosis of sesamoid injury constitutes a spectrum of abnormality rather than an isolated injury. [165] Depending on the stage at which the patient is first seen, the diagnosis may range from bursitis over the tibial sesamoid to the vague entity sesamoiditis, which encompasses both osteochondrosis and osteochondritis. In later stages, the diagnosis may include stress fracture, traumatic or degenerative arthritis, and chondromalacia. Hematogenous osteomyelitis of the sesamoid also should be included in the differential diagnosis, and sesamoid periostitis may occur in athletes with one of the rheumatoid variants, such as psoriasis, Reiter syndrome, and ankylosing spondylitis. Typically, patients have pain and tenderness to palpation over the involved sesamoid, without swelling or erythema that would indicate infection or bursitis. An axial radiograph or CT may show an enlarged or deformed sesamoid with irregular areas of increased bone density, mottling, and fragmentation. [168] Comparison views of the opposite foot are helpful to distinguish a bipartite sesamoid from a fracture.

AUTHORS' PREFERRED METHOD

Initial treatment is nonoperative, including NSAIDs, activity modification, and full-length shoe orthoses with a metatarsal pad and a relief beneath the first metatarsal pad. If no relief is obtained, a period of cast immobilization is tried. Persistent symptoms are an indication for sesamoid excision. Only the symptomatic sesamoid should be excised because of the likelihood of creating an intrinsic minus cock-up (hallux extensus) deformity with excision of both sesamoids. Even excision of a single sesamoid may result in hallux varus deformity, exacerbation of hallux valgus, or overload of the metatarsal head. Removal of both sesamoids results in about a 30% decrease in flexion strength of the hallux. [1822] [1823] Return to athletic activity usually is possible at 6 weeks after excision of one of the sesamoids; competitive sports are not allowed for 12 weeks after excision of both sesamoids.

CRITICAL POINTS

- Important differences exist related to the mechanism of injury for medial and lateral talar dome lesions, and these must be recognized and considered in choosing treatment methods.
- Most OLT, including posterior ones, can be treated arthroscopically, but accessory portals often are required.

- The shorter the interval between the diagnosis of MRI stage II and IV OLT and operative treatment, the better the results.
- OLT and fractures of the talar and calcaneal processes can be mistaken for ankle sprains and should be suspected when there is an inability to bear weight on the foot and ankle, severe swelling, or continued pain 3 to 4 weeks after “ankle sprain.”
- Small, minimally displaced fractures of the talar and calcaneal processes usually can be successfully treated nonoperatively, but larger fragments (>1 cm) or displaced fragments (>2 mm) generally require open reduction and internal fixation.
- Ankle impingement syndromes usually occur after an inversion ankle injury or repetitive ankle flexion to extremes (e.g., sprinters, ballet dancers) and can be caused by bony lesions (i.e., osteophytes, os trigonum, Stieda process) or soft-tissue lesions (i.e., thickened ATFL [Basset ligament], deltoid ligament injury, tenosynovitis, meniscoid lesion).
- Pain with palpation and dorsiflexion suggests anterior impingement (most common), whereas pain with forced plantar flexion is indicative of posterior impingement.
- Initial treatment of ankle impingement is nonoperative; if symptoms persist, arthroscopic treatment (débridement, synovectomy, excision, decompression) is successful in 90% to 95% of patients.
- Sever, Köhler, Buschke, Freiberg, and Iselin diseases; accessory navicular; and osteochondritis of the sesamoids are all relatively rare conditions that may be confused with traumatic injury; initial treatment is always nonoperative, which is successful in most patients.

Figure 25I-36 **A,** Osteochondrosis of the tibial (medial) sesamoid in a runner. **B,** Note fragmentation of sesamoid.

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SECTION J Etiology of Injury to the Foot and Ankle

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The etiology of injury to the foot and ankle is of utmost importance in sports medicine. The capacity to recognize the injury-causing conditions and the ability to control them may help prevent these same injuries. Research has shown that when etiologic factors are addressed, it is possible to decrease injury rates significantly. [1825] [1826] [1827]

This chapter reviews predisposing conditions for injury of the foot and ankle and the variables that may have a protective value. The primary areas of importance are (1) joint flexibility, (2) shoewear, and (3) the quality of the playing surface.

Injury variables can be divided into intrinsic and extrinsic factors. [1828] [1829] [1830] Intrinsic factors include both an athlete's individual physical and personality characteristics. The physical characteristics include age, sex, genotype, somatotype, strength, speed, agility, coordination, fitness, flexibility, malalignment, muscle composition, previous injury, and residual structural inadequacies. [1828] [1829] [1831] [1832] [1833] [1834] [1835] [1836] [1837] [1838] [1839] [1840] [1841] [1842] [1843] [1844] [1845] [1846] [1847] [1848] [1849] [1850] [1851] [1852] [1853] [1854] [1855] [1856] [1857] [1858] [1859]

Personality characteristics such as extroversion, anxiety level, conscientiousness, self-confidence, determination, responsiveness to coaching, discipline, dependency, sensitivity, and others may play a role in whether an individual is injury prone. [1828] [1841] [1850] [1860] [1861] [1862] [1863] [1864] [1865] The contribution of these variables constitutes the individual athlete's intrinsic risk for injury.

Extrinsic factors include the kind of sports activity, the training methods, the knowledge and skill of the coach, the level of competition, the environmental conditions, the type of shoewear used, the playing surfaces, and the equipment. [1828] [1829] [1832] [1833] [1834] [1842] [1846] [1850] [1851] [1852] [1856] [1866] [1867] [1868] [1869] [1870] [1871] [1872] [1873] [1874] [1875] [1876] [1877] [1878] [1879] [1880] [1881] [1882]

There is, and always has been, an inherent risk for injury associated with athletic endeavors. The kind of athletic activity can further be separated into high, medium, or low risk for foot and ankle injuries (Box 25J-1). [1883] [1884] [1885] In addition, each sport poses a different risk to a particular body part. For example, running carries a low risk for shoulder injuries but a high risk for foot or knee injuries. Conversely, swimming may pose a high risk for shoulder injuries but a low risk for foot and ankle injuries. Certain other variables play a role in the risk for injury, including playing time, level of competition, the movements required, equipment used, individual versus team play, the level of fitness, and the enforcement of the rules and regulations. [1884] [1886] [1887] Collins and colleagues examined athletes at 100 U.S. high schools and found that nationally 6.4% of all high school sports–related injuries were related to breaking of the rules of the sport. [64]

Practice methods also have a definite impact on injury statistics. [1834] [1869] [1889] [1890] In competitive running, training techniques such as high-mileage workouts, excessive use of plyometrics, and interval work have been implicated in the incidence of overuse problems. [1830] [1891] [1892] [1893] Certain factors frequently lead to overstress: (1) when unprepared athletes are asked to cross-train with long-distance running for such sports as swimming, basketball, or volleyball; (2) when athletes are out of shape because it is early in the season; and (3) when it is the transition period between seasons when a player shifts from one sport to another without recognizing the different stresses introduced by the variation in performance requirements, shoes, and playing surfaces involved. Running programs that involve excessive mileage or, more commonly, "too much too soon" are responsible for stress fractures or other overuse syndromes. [1832] [1852] Taunton and coworkers found that being active for less than 8.5 years was positively associated with injury in both sexes for tibial stress syndrome; and women with a body mass index of less than 21 kg/m [2] were at a significantly higher risk for tibial stress fractures. [70] A lack of stress adaptation frequently results in overload problems and injury.

Other training techniques may increase an athlete's risk for injury. Plyometric training is promoted to improve jumping ability and speed [71] ; however, such exercises as bench jumping and bounding can overload the system. [72] This is particularly true when stress accommodation is not allowed and when shoes and surfaces are inappropriate for these activities.

Stress tolerance varies widely from individual to individual. This must be recognized in the design of preseason, in-season, and off-season training programs. Unnecessary exposure to injury can be attributed to coaching methods. Uncontrolled scrimmages and excessive contact work increase injury rates in football. [10] Gymnasts who perform complex maneuvers without adequate spotting or preparation have a higher risk for injury. [22] Baseball and softball players who practice sliding into fixed bases are unnecessarily exposed to foot and ankle trauma (Fig. 25J-1). [1871] [1872] Finally, the “no pain, no gain” coaching philosophy can pressure an athlete to return to play after an injury without adequate rehabilitation and increase the potential for injury: an ankle syndesmosis injury treated functionally as a typical lateral ankle sprain can worsen considerably. In these times of astronomical salaries for professional athletes, there is increasing emphasis on players returning to sport after injury in the shortest amount of time possible.

The environment may affect injury risk: weather conditions can alter the playing surface, the shoes worn, the interface characteristics between shoe and surface, and the attitudes of players and coaches. The complex interplay between extrinsic and intrinsic elements makes statistical analysis difficult from a precise epidemiologic perspective. As scientists, we must recognize that injury statistics are only as good as the integrity of the investigator, the statistical methods, and the research design. [1897] [1898] [1899] [1900]

The use of performance-enhancing drugs and nutritional supplements has been rearing its ugly head for more than a century. [1901] [1902] In 1886, Arthur Linton, a 24-year-old Welsh cyclist, died during a race from Bordeaux to Paris. He was believed to have taken the stimulant trimethyl. Shortly thereafter, Charles Edouard Brown-Séquard, in 1889, extracted testosterone in dogs and injected himself with the extract, claiming that it made him feel younger. From 1940 to 1945, the Germans tested anabolic steroids in prisoners, and Hitler himself may have used them. Some believe his behavior late in life exhibited the characteristics of steroid use: aggressiveness and violent behavior with bouts of depression and suicidal ideation. In 1954, the Soviet weightlifting team dominated the sport, and their team doctors revealed the use of injected testosterone. At the 1976 Montreal Olympics, the East German women’s swim team dominated with 11 of 13 individual gold medals, setting 8 world records in the process. Much later, the German team coaches admitted to steroid use. Based on the lawsuits filed in German court, it is believed that up to 2000 of the East German athletes who have used anabolic steroids are now suffering from serious health problems, including liver tumors, heart disease, testicular and breast cancer, infertility, eating disorders, depression, and birth defects. The list of world-famous athletes who used, or are accused of using, performance enhancing drugs continues to grow: Canadian sprinter Ben Johnson, NFL defensive end Lyle Alzado, baseball’s 1996 National League MVP winner Ken Caminiti (who died of a heart attack at age 41 years), Major Leaguers Barry Bonds and Jose Canseco, Tour de France Winner Floyd Landis, Sydney Olympic Champion Marion Jones—who had her five medals stripped by the International Olympic committee, and most recently Roger “The Rocket” Clemens. [78] Unfortunately, with the desire to win, sometimes at all costs, this list of famous athletes may continue to grow. Quite clearly, there are negative effects associated with the use of performance-enhancing drugs. They are illegal, constitute an unfair advantage, and place the athlete at an unnecessary and serious risk. [1901] [1903] [1904]

Injury risk is a natural and accepted part of sports participation. Different sports with different performance factors naturally provide varying degrees of exposure to injury. [1850] [1884] The broad categories of analysis in the injury equation are intrinsic and extrinsic factors. For the foot and ankle, the primary areas of importance are joint flexibility, shoewear, and the quality of the playing surface. These can be related to a classification of sports activities based on six basic motions: (1) stance, (2) walking, (3) running, (4) jumping, (5) throwing, and (6) kicking. [1846] [1905] Running, jumping, and kicking seemingly pose the greatest risk for injury to the foot and ankle. By using these types of categorization and classification, combined with scientific analysis of biomechanical stresses, it may be possible to analyze injury potential more accurately and to design effective prevention strategies.

BOX 25J-1

HIGH-, MEDIUM-, AND LOW-RISK SPORTS FOR FOOT AND ANKLE INJURY

High Risk

- Ballet
- Basketball
- Dance
- Ice skating
- Mountaineering
- Running
- Skateboarding

- Snowboarding
- Soccer

Medium Risk

- Aerobics
- Baseball
- Football
- Gymnastics
- Ice hockey
- Lacrosse
- Racquetball, squash
- Roller skating
- Rugby
- Tennis
- Volleyball
- Water-skiing

Low Risk

- Archery
- Boating
- Bowing
- Cycling
- Equestrian
- Fishing
- Golf
- Parachuting
- Rodeo
- Skiing
- Weight training
- Wrestling

Adapted from Sports Injuries. Accident Facts. Report of the National Safety Council, 1990, p 88; and from Table 26-1 in Clanton TO: Athletic injuries to the soft tissues of the foot and ankle. In Coughlin MJ, Mann RA (eds): Surgery of the Foot and Ankle, 7th ed. St. Louis, Mosby, 1999.

Figure 25J-1 A, Potential for injury created by sliding into a fixed base. B, A 15-year-old boy who injured his ankle sliding into second base. C, Distal fibula fracture of 15-year-old male baseball player. *(A, Photograph courtesy of Rice University Sports Information Office; B, photograph by Christopher B. Hirose.)*

INCIDENCE

Injuries to the lower extremities account for most sports injuries. [82] Between 55% and 90% of all sports injuries occur from the hip region down to the toe. [83] Running, jumping, and kicking sports are associated with injuries to the foot and ankle. When combined with cutting and sliding maneuvers, the injury statistics increase dramatically. It has been suggested that the sprained ankle is the single most common injury in sports, and many ankle sprains go unreported and untreated (Fig. 25J-2). [1908] [1909] [1910] [1911] [1912] [1913] Nevertheless, epidemiologic analysis of sports injuries occurring around the world indicates an overwhelming preponderance of injuries to the lower extremities, and most of these are sprains, strains, and contusions. [1870] [1908] [1910] [1911] [1913] [1914] [1915] [1916] [1917] [1918] [1919] [1920]

Figure 25J-2 Ankle ligamentous examination at the University of Texas. *(Photograph by Thomas O. Clanton.)*

Studies of sports-related injuries in the running and jumping sports have suggested an incidence of injury of 10% to 15% for the ankle and 3% to 15% for the foot. [1910] [1914] [1916] [1917] [1920] In their study of more than 12,000 injuries occurring in 19 different sports in a university sports clinic, Garrick and Requa found that 25% of these injuries involved the foot and ankle. [86] Patients participating in running, tennis, and dance were seen most frequently in this clinic setting. Kannus and colleagues reported on the injuries seen in their European sports medicine clinic, where the most common sports were soccer, running, and orienteering. [92] Ankle injuries accounted for 9% of the visits to the clinic during a 6-month period. Achilles tendon problems and heel pain accounted for another 7%. DeHaven and Lintner, from the University of Rochester Section of Sports Medicine, surveyed injuries seen over a 7-year period, including patients from unorganized sports all the way through the professional level. [90] Of the 3431 cases studied, 12% involved the ankle, and 3% involved the foot. The three sports most commonly producing injury in this clinical setting were football, basketball, and soccer. In 1980, Zaricznyj and associates reported a thorough analysis of the causes and severity of sports injuries in a population of school children from elementary through high school. [96] This study documented all injuries in a population of 25,512 school children. Of these, 11.4% occurred to the ankle. The foot and toes were injured in 6%.

A thorough analysis of the incidence of injury in different sports reveals the magnitude of the problem. Soccer claims anywhere from 40 to 120 million participants worldwide and about 13 to 16 million in the United States. [1921] [1922] It has an injury rate of 22% to the ankle and 8% to the foot. [82] Football involves an estimated 17,400,000 participants in the United States. [98] The overall likelihood of player injury in football has been estimated to be anywhere from 10% to 80% of participants. Combining these numbers with the estimated injury rate to the foot and ankle of 15% to 25%, the total injuries to the foot and ankle in football in this country range from a low of about 261,000 to a high of 3,480,000. [1833] [1923] [1924] [1925] [1926] [1927] The estimated injury rates for the foot and ankle in other sports are listed in Table 25J-1 . When combined with estimates of participation numbers in those sports, [98] the magnitude of the athletic injury problem to the foot and ankle and the need for better prevention become evident. Whether the sport is a team or individual sport, organized or unorganized, professional or nonprofessional does not appear to make a major difference in the types of foot and ankle injuries that occur and has only a minor influence on the overall injury rate. This suggests that the intrinsic movements and extrinsic load characteristics of the foot and ankle required by sports participation involve a certain basic risk for injury.

TABLE 25J-1 -- Injury Rates Calculated for the Foot and Ankle in Various Sports from a Review of the Literature

Sport/Study	Skill Level	Ankle Injury (%)	Foot Injury (%)
Aerobics			
Rothenberger	Recreational	12	5
Garrick	Recreational	11	18
Ballet			
Garricki	Professional and nonprofessional	17	22
Sohl	Review of literature	14	15
Baseball			
Garfinkel	Professional	19	4
Basketball			
Zelisko	Professional	19	4
Henry	Professional	18	6
Moretz	High school	31	8
Cycling			
Davis	N/A	F&A	8
Kiburz	Club	F&A	14
Dance (general)			
Washington	Various levels	17	15
Rovere	Student	22	15
Equestrian			
Bermhang	Top class	F&A	13
Bixby-Hammett	Top class	F&A	6
Football			
Blyth	High school	15	2
Culpepper	High school	11	4
DeLee	High school	18	2
Zemper	College	16	4
		AE = 1	AE = 0.25
Canale	College	11	2
Golf			
McCarroll	Professional	2	3
McCarroll	Amateur	3	2
Gymnastics			
Caine	Club	21	3
Garrick	High school	10	8
Ice hockey			
Sutherland	Amateur	0	0
	High school	0	0
	College	7	10
	Professional	0	0

Sport/Study	Skill Level	Ankle Injury (%)	Foot Injury (%)
Park	Junior	4	1
Lacrosse			
Muller	College	15	4
Nelson	College	14	4
Mountaineering			
McLennan	N/A	41	8
Tomczak	N/A	40	35
Parachuting			
Petras	Military	7	0.3
Rodeo			
Meyers	College	6	1
Roller skating			
Ferkel	College	10	N/A
Perlik	N/A	8	2
Rugby			
Micheli	College/club	8	2
Running			
Cottlieb	Recreational	19	11
Walter	Recreational	15	16
Temple	N/A	26	26
Marti	N/A	30	10
Ice skating			
Brown	National males	8	8
Smith	Age 14-19 yr	29	8
Skiing			
Downhill			
Johnson	Various	9	N/A
Blitzer	Youth	F&A	8
Freestyle			
Dowling	USSA	8	N/A
Snowboarding			
Pino	Recreational	26	3
Soccer			
Ekstrand	Swedish senior male division	17	12
Nilson	Various	36	8
Bareger-Vachon	Amateur leagues (France)	20	N/A
Squash/racquetball			
Berson	Recreational	21	2
Soderstrom	N/A	20	7
Tennis			
Winge	Elite	11	9

Sport/Study	Skill Level	Ankle Injury (%)	Foot Injury (%)
Volleyball			
Schafle	National amateur	18	6
Water skiing			
Hummel	N/A	4	15
Weight training			
Kulund	Elite, Olympic	2	0
Wrestling			
Roy	College	10	3
Lok	Olympic	10	0
Snook	College	4	0
Requa	High school	3.8/100 wrestlers	

From Clanton TO: Athletic injuries to the soft tissues of the foot and ankle. In Coughlin MJ, Mann RA (eds): Surgery of the Foot and Ankle, 7th ed. St Louis, Mosby, 1999, pp 1093-1094.

AE, number of injuries per athletic exposure; F&A, foot and ankle; N/A, not available; USSA, United States Ski Association.

FLEXIBILITY AND STIFFNESS

Flexibility is one of the components of physical fitness that has been judged to be critical for injury-free performance. [1928] [1929] Its effect on injury to the foot and ankle has been discussed primarily in relation to (1) ankle joint stiffness affecting the incidence of ankle sprains and (2) flatfeet or hyperpronated feet as a source of injury or pain.

Definitions

Flexibility is the range of motion commonly present in a joint or group of joints that allows normal and unimpaired function. [1930] [1931] It can be subdivided into static and dynamic flexibility. Static flexibility is the maximal range of motion that a joint can achieve with an externally applied force, such as gravity. Dynamic flexibility is the range of motion that an athlete can produce and the speed at which he or she can produce it. Dynamic flexibility is important in high-velocity movement sports such as throwing, sprinting, or jumping. [1928] [1931] Having excellent static flexibility does not mean that one possesses excellent dynamic flexibility. Beighton and coworkers noted that articular range of motion is a spectrum, where one end of the spectrum includes considerable joint laxity. [108] When flexibility exceeds the normal range of movement in multiple joints, an individual is considered loose-jointed or hypermobile. Hypermobility has been shown to have multiple inheritance patterns and can occur in both benign and pathologic forms. [1933] [1934] [1935] [1936] [1937] Box 25J-2 shows characteristics of joint mobility patterns and changes with age and gender and ethnicity.

BOX 25J-2

FLEXIBILITY GENERALIZATIONS

- Inherited characteristic
- Individual variability
- Joint specific
- Females more flexible then males
- Decreases with age
- Can be acquired through training
- Strength training does not necessarily reduce flexibility
- Little relationship with body proportion or limb length

- Little relationship with injury rate

Data from references 13, 107, 108, 116, 118, 121, 126, and 127.

Stiffness is the physical measurement of a reduced range of motion of a joint or a group of joints. The loss of flexibility during rapid growth in children causes both muscle tendon imbalance and increased apophyseal traction. Resultant overuse injuries include traction apophysitis and tendinitis. Sever's disease is a common cause of heel pain in adolescents.

Little discussion in the medical literature centered on hypermobility until it was proposed as a heritable cause of joint problems by Finkelstein in 1916. [114] Connective tissue diseases such as Ehlers-Danlos syndrome, Marfan syndrome, Larsen's syndrome, Down syndrome, hyperlysinemia, homocystinuria, and osteogenesis imperfecta are known to be associated with joint hypermobility and resultant subluxation and dislocation of joints, although this rarely affects the foot and ankle joints. [1935] [1939] [1940] [1941]

Hypermobility is a trademark of certain sports such as diving, gymnastics, and ballet. Ballet dancers are particularly noted for excessive motion in their spine, hips, and ankles. [118] To a certain degree, individuals with these traits of flexibility are selected through the intense training that is required in competitive dancing. It seems that inherited hypermobility gives these individuals an advantage during the training years. [118] There is also a negative side: studies have shown that hypermobile dancers have a higher incidence of injuries than those who are not hypermobile. [1889] [1943]

Although Nicholas cited hypermobility as a causative factor in ligamentous injuries in athletes in 1970, [120] this relationship was not confirmed in follow-up studies. [1862] [1945] [1946] [1947] The connection between hypermobility, particularly when it is familial, and dislocation of one or more joints is relatively well established, but it has not included the other joints of the foot and ankle. [1940] [1945] The occurrence of joint effusions in the knees and ankles in the absence of trauma or other known inciting causes was attributed to hypermobility by Sutro in 1947. [124] Additionally, there have been some reports suggesting an association between hypermobility and osteoarthritis. [1940] [1942] [1945] [1949] Although the presence of flatfeet is frequently included among the characteristic findings of individuals who are hypermobile, it is quite clear that not all individuals with flatfeet are hypermobile. [1950] [1951] There is considerably less evidence implicating hypermobility with other pathologic conditions of the foot and ankle.

Criteria for the diagnosis of hypermobility were first proposed by Carter and Wilkinson [128] and later modified by Beighton and colleagues. [108] The tests commonly used include (1) passive thumb apposition to touch the forearm, (2) passive little finger hyperextension of more than 90 degrees, (3) elbow hyperextension of more than 10 degrees, (4) knee hyperextension of more than 10 degrees, and (5) forward flexion of the trunk with the knees straight and the palms of the hands resting flat on the floor (Fig. 25J-3). Hypermobility is diagnosed in individuals who can perform three or more of these tests. [117]

Figure 25J-3 Tests for hypermobility. A, Passive thumb apposition to touch forearm. B, Passive little finger hyperextension. C, Forward flexion of the trunk so that palms rest on the floor. *(Photographs by Christopher B. Hirose.)*

The distinction between hypermobility, or laxity, and instability is an important one. Hypermobility is normal movement carried beyond the range found in most individuals. This hypermobility is primarily a function of the stiffness within the muscles, ligaments, and tendons coupled with the bony configuration of the joint. Instability is a symptom-producing phenomenon that is related to the ligamentous and bony integrity of joint as well as compressive joint forces and neuromuscular control mechanisms and their opposition to the forces of shear, distraction, and angulation. Instability may be further subdivided into functional and mechanical instability.

The idea of functional instability as proposed by Freeman is "… the occurrence of recurrent joint instability and the sensation of joint instability due to the contributions of any neuromuscular deficits." [1825] [1953] Such deficits would primarily be related to injury to the joint mechanoreceptors and afferent nerves resulting in combinations of impaired balance, reduced joint position sense, and slower firing of the peroneal muscles in response to inversion stress.

Mechanical stability, in contrast, is defined as "laxity of a joint due to structural damage to ligamentous tissues which support the joint." [129] Structural damage also includes damage to the bony support of the joint. This type of instability is not always evident on physical examination. The varus stress test applied to the ankle to produce a talar tilt demonstrating lateral ligamentous injury at the ankle can be difficult to interpret in patients who have increased subtalar motion. Adding to the difficulty of such an assessment, as of this writing, there are no scientifically based anatomic and biomechanical definitions that are universally accepted.

Historical Perspective on Flexibility

Hypermobility is often seen in athletes and may be more frequently symptomatic than in the nonathletic population. This is likely due to the added stress of sports participation. According to Grahame, Hippocrates first mentioned hypermobility as a source of difficulty for athletes as early as the 4th century BC. [116]

The importance of flexibility in athletic performance and the prevention of injury are of rather recent origin according to Corbin. [107] The immobility caused by wartime injuries, together with the epidemic of poliomyelitis, instigated research efforts in this field in the past century. Cureton emphasized flexibility as an important component of physical fitness as early as 1941 after his work with swimmers during the 1932 Olympic games. [1954] [1955] Kraus' work led to the formation of the President's Council on Physical Fitness and Sports and fostered much of the subsequent research on flexibility. [107] In a classic work, DeVries proved the value of passive stretching in improving flexibility. [1956] [1957] Furthermore, the work of Salter and associates in Toronto has shed new light on the importance of maintaining flexibility and motion postoperatively in patients who have undergone musculoskeletal procedures. The benefits of continuous passive motion to the joints and to the surrounding musculotendinous and ligamentous structures are well established. [1958] [1959] [1960]

Although the natural inclination and the accepted teaching for many years in the fields of sports medicine and exercise physiology has been that stretching is a preventive measure for athletic injury, there is little conclusive epidemiologic evidence to support this idea, and studies have been contradictory and inconclusive. [1869] [1961] [1962] Two well-designed studies of running-related injuries failed to show a significant relationship between stretching or its absence and the frequency of injury. [1856] [1875] Conversely, a study of military basic trainees showed a reduction in overuse injures with an effective hamstring flexibility program. [139] Research from Duke University has provided scientific groundwork on the preventive aspects of stretching and warm-up periods by showing that greater tension is required to rupture a muscle that has been stretched. [1964] [1965] There is the possibility that a degree of tightness protects against injury when joints are stressed. This implies that stretching beyond a certain point may reduce the load-sharing ability of the musculotendinous units or the capsuloligamentous complex, which are responsible for joint stability. [137] Ingraham believes that the current evidence suggests that stretching and increasing the range of motion beyond function are not beneficial and may actually cause injury and decrease performance. [142] If this is indeed the case, excessive stretching or hypermobility could result in increased stress on the ligaments, bone, and cartilage at the joint, leading to injury or arthritis. [1965] [1967] [1968] This may be the situation when ballet dancers force ankle plantar flexion to such an extent that it creates posterior impingement symptoms and reactive bone formation (Fig. 25J-4). [1889] [1943] [1969] [1970] [1971]

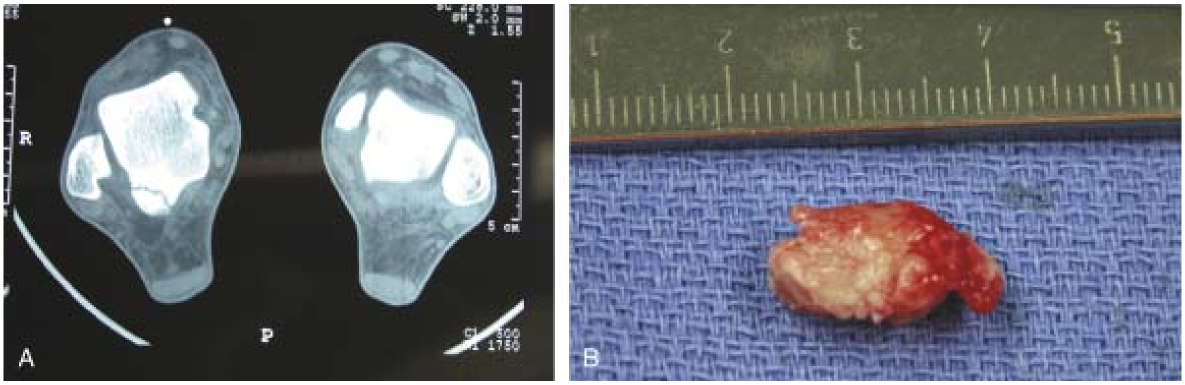

Figure 25J-4 A, Computed tomographic scan demonstrating bony posterior impingement. B, Excised bony fragment. *(Photographs by Christopher B. Hirose.)*

The balance between adequate and excessive stretching is further demonstrated in studies of runners. Research has shown that during level running a great deal of energy is stored in the muscle-tendon unit. [1837] [1972] Several laboratories have shown that less flexible individuals use less oxygen to cover the same distance while running at the same speed than more flexible individuals. [149] This interesting fact may explain why runners as a group are relatively inflexible unless specific stretching exercises are pursued. The importance of muscle stiffness in athletic injury remains an area for continued study because there is no scientifically based program for flexibility training with statistically reproducible results in lowering injury rates or improving performance. [13]

In summary, there appears to be an optimal range of motion for each individual and for each of his or her joints that may be sport specific. Warming up and stretching to obtain or maintain this range may or may not prevent injury, but stretching beyond this range is potentially harmful. Certain sports require a particular range of motion and flexibility. Athletic activity and the competitive process tend to select naturally those who can attain the movement criteria with the most efficiency and without a subsequent increase in injury rate. [150]

Joint Motion

We define joint motion based on the position of the human body in relation to the three cardinal planes: the sagittal, frontal, and horizontal planes (Fig. 25J-5 and Table 25J-2). [151] This system considers pronation and supination as a triplane motion. Pronation consists of eversion, abduction, and dorsiflexion, and supination consists of inversion, adduction, and plantar flexion. [1975] [1976] It is apparent that motion in almost all joints and specifically in the ankle, subtalar, and transverse tarsal joints is actually a triplane motion to one degree or another. [1976] [1977] [1978] [1979] [1980] As an example, dorsiflexion of the ankle produces some degree of foot abduction as well as external rotation. Similarly, plantar flexion produces some adduction and internal rotation.

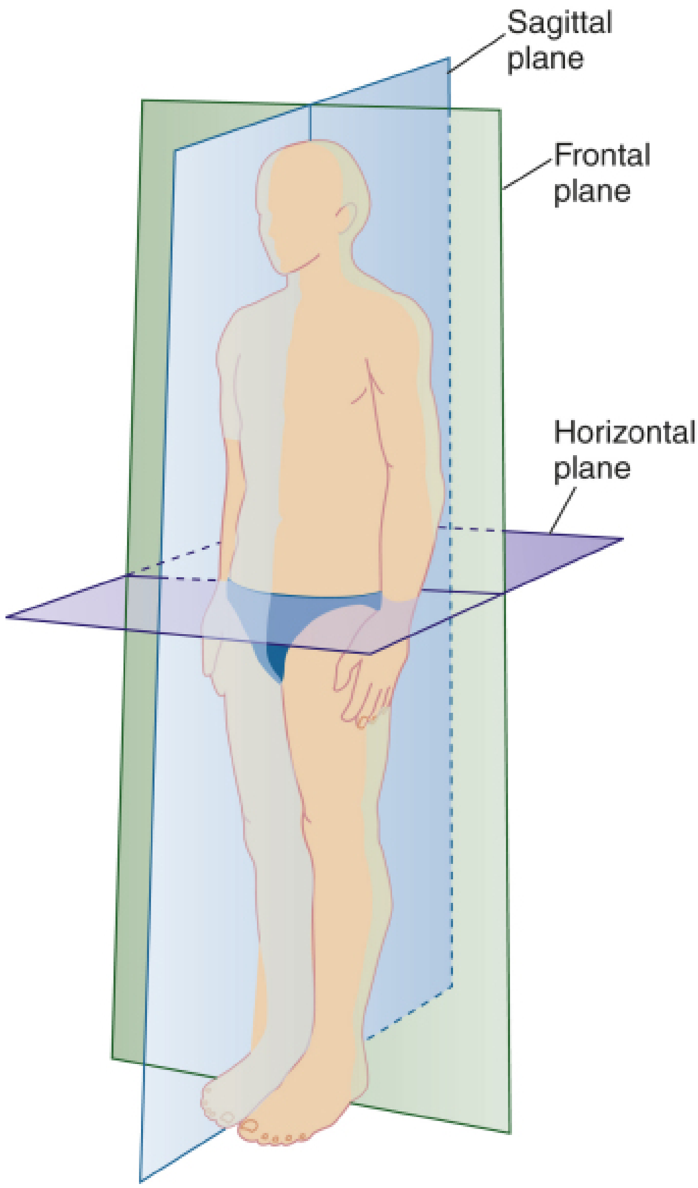

Figure 25J-5 The cardinal planes of motion. *(Redrawn with permission from Women in Sports. Sport Research Review. Beaverton, Ore, Nike Sport Research Laboratory, Mar/Apr, 1990; and Oatis C: Biomechanics of the foot and ankle under static conditions. Phys Ther 68:1815-1821, 1988.)*

TABLE 25J-2 -- Terminology Used for Motion, Instability, and Deformity

Plane	Motion	Position	Deformity
Sagittal	Dorsiflexion	Dorsiflexed	Calcaneus
	Plantar flexion	Plantar flexed	Equinus
Frontal	Inversion	Inverted	Varus
	Eversion	Everted	Valgus
Transverse	Adduction	Adducted	Adductus
	Abduction	Abducted	Abductus
Triplane description	Pronation	Pronated	Pronatus
	Supination	Supinated	Supinatus

There are different norms for range of motion of the ankle, subtalar, and first metatarsophalangeal joints (Tables 25J-3 to 25J-5 [0230] [0235] [0240]). This is due to the degree of anatomic constraints around these joints for each individual. A review of the literature shows that the average ankle range of motion is calculated as 53 degrees, with average dorsiflexion equaling 18 degrees and average plantar flexion equaling 35 degrees. [1979] [1980] [1981] [1982] [1983] Variability in ankle motion measurements is introduced depending on the methodology of measurement: radiographic or clinical using flexometry, goniometry, or electrogoniometry; selection of landmarks; and measurement of specific tibiotalar movement or combined ankle-foot motion. [155] Timing with regard to a warm-up period and geographic consideration may also be important. [1984] [1985] [1986]

TABLE 25J-3 -- Ankle Joint Motion [*]

Study	Method	Dorsiflexion (Extension) (degrees)	Plantar Flexion (degrees)	Total (degrees)
AAOS[159]	NS	20	50	70
Bonnin[406]	NS	10 to 20	25 to 35	35 to 55
Boone and Azen[158]	A	12.6 ± 4.4	56.2 ± 6.1	66.8 ± 5.5
Sammarco[175]				
WB	P	21 ± 7.21	23 ± 8	44 ± 7.6
NWB	P	23 ± 7.5	23 ± 9	46 ± 8.25
Weseley[169]	P	0 to 10 (max 23)	26 to 35 (min 10) (max 51)	26 to 45 (min 51) (max 84)
Lundberg[155]				
Review/summary	NS	13 to 33	23 to 56	36 to 89
Personal study	NS	24.9 ± 3.0	28.5 ± 7.5	53.4 ± 5.25
AMA[407]	A or P	20	40	60

A, active; NS, not specified; NWB, non-weight-bearing; P, passive; WB, weight-bearing.

* When a range is given, it signifies the range of greater concentration.

TABLE 25J-4 -- Subtalar Joint Motion

Study	Inversion (degrees)	Eversion (degrees)		Arc (degrees)
AAOS[157]	5	5		10
Inman[154]	—	—		44 ± 7
Sammarco[175]	20	5		25
DeLee[408]	—	—		35
McMaster[409]	25	5		30
Brantigan[410]	—	—		38 ± 6
James[251]	23 ± 6	8 ± 4		31 ± 7
Method	**R**	**L**	**R**	**L**
ASOS	2.0 ± 7.4	31.2 ± 7.7	3.9 ± 4.1	4.0 ± 4.2
Cailiet	21.4 ± 5.4	21.6 ± 5.5	3.4 ± 3.1	3.2 ± 3.3
James	18.4 ± 5.2	18.4 ± 5.2	6.4 ± 3.7	6.7 ± 4.2

TABLE 25J-5 -- First Metatarsophalangeal Joint Motion

Study		Dorsiflexion (degrees)		Plantar Flexion (degrees)
AAOS [157]		70		45
AMA [407]		50		30
Sammarco [175]		90		30
Joseph [176]	Standing	Active	Total	
	16	51	74	23
		(40 to 100)		(3 to 43)
Clanton [173]		72.3 ± 12.4		35.0 ± 10.2

As one moves distally, range of motion of the joints of the foot and ankle becomes increasingly difficult to measure objectively. This is particularly true of subtalar motion, for which numerous methods of measurement have been described. No consensus for normal motion has been reached (Fig. 25J-6 ; see Table 25J-4). This absence of a consensus creates considerable difficulty when trying to determine whether or not subtalar instability exists. [163] It is clear that there is intersubject variability in measuring subtalar motion. At the current state of knowledge, subtalar motion is generally described as movement in the frontal plane of 10 to 59 degrees, with an average of 24 degrees. [1988] [1989]

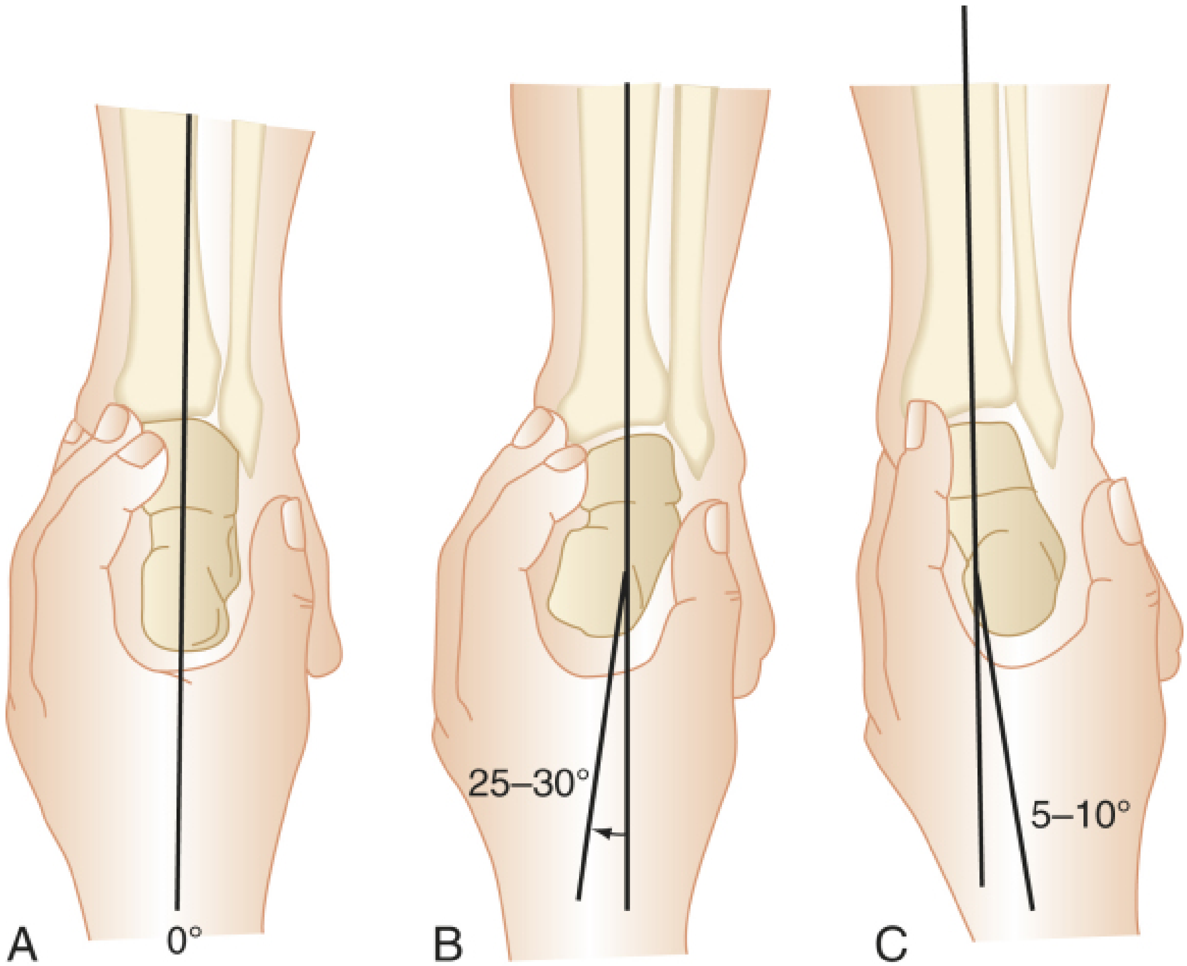

Figure 25J-6 Method of measuring subtalar motion with patient prone and knee flexed. A, Neutral position at 0 degrees on goniometer. B, Inversion. C, Eversion. *(Redrawn with permission from American Academy of Orthopaedic Surgeons: Joint Motion-Method of Measuring and Recording. Chicago, American Academy of Orthopaedic Surgeons, 1965; and Oatis C: Biomechanics of the foot and ankle under static conditions. Phys Ther 68:1815-1821, 1988.)*

Transverse tarsal joint motion plays an important role in lower extremity kinematics. Its instability has been described as *medial swivel syndrome*. [1990] [1991] There has even been one report of surgical treatment of patients with transverse tarsal joint instability. [168] Average motion in the transverse tarsal joint depends on the position of the hindfoot, or subtalar joint. As the subtalar joint undergoes eversion early in the gait cycle, the axes of the transverse tarsal joint are aligned so that they become parallel in this everted position of the heel, and this permits increased motion. This is about 22 degrees. [153] Conversely, when the heel is inverted as occurs near the end of stance phase, the axes of the transverse tarsal joint become more divergent and less mobile, averaging 8 degrees of motion. [153] This fact is important when assessing range of motion about the tibiotalar joint. If range of motion of the tibiotalar joint is tested with the hindfoot in an everted position, one gets a falsely increased range of motion due to a combination of motion through the tibiotalar joint and transverse tarsal joint. With the heel inverted, a truer measure of tibiotalar joint motion can be obtained because a minimal amount of motion is taking place through the transverse tarsal joint. Large variations in motion exist in this region, and this makes the job of defining separate motions for the talonavicular and calcaneocuboid joints difficult. [155] The literature indicates that 13 to 15 degrees of dorsiflexion or plantar flexion movement occurs in the midfoot. [169]

Motion in the tarsal-metatarsal joints is also a triplane motion, but it occurs primarily in a single plane. [1977] [1980] This is most obvious in the first and fifth ray, where we describe dorsiflexion and plantar flexion movement. With dorsiflexion of the first ray, however, some degree of abduction and external rotation occurs, and with plantar flexion, there is adduction and internal rotation. [170] Some believe that the first ray, like the transverse tarsal joint, has a locking mechanism. Perez and colleagues have demonstrated that the frontal plane position of the first ray affects the sagittal plane motion. [55] An everted position, compared with an inverted position, has the least mobility in the sagittal plane. Average dorsiflexion and plantar flexion in the first through fifth tarsal-metatarsal joints are 3.5, 0.6, 1.6, 9.6, and 10.2 degrees, respectively. [171] The triplane motion of supination and pronation was also described as 1.5, 1.2, 2.6, 11.1, and 9.0 degrees, respectively. This motion is used to advantage in the Lapidus procedure for treating hallux valgus and metatarsus primus varus in the hypermobile foot. Translational movements are abnormal in this area, and excessive forces are a cause of pathologic conditions ranging from stress fractures at the base of the second metatarsal to mild diastasis between the first and second metatarsals (Fig. 25J-7) and on to the more severe forms of Lisfranc's fracture-dislocation (Fig. 25J-8).

Figure 25J-7 A, Non–weight-bearing radiograph with small fleck sign near the base of the second metatarsal. B, Weight-bearing radiograph with subtle diastasis. *(Photographs by Christopher B. Hirose.)*

Figure 25J-8 A, Photograph of a swollen midfoot and forefoot in a patient with a Lisfranc injury. B, Radiograph of diastasis and fracture of the second metatarsal. *(Photographs by Christopher B. Hirose.)*

The normal range of motion in the first metatarsophalangeal joint is quite variable (Fig. 25J-9). According to Joseph, the average range of motion is 51 degrees of active dorsiflexion and 74 degrees of active plus passive dorsiflexion using the axis of the first metatarsal as the neutral line. [1996] [1997] [1998] The natural position for this joint in the standing posture is 16 degrees of dorsiflexion. Active plantar flexion varies between 23 degrees and 45 degrees. [1996] [1997] [1998] In performing these measurements, some variability occurs from the positioning of the ankle and subtalar joints. Motion is reduced when the ankle is in dorsiflexion and the subtalar joint is inverted. Motion is also reduced with advancing age. [172] When maximal dorsiflexion is reached in the first metatarsophalangeal joint, the normal gliding motion of the proximal phalanx on the metatarsal head ceases, and impingement can occur between the proximal phalangeal base and the first metatarsal head. [175] This may be a source of some of the problems seen in turf toe injury as well as hallux rigidus. First metatarsophalangeal joint dorsiflexion is important because of its relationship to gait and to stability of the skin of the metatarsal pad. [176]

Figure 25J-9 Variability of first metatarsal phalangeal joint dorsiflexion. *(Photographs by Christopher B. Hirose.)*

Motion in the lesser metatarsophalangeal joints and interphalangeal joints has been studied less thoroughly. Joseph's study included motion at the interphalangeal joint of the great toe. Average motion was 31 degrees of total extension and 46 degrees of active plantar flexion. Recorded norms for lesser metatarsophalangeal joint motion has a wide range from 40 degrees to 90 degrees of dorsiflexion and from 35 degrees to 50 degrees of plantar flexion. [1981] [1999] Excessive movement or stress to the lesser metatarsophalangeal joints can be a rare source of disease. Isolated synovitis and instability of the lesser metatarsophalangeal joints, however, are becoming increasingly recognized as a source of forefoot pain in patients, including athletes. [2001] [2002] Interphalangeal joint motion in the lesser toes is even less well defined, with 0 degrees being the standard for extension and 35 to 40 degrees for plantar flexion. [157] Loss of motion in these interphalangeal joints rarely creates symptoms unless there is a fixed flexion deformity, such as a hammer toe or mallet toe (Fig. 25J-10).

Figure 25J-10 Proximal interphalangeal joint flexion. *(Photograph by Christopher B. Hirose.)*

Knowledge of the normal range of motion allows one to determine the etiology of specific injuries to athletes, while being mindful that a considerable degree of variation exists that obscures a precise cause-and-effect relationship.

Etiologic Role of Flexibility

With a more scientific understanding of joint motion and flexibility, we will examine foot and ankle injuries related to flexibility or its absence. Turf toe injuries provide one of the best examples of disease related to loss of flexibility. It was initially believed that turf toe injuries were related to lack of flexibility in the first metatarsophalangeal joint. [179] We might expect a greater frequency of hyperextension injuries in athletes who have less natural dorsiflexion motion in the first metatarsophalangeal joint. All research to date, however, has failed to confirm an etiologic relationship between the loss of dorsiflexion of the first metatarsal phalangeal joint and turf toe injuries. [1997] [1998] [2004] [2005] An initial first metatarsal phalangeal joint injury can cause long-term morbidity, and hallux rigidus and hallux valgus are two specific long-term sequelae. [1997] [2006] In hallux rigidus, a loss of motion occurs in the first metatarsophalangeal joint that produces pain (Fig. 25J-11). [183]

Figure 25J-11 Hallux rigidus. *(Photograph by Christopher B. Hirose.)*

Lack of flexibility in the lesser metatarsophalangeal joints or interphalangeal joints is rarely linked with injuries or pathologic conditions in athletes. The loss of interphalangeal joint motion is a key component in mallet toes, hammer toes, and claw toes, and flexibility exercises are often advocated in the treatment of these conditions. A lack of flexibility in such toes can result in painful calluses at the tips of the toes and painful corns over the dorsum of the interphalangeal joints.

A lack of dorsiflexion in a lesser metatarsophalangeal joint can be a source of pain and disability, particularly when this involves the second toe. There is usually an iatrogenic or post-traumatic cause. Conversely, increased motion appears to be a factor in conditions involving the second metatarsophalangeal joint, including transient synovitis, crossover second toe, and subluxation and dislocation of the second toe. [2001] [2002] [2008] Flexibility studies on the relation of these conditions to injury of the lesser metatarsophalangeal joints are sparse.

Hypermobility of the first ray has been implicated as a source of problems in the foot such as stress fractures, but confirmatory studies using accepted statistical techniques are lacking. [2009] [2010] Simkin and colleagues have suggested an association between low arches and metatarsal stress fractures. [187] Gross and Bunch measured stresses in 21 distance runners and found that the greatest force occurred under the first and second metatarsal heads. [188] Using the model of the metatarsals as proximally attached rigid cantilevers, the authors showed that the greatest bending strain and shear force occurred in the second metatarsal, where stress fractures are most common. These two studies provide circumstantial evidence that increased first ray flexibility is an anatomic factor in stress fractures of the second metatarsal shaft.

Studies of the length of the metatarsals in dancers and risk for second metatarsal stress fractures note contradictory and inconclusive findings. [2013] [2014] [2015] [2016] Some believe that a longer second metatarsal predisposes dancers to an increase risk for second metatarsal stress fracture [2017] [2018] [2019] because a long second metatarsal is believed to be the recipient of added stress, especially in the en pointe position. [196] Others note no association between the relatively long second metatarsal and increased risk for stress fracture. [2015] [2021]

We may expect that the best source of a direct link between the lack of flexibility and injury would come from the ankle because of the high frequency of ankle injuries in sport. The tight Achilles tendon has been blamed for numerous conditions, including bunions, [198] turf toe, [181] midfoot strain or plantar fasciitis, [2023] [2024] [2025] ankle sprains, [202] Achilles tendinitis, [68] calf strains, [2027] [2028] and hyperpronation. [2029] [2030] [2031] Although there are numerous studies, evidence remains scarce for a causal relationship. Walsh and Blackburn suggested heel cord stretching as a preventive maneuver to decrease the incidence of ankle sprains, but they reported no results from this program. [202] Mahieu and colleagues determined that increased ankle dorsiflexion excursion and reduced plantar flexion strength may be linked with Achilles tendon overuse injuries. [208] The strength of the plantar flexors and amount of dorsiflexion excursion were identified as significant predictors of an Achilles tendon overuse injury. A plantar flexor strength lower than 50 Nm and dorsiflexion range of motion higher than 9.0 degrees were possible thresholds for developing an Achilles tendon overuse injury. [208] It seems somewhat paradoxical that on the one hand, the athletic trainer works hard to improve the athlete's flexibility at the ankle, and on the other hand, the trainer tapes or braces the ankle before practice or games to restrict motion.

Hyperpronation has been blamed for many problems known to runners. [2029] [2030] [2031] Many runners come to their local orthopaedist, podiatrist, or running shoe salesperson with the self-made diagnosis of "pronated feet" and ask for a shoe or an orthotic device to cure their shin splints, knee pain, or arch pain. Running shoes have been made and marketed specifically for pronated feet or for the supinated, rigid, cavus foot. [2033] [2034] [2035] The complex movements in the foot and ankle associated with pronation produce a more flexible foot at the time of weight transfer. This can produce problems in one of two ways. First, the normal foot [2036] [2037] goes through 6 to 10 degrees of subtalar eversion in the frontal plane during gait, and the flatfoot or pronated foot may have 12 to 15 degrees of motion. [2038] [2039] [2040] [2041] This increased motion will produce a corresponding increase in transverse plane motion. [218] Second, the speed with which the rearfoot angle changes seems to be important. [2043] [2044] The pronated foot goes through the available pronation range more quickly, and this force results in increased load transmission. [2038] [2045] Both of these movements are important components of load absorption in running. In addition, the duration of foot pronation may also have a protective effect on tibial stress fractures.

In the cavus foot, there is more rigidity in the joints of the foot. [2043] [2046] This means that loads are not dampened as effectively, and higher stress is applied at each level, which can become symptomatic. So far, treatment has centered on the use of orthotic devices, but their success in the athlete with a cavus foot is limited. [2031] [2046] [2047] [2048]

As with all areas of science, studies of the etiology of foot and ankle injuries produce more and more questions. Because of the interrelationship of many factors and the extent of individual variability, it has been difficult to impose the proper degree of control in open systems and in vivo studies to come to solid conclusions. Current studies have not provided a reliable prediction of the influence of the pronated or cavus foot on the risk for injury. [1852] [2011] [2039] [2049] [2050] [2051] [2052] [2053] A 1999 study from the Mayo Clinic looked at the effect of foot structure and range of motion on musculoskeletal overuse injuries. [230] The study group was a well-defined cohort of 449 naval trainees. They were tracked prospectively for injuries throughout training. The risk factors that predisposed trainees to overuse injuries in this study were dynamic pes planus, pes cavus, restricted ankle dorsiflexion, and increased hindfoot inversion.

SHOEWEAR-RELATED INJURY

Although the role of flexibility in sports injuries of the foot and ankle remains somewhat unclear, more evidence exists implicating shoewear and playing surfaces. The intimate relationship between these two makes separation into individual components difficult. Many studies of sports injuries look at these in a combined manner as the *shoe-surface interface.* [2055] [2056] [2057] [2058] [2059] [2060] [2061] [2062] The shoe can be a factor in athletic injuries in other ways, such as improper fit, lack of cushioning, inadequate support, and abnormal force generation. For this reason, shoewear and playing surfaces have been included as separate etiologic factors here, but the reader should maintain an awareness of their interdependency.

History

Barefoot participation in sports was the norm in ancient times. Perhaps the first recorded injury in shod feet was noted by the Greeks. [61] Today, shoes can cause problems for athletes in so many ways, one has to wonder whether it might be better for athletes to participate without shoes. An advantage to barefoot play can be seen in young children who remove their shoes to race. More recently, Zola Budd and Abebe Bikila achieved Olympic fame without shoes (Fig. 25J-12). The symptom-free nature of peoples who trod without shoewear (regardless of their degree of pes planus or pes cavus) also makes one question the true value of modern shoewear. [2063] [2064] [2065] [2066]

Figure 25J-12 Zola Budd running in 1984 Olympic 3000-meter race against Mary Decker. *(© 1991 David Madison.)*

Numerous studies have demonstrated that the least amount of pronation occurs during barefoot running. [2038] [2044] [2067] [2068] This finding stimulates an inquiry into the role of pronation in injury and whether a shoe designed for overpronators is protective. Robbins and colleagues championed the role of sensory feedback from the plantar surface of the foot in modifying load and protecting the runner from stress-related injury. [2069] [2070] [2071] They suggested that this important system is impaired by modern footwear, creating a "pseudo-neuropathic" condition. In support of this, they note that studies have shown no trend toward a reduction in injury from the use of modern athletic footwear. DeWit and colleagues looked at the biomechanics of the stance phase during barefoot and shod running. [248] Barefoot running is characterized by a significantly larger external loading rate and a flatter foot placement at touchdown. This flatter foot placement correlates with lower peak heel pressures. It was postulated that runners adopt this altered foot placement position in order to limit the local pressure beneath the heel. Divert and colleagues studied the biomechanics of barefoot compared with shod runners. [209] Barefoot runners showed lower contact and flight time and lower passive peak than shod. They concluded that barefoot running leads to a reduction of impact peak in order to reduce the high mechanical stress occurring during repetitive steps. They then studied why athletes have a higher oxygen consumption and lower net efficiency when running shod compared with running barefoot. Many believe that this effect is due to the additional mass of the shoe, and their results show that there is a significant mass effect and increasing oxygen consumption. However, stride frequency, vertical stiffness, leg stiffness, and mechanical work were significantly higher in barefoot condition. The lower net efficiency reported in shod running may also be due to the impact-dampening properties of the shoe as the foot strikes the ground. [249] Even though these studies suggest that shoes have no protective effect, most athletes still use shoes for competition and daily life. We explore a critical analysis of shoes in this chapter and discover how they fall short in their role as protective equipment for the sports participant.

Incidence

The exact incidence of injury attributable to athletes' shoes is unknown, but several studies have included shoes as a separate factor in injury rates. [250] James and colleagues included shoes and surfaces as one of three categories under cause of injury in runners in addition to training errors and anatomic factors. [251] Nineteen percent of the 180 runners in their series were treated by a change or modification in shoewear. Lysholm and Wiklander studied 60 runners with 55 injuries during a 1-year period. [252] Surface or shoe problems were the primary sources of injury in 3 cases and one of multiple factors in 10 others. Inferior footwear was the etiologic factor blamed in 34 of 318 injuries in soccer players.

Football has provided the best perspective on shoewear and its relationship to knee and ankle injuries. Torg and Quedenfeld published an extensive study in 1971 relating the incidence and severity of knee and ankle injuries to shoe type and cleat length in Philadelphia high school football players. [253] Rates of injury to the ankle were reduced from 0.08 per team per game to 0.01 per team per game by switching from conventional cleated football shoes to soccer-style shoes with multiple shorter cleats. This result was substantiated by the work of Mueller and Blyth, who noted a reduction in knee and ankle injuries by resurfacing the playing field (30.5% reduction), changing from regular cleats to soccer shoes (22.3% reduction), or making both changes (46% reduction). [254] Although these studies do not consider such shoe-related problems as cleating-induced contusions or lacerations or turf toe, they do provide a framework for studying the role of shoes in sports injury.

Mechanical Factors

Shoe Fit

The most obvious problem with shoes that plagues the shod people of the world regardless of whether they are athletes is the proper fit. Even the smallest problem with fit can prevent athletes from performing to the best of their capabilities. For example, a web corn can become so painful that each step is agony. Metatarsalgia can result from a shoe that is too tight across the forefoot. Similarly, a narrow shoe often aggravates a Morton's neuroma (Fig. 25J-13). The black toe of long-distance runners can be a sequela of improper shoe fit. Toe deformities such as hammer toes, claw toes, and overlapping fifth toes may become symptomatic in athletes whose shoes have toe boxes that are either too narrow or of insufficient depth. Calluses and blisters are inherent in most sports participation (Fig. 25J-14). Improperly fitted shoes that rub the skin excessively or allow the foot to move or slide disproportionately in the shoe enhance the frequency of calluses and blisters. Other irritants include the insole edge, penetrating cleats, prominent seams, and orthotic device edges.

Figure 25J-13 Morton's neuroma excised from the third web space. *(Photograph by Christopher B. Hirose.)*

Figure 25J-14 Calluses in a long-distance runner. *(Photograph by Christopher B. Hirose.)*

Cushioning

The importance that cushioning protects athletes from injury has been promoted largely by the running shoe industry and those whose research it supports. [255] The running literature has seen a proliferation of articles addressing impact forces, shoe cushioning, shock absorption, and the effects of various alterations in shoe construction. The logical assumptions have been that load on the human body is directly attributable to impact forces at foot strike and that these forces are naturally altered by the cushioning properties of the shoe. [2067] [2079] [2080] [2081] [2082] These assumptions foster the belief that changing the shoe's material properties (e.g., midsole thickness) can influence impact load, thereby changing rates of injury.

Impact forces are a critical feature in the etiology of sports-related pain and injury whether acute or chronic. [27] The bionegative effects of impact loads are evident from the damage produced to articular cartilage by high-impact loads. [2083] [2084] Radin and coworkers showed that deleterious changes occurred in the biochemical and biomechanical properties of articular cartilage of sheep walking constantly on concrete as opposed to those walking on wooden chips. [2083] [2084] Impact force and shock wave transmission play a role in the etiology of experimentally induced osteoarthritis. [2085] [2086] Shock-absorbing insoles in the boots of South African military recruits produced a 9% reduction in their incidence of overuse injuries. [263] In addition, the German armed forces studied the properties of cushioned insoles. The aim was to assess metatarsal head loading in combat boots with respect to the prevention of metatarsal stress fractures comparing cushioned with standard conventional insoles. The cushioned insoles were superior to the conventional insoles with respect to the plantar pressure distribution. [264] These studies support the belief that impact loads are an etiologic factor in certain injuries, but careful analysis leaves the impression that this load plays a less than consequential role.

Ground reaction forces are the primary external force acting on the human body during running. [1851] [2089] [2090] [2091] [2092] These forces increase as running velocity increases. Higher ground reaction forces are seen in the progression from walking to jogging to sprinting to jumping. Estimates vary from 1.2 times body weight (BW) for walking to 2.5 times BW for jogging. Sprinting increases load by 3 to 6 times BW, whereas jumping multiplies this force by 6 to 8 times BW [27] (Table 25J-6). Impact force amplitude is reduced when soft materials are used in the shoe or running surface. [269] This reduction is achieved by increasing the deceleration distance of the foot. Calculations of impact peaks can be made from the following equation: $F_{max} = F_{xi} = v\ fm,$

TABLE 25J-6 -- Variations in Ground Reaction Force from Walking to Jogging to Sprinting to Jumping

Study	Movement	Velocity (m/sec)	Footwear	F_{max} (N)	F_{max} (BW)
Cavanagh, 1981	Walking heel-toe	1.3	Barefoot	—	0.6
Cavanagh, 1981	Walking heel-toe	1.3	Casual shoes	—	0.3
Clarke, 1982	Running heel-toe	2.7	Running shoe	—	2.8
Frederick, 1981	Running heel-toe	3.4	Barefoot	1365	2.0
Frederick, 1981	Running heel-toe	3.8	Barefoot	1590	2.3
Cavanagh, 1980	Running heel-toe	4.5	Running shoe	—	2.2
Frederick, 1981	Running heel-toe	4.5	Barefoot	1963	2.9
Nigg, 1981	Running heel-toe	5.5	Running shoe	2350	3.6
Nigg, 1978	Running jump	6.0	Spikes	4000	5.3
Nigg, 1981	Running jump	8.0	Spikes	5500	7.9

From Nigg BM: Biomechanical aspects of running. In Nigg BM (ed): Biomechanics of Running Shoes. Champaign. Ill, Human Kinetics, 1986, p 21. Copyright 1986 by Benno M. Nigg.

BW, body weight; N, Newtons.

where F_{max} is the maximal force occurring in the vertical direction (F_{xi}) and is proportional to velocity (v), the mass of the body (m), and the spring constant for that body (f). From this equation it is apparent that impact velocity has the greatest influence on vertical load. It is also reduced by reducing the mass (e.g., body weight) or by reducing the spring constant (e.g., knee flexion angle). [2070] [2094] [2095] [2096]

As an etiologic factor in injury, shock absorption has not proved to be the critical factor that advertising would lead us to believe. Although changing from a new shoe with good cushioning properties to an old shoe lacking these properties can produce injury, an injury may come from changing to a newer shoe as well. It may be that change itself is the critical factor in an injury that occurs during a repetitive activity. Reinschmidt and Nigg have noted that for running shoes, at least, pronation control and cushioning are still considered the key concepts for injury prevention despite the fact that conclusive clinical and epidemiologic evidence is missing for these design strategies. [273] In addition, recent running shoe research has suggested that cushioning may not be related to injuries and that cushioning during the impact phase of running may be more related to aspects like comfort and muscle fatigue. A more recent study refuted the shock-absorbing qualities of insoles in injury prevention. This study was a randomized controlled trial of 1205 Royal Air Force recruits to assess the differences, if any, in the efficacy of two commonly available shock-absorbing insoles. Similar rates of lower limb injuries were observed for all insoles (shock-absorbing and non–shock-absorbing) in the trial. [274]

One case example for the role of poor cushioning is that of a 26-year-old runner who sustained bilateral stress fractures of the fibulae after he changed from his usual pair to an older pair of shoes for a 14-km race. [275] Instrom testing of the original running shoe and the ipsilateral older shoe demonstrated that the original running shoe had twice as much energy absorption and 5 times the deformation as the older pair. Although lack of shock absorption is the postulated cause of injury in the aforementioned case, other factors might have played a role as well. These include the change itself, the altered muscle activity causing increased bending moments on the fibula, increased muscle activity resulting in muscle fatigue and loss of the protective function of the muscle, and variations in foot geometry leading to increased pronation. [2099] [2100] [2101] [2102] [2103] [2104] [2105] [2106] [2107]

Cook and coworkers have demonstrated by mechanical testing and in vivo experimentation that different shoe models vary by as much as 33% in shock-absorbing characteristics, and all show significant decreases with mileage. [2108] [2109] Initial shock absorption values were reduced by 60% or more after 250 to 500 miles of machine-tested running. Other variables that reduced shock absorption were environmental conditions, such as moisture content of the shoe (e.g., perspiration or rain) and hardness of the testing surface (e.g., asphalt versus grass). Some running authorities have suggested that running shoes may have time-related life spans. The longevity of the shoe is related to the mileage run and usually falls in the 500- to 1000-mile range (Fig. 25J-15). [243] One study examined heel pad stresses during heel strike with simulated wear of the ethylene vinyl acetate foam commonly found in modern sport shoes. Heel pad stresses were consistently increased when the ethylene vinyl acetate thickness was decreased, suggesting that the age of the shoe affects the viscoelastic properties of the shoe. [286]

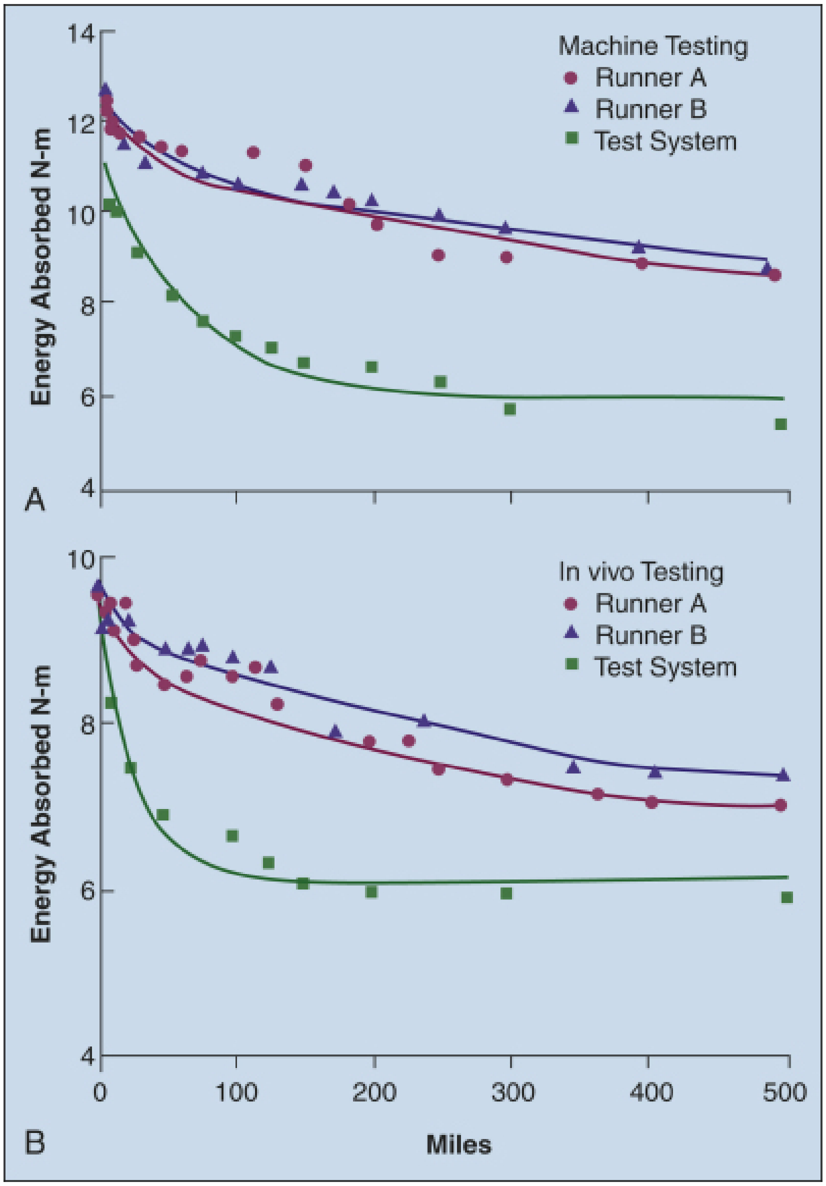

Figure 25J-15 Life span of running shoes related to mileage as demonstrated by reduction in retention of initial shock absorption with increasing mileage run in the shoes. A, Machine testing. B, In vivo testing. *(Redrawn with permission from Cook SD, Kester MA, Brunet ME: Shock absorption characteristics of running shoes. Am J Sports Med 13:248-253, 1985.)*

Whittle looked at transient impulses beneath the foot and how they are generated and attenuated. [287] He found that there is a "shock-wave" that passes up the limb that may lead to degenerative joint disease. Limb positioning, materials used in shoe construction, and the use of orthoses can alter these forces. In addition, the intrinsic shock absorption system of the body produces behavior modification to control load magnitude. Impact forces are dramatically reduced by increasing knee and hip flexion at ground contact. [288] Several studies have proposed that cushioned shoes lead to negligible decreases in load because subjects decrease flexion to accommodate the instability produced by softer surfaces. [1836] [2112] [2113] [2114] In reality, the human body accommodates load using complex strategies, and the material used for cushioning in shoes is most likely a very small element in this shock absorption system. Milani and colleagues' study supports this hypothesis. [291] The perceptual ratings of eight identical running shoes with a relatively close range of midsole stiffness was examined. Their findings suggest that the body's sensory systems differentiate well between impacts of different frequency content, and that subjects adapt their running style to avoid high heel impacts. [291]

Experimental results do not universally confirm that a lack of shock absorption is the major cause of injury. [2049] [2116] [2117] Softer materials of inadequate thickness have a tendency to reach maximal compressibility and then transmit greater amounts of load. [220] To reduce impact forces, material thickness must be of sufficient height to prevent maximal compressibility. In one study, however, a change in midsole hardness from soft to hard did not alter the impact force peaks measured in 14 test subjects at four different running velocities. [294] These results appear to be related to the variation in the point of application on the foot of the ground reaction force. Harder materials impose a larger lever arm in the force equation by moving the effective point of application farther lateral from the subtalar joint axis. This produces an increase in both initial pronation and initial pronation velocity, corresponding to an increase in deceleration distance over time and a resulting decrease in the impact force. [2093] [2118] The foot-shoe-surface interaction is so complex that investigators are still debating the proper methodology for testing it. There are dramatic differences between machine testing and subject testing, between internal forces and external forces, and between subjects as well as in the same subject under varying test conditions. [2044] [2049] [2116] [2119] [2120] [2121] [2122] Bates and coworkers have even shown that the same test in the same runner can produce different results between trials. [225,295] With all these confounding data, it is hardly surprising that investigators in this field frequently disagree and have occasionally produced contradictory results.

In the final analysis, the question remains whether the cushioning provided by the shoe is a critical factor in the etiology of injury in the athlete. The sport shoe industry and its related research have concentrated heavily on improving the shock-absorbing characteristics of its shoes. We have seen a plethora of innovative cushioning designs such as air soles, gel shoes, variable density and encapsulated soles, and soles that will automatically vary cushioning according to the runner's weight or speed. As remarkable as these designs are, there is still no universally accepted scientific study that proves better cushioning in the shoe lowers the incidence of injury in runners. Robbins and Waked noted that deceptive advertising creates a false sense of security with users of expensive athletic shoes, inducing attenuation of impact-moderating behavior, increased impact, and injury. [299] In conclusion, it can be stated that a lack of cushioning may be a factor in producing injury but probably not to the degree to which some researchers and shoe manufacturers might lead us to believe.

Shoe Control

For the running shoe, control or support is primarily interpreted in the context of rearfoot control. This is defined as the shoe's ability to limit the amount or rate of pronation occurring through the subtalar joint at heel strike. [214] A less well-defined component of shoe support is its control of take-off supination, which occurs at the lateral forefoot during toe-off. [220] Foot support also consists of the relative flexibility of the shoe, most often measured as forefoot flexibility, which allows the shoe to bend at the metatarsal phalangeal joints. [243] Greater flexibility has been considered desirable, but shoe flexibility can be a major factor in turf toe injuries. [2003] [2058] Conversely, stiffening the sole in the area of the metatarsophalangeal joint may decrease energy loss and improve performance. [300] Control of movement in the sagittal plane by the shoe remains a subject for further investigation.

Support for lateral motion and medial and lateral shear is a concern in court shoes: tennis, basketball, and aerobic shoes. [2125] [2126] [2127] [2128] [2129] Lateral stability, torsional flexibility, cushioning, and traction control appear to be important design strategies to decrease the risk for injury. [273] The support provided for the ankle by high-top shoes has been an im-portant consideration in preventing injury, particularly in basketball and football (Fig. 25J-16). [2129] [2130] [2131] [2132] [2133] [2134] Brizuela and associates examined the performance of basketball shoes with increased ankle support compared with a shoe with no ankle support. [311] The high support shoes resulted in higher forefoot impact forces and lower shock transmission to the tibia. The use of high support shoes also resulted in lower ranges of eversion and higher ranges of inversion for the ankle on landing. In the motor performance tests, the high support shoes reduced the height jumped and increased the time to complete the running course compared with low support shoes. This study underlines the importance of designing shoes that will maximize performance without compromising safety. Johnson and associates tested torsional stiffness of high-top football shoes using a special chair and measurement apparatus and found that high-top shoes were 50% stiffer than low-cut models. [307] Theoretically, the high-top shoe should stimulate the proprioceptive feedback mechanism, resulting in greater sensitization of the peroneal muscles and improved stability for the ankle. [312] Potential negatives induced by the high-top shoe include the reduction in load carried by the collateral ligaments of the ankle and the limitation in subtalar motion restricting the foot's ability to adapt to surface irregularities. Handoll and colleagues believe that the protective effect of high-top shoes still remains to be established. [313] They studied various interventions, including external ankle supports in the form of a semirigid orthosis, air-cast braces, high-top shoes, ankle disk training, taping, muscle stretching, boot inserts, and controlled rehabilitation. The main finding was a significant reduction in the number of ankle sprains in people allocated external ankle support and no statistical difference in those athletes wearing high-top shoes. [313] Although some evidence supports the ability of high-top shoes to lower the rate of injury to the ankle, [2104] [2138] [2139] other studies have found no difference in ankle sprain incidence related to shoe type. [2140] [2141] [2142] In related work, the effect of ankle bracing and taping on athletic performance and injury prevention remains unclear despite numerous studies. [2139] [2143] [2144] [2145] [2146] [2147] [2148] [2149] Control characteristics are essential elements in modern sports shoes designed for prevention of injury, but not all features are beneficial.

Figure 25J-16 Examples of a current high-top and low-top athletic shoe. *(Photographs by Christopher B. Hirose.)*

Rearfoot stability provided by the athletic shoe has become a critical ingredient in managing the overuse problems attributed to overpronation (Fig. 25J-17). [2150] [2151] Running shoes are designed to accomplish this control. Included are such creative concepts as cantilever soles with imbedded plastic stability devices, plastic torsion bars, composite plates, gel pods, air chambers, progressive-rate polymer columns, variable-density-foam midsole platforms, metal springs buried inside the heel, and even magnetic impact-sensing systems, materials of variable hardness in areas of the midsole, thermoplastic heel counters, heel flares, external stabilizers, and combination lasts. The importance attributed to rearfoot control is derived from the assumption that overpronation produces injuries in runners. This assumption has been extrapolated from studies of runners beginning with the classic study by James and colleagues. [251] Fifty-eight percent of those injured were classified as pronators on biomechanical examination. [2075] [2152] The ability of the shoe or an orthosis to control excessive pronation is supported by the work of Bates and associates, Cavanagh, Nigg and Morlock, and Smith and coworkers (Fig. 25J-18). [2067] [2153] [2154] [2155]

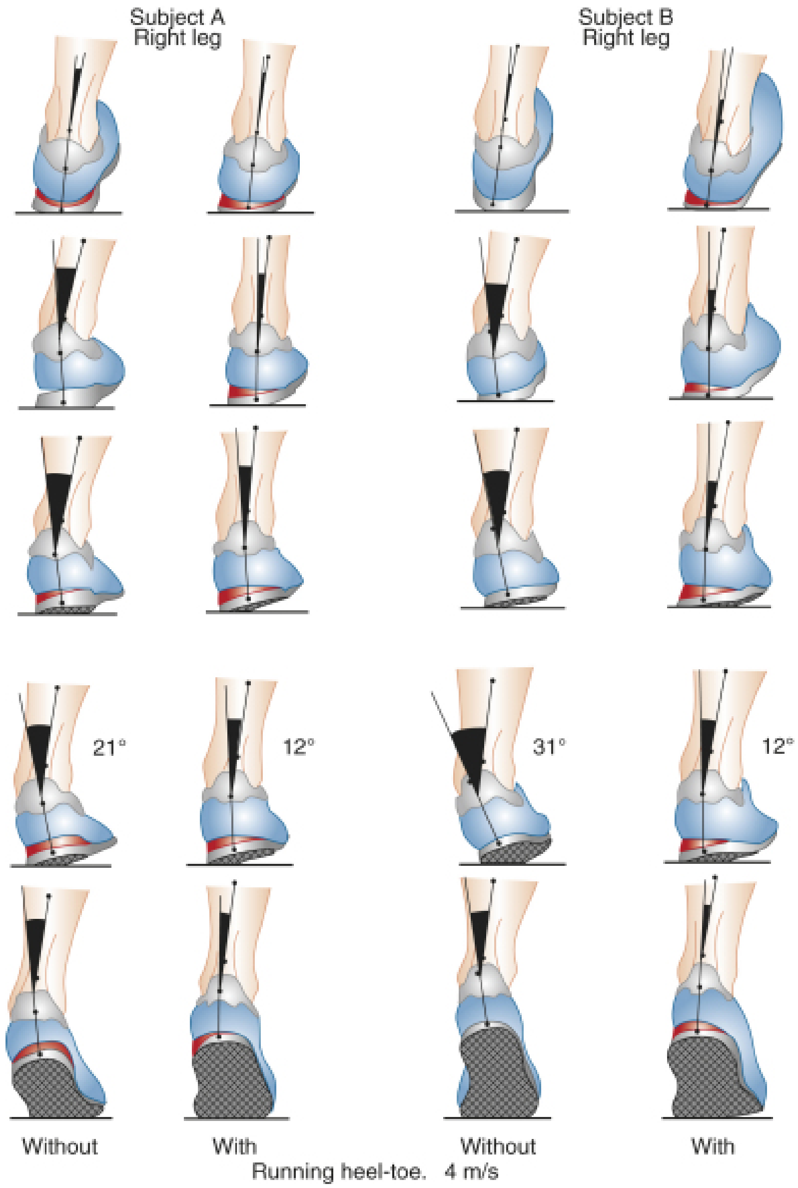

Figure 25J-17 Rear foot stability provided by athletic shoewear. Illustration of two runners switching between their commonly used shoes without control features and shoes with special control features provided in the laboratory. *(Redrawn with permission from Nigg BM, Bahlsen AH, Denoth J, et al: Factors influencing kinetic and kinematic variables in running. In Nigg BM [ed]: Biomechanics of Running Shoes. Champaign, Ill, Human Kinetics Publishers, 1986, p 157. © 1986 by Benno M. Nigg.)*

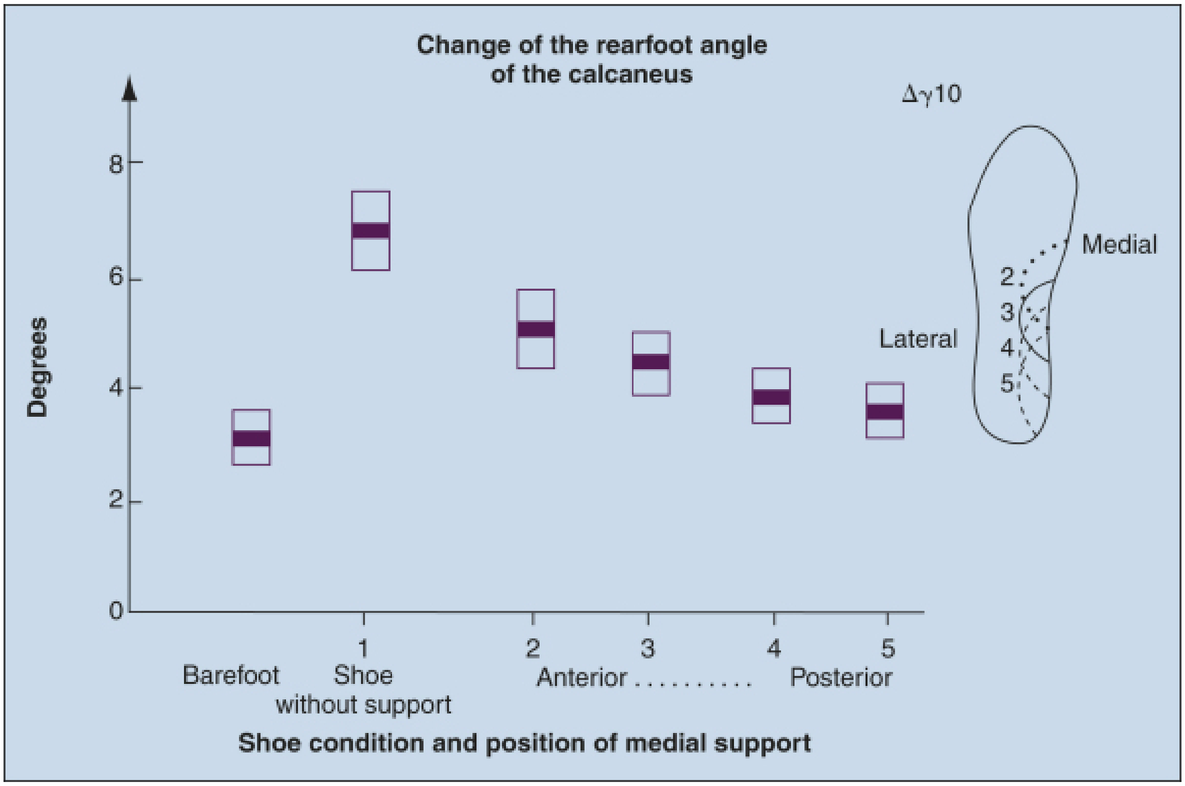

Figure 25J-18 Shoe control of excessive pronation as demonstrated by change in rearfoot angle ($\Delta\gamma_{10}$) resulting from systematic alterations in location of medial support (positions 2 to 5) in the running shoe compared with support while barefoot and in shoe without support. *(Redrawn with permission from Nigg BM, Bahlsen AH, Denoth J, et al: Factors influencing kinetic and kinematic variables in running. In Nigg BM [ed]: Biomechanics of Running Shoes. Champaign, Ill, Human Kinetics Publishers, 1986, p 152. © 1986 by Benno M. Nigg.)*

Take-off supination has been blamed for Achilles tendon strain and reduced performance. [220] Take-off supination corresponds to the take-off angle, which should be close to 180 degrees. Angles between 160 and 170 degrees are indicative of increased take-off supination. Interestingly, take-off supination is seldom seen in people engaged in barefoot running and is primarily a product of shoewear. It is unaffected by midsole hardness and running velocity but is heightened by additional medial support, especially when such support is located more posterior in the shoe. Nigg and coworkers have shown that geometric solutions are possible by adding lateral forefoot support or midsole grooves. [220] Little clinical information is available to support or refute this concept in shoe control. [332]

The next category to be assessed in the etiology of athletic injury is shoe flexibility. This quality can be assessed by the amount of force necessary to flex the shoe's forefoot 40 to 50 degrees. [2157] [2158] According to Ryan, the running shoe should be "moderately flexible" to allow proper foot mechanics. [335] A shoe with greater flexibility is typically preferred by runners and is even advocated for those with rigid cavus feet.

More convincing clinical and experimental evidence exists to implicate excessive flexibility of shoes in causing football injuries. In 1964, artificial grass was introduced, and a flexible sole soccer shoe replaced the traditional stiff, cleated football shoe. [2003] [2058] A new symptom complex appeared that was attributed to the use of these more flexible shoes on the harder surface, which was nicknamed *turf toe*. [234] Two cases of turf toe were found in track athletes whose sprains occurred while wearing flexible racing spikes. [179] Further characterization of this injury has made turf toe a well-defined clinical entity and has incriminated overly flexible shoewear conclusively in its etiology (Fig. 25J-19). [1997] [2003] [2004] [2005] To their credit, shoewear manufacturers have responded by either stiffening the forefoot or providing shoe inserts to stiffen the forefoot. [336] These examples support the view that the shoe flexibility plays a critical role in the etiology of injuries to the forefoot.

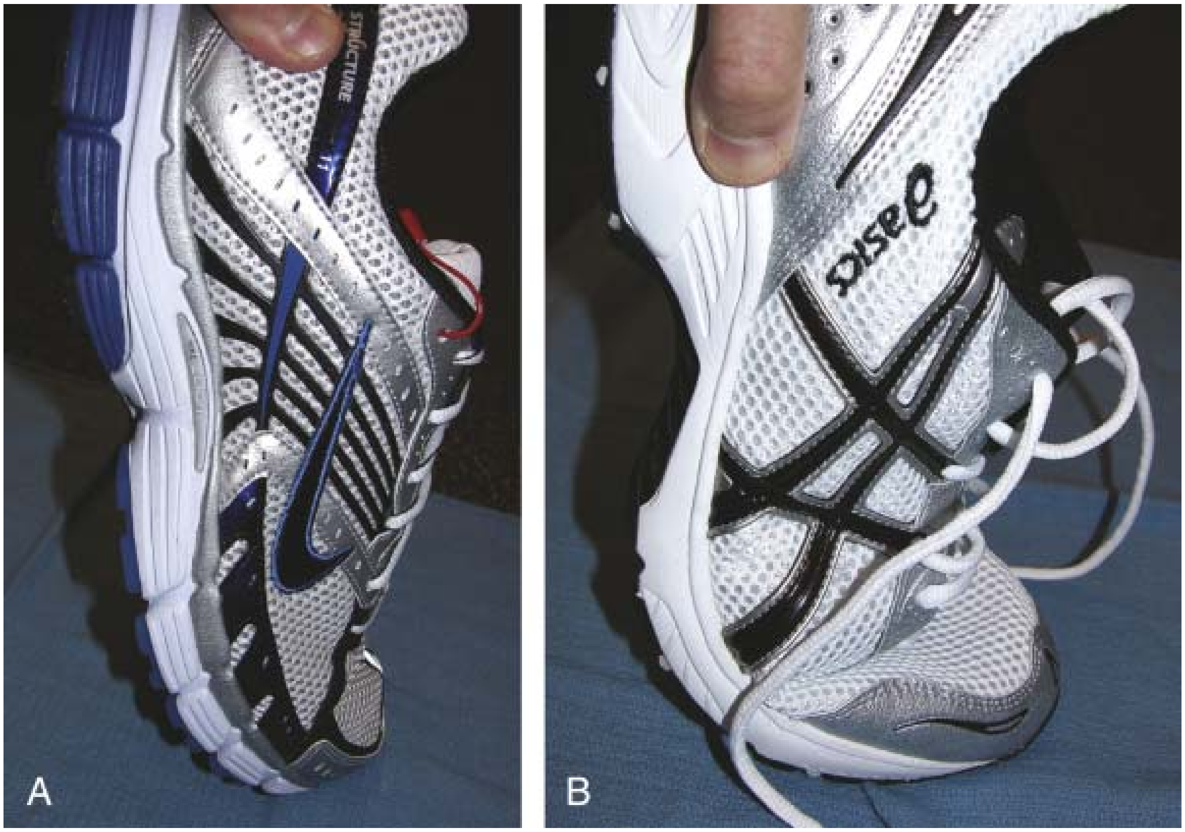

Figure 25J-19 Variability of the forefoot stiffness of two different running shoes. *(Photographs by Christopher B. Hirose.)*

Orthotics

Is the runner with a hyperpronating foot truly more susceptible to injury? Does a rigid orthosis holding the foot in a subtalar neutral position reduce the likelihood of injury? What role do orthoses have in injury reduction or production? Further evidence corroborating the effect of pronation control is provided by the results of treatment of injury by the use of orthoses, which led to improvement and resumption of training in 70% to 90% of those treated. [1976] [2161] [2162] [2163] Nigg believes that there is some evidence that orthotics reduce movement-related injuries, but that the orthotic functions as a second filter of force input, the first being the shoe and the third being the athlete's foot. [340] An orthotic can optimally function by simply reducing muscle activity, increasing comfort, and ideally increasing performance. Nigg and associates looked at the effect of shoe insert construction on foot and leg movement. [341] They found that the changes resulting from the use of all inserts in total shoe eversion, total foot eversion, and total internal tibial rotation were smaller than 1 degree when compared with the no-insert condition. Also, they found that the soft insert construction was more restrictive than the harder inserts. They concluded that it is important to match specific feet and shoe inserts optimally. Kulcu and colleagues studied over-the-counter insoles and gait patterns with those who have a flexible flatfoot. [342] They concluded that over-the-counter insoles have no beneficial effect in normalizing forces acting on the foot and on the entire lower extremity. Crosbie and Burns studied orthotics in those with pes cavus deformities and found that the mechanisms by which orthotic intervention is effective in improving pain and function in painful, idiopathic pes cavus remain unclear and equivocal. [343] In addition, Krivickas studied overuse injuries and bony alignment of the extremities and noted that although malalignment of the lower extremities is frequently cited as predisposing to knee extensor mechanism overuse injuries and foot injuries, orthotics do not have any effect on knee alignment, and although they can alter subtalar joint alignment, the clinical benefit of this remains unclear. [20] Despite the improvements in rearfoot control offered by current athletic shoewear, it has been difficult to relate this to any evidence for reduction in incidence of running injuries.

Cleats and Sole Modifications

Traction is a crucial ingredient in efficient performance in all sports and is related to the design features of athletic shoewear. It can also be influenced by the playing surface and by the weight of the participant. Factors to be considered in the analysis of traction related to shoewear include the outsole materials used, the sole pattern, and the presence of cleats as well as their size and configuration. Load related to traction is studied primarily with regard to torque and friction. [1962] [2055] [2062] [2157] [2168]

Outsoles are the bottom-most layer of the shoe that has direct contact with the ground. The most frequently used materials are carbon rubber, styrene butadiene rubber, ethylene vinyl acetate, and polyurethane. [2169] [2170] In general, if the material is harder, its durability is greater. Traction considerations must encompass the interaction of these materials with a variety of playing surfaces and conditions, including the artificial surfaces used indoors and outdoors as well as the amount of moisture or dust on the field or court. [347] Major differences occur in the coefficients of static and sliding friction for various shoe-surface combinations (Table 25J-7). [348]

TABLE 25J-7 -- Friction Coefficients for Several Floor Combinations Tested under Laboratory Conditions

	Surface				
Shoe	**Carpet**	**Synthetic Granular**	**PVC**	**Sand**	**Asphalt**
Sliding Friction Coefficients					
All-around shoe	1.05-1.15	0.95-1.05	1.00-1.20	0.40-0.60	0.70-0.80
Little profile					
All-around shoe	0.95-1.05	0.80-0.95	0.80-0.90	0.30-0.55	0.60-0.75
Treaded					
Profile					
Tennis shoe	0.50-0.60	0.75-0.90	0.40-0.50	0.30-0.50	0.65-0.75
Indoor					
No profile					
Static Friction Coefficients					
All-around shoe	1.15-1.25	1.05-1.15	1.00-1.10	0.50-0.60	0.70-0.80
Little profile					
Jogging shoe	105-1.15	0.95-1.05	0.80-0.90	0.40-0.60	0.70-0.80
Treaded					
Profile					
Tennis shoe	0.60-0.70	0.80-0.90	0.40-0.50	0.40-0.50	0.75-0.85
Indoor					
No profile					

PVC, polyvinyl chloride.

Torque is the component of traction that is measured as the tendency of a force to rotate an object around an axis. It correlates with the static coefficient of friction. Laboratory experiments have established large differences in torque between different shoe-surface combinations, as illustrated in Figure 25J-20 . [348] Rheinstein and colleagues performed similar experiments supporting this difference in torque as related to outsole material, outsole hardeners, player weight, and playing surface. [347] They found greater maximal torques with the softer outsoles combined with artificial and clean hardwood flooring in heavier players. Rubber-soled shoes demonstrated much more sensitivity to dust than the polyurethane soles when analyzed for loss of traction. This research has considerable implications in sports medicine because higher torque means higher load transmission to the body and an increased potential for injury. Alternatively, lack of traction implies sliding, slipping, falling, and poor performance. Where is the proper balance? This question may be unanswerable but deserves attention.

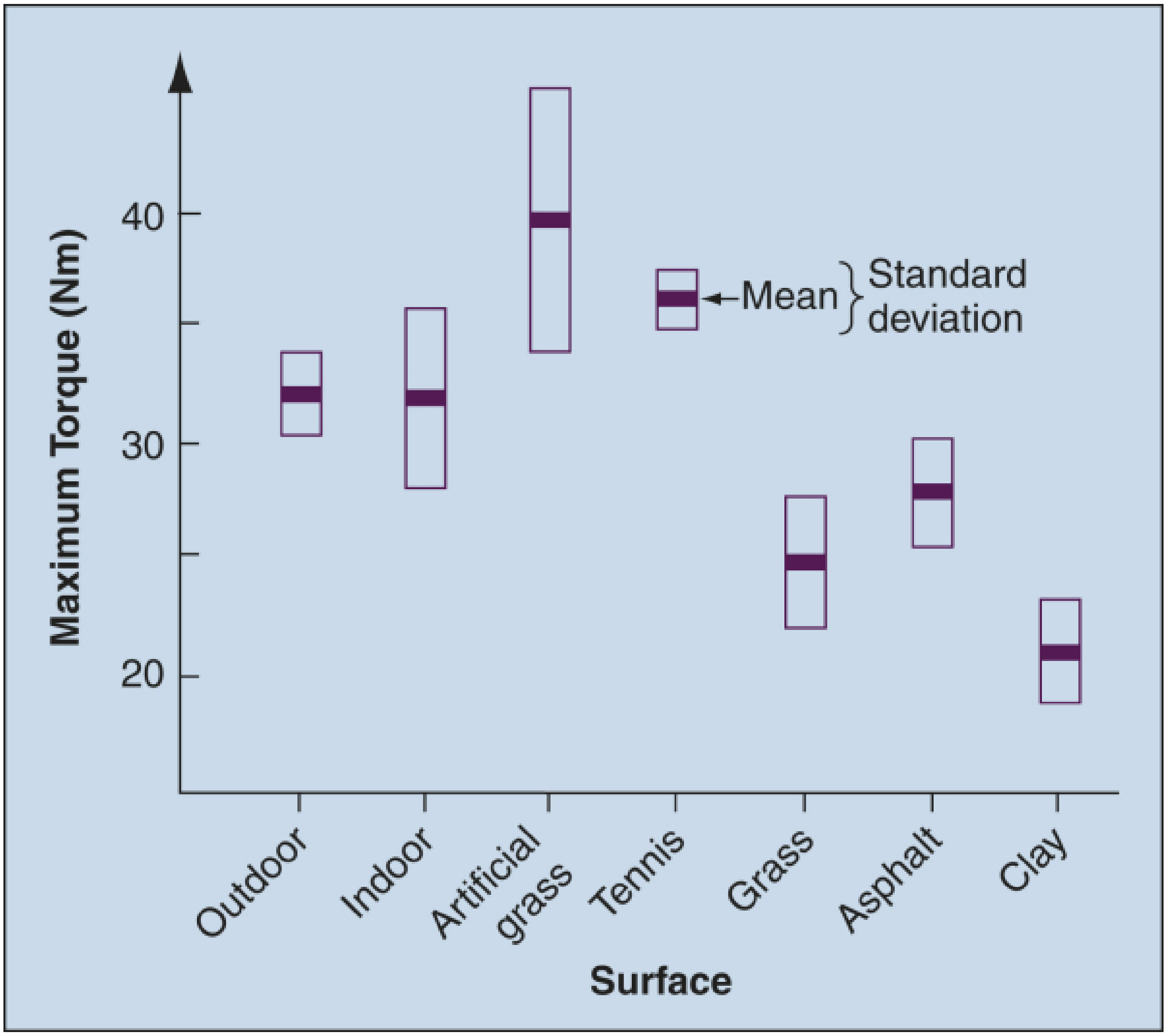

Figure 25J-20 Mean values and standard deviations for maximal torque for 12 subjects on seven surfaces in eight different shoes. *(Redrawn with permission from Nigg BM, Denoth J, Keir B, et al: Load sport shoes and playing surfaces. In Frederick EC [ed]: Sport Shoes and Playing Surfaces. Champaign, Ill, Human Kinetics Publishers, 1984, p 12. © Nike, Inc.)*

Outersole tread design has developed from the flat rubber of the traditional canvas tennis shoe of yesteryear to the high-technology multiple-patterned sole we see in running shoes and court shoes today (Fig. 25J-21). [2169] [2173] These tread designs theoretically alter the mechanical properties of the shoe by enhancing performance or preventing injury by determining the flexion path for the shoe, the proper break point, and the pivot point, as well as by affecting traction and shock absorption. [346] The tread design has been used in marketing certain running shoes, including the original Nike waffle sole (Fig. 25J-22). The tread design can clearly alter the traction characteristics of the shoe, but its relationship to foot function and injury prevention remains unproved.

Figure 25J-21 Different tread designs: A, Track shoe. B, Football cleats. C, Baseball spikes. D, Running shoe. *(Photograph by Christopher B. Hirose.)*

Figure 25J-22 The original Nike Waffle shoe. *(© Nike, Inc.: www.nike.com .)*

Efficiency of movement is an important factor in athletic performance, and proper traction ensures that internal forces generated by the body's muscles are efficiently converted into movement. A natural byproduct of this relationship was the introduction of cleats to the outsole of the shoe to improve traction. Introduced about 100 years ago for sports, they have been a source of controversy ever since. Rule changes in sports banned pointed cleats and placed size limits on cleats. The polemics continued with the concept that foot fixation was a leading cause of injury of the ankle and knee in sports. [2170] [2174] [2175] Dr. Daniel Hanley of Bowdoin College championed this attack on rigid cleating, particularly in the heel area, after he observed the incidence of significant noncontact injuries to the knee at Bowdoin. [2169] [2175] [2176] Cleat modifications followed, including plastic heel disks, [237] lower profile oval cleats, [345] soccer cleats, [353] and cleats attached to a rotating turntable (Fig. 25J-23), [2069] [2174] as well as cleats with a circular design for artificial grass, the Tanel 360 (Fig. 25J-24). [2178] [2179] This design concept theoretically allows pivoting with adequate traction but without the problem of foot fixation. Queen and colleagues examined the effect of different cleat plate configurations on plantar pressure. [356] They noted significant differences in forefoot loading patterns among cleat types, but no definitive conclusions were drawn in regard to injuries.

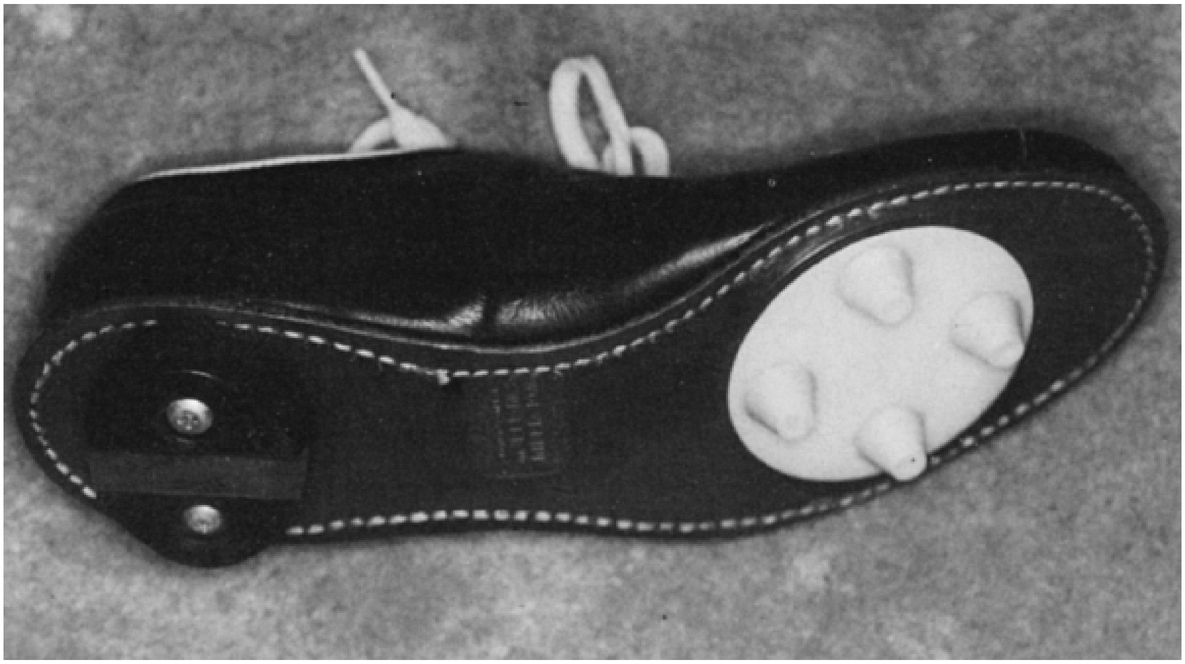

Figure 25J-23 Dr. Bruce Cameron's swivel shoe alternative cleat design. *(Photograph by Thomas O. Clanton.)*

Figure 25J-24 Alternative cleating patterns in the Tanel 360 football shoe. *(Photograph courtesy of Tanel Corporation.)*

Research has documented the relationship between cleats and sports injuries. Rowe studied the effect of different shoes and cleats on knee and ankle injuries in the New York State Public High School Athletic Association during the 1967 and 1968 seasons. [353] He found a reduction in injuries with the use of a low-cut disk heel shoe compared with low- and high-top shoes with heel cleats and an even more substantial reduction with short soccer-type cleats used by athletes playing on natural grass. Although Rowe's findings were not subjected to statistical verification, ankle injuries were reduced from a high of 77 per 100,000 hours of participation to 34 per 100,000 by changing from the low-cut conventional heel to the soccer shoe. Rates of injury with the soccer shoe were lower than those seen with either type of high-top shoe. Torg and Quedenfeld found a similar reduction in ankle injuries from 0.45 per team per game with conventional cleats to 0.23 per team per game with a soccer-type shoe. [253] Cameron and Davis found a progressive reduction in ankle injuries from 8.46% with a cleated shoe to 7.69% with a heel plate to 5.64% with a soccer shoe to 3.00% with their swivel shoe design. [350]

An exhaustive study of injuries in North Carolina high school football players by Mueller and Blyth emphasized the critical role of the playing surface in evaluating the role of cleats. [254] They reported a reduction in knee and ankle injuries from 14.8% to 11.5% by changing from traditional cleats to soccer-type shoes on properly maintained fields. Bonstingl and colleagues concluded that there was a positive relationship between torque and injury. [2181] [2182] The highest torque occurred with a conventional football shoe, which contained seven ¾-inch high cleats. A substantial reduction in torque occurs in circular pattern outsole design (e.g., the original Tanel 360 shoe). Other studies have confirmed these differences in friction and torque between various outersoles, cleats, and playing surfaces. [2055] [2057] [2168] [2181] [2183] This is one of the most obvious areas in which basic science and clinical research in the field of sports medicine have had an impact on sports equipment designed to prevent injury.

PLAYING SURFACES AND INJURY

Of all the etiologic factors involved in foot and ankle injuries, the playing surface may be the most important and, at the same time, the least understood. If it is true that load on the human body is the common ground for discovering clues to the causes of athletic injury, the influence of forces and moments intrinsic to the surface of play must be critically analyzed. [2172] [2184] Traditional sports surfaces have been composed of natural materials: wooden basketball courts, clay and grass tennis courts, cinder tracks, grass and dirt baseball diamonds, and natural grass football and soccer fields. With advancing technology, there was a move to replace these with more durable low-maintenance synthetic surfaces. Although such surfaces had some attractive advantages, they were not universally accepted and were subjected to quick criticism that followed their short-lived popularity.

Sports surfaces should accomplish three functions: (1) protection, (2) performance, and (3) maintenance. [293] The first is concerned with the protection of the athlete from excessive forces and injury. The sports performance function relates to the optimization of the athletic experience through the qualities of the surface. Maintenance refers to the durability and conservation of the surface and preserving the former two qualities. The task of reviewing all sports surfaces is overwhelming, considering that wrestling mats, gymnastic beams, ice-skating rinks, and snow-packed slalom courses could all be included. To gain some understanding of this broad topic, we divide surfaces into indoor and outdoor surfaces and into natural and synthetic surfaces. There is some overlap between surfaces in sports, and multiple uses are the rule in most sports facilities. As the characteristics of a particular surface used in one sport are delineated, the reader is reminded that the surface characteristics are applicable in other sports, but that protective, technical, and performance characteristics may vary among sports, types of athletes, shoewear, and environmental situations.

History

Since ancient times, humans have competed athletically. The surface chosen was that which occurred naturally, most commonly grazed fields and dirt areas freed of rocks and obstacles. As culture progressed, so did the playing fields, and stadiums were created for competition in ancient Greece. Improvements in these outdoor facilities were accomplished by maintaining the fields, using developments in soil and grass technology, crowning to improve drainage, and limiting play to allow the surface to recover. It was not until 1964 that synthetic grass, produced by Monsanto, was introduced and installed on the playing field at Moses Brown School in Providence, Rhode Island. [1875] [2185] [2186] Developed as a substitute for grass in a place where its natural growth was difficult, this artificial surface created little impact until the 1960s. On April 9, 1965, the "eighth wonder of the world," the Astrodome, was completed amid much hype in a city known for space-age technology: Houston, Texas. [363] A special grass was developed to grow inside the Astrodome: Tifway 419 Bermuda. [363] Even the foundation soil required a unique design. Unfortunately, when the clear Lucite roof panels were darkened to eliminate glare and lost fly balls, the grass withered, and management scrambled to find a suitable substitute. The following year, the natural grass was replaced by Monsanto's synthetic grass, and this became known as *AstroTurf.*

Now, grass playing fields in natural and custom-installed varieties compete with synthetic surfaces. [364] Entire publications advise facility managers in the most intricate detail of how to maintain playing fields. [32] The debate about which is the better surface has involved both the public and the scientific community, and more than 50 articles have appeared without a summary conclusion. [1875] [1881] [1984] [2184] [2189] [2190] [2191] [2192] [2193] [2194] [2195]

Perhaps the key deficiency in these studies is their poor control of other key factors in the turf equation such as the shoe, field maintenance, and weather. Synthetic grass manufacturers have been forced to make improvements in their fibers, mats, underpads, and drainage systems, and advocates of natural grass have used modern methods to introduce advances of their own. As long as prevention of injury is a prime concern in these advances, both the individual athlete and society as a whole will benefit.

This same revolutionary process has occurred in other outdoor surface sports, such as tennis and track. Tennis originated from a French handball game called *jeu de paume* at about the 13th century and was played in courtyards over a fringed net. [372] Major Walter Wingfield of North Wales invented a game in 1873 from which modern outdoor tennis has evolved. Its popularity spread so rapidly that the All England Croquet Club added the name Lawn Tennis to its title and sponsored the first championship in 1877. As the name indicates, grass was the original surface used for play. As the popularity of tennis increased, other playing surfaces were needed to allow winter play and to bypass the problems inherent in growing a playing field. In 1909, Claude Brown introduced the clay court. Although these surfaces remain popular, new court surfaces have flourished to such a degree that modern tennis has the widest choice of playing surface of any major sport (Table 25J-8). Only recently have the surface characteristics of these courts been subjected to laboratory and subject tests in an attempt to establish their safety.

TABLE 25J-8 -- Chart Comparing Various Tennis Court Surfaces

Court Type	Repairs May	Glare	Initial Cost	Including Base [*]	Maintenance	Avg. Time before	Resurfacing Cost (1992)	Surface Hardeness	Ball Skid Length

	Costly		**per Court (1992) prices**		**Resurfaceing**	**prices)**		
Porous								
Fast dry	No	No	14,000-18,000 [†]	Daily and yearly	Annual	1000-3000	Soft	Short if damp cou
With subirrigation			24,000-26,000			Top dressing		
Clay	No	Generally	9,000-11,000	Daily and yearly	5 yr	1000-1500	Soft	Short if damp cou
Grass	No	no	15,000-17,000	Daily and yearly	Indefinite	Varies	Soft	Moderatel long
Sand-filled synthetic turf [‡]	Yes	no	25,000-30,000	Daily and yearly	Indefinite	N/A	Soft	Short
Porous concrete	Yes	yes	27,000-31,000	Minor	Indefinite	N/A	Hard	Short
Nonporous Noncushioned								
Concrete post-tensioned	Yes	No (if colored)	22,000-25,000	Very minor	5 yr (if colored)	3000-3500	Hard	Controllab
Concrete reinforced	Yes	No (if colored)	19,000-21,000	Very minor	5 yr (if colored)	3000-3500	Hard	Long if glossy finish, medium if gritty finis
Asphalt plant mix (colored)	No	No	16,000-18,000	Very minor	5 yr	2500-3000	Hard	
Emulsified asphalt mix	No	No	19,000-21,000	Very minor	5 yr	3000-3500	Hard	Long if glossy finish, medium if gritty finis
Asphalt penetration macadam	No	No	15,000-17,000	Very minor	5 yr	3000-3500	Hard	
Nonporous Cushioned								
Asphalt bound system (colored)	No	No	19,000-23,000	Very minor	5 yr	2500-3500	Soft	Long if glossy finish, sho if gritty finish
Liquid applied synthetic	Yes	Possible	30,000-40,000	Very minor	5-10 yr	2500-3500	Soft	Varies shortest tc longest
Textile [§]	No	No	25,000-28,000	Very minor	Varies	Varies	Soft	Short
Modular [§]	No	No	22,000-26,000	Very minor	Varies	Varies	Soft	Medium tc short

Removable [§]	No	No	25,00-30,000	Very minor	Varies	Varies	Soft	Varies shortest tc longest

Reprinted from Tennis Courts 1992-1993 with the permission of the United States Tennis Association, 707 Alexander Road, Princeton, NJ 08540.

* Prices vary regionally, do not include site preparation or fencing, and will be somewhat reduced when building or resurfacing batteries of courts.

† Including sprinkler system.

‡ Damaged areas may be readily repaired.

§ Including base construction and structurally sound surface.

Track and field is unquestionably the oldest form of organized sports activity, dating back 3500 years to ancient Greece. The religious festival at Olympia featuring this activity became an event of such proportions that the Greeks dated events with reference to the year of the Olympiad. [2197] [2198] Records dating back to 776 BC indicate that a race covering 192 m took place on a track about 32 m wide. [374] These early contests were held on grass, and later races took place on public thoroughfares.

Improvements in track design led to the use of cinders and a mixture of cinders and clay by the late 1800s. Synthetic surfaces began to appear in the 1960s with the production of a prototype track by the 3M Corporation for Macalester College in 1965. [361] The synthetic track gained wide appeal after its use in the 1968 Olympics in Mexico City, where numerous records were set. Although athletes have long known the difference between “fast tracks” and “slow tracks,” it remained for McMahon and Greene from Harvard to produce a track engineered to optimize running speed. [2199] [2200] [2201] [2202] This was an indoor track that theoretically and practically enhanced speed by about 2% (or 5 seconds per mile), and the physics have since been incorporated into outdoor track surfacing technology. Although the cinder track has been virtually eliminated from elite competition, its value in reducing injuries remains clear, and some schools have preserved cinder tracks for use in training. Tracks for elite competition are generally made from rubber or synthetic materials such as polyurethane. Corporate research and development departments working in conjunction with bioengineers at universities in locations as far apart as Calgary, Zurich, Rome, and Boston design these surfaces to maximize performance and reduce injury potential. [379]

The origin of the movement of sports events from outdoors to inside can also be traced to ancient Greece. The name *gymnasium* is derived from the Greek word meaning “school for naked exercise.” [380] Exercise was an integral part of Greek society, but the gymnasium also served as a public facility for the training of male athletes to participate in the aforementioned public games. From its beginning as a room that served as a gathering place for exercise, the gymnasium grew in proportion to accommodate baths, dressing quarters, rooms for specialized purposes, and larger areas for contests and spectators. [380] The first modern gymnasium opened in Copenhagen in 1799. [381] Pestalozzi, Ling, and Spiess stimulated further development, and groups such as the Young Men’s Christian Association and the Young Men’s Hebrew Association included physical exercise in their activities in the mid-1800s. The international Young Men’s Christian Association training school in Springfield, Massachusetts was the site where James Naismith introduced the new game of basketball to the world in 1891. [382] Wooden floors became the traditional gymnasium surface and continued to predominate for basketball courts as well as for other indoor courts and dance studios. Contemporary surfaces have been said to have advantages ranging from improved durability to noise reduction to better use of space, but seldom have their supporters argued for improved safety. In the study of sports surfaces, whether indoor or outdoor, it is evident that many factors affect the safety value of the surface. These include not only the visible surface and the top finish but also, and equally important, the undersurface.

Injury Incidence

Although there is consensus that sports surfaces play a critical role in causing athletic injuries, it has been quite difficult to establish the incidence of injury in a sport that can be attributed solely to the playing surface. Even in the area of traditional grass football fields, there have been studies that have suggested differences in incidence of injury from field to field, although the surface material is the same. The 1992 study by Powell and Schootman is one of the best-controlled studies demonstrating that football players were at significantly higher risk for knee ligament injury when playing on AstroTurf as compared with natural grass. [383] Unfortunately, their study does not address injuries outside the knee. A study of North Carolina football players conducted by Mueller and Blyth [254] showed that a reduction in the injury rate resulted simply from resurfacing and maintaining the game and practice fields. Injury rates plummeted from 29.3% on unresurfaced fields in 1969 to 14.8% on resurfaced fields in 1972, a 30% reduction. Poor field conditions were considered a factor in 8 of 34 soccer injuries in the paper by Sullivan, [95] in 14 of 18 outdoor soccer injuries in Hoff and Martin’s series, [46] and in 62 of 318 injuries in the Swedish study of Ekstrand and Gillquist. [91] These studies draw attention to the playing field and its importance and the need for further work on proper playing surfaces and their maintenance to reduce injury rates in sports. FieldTurf was developed to duplicate the playing characteristics of natural grass. Meyers and associates studied the differences in injuries of high school football players between the two surfaces and found that the types of injuries were different between the two surfaces. The natural grass surface produced more head and neural traumas and ligamentous injuries. The FieldTurf produced more injuries during higher temperatures, muscle-related trauma, and epidermal injuries. [384]

Studies of injuries and injury rates in sports ranging from dance [2013] [2209] to ice hockey [2210] [2211] to tennis [388] have mentioned the sport's surface as a factor. For example, Pasanen and colleagues studied the injury risk in pivoting indoor sports between artificial floors and wooden floors. [389] They found that the risk for a traumatic injury in pivoting indoors sports is two-fold higher on artificial floors when compared with wooden floors, largely due to a higher shoe-surface friction level. Although the surface is frequently named as a source of problems in runners, there have been no studies that have unequivocally confirmed this. [28] The Ontario cohort study found no association between running surface and injury, whereas the companion study from South Carolina showed a statistically meaningful relationship only for females running on concrete. [228]

In baseball, if the base is included as a part of the playing field surface, several studies can be cited indicating that this is the primary factor in ankle injuries in baseball and softball. Janda and coworkers found that 71% of the recreational softball injuries in their study were related to sliding into bases. [47] A follow-up to this study showed the implications of an injury-prevention method such as the use of a breakaway base on these surface-related injuries. [48] Janda and associates estimated that breakaway bases could reduce the incidence of serious injury in softball by 96% and result in a $2 billion per year savings in acute medical care costs. [390]

In the area of children's playground injuries, statistics suggest that falls to the ground account for 60% of playground equipment injuries, yet barely half of day care playground equipment is installed on impact-absorbing surfaces. [2070] [2215] Impact studies of five types of loose-fill playground surfaces at a variety of drop heights, material depths, and conditions suggested that shredded rubber was the best performer, and there was little difference between sand, wood fibers, and wood chips; and pea gravel had the worst performance, making it a poor choice for playground surfacing. [392] A separate study supported these findings: children sustained significantly more injuries in playgrounds with concrete surfaces than in those with bark or rubberized surfaces. Playgrounds with rubber surfaces had the lowest rate of injury, with a risk half that of bark and a fifth of that of concrete. Rubberized impact-absorbing surfaces are safer than bark. [393] Certainly the playing surface is an important consideration in the prevention of injury.

MECHANICAL FACTORS

Regardless of the surface, the underlying question to be answered is how the surface affects load in the individual participant. [2122] [2172] For this purpose, attention has been focused on certain material properties of the sports surface such as hardness and friction together with performance properties such as energy loss or resilience. Nigg has reviewed the methods by which a playing surface may be characterized and specified some of the tests that are important in determining these properties. [394]

Hardness

One of the most obvious differences between surfaces detected by casual observation as well as by sophisticated testing is the relative hardness or softness. Hardness is related to the ground reaction force. In conformity with Newton's first law (for every action there is an equal and opposite reaction), the vertical reaction force responds to the vertical force component applied by the individual at foot strike. Its amplitude is affected by the shock-absorbing qualities of the surface to which the force is applied. The time needed for force absorption and reaction is the key to the amplitude of the reaction force and relates to the compliance of the surface material. Hard materials deform less than soft materials during identical impact testing conditions: a well-maintained grass lawn is softer than a concrete sidewalk. When impact occurs between an object such as a leg and a surface such as grass or concrete, it is the surface hardness that limits the time of impact while increasing the amplitude of the reaction force (Fig. 25J-25). Kerdok and colleagues built experimental platforms with adjustable stiffness to examine the leg stiffness and metabolic cost of the athlete. [395] The 12.5-fold decrease in surface stiffness resulted in a 12% decrease in the runner's metabolic rate and a 29% increase in their leg stiffness. They concluded that an increased energy rebound from the compliant surfaces studied contributes to the enhanced running economy. It takes little imagination to determine that sports participation on a soft surface such as a gymnastics mat carries less risk for certain impact-related injuries than a similar activity performed on a wooden floor. By the same token, athletes running on shock-absorbing gymnastics mats compared with a tuned track would set few records. [378] From these mundane examples, we can conclude that there are some opposing factors confronting us in this analysis. Although a harder surface may provide a better surface for performance needs in certain sports, it may create simultaneously an increased exposure to injury. This fact appears to be borne out most obviously by synthetic tracks.

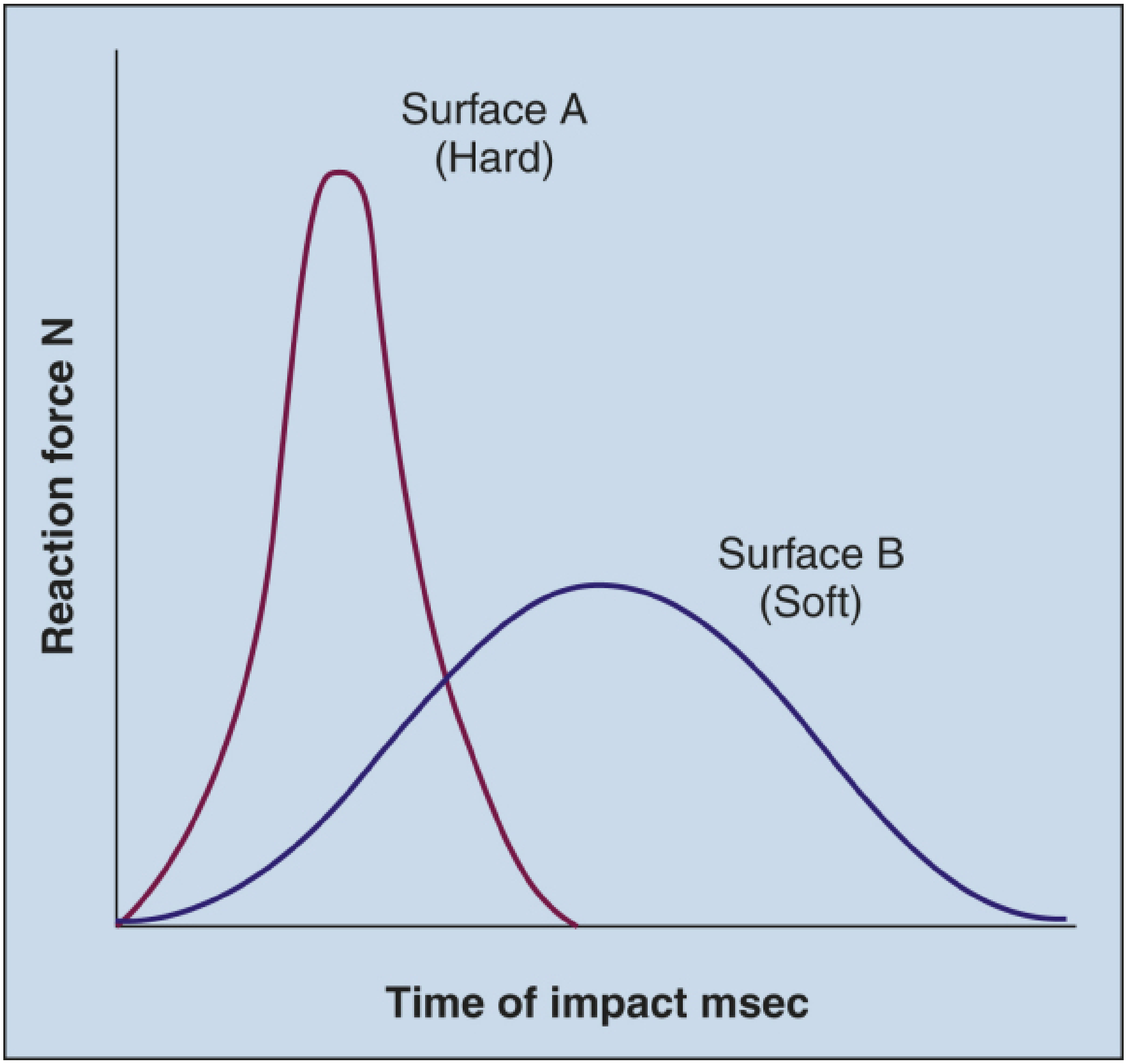

Figure 25J-25 Example of relationship between time of impact and type of surface. Hard surface (A) has a short time of impact but a high reaction force, whereas soft surface (B) has a longer time of impact and a lower reaction force.

Hardness is the resistance generated by a material during deformation in response to an externally applied force. [2172] [2220] In the study of various materials, their behavior is described by means of a stress-strain curve or stress-deformation diagram. The plastic or elastic behavior of a material is determined by the remaining deformation once the acting force is removed. When the deformation persists, the material is described as plastic, and when the material returns to its original shape, it is elastic. A sports surface may be further characterized as being either area elastic or point elastic. [397] Owing to their high bending strength, area elastic surfaces distribute forces over a wide area. Point elastic surfaces have low bending strength and therefore deform only in a very confined area (Fig. 25J-26). This fact has implications for both performance and health.

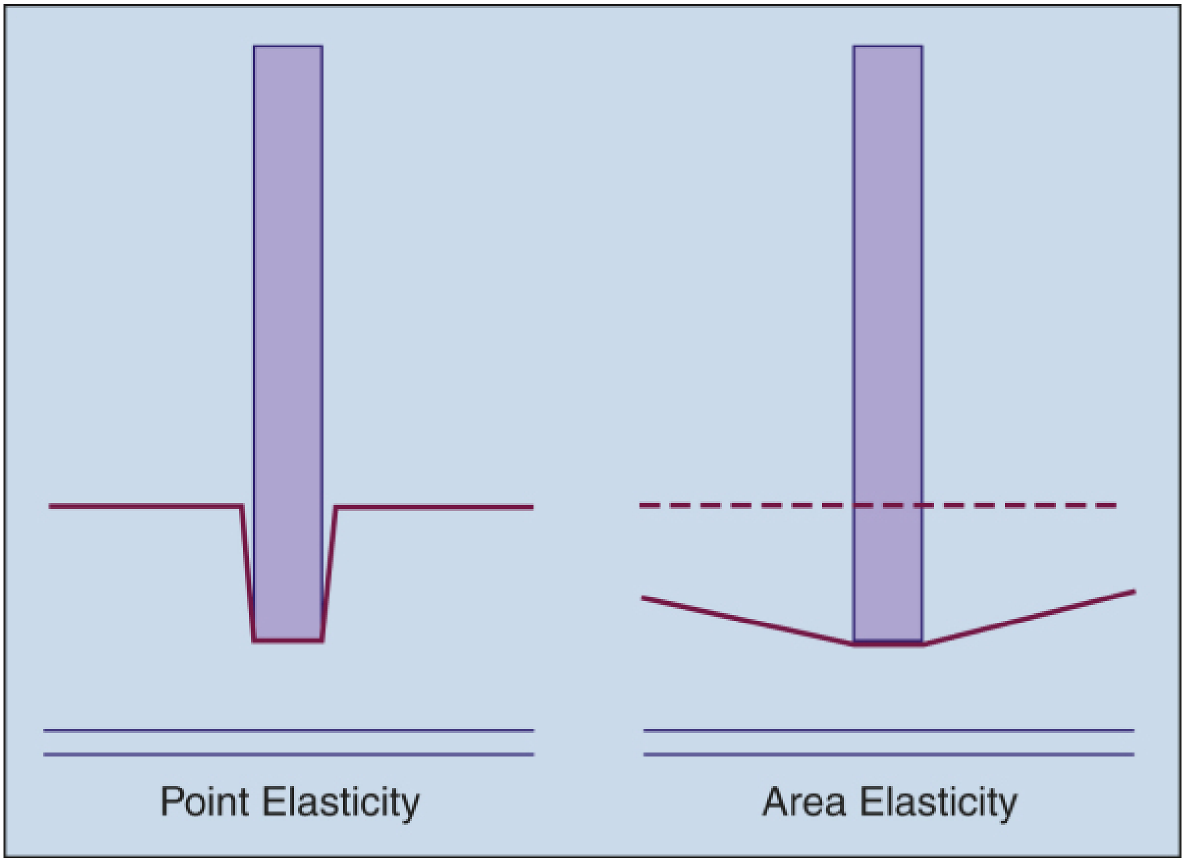

Figure 25J-26 Example of point elastic surface and area elastic surface. *(Redrawn with permission from Denoth J: Indoor athletic playing surfaces—floor vs. shoe. In Segesser B, Pförringer W [eds]: The Shoe in Sport. Chicago, Year Book Medical Publishers, 1989, pp 65-69.)*

From the clinical standpoint, there is a widespread belief that harder surfaces are associated with a higher incidence of injury for any given activity. This conclusion seems obvious, but it is difficult to support scientifically. Such conditions as shin splints, stress fractures, tibial stress syndrome, turf toe, bursitis, arthritis, and even acute fractures have been associated with the higher loads imparted by surfaces with limited compliance. Bowers and Martin demonstrated the existence of reduced shock-absorbing characteristics in 5-year-old AstroTurf compared with new AstroTurf, describing this as "clearly detrimental to player safety." [398] Unfortunately, they did not show a clear relationship between this lack of impact absorption and an increase in either acute or chronic injuries. Larson and Osternig [399] reported one of the few clinical studies implicating surface hardness as a source of specific athletic injury in 1974. In a survey of Pacific-8 Conference athletic trainers after the 1973 football season, they showed that the incidence of prepatellar and olecranon bursitis was increased on artificial grass compared with natural grass and attributed this increase to the hard underlying sub-base. Anecdotally, considerable evidence of problems with harder playing surfaces exists because players commonly complain of aching feet and legs after standing and practicing on older synthetic fields.

The injury most frequently associated with sports participation on artificial grass is turf toe. [2003] [2005] [2058] This injury is a sprain of the first metatarsophalangeal joint that has been inextricably linked to the artificial playing surface. Turf toe is a distinct clinical entity related to the combination of a relatively flexible shoe and a hard artificial surface. [234] Despite the weight of clinical evidence pointing to the relationship between turf toe and the artificial surface, little statistical support exists implicating surface hardness as a major factor. The injury does indeed occur on natural grass and probably has more to do with the flexibility of the shoe and frictional characteristics of the surface than its hardness.

Impact forces, skeletal transient forces, and excess load produced by hard surfaces have been emphasized as etiologic factors in many other conditions ranging from osteoarthritis to shin splints to stress fractures. Statistical verification of this relationship, however, remains absent. An increased incidence of tibial periostitis, Achilles tendinitis, Achilles tendon rupture, and muscle rupture are postulated to be the result of hard surface synthetic tracks. [53] Haberl and Prokop have related these conditions to what they call *Tartan syndrome* and propose that surface hardness producing initial decelerations as high as 80 G force (G) is a primary etiologic factor. [400] The relationship between surface hardness and stress fractures has been mentioned in several studies. [1896] [1983] [2012] [2104] [2105] [2106] [2225] With this as background, it is apparent that surface hardness is important in the study of athletic injury, although its relative contribution among the interrelated factors is imprecise.

Friction

Without friction, human locomotion would be impossible. The frictional properties of the surface are the second critical mechanical factor of sport surfaces related to sports performance and injury. Whereas hardness is defined by a high vertical stiffness, friction relates to horizontal stiffness. The static coefficient of friction (μ) is the inherent property of the two contacting materials. It can be calculated from the equation $\mu = F/W$, where W is the weight of the object being moved over a surface and F is the force required to move the object. [2168] [2226] This equation applies to smooth, uniform surfaces, but not to the shoe-turf interface. Therefore, a similar term has been described as the release coefficient (r). It is expressed as $r = F/W$, where W is the weight in the shoe and F is the force necessary to release the shoe-turf interface when engaged. [344] The difficulties imposed by reduced friction are well known to the novice ice skater. High friction between the shoe and the surface translates into good traction for the athlete and improved performance. Unfortunately, this factor also means greater load on the body, which may exceed physiologic limits. Therefore, a trade-off exists between performance-enhancing qualities and safety considerations.

Friction can be viewed in several ways that are important to sports biomechanics. There are two types of friction to be considered: static and kinetic. [403] Static friction is the resistance to movement between two objects that are not moving relative to each other. It is a surface property of the contact surfaces. Kinetic friction occurs when two objects are moving relative to each other, rub together, and typically slow down one or both of the objects. Friction can also be viewed in terms of horizontal or rotational friction. [2172] [2217] [2227] Functionally, the former corresponds to the force resisting the foot sliding or moving sideways, whereas rotational friction relates to torque generated in activities such as turning. Torque is considered one of the primary etiologic factors in injuries to the knee and ankle. [2055] [2181]

Energy Loss

A separate but equally important property apart from hardness is the energy loss that occurs when a material is loaded. This property varies over a wide range from surface to surface and carries major implications in the field of sports biomechanics. As depicted in Figure 25J-27 , materials can exhibit similar elastic behaviors on the stress and strain diagram and yet have very different responses to the effects of loading and unloading. In this figure, material A shows no loss of mechanical energy, whereas material B depicts a loss of mechanical energy equivalent to the shaded area. This energy loss is not a true material property because it depends on the loading rate and other variables. When a material has the quality of variable deformation dependent on velocity of deformation, it is described as viscoelastic. If energy applied to a surface is lost in the surface deformation, performance may suffer. [378] Surfaces that deform to greater degrees are called *compliant* and result in increased contact time. This is the means by which cushioning occurs. By increasing the time of collision, the force between the colliding bodies is decreased. The final property of importance in athletic performance on a given surface is its resilience. High resilience indicates that the energy stored in the surface owing to its stiffness is returned to the athlete. This has implications for enhancing performance as well as lessening fatigue. [2202] [2221]

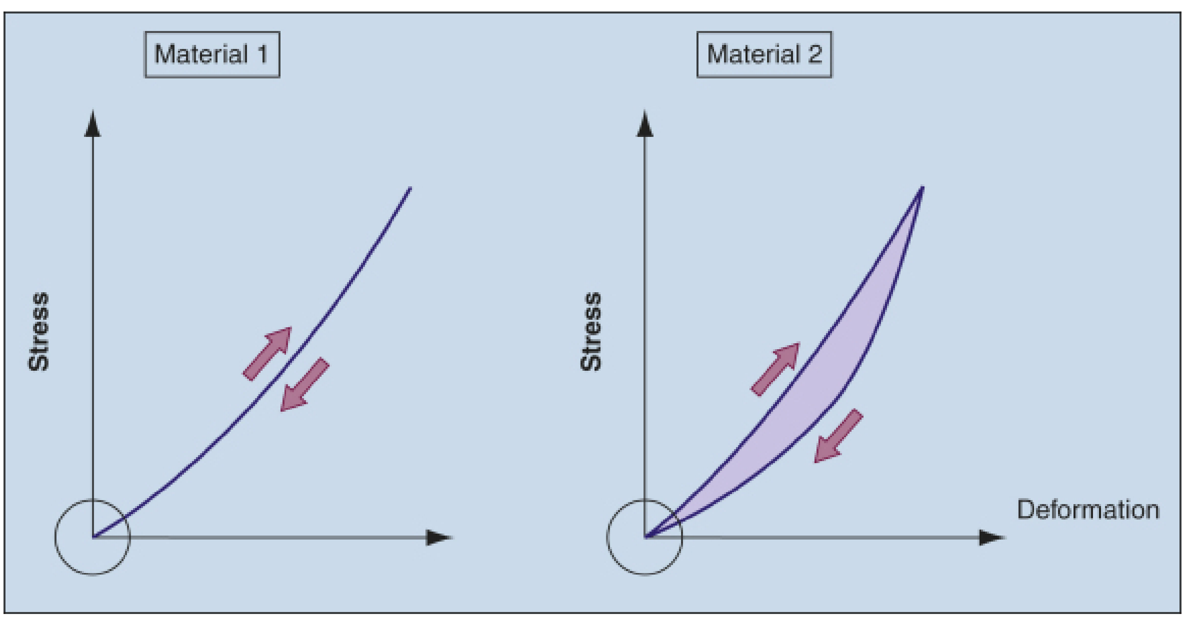

Figure 25J-27 Differences in response of materials to loading and unloading despite similar elastic behavior. *(Redrawn with permission from Denoth J: Load on the locomotor system and modelling. In Nigg BM [ed]: Biomechanics of Running Shoes. Champaign, Ill, Human Kinetics Publishers, 1986, p 96. © 1986 by Benno M. Nigg.)*

Experimental Work

Because of a presumed association with injuries, frictional properties of various sport surfaces and athletic shoes have been the subject of studies by several authors. Torg and Quedenfeld published pioneering work in the field of sports medicine in 1971 aimed at reducing injury rates by targeting the shoe-surface interface.[253] The study was done on grass and concentrated on the relationship between the number and size of shoe cleats and the incidence and severity of knee injuries in high school football players. This investigation showed a reduction in ankle injuries from 72 to 36 and in ankle fractures from 13 to 7 by changing from a traditional seven-cleated football shoe to a multicleated soccer shoe. Continuing this study, Torg and colleagues performed laboratory studies to determine the torque necessary to release an engaged shoe-surface interface.[2060] [2061] Twelve shoes and nine surface conditions were tested for a total of 108 release coefficients. These are shown in Tables 25J-9 and 25J-10 [0260] [0265]. Coefficient differences of 0.05 were determined to be significant. The release coefficients ranged from a high of 0.55 ± 0.06, with a conventional seven-cleated football shoe on dry grass, to a low of 0.20 ± 0.02, with a conventional shoe that had an uncleated disk heel (Bowdoin modification) on dry Polyturf. The use of wet versus dry conditions was based on the study by Bramwell and colleagues in 1972, which suggested that fewer injuries were sustained on wet synthetic fields than dry synthetic fields.[404] From their study, Torg and coworkers concluded that the release coefficient varies with (1) the number, length, and diameter of the cleats; (2) the type of surface—natural or artificial; (3) the condition of the surface—wet or dry; and (4) the outsole material of the shoe—polyurethane or soft rubber. They classified shoes as safe for a particular surface when the release coefficient was 0.31 or below.

TABLE 25J-9 -- Results of Torque Testing in 12 Shoes for Nine Surface Conditions

	Release Coefficients							
Shoe	**Grass**	**Grass Wet**	**AstroTurf**	**Astroturf Wet**	**Tartan Turf**	**Tartan Turf Wet**	**Poly Turf**	**Poly Turf Wet**
Group I	0.55 ± 0.06		0.35 ± 0.03		0.34 ± 0.03		0.29 ± 0.3	
Group II	0.44 ± 0.04		0.31 ± 0.03		0.32 ± 0.02		0.26 ± 0.02	
Group III	0.37 ± 0.04		0.26 ± 0.02		0.26 ± 0.03		0.20 ± 0.02	
Group IV	0.36 ± 0.03	0.32 ± 0.04	0.41 ± 0.03	0.26 ± 0.03	0.34 ± 0.03	0.23 ± 0.02	0.33 ± 0.02	0.23 ± 0.02
Group V	0.28 ± 0.03	0.27 ± 0.03	0.29 ± 0.03	0.29 ± 0.03	0.27 ± 0.03	0.24 ± 0.02	0.26 ± 0.02	0.23 ± 0.02
Group VI			0.40 ± 0.01		0.36 ± 01		0.41 ± 0.02	
Group VII			0.45 ± 0.04		0.37 ± 0.02		0.45 ± 0.02	
Shoes in groups I-V have plastic or polyurethane soles.								
Shoes in groups VI-VII have rubber soles.								
Group I: Conventional 7-posted football shoe, ¾-inch cleat length, ⅜-inch tip diameter, plastic sole								
Group II: Conventional 7-posted football shoe, ½-inch cleat length, ⅜-inch tip diameter, polyurethane sole								
Group III: Conventional shoe with five ¾-inch cleats, ⅜-inch tip diameter, Bowdoin heel, polyurethane sole								
Group IV: Soccer style, 15 cleats with ⅜-inch tip diameter, polyurethane sole								
Group V: Soccer style, 15 cleats with ½-inch tip diameter, polyurethane sole								

	Release Coefficients							
Shoe	**Grass**	**Grass Wet**	**AstroTurf**	**Astroturf Wet**	**Tartan Turf**	**Tartan Turf Wet**	**Poly Turf**	**Poly Turf Wet**
Group VI: Soccer style, 12 cleats (ten ⅜-inch length and two ½-inch length), ½-inch tip diameter, rubber sole								
Group VII: Soccer style, 49 to 121 cleats (⅜-inch or 5⁄16 -inch length), ½-inch tip diameter to pointed tips, rubber sole								

From Torg JS, Quedenfeld TC, Landau S: The shoe-surface interface and its relationship to football knee injuries. J Sports Med 2:261-269, 1974.

TABLE 25J-10 -- Relationship of Shoe-Turf Interface Release Coefficient to Incidence of Football Knee Injuries

Release Coefficient	0.60—	Not safe	
	0.50—		0.49
		Probably safe	
	0.40—		
		Probably safe	
	0.30—		0.31
		Safe	
	0.20—		
	0.10—		

From Torg JS, Quedenfeld TC, Landau S: The shoe-surface interface and its relationship to football knee injuries. J Sports Med 2:261-269, 1974.

Cawley and colleagues recently studied nine shoes by three manufacturers, which were characterized as turf, court, molded cleat, or traditional cleat and tested on both natural grass and synthetic turf. [405] They found that the cleated shoes (both traditional and molded) generated the highest frictional and torsional resistance on the grass surface when compared with the other categories of shoes. Grass generated higher peak moments than turf for the cleated shoes. These results demonstrate the considerable differences between laboratory and physiologic conditions and that the increase in frictional resistance is nonlinear with increasing loads.

Stanitski and associates determined the static coefficient of 16 different shoe-surface combinations. [402] They used a drag test of a size 13 shoe with a 25-pound load pulled both with and against the grain, across various sections of football fields. Their results are shown in Table 25J-11 . The coefficient of friction ranged from a high of l.54 for a Riddell At-31 (standard last, leather upper, molded plastic sole, 20⅜-inch conical cleats) on Polyturf to a low of 0.92 for the Puma 1430 ("soft last," molded rubber sole, 23⅜-inch cylindrical cleats with central indentations) on Tartan Turf and grass fields. These investigators found no grain effect and "essentially no change" when the surface was wet.

TABLE 25J-11 -- Friction Coefficients for Various Surfaces and Shoes

	Coefficient of Sliding Friction Surface			
Shoe	**Poly Turf**	**Astro Turf**	**Tartan Turf**	**Grass**
A	1.49	1.34	1.42	1.23
B	1.54	1.31	1.16	1.21
C	1.33	1.23	1.13	1.07
D	1.38	1.16	0.92	0.92

From Stanitski CL, McMaster JH, Ferguson RJ: Synthetic turf and grass: A comparative study. J Sports Med 2:22-26, 1974.

Bowers and Martin continued the study of cleat-surface friction, adding the new parameter of a new versus a worn synthetic surface. [233] The cleats from two different shoes were studied. One cleat was evaluated in both a slightly worn and a very worn state. Three similar shoes were mounted in a triangular pattern on a platform weighted from 2 to 14 pounds that was pulled across the new or worn turf. This study showed 16% more friction against the grain of a

5-year-old AstroTurf field when wet and 22% more friction when dry (Fig. 25J-28). From this study, the authors provided a formula for calculating coefficients of friction for individual shoes and surfaces that increased linearly with the number of cleats. They suggested that changes in friction altered player performance (e.g., smaller slip angles on wet turf) and that increasing friction could produce “foot lock” and result in increased injuries.

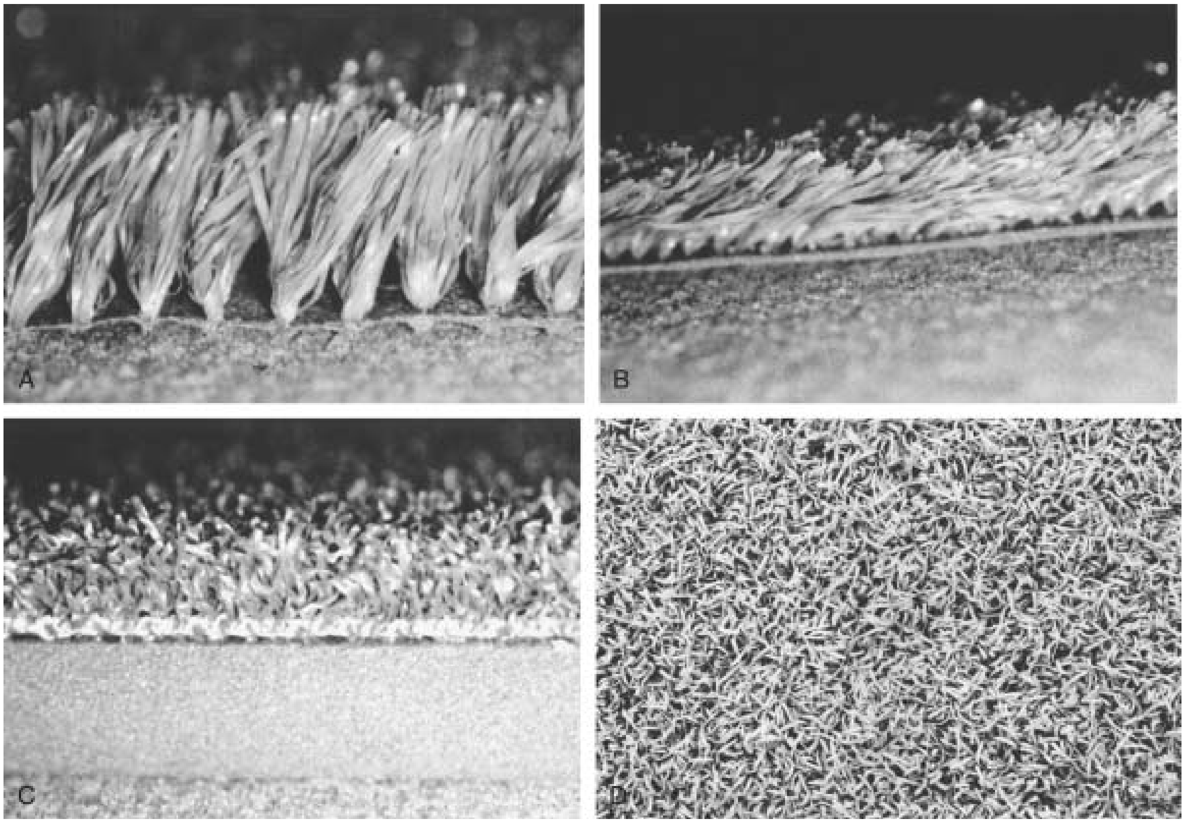

Figure 25J-28 AstroTurf evolution. A, Original AstroTurf (new) in its 1966 design with long nylon ribbon. B, Original AstroTurf showing its grain effect. C, Newer AstroTurf 8. D, Weave pattern of AstroTurf designed to prevent a grain effect. *(Photographs by Thomas O. Clanton.)*

Bonstingl and coworkers expanded the study of shoes and surfaces by looking at dynamic torque for 11 shoe types on four dry turf samples at two different player weights.[357] They used the swivel shoe of Cameron’s design, five styles of multicleated soccer shoes, four styles of noncleated basketball shoes, and a conventional football shoe for grass (seven ¾-inch plastic screw-on cleats with metal tips). The four surfaces were AstroTurf, Tartan Turf, Polyturf, and grass. The two player weights used for the normal force were 170 pounds (77 kg) and 200 pounds (91 kg). All combinations were tested for both toe stance and foot stance positions. They found that all shoes except the swivel shoe developed about 70% more torque in foot stance than in toe stance and that the higher player weight resulted in more torque. The conventional shoe tested on grass had among the highest torque. Although noncleated shoes generally had less torque for all playing surfaces, this was not absolutely true for all surfaces. This study proved that torque applied to an athlete’s leg depends on (1) the type and outsole design of the shoe, (2) the playing surface, (3) the player weight being supported, and (4) the foot stance assumed.

Culpepper and Niemann continued the study of the shoe-turf interface in 1983 by looking at the release coefficient for torque in several shoe-surface combinations.[344] They tested five different soccer-style shoes of variable cleat number and configurations on old and new Polyturf and new AstroTurf under wet and dry conditions. Loads ranging from 10 to 90 pounds were transferred down a metal shaft to a prosthetic foot on which the different shoes were mounted (Fig. 25J-29). The release coefficient of torque was calculated for 30 different conditions (Table 25J-12). The authors found that although a specific shoe did vary in its release coefficient ranking on different surfaces, *a shoe that had a low coefficient on one surface under one condition was generally lower on all three surfaces whether wet or dry, whereas a shoe that ranked higher did so for all conditions*.

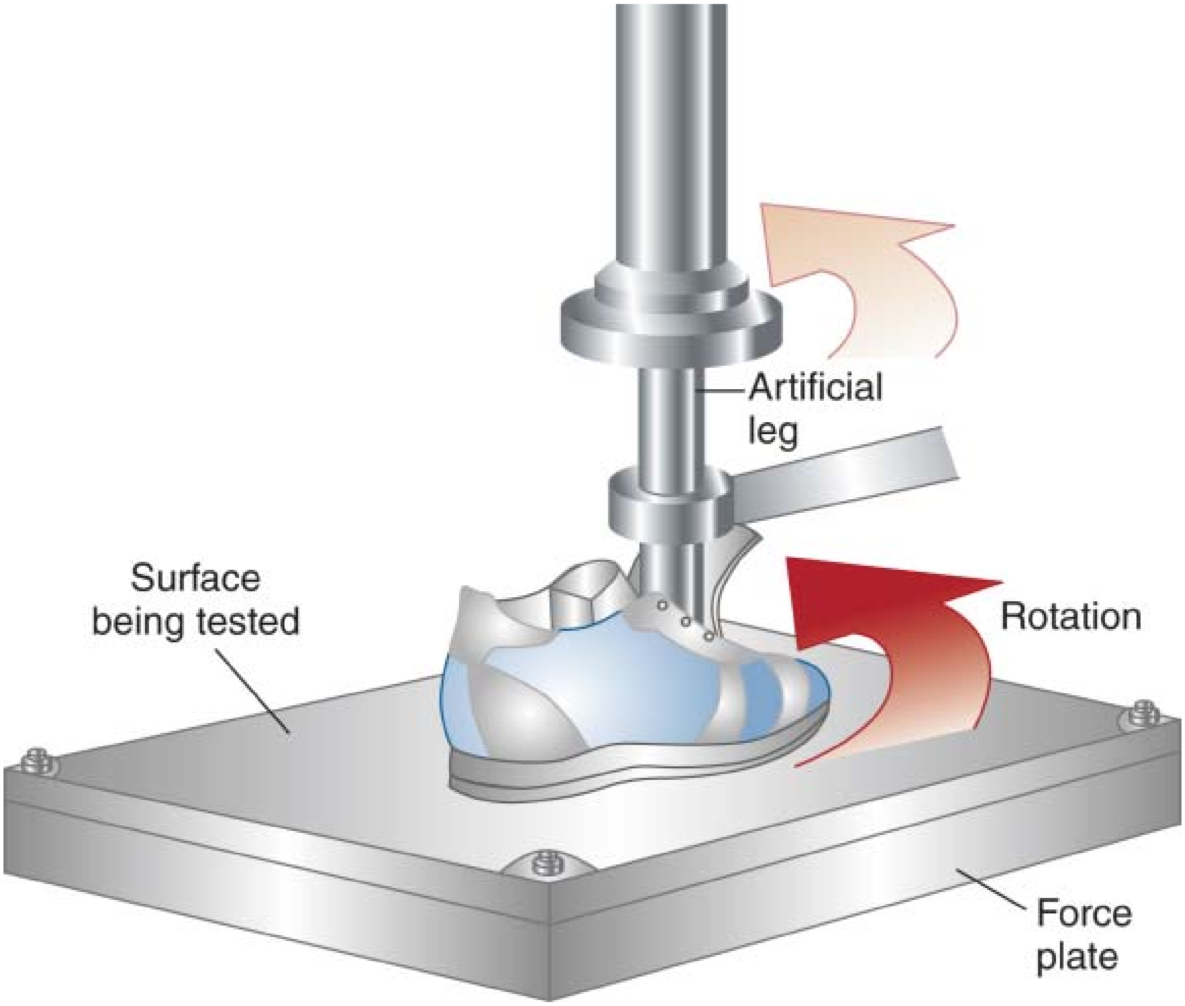

Figure 25J-29 Example of device for testing torque. *(Redrawn with permission from Physical Tests. Sport Research Review. Beaverton, Ore, Nike Sport Research Laboratory, Jan/Feb, 1990; and Van Gheluwe B, Deporte E, Hebbelinck M: Frictional forces and torques of soccer shoes on artificial turf. In Nigg BM, Kerr BA [eds]: Biomechanical Aspects of Sport Shoes and Playing Surfaces. Calgary, University of Calgary Press, 1983, pp 161-168.)*

TABLE 25J-12 -- Release Coefficients of 30 Shoe-Surface Combinations

	New Poly Turf		Old Poly Turf		New Astro Turf	
Shoe	**Dry**	**Wet**	**Dry**	**Wet**	**Dry**	**Wet**
A	0.27 ± 0.01	0.21 ± 0.01	0.24 ± 0.02	0.24 ± 0.02	0.19 ± 0.01	0.24 ± 0.01
B	0.35 ± 0.03	0.29 ± 0.01	0.33 ± 0.05	0.31 ± 0.01	0.38 ± 0.03	0.35 ± 0.02
C	0.21 ± 0.01	0.23 ± 0.01	0.29 ± 0.05	0.00 ± 0.01	0.19 ± 0.01	0.22 ± 0.02
D	0.32 ± 0.03	0.24 ± 0.01	0.32 ± 0.02	0.31 ± 0.01	0.34 ± 0.03	0.26 ± 0.01
E	0.28 ± 0.01	0.29 ± 0.01	0.30 ± 0.01	0.25 ± 0.02	0.26 ± 0.01	0.28 ± 0.01

Date from Culpepper MI, Niemann KMW: An investigation of the shoe-turf interface using different types of shoes on Poly-turf and Astro-turf: Torque and release coefficients. Alabama J Med Sci 20:387-390, 1983.

Another study of both static friction and torque appeared in 1983, when Van Gheluwe and colleagues tested nine different shoes on three varieties of artificial turf in both the toe stance and foot stance positions. [238] In their analysis, the authors demonstrated higher values for friction and torque for AstroTurf compared with the other surfaces (AstroTurf scored highest in 22 of 36, or 61%, of the test conditions). They attributed this result to the nylon fiber of AstroTurf compared with the polypropylene fiber used in the other surfaces. This confirmed the work of Bonstingl and colleagues; however, the results violate the law of physics for dry friction, which predicts the linear relationship for friction between two surfaces. This contradiction is significant when interpreting the results of other studies that have assumed this linear relationship.

Andreasson and coworkers in Sweden published a more detailed study in 1986 evaluating torque and friction in a dynamic mode on an artificial surface.[231] This was the first study to specifically add dynamic torque by using a rotating disk on which the surface was applied to simulate speed from walking to running. Twenty-five different shoes were tested, including running shoes, tennis shoes, soccer shoes for artificial turf, and soccer shoes for natural turf. Torque in toe stance varied from a low of less than 10 Nm for one of the running shoes and one of the multicleated soccer shoes to a high of more than 50 Nm for another multicleated soccer shoe placed against the grain of the Poligrass test surface. The authors showed that the frictional force is independent of speed between 1 and 5 m/second, but this is below the 7 to 10 m/second speeds that occur in modern football and soccer. Torque was generally lower for shoes with polypropylene outsoles compared with polyurethane and rubber-like soles.

Nigg and Segesser reported in 1988 on the variation in friction related to a change in load.[360] They found when studying the six different surfaces considered for the Toronto SkyDome playing surface that the static coefficient of friction changed from lows of 1.13 for 280 N normal loads to highs of 3.48 for 769 N loads (Table 25J-13).[360] The tested surfaces ranked differently for the two load conditions. This result indicates the complexity of using material tests for choosing a playing surface because the individuals playing vary by a factor of 2 or more in weight and generate forces that may be well beyond those studied to date in laboratory tests.[400]

TABLE 25J-13 -- Variation in Static Coefficient of Friction for Translation with Variation in Vertical Load Using Football Shoes on Six Different Playing Surfaces (A through F)

	Static Coefficient of Friction (Translational)					
280	**1.13**	**1.42**	**1.30**	**1.30**	**1.56**	**1.51**
769	3.15	2.90	2.57	2.57	3.48	3.15

From Nigg BM, Segesser B: The influence of playing surfaces on the load on the locomotor system and on football and tennis injuries. Sports Med 5:375-385, 1988.

Just as there are differences between natural and artificial grass that affect the frictional properties of the playing surface, similar differences have been known for other sport surfaces. Rheinstein and colleagues looked at static drag and dynamic torque characteristics of different shoes on two basketball surfaces (Fig. 25J-30).[347] The three playing surfaces tested were clean hardwood, dusty hardwood, and clean artificial flooring (Tartan indoor surface manufactured by the 3M Corporation). Polyurethane soles produce less torque than the elastomer outsoles for all weight, floor, and surface condition parameters. Torque also decreased with increasing sole hardness for the elastomer outsoles on the clean hardwood and artificial surface. As expected, greater player weight increased torque, and dust on the hardwood flooring cut torque almost in half. The polyurethane soles were considerably less affected by dust than the elastomer outsoles. One would expect that high friction and torque could overload the athlete and result in injury. These findings have obvious implications for performance as well as prevention of injury in sports medicine.

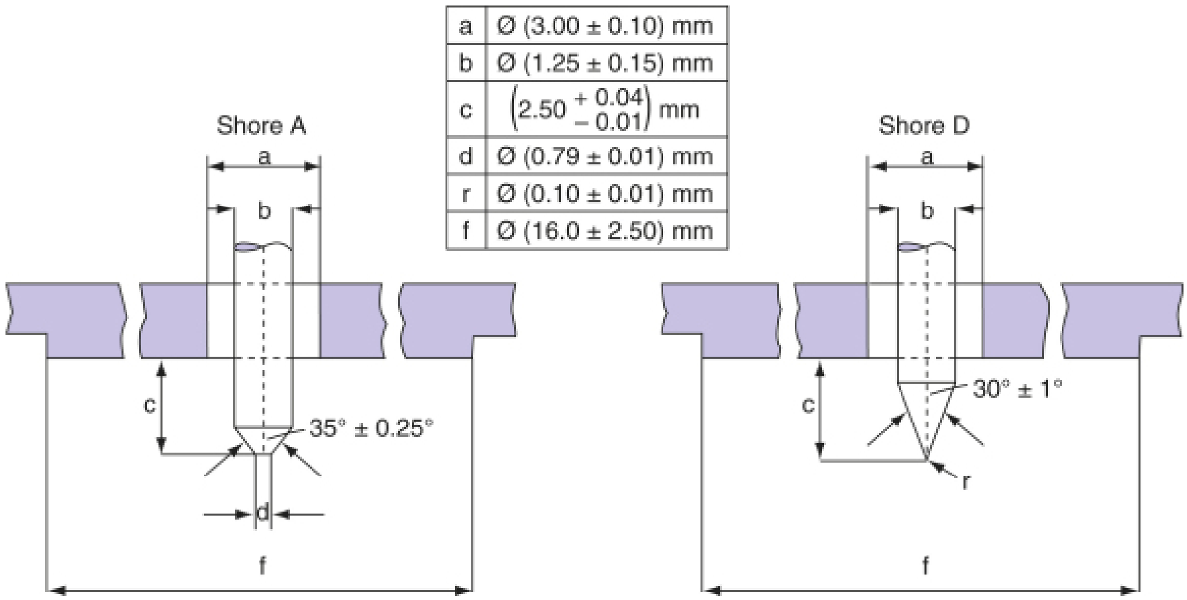

Figure 25J-30 Methods for measuring shore hardness. *(Redrawn with permission from Denoth J: Load on the locomotor system and modelling. In Nigg BM [ed]: Biomechanics of Running Shoes. Champaign, Ill, Human Kinetics Publishers, 1986, p 97. © 1986 by Benno M. Nigg.)*

Nigg and associates measured the coefficients of static and sliding friction in 1980 for five different surfaces and three styles of shoewear. [348] The dynamic, or sliding, coefficient of friction ranged from a low of 0.3 on sand (clay) with an indoor tennis shoe or a treaded jogging shoe to a high of 1.20 on a polyvinylchloride floor with an all-around shoe (see Table 25J-7). Nigg expanded on this work in 1986 and has used these studies to emphasize the dominant role of the playing surface on the translational friction coefficient, although he acknowledged the open question of whether the structure or material, or both, are most responsible for the result. [218]

In a separate study, Nigg and colleagues measured torque for 12 subjects rotating 180 degrees on one leg on seven different sport surfaces and with eight different types of shoes. [303] Mean values for the different surfaces are shown in Figure 25J-20 . The range varied from 20 Nm to 38 Nm, the highest torque being found on artificial grass. In an expanded study using average torque for five tested surfaces (10 Nm to 20 Nm) and average torque for 10 tested shoes (13 Nm to 18 Nm), Nigg proposed that torque was shoe and surface codependent to a greater degree than translational friction. [218] In attempting to correlate the material tests for translational friction with the subject tests for rotational friction, Yeadon and Nigg found no relationship. [397] This study demonstrates the difficulty of combining material and subject tests, as occurs also in tests of impact load, in which the subject's response to a test condition may alter the result. [394]

Synthetic playing surfaces with rubber or sand infill are now used on many athletic fields such as soccer, football and rugby. Although these surfaces may come closer to the mechanical characteristics of a true grass playing surface than the older turf designs, their potential effects on lower extremity biomechanics and related injury rates necessitate further study.

With the continued introduction of different playing surfaces, the relentless study the shoe-surface interaction can only help improve athlete safety. It does, however, raise some interesting questions. Does this put schools that cannot afford multiple types of player surfaces at a measurable disadvantage? Does it put athletes at greater risk for injury when they are not afforded the best playing surface for a given game condition? Does the use of a shoe on surfaces for which it is not designed carry liability exposure for the school? Does there exist a shoe or surface that is "the best" for a particular sport?

Clinical Relevance

In 1980, Nigg and associates reported the results of a retrospective study of tennis injuries using a questionnaire. [348] Using one tennis player during one 6-month season as a single case, they analyzed 2481 cases to determine the relationship between injury and playing surface. The foot and ankle were the most commonly affected area when ankle joint, Achilles tendon, heel, and sole cases were combined (Fig. 25J-31). The authors factored in the variation and frequency of play for the different surfaces by determining the relative frequency of pain per hour per week (Table 25J-14). [360] Their data showed that the lowest frequency of injury occurred on clay and synthetic clay surfaces, and a lower frequency of pain occurred on asphalt or concrete than on carpet or synthetic grill. Using this information, Nigg and Segesser speculated that the compliance of a surface is less important in tennis injuries than its frictional properties. [360]

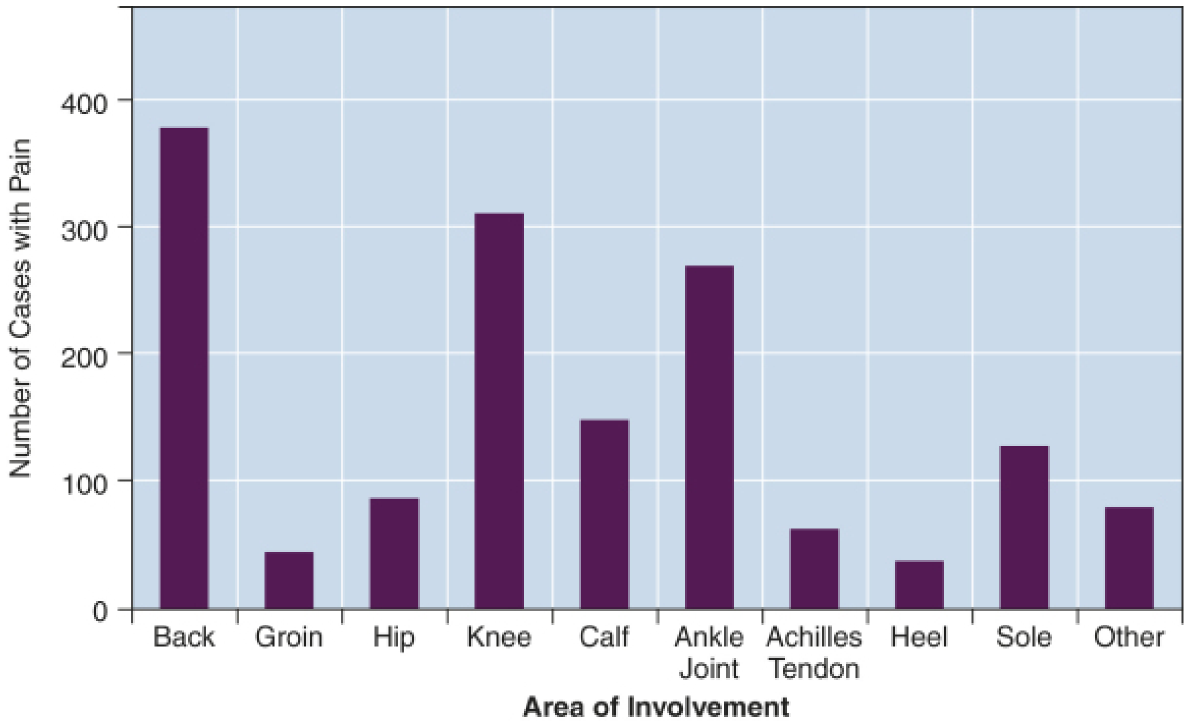

Figure 25J-31 Number of cases of tennis players with back and lower extremity pain by area of involvement. *(Redrawn with permission from Nigg BM, Segesser B: The influence of playing surfaces on the load on the locomotor system and on football and tennis injuries. Sports Med 5:375-385, 1988.)*

TABLE 25J-14 -- Frequency and Relative Frequency of Pain in Tennis Players Related to Six Different Playing Surfaces

Surface	Frequency of Pain (%)	Relative Frequency (%/hr/wk)
Clay	2.2	0.5
Synthetic sand	3.0	1.6
Synthetic Surface	10.7	3.0
Asphalt	14.5	3.9
Felt carpet	14.8	4.8
Synthetic grill	18.0	3.8

From Nigg BM, Segesser B: The influence of playing surfaces on the load on the locomotor system and on football and tennis injuries. Sports Med 5:75-385, 1988.

Studies of the frictional properties of track surfaces have also yielded interesting insight on performance and injury. Stucke and colleagues pointed out the importance of static friction coefficients for starting efficiency in running events. [403] When the static coefficient of friction is small, shorter steps and less body lean are used. The authors measured the static coefficient of friction for starting, stopping, and turning during 100 trials using five subjects wearing the same shoe type. They found that the cinder track had an intermediate value between 0.65 and l.72, compared with the synthetic outdoor surfaces' values of 0.8 to 2.22 and the synthetic indoor surface values of 0.54 to 1.47 (Fig. 25J-32). This study emphasizes the variability between surfaces by pointing out that an artificial surface does not automatically indicate a surface with greater frictional stresses. Automatic changes in movement technique are influenced by the variation in surface friction properties. The authors speculated that the use of surfaces of varying frictional properties during training and competition is disadvantageous because of the time necessary to perfect a repertoire of movement skills to meet the requirements of different sport surfaces. This conclusion begs the question of whether it is preferable to train on one surface (e.g., natural grass) to reduce injury exposure when contests will be held on a different surface (e.g., artificial grass), or to train on the same surface on which the contest will be conducted with scheduled training in a sequence of graduated stress to allow proper adaptation by the body.

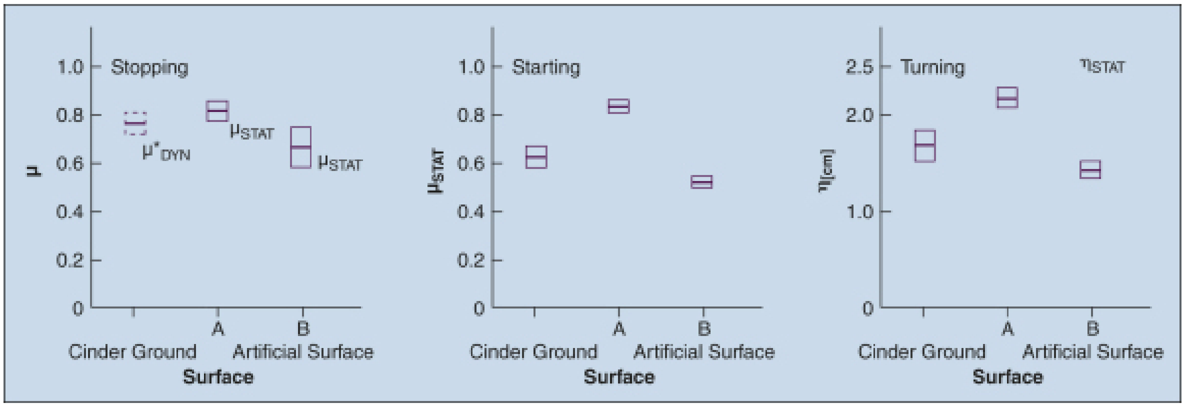

Figure 25J-32 Variation in coefficient of friction between three different track surfaces for starting, stopping, and turning. μ, coefficient of friction for translation; η, coefficient of friction for rotation; DYN, dynamic; STAT, static. *(Redrawn with permission from Stucke H, Baudzus W, Baumann W: On friction characteristics of playing surfaces. In Frederick EC [ed]: Sport Shoes and Playing Surfaces. Champaign, Ill, Human Kinetics Publishers, 1984, pp 91-96. © Nike, Inc.)*

When frictional properties of a surface are too low, slipping can occur, and injuries may result. [360] When the frictional resistance is too high, the load transference to the body may exceed its range of tolerance, resulting in injury. An optimal range between excessive frictional overload and lack of traction exists to prevent injury. Nigg has suggested optimal ranges for the coefficient of translational friction for various sports. 360 He based his recommendations on both objective and subjective assessments, and the range was always between 0.5 and 0.7. Stussi and colleagues calculated the coefficients of static friction to be 0.6 and sliding friction to be 0.5 for clay; values on fabric courts approached 1.0. [305] Ankle strain is reduced, according to Stussi and colleagues, during braking maneuvers on sandy courts. They acknowledged that greater performance demands might require increasing coefficients of friction and suggested that more stable shoewear (e.g., with improved ankle support) might allow the player to tolerate the greater strain of the higher friction surfaces. Owing to the epidemiologic flaws in the study of tennis injuries by Nigg and coworkers, [360] the final answer on the relationship between the frictional properties of playing surfaces and athletic injuries has not been determined, but the stage is certainly set for such a study.

SUMMARY

This discussion of the etiologic factors involved in the foot and ankle injuries should introduce the concepts necessary to understand the injuries discussed in the other sections in this chapter. It is only after one understands the underlying causes of a problem that solutions are forthcoming. In the foot and ankle, as in no other area of the body, there is a direct interaction between anatomy and the environment—between the foot and ankle and shoewear, and between shoewear and the playing surface. As the reader investigates the specific injuries and pathologic conditions that beset athletes in sports, he or she should keep in mind the individual nature of these injuries and their potential risk factors. When causes are discovered, prevention is only a step behind.

Critical Points

- It is our job as physicians to act as educators for coaches, therapists, and athletic trainers so that they understand the benefit of returning an athlete to sport participation at the appropriate time after injury. Likewise, we should point out the risks involved when criteria for return to competition are ignored.

- Warming up and stretching to obtain or maintain this range may or may not prevent injury, but stretching beyond this range is potentially harmful.
- The role of flexibility in foot and ankle injuries is unclear.
- A lack of cushioning *may* be a factor in producing injury but probably not to the degree to which some researchers and shoe manufacturers might lead us to believe.
- There is more evidence supporting the link between shoewear control and injury incidence than that of shoewear cushioning.
- The compliance of a surface may be less important in injuries than the frictional properties of a surface.
- The optimal ranges for the coefficient of translational friction for a playing surfaces is likely between 0.5 and 0.7.

SUGGESTED READINGS

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