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Overuse injuries: tendinopathies, stress fractures, compartment syndrome, and shin splints

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Approximately 50% of all sports injuries are secondary to overuse [1]. The frequency of overuse injuries evaluated in primary care sports medicine clinics is even greater, reportedly up to twice the frequency of acute injuries [2]. The majority of injuries evaluated in running injury clinics are related to overuse [3,4], and approximately half of these involve the lower leg (20%), ankle (15%), and foot (15%) [5,6].

Overuse injuries result from repetitive microtrauma that leads to local tissue damage in the form of cellular and extracellular degeneration, and are most likely to occur when an athlete changes the mode, intensity, or duration of training—a phenomenon described as the ''principle of transition'' [7,8]. Physical training uses prescribed periods of intense activity to induce the desired goal of ''supercompensation'' or performance improvement. A mismatch between overload and recovery can lead to breakdown on a cellular, extracellular, or systemic level, however. At the cellular level, repetitive overload on tissues that fail to adapt to new or increased demands can lead to tissue breakdown and overuse injury. It is important to realize that, in theory, this subclinical tissue damage can accumulate for some time before the person experiences pain and becomes symptomatic. On the systemic level, rapid increases in training load without adequate recovery may cause a global ''overtraining syndrome.'' Strong predictors of overuse musculoskeletal injury include a previous history of injury as well as walking or running more than 20 miles per week [9].

Both intrinsic and extrinsic factors contribute to overuse injuries. Intrinsic factors are biomechanical abnormalities unique to a particular athlete and include such features as malalignments, muscle imbalance, inflexibility, weakness, and instability. High arches, for example, have been demonstrated to predispose to a

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greater risk of musculoskeletal overuse injury than low arches (''flat feet'') in military recruits [10]. Extrinsic (avoidable) factors that commonly contribute to overload include poor technique, improper equipment, and improper changes in the duration or frequency of activity. These improper changes in activity duration/frequency or ''training errors'' are the most common causes of overuse injuries in recreational athletes. Vulnerability to extrinsic overload varies with the intrinsic risk factors of an individual athlete [7].

Sports-acquired deficiencies, categorized as an extrinsic risk factor, actually represent the product of biomechanical abnormalities and training errors. Because sports activity can overload an athlete's musculoskeletal system in predictable ways, athletic repetition without proper conditioning can propagate muscular imbalance and flexibility deficits.

Injuries are often related to biomechanical abnormalities removed from the specific site of injury, underscoring the importance of evaluation of the entire kinetic chain [11].

Common overuse injuries of the lower leg, ankle, and foot include tendinopathies, stress fractures, chronic exertional compartment syndrome, and shin splints.

Tendinopathies

Tendinopathies of the foot and ankle are relatively common and encompass a wide spectrum of maladies ranging from tendinitis (acute inflammation of the tendon) to tendinosis (chronic degeneration) to tenosynovitis (inflammation of tendon sheath) to partial and complete ruptures. Each one of these disorders is distinct, although they may be seen in combination. Four tendons in the foot and ankle—the Achilles, posterior tibialis, peroneal brevis, and peroneal longus tendons—are most often involved. In contrast to acute traumatic tendinous injury, sport-related injuries most often involve repetitive submaximal loading of the tissues, resulting in repetitive microtrauma. An understanding of the anatomical pathophysiologic basis of these maladies is critical to their diagnosis and management.

Histopathology

The debate over the nomenclature of chronic tendon injury highlights some of the unresolved issues in the histopathology, etiology, and management of tendinopathies of the foot and ankle. Although the pathological label ''tendinosis'' has been in use for more than 25 years to describe collagen degeneration in tendinopathy [12], many clinicians still use the term ''tendinitis'' to describe painful chronic overuse injury, implying that the fundamental problem is inflammatory [13]. Maffulli, Khan, and Puddu advocate the use of the term tendinopathy as a generic descriptor of clinical conditions such as pain, swelling, and impaired performance in and around tendons arising from overuse, with the labels tendinosis and tendinitis most appropriately applied after histopathological examination [14].

This nomenclature separates chronic degeneration of tendons from acute and mainly inflammatory processes, with implications for treatment and management.

Tendinosis has been described as a failure of cell matrix adaptation to trauma because of an imbalance between matrix degeneration and synthesis [8]. The classic pathology is a loss of the normal collagenous architecture and replacement with an amorphous mucinous material that lacks the parallel, longitudinal architecture of normal tendon [15,16]. Histological examination reveals intratendinous collagen degeneration with fiber disorientation and thinning, hypercellularity, scattered vascular ingrowth, increases in the amount of ground substance and the proteoglycan concentration of ground substance, and a decrease in the ratio of Type I to Type III collagen [15,17,18]. Any inflammatory response or the presence of inflammatory cells is notably lacking in tissue samples of tendinopathy, differentiating tendinosis, or chronic overuse pathology from acute injury and tendinitis. Astrom and Rausing describe the major lesion in chronic Achilles tendinopathy as ''a degenerative process characterized by a curious absence of inflammatory cells and a poor healing response'' [18]. Similar histopathological findings in posterior tibial tendon dysfunction of a degenerative tendinosis with mucinous degeneration, fibroblast hypercellularity and neovascularization [19], and higher proportions of collagen Type III at the expense of collagen Type I [20] support the notion of a common disease process in overuse tendon injury, leading ultimately to tendon degeneration and an insufficient repair response.

Functionally, whereas the healing response to an acute tendon injury involves an organized triphasic response of inflammation, proliferation, and maturation, the response to an overuse injury involves an inadequate, incomplete, and disorganized repair mechanism resulting in a substantially defective ''repaired'' tendon lacking in extracellular tissue organization, with decreased resistive strength and more vulnerability to further injury. Although the exact role of overuse in the pathogenesis of chronic tendon injuries and disorders is not completely understood, it is speculated that fatigued tendon loses its basal reparative ability with intensive repetitive activity, often eccentric in nature, leading to cumulative microtrauma that further weakens the collagen cross-linking and noncollagenous matrix and disturbs the micro- and macrovasculature of the tendon [21]. Ensuing local tissue hypoxia and impaired nutrition and energy metabolism likely play an important role in the sequence of events leading to tendon degeneration [13,21]. Leadbetter has called this the ''tendinosis cycle'' [8]. One of the first animal models of tendinopathy, developed by Soslowsky et al [22,23], has shown persistent microscopic changes of tendinosis in rat rotator-cuff supraspinatus tendon after exposure to multiple factors, including impingement and overuse [23]. Neither impingement alone nor overuse alone produced the same degree of changes, implying a multifactorial etiology for the pathologic effects of overuse on rotator cuff tendon.

Current thinking supports the belief that a spontaneous tendon rupture is a typical end-state manifestation of degenerative processes in the tendon tissue [21], with partial macroscopic tears as a stage in the continuum of tendon degeneration [14]. Analysis of surgical specimens of Achilles tendons reveals that, although ruptured and tendinopathic tendons are histologically significantly more degen-

erated than control tendons, the general pattern of degeneration seen is common to the ruptured and tendinopathic tendons, suggesting the possibility of a common, as yet unidentified, pathological mechanism acting on both tendon populations [24].

Laboratory and molecular analyses of tendinopathy have begun to reveal strategies that may guide future clinical management of overuse tendon injury. It has been hypothesized in the past that tendon degeneration may be preceded by acute and then chronic phases of inflammatory "tendinitis" [12,25,26]. Although no inflammatory infiltration has been observed in multiple studies of biopsy specimens of tendinopathic tendons, recent in-vitro work demonstrates that a "molecular inflammation cascade" mediated by IL-1 beta in human tendon cells can induce connective tissue cell expression of cytokines that further induces known matrix destructive enzymes such as matrix metalloproteinases (MMP-1 and MMP-3) [27]. Clinically, the activity of metalloproteinases in tendon destruction and degeneration is the target of the use of injectable aprotinin, a metalloproteinase inhibitor, in the setting of patellar and Achilles tendinopathy as an alternative to corticosteroid therapy [28,29]. Apoptosis, mediated by overuse-induced, stress-activated protein kinases, may also play a role in tendon degeneration and weakening, presenting another set of molecular targets for future therapies aimed at preventing or treating tendinopathy more effectively [16,30,31].

Achilles tendon disorders

Commonly and inappropriately generalized as "Achilles tendinitis" by many clinicians, posterior heel pain in the setting of an overuse injury of the foot and ankle actually encompasses a spectrum of distinct and often coexistent pathological disorders with both inflammatory and degenerative etiologies [32]. The classification system set forth by Puddu et al separates degenerative conditions of the tendon itself (tendinosis with or without partial rupture) from inflammation of the paratenon (paratenonitis), inflammation of the tendon substance at its insertion (insertional tendonitis), and inflammation of the commonly afflicted bursa anterior to the insertion of the Achilles tendon on the calcaneus (retrocalcaneal bursitis) from complete tears caused by acute injury [12].

Puddu reserves a mixed category for paratenonitis with tendinosis including degeneration, partial tears, and calcification within the tendon [12], and Maffulli et al agree that clinically observed tendinopathy should include both of the histopathological entities peritendonitis and tendinosis [14]. According to Paavola et al, this suggestion has a sound basis because the clinical rationale to differentiate the histopathologic entities of Achilles peritendinitis and tendinosis is an uncertain one and there have been no randomized controlled studies comparing the outcomes of treatment or the natural history of these two conditions [25]. In clinical studies, the most common diagnosis of Achilles disorders was tendinopathy (55%–66%), followed by insertional problems (retrocalcaneal bursitis and insertional tendinopathy) (20%–25%) [25,33–36].

Achilles tendon disorders occur most often in athletes, and most often in those involved in running sports. An annual incidence of Achilles tendon disorders of 7%

to 9% in top-level runners has been reported [6,37]. Kvist reviewed cases of 455 athletes with Achilles tendon disorders [33]. Eighty-nine percent of the athletes studied were men, with 53% involved in running sports and 11% involved in soccer. The rest of the patients were involved in other sports in which running was an important training means [13,33]. Interestingly, malalignment of the lower extremity was found in 60% of the athletes with Achilles tendon disorders [25,33,36].

Many intrinsic and extrinsic etiologic factors have been proposed to account for the development of Achilles tendon disorders. Common intrinsic etiologies invoked include various alignment and biomechanical faults, including hyperpronation of the foot, limited mobility of subtalar joints and limited range of motion of the ankle joint, leg-length discrepancy, varus deformity of the forefoot and increased hindfoot inversion, decreased ankle dorsiflexion with the knee in extension, poor vascularity, genetic makeup, and gender, age, endocrine, or metabolic factors [33,34,38–40]. Changes in training pattern, poor technique, monotonous, asymmetric, and specialized training, previous injuries, footwear, and environmental factors such as training on hard, slippery, or slanting surfaces have been cited by many authors as extrinsic factors which may predispose the athlete to tendinopathy. Training errors have been reported to be involved in 60% to 80% of runners who have tendon overuse injuries [25]. Training errors cited include running a distance that is too long, running at an intensity that is too high, increasing distance too greatly or intensity too rapidly, and performing too much uphill or downhill work [25,34,40,41]. Training errors, alignment, biomechanics, and extrinsic factors such as footwear and training surfaces can create microtrauma resulting from nonuniform stress within the Achilles tendon from different individual force contributions of the gastrocnemius and soleus, producing abnormal load concentrations within the tendon and frictional forces between the fibrils, and leading to localized fiber damage [13,42]. In this manner, excessive motion of the hindfoot in the frontal plane, especially a heel strike with excessive compensatory pronation, is thought to cause a "whipping action" on the Achilles tendon and predispose it to tendinopathy [13,43].

A complete discussion of the etiological factors of Achilles tendon disorders must include the caveat that the exact pathogenesis of Achilles tendinopathy and other disorders remains largely unknown [25]. In addition to the epidemiological studies showing Achilles tendon disorders in athletes, other studies have shown a significant incidence of Achilles tendinopathy in nonathletes and middle aged men with sedentary lifestyles [18,44]. In the absence of a true inflammatory reaction in chronic Achilles tendinopathy, the etiology of pain, the most limiting factor and usual chief complaint of patients with Achilles tendon disorders, is not well understood [45,46]. Puddu et al have shown that long-standing degeneration of the tendon can occur without clinical symptoms or pain, and yet tendinopathy can become symptomatic with the introduction of heavy training [12].

History and physical examination play a key role in the diagnosis of Achilles tendon injury. The onset of pain, its duration, and aggravating factors should be documented. A classic history involves an insidious and gradual increase in pain

located 2 cm to 6 cm proximal to the insertion of the tendon and felt after exercise within days of a change in activity levels or training techniques. Rest often relieves symptoms, but return to activity reactivates the pain, generally within a few training sessions. In patients with advanced tendinopathy, pain may occur during exercise, and when severe, may interfere with the activities of daily living. Runners typically experience pain at the beginning and at the end of a training session, with a period of diminished discomfort in between.

Clinical examination should start by the exposure of both legs from above the knees, and the patient should be examined standing and prone. Careful inspection should reveal malalignment, deformity, areas of swelling, obvious asymmetry in the size of the tendon, localized thickening, erythema, and any previous scars. Palpation should document contours of the tendons, tenderness, thickening, palpable tendon nodules or defects, crepitation, and warmth. Biomechanics of the foot, ankle, and leg during walking and running, including slow motion analysis, should be evaluated in athletes. All patients should be examined for evidence of ankle instability [25]. The ''painful arc'' sign may help to distinguish between lesions of the tendon and paratendon [47]. Whereas peritendinitis is characterized by crepitus, exquisite tenderness, and swelling that does not move with tendon action, chronic Achilles tendinopathy is notable for absence of crepitation and swelling, with focal tender nodules that move as the ankle is dorsiflexed and plantar flexed [47]. The VISA-A scale is a subjective, quantitative scale of symptoms and dysfunction in the Achilles tendon and may be a useful tool to assess and follow symptomatology over time [48].

Both ultrasound and magnetic resonance imaging (MRI) play a role in the diagnosis of Achilles tendon disorders. Ultrasonography provides an inexpensive, sensitive analysis of the pathology of the Achilles tendon, with data regarding tendon width, water content within the tendon and peritendon, and collagen integrity. Abnormal tendons may have increased tendon diameter, focal hypoechoic intratendinous areas (areas of increased water content which at surgery have been shown to be degenerated tissue), localized tendon swelling and thickening, collagen discontinuity, and tendon sheath swelling or calcifications [25]. In the acute phase, ultrasound examination may reveal fluid surrounding the tendon. In the chronic phase, thickening of the hypoechoic paratenon may be seen, although ultrasonography has not been shown to reliably differentiate focal tendinosis from partial rupture [49]. Abnormalities detected by ultrasonography in an asymptomatic Achilles tendon can accurately presage the development of Achilles tendinopathy [50].

Magnetic resonance imaging provides extensive information on the internal morphology of the tendon and the surrounding structures and is used often for evaluation before surgical intervention [32]. MRI can also help characterize retrocalcaneal bursitis and insertional tendinitis. In patients with chronic tendinopathy, MRI often reveals tendon thickening and increased signal within the Achilles tendon. Areas of mucoid degeneration are shown on MRI as a zone of high signal intensity on T1- and T2-weighted images. Two caveats on MRI interpretation in Achilles tendon disorders include the unreliability of MRI to demonstrate

changes of paratenonitis [51] and the temptation of the clinician to mistake areas of increased signal on MRI for pathologic, clinically significant foci rather than asymptomatic areas of degeneration [32].

The goals of treatment in Achilles tendinopathy are threefold: (1) to minimize the pain, (2) to prevent further degeneration, and (3) to allow return to baseline activity. In athletes, an additional demand is that the recovery time should be as short as possible [25]. Initial conservative management aims to relieve symptoms and correct factors causing load imbalance and repetitive strain on the tendon and surrounding structures. This includes a combination of strategies aimed at controlling inflammation and correcting training errors, limb malalignment, decreased flexibility, and muscle weakness, and the use of appropriate equipment during sports [25,52]. The role of anti-inflammatory therapy such as oral nonsteroidal anti-inflammatory drugs (NSAIDS) or steroid pain relievers to control inflammation remains controversial. Although no inflammatory infiltrate has been documented in histological analyses of tendinopathic samples, anti-inflammatory medication does help to diminish pain and facilitate rehabilitation in cases of chronic tendinopathy and most certainly has a place in the management of retrocalcaneal bursitis and insertional tendinitis [17,53]. Cryotherapy has also been shown to be useful to help control inflammation and facilitate therapy in tendinopathy [54]. Occasionally, complete rest or cessation of the training that caused the symptoms may be required for a short time to settle severe symptoms. Because the repair and remodeling of collagen fibers are stimulated by loading of the tendon, only very short courses of complete rest should be prescribed. Heparin may be injected to prevent fibrin exudate in the paratenon region [17]. Recently, Ohberg and Alfredson have described successful ultrasound-guided injection of polidocanol, a sclerosing agent, to decrease the neovascularization and symptomotology of chronic midportion Achilles tendinosis [55].

Appropriate and progressive exercises using eccentric exercise programs targeting specific muscle hypertrophy, speed, strength, and endurance requirements represent the gold standard for Achilles tendon rehabilitation and appear to be effective in most athletes [56]. Mafi et al have shown prospectively that a program of eccentric calf muscle training was superior to concentric training in Achilles tendinosis [57]. Correction of biomechanical imperfections is clinically important, even if their effects on tendonitis are unclear. Interventions improving flexibility of the ankle joint, flexibility of calf muscles, amount or speed of foot pronation, and foot and ankle mechanics (with orthotics) have been implicated in ameliorating symptoms of tendinopathies.

Operative treatment is recommended for patients who do not respond adequately to a 3- to 6-month trial of appropriate conservative treatment. To date, no prospective randomized controlled studies comparing operative and conservative treatment of Achilles tendinopathy have been published [25]. Surgery for overuse tendinopathies usually involves excision of fibrotic adhesions and degenerated nodules, or decompression of the tendon by longitudinal tenotomies. Reconstructive procedures may be necessary if large nodules and lesions are excised. Some authors have used open or percutaneous multiple longitudinal incisions of the

tendon [58]. In most studies, satisfactory results in 75% to 100% of the patients have been reported after operative intervention of Achilles tendinopathy [25]. Generally, long-standing tendinopathies are associated with poorer surgical outcomes [51,58]. Recently, an overall complication rate of 11% was documented in a series of 432 consecutive patients [59].

Although much is known about Achilles tendon disorders, and much more is known about Achilles tendinopathies than about other tendinopathies of the foot and ankle, ample opportunity exists for better controlled studies to examine operative and conservative treatment regimens, postoperative rehabilitation protocols, and long-term follow-up of clinical interventions. In addition, further study of the etiologies of Achilles tendon disorders, the causative factors in the pain of chronic tendon injury, and the factors contributing to the histopathology of tendinopathy could help identify therapeutic targets for molecular medicine.

Posterior tibial tendon dysfunction

Posterior tibial tendon dysfunction (PTTD) is a common cause of painful acquired flatfoot deformity in adults and is associated with substantial functional problems resulting in significant morbidity. These patients typically have a loss of hindfoot inversion, inability to negotiate uneven ground, climb, and descend stairs [60]. As acquired flatfoot syndrome advances, progressive collapse of the medial longitudinal arch, hindfoot valgus, and forefoot abduction abnormalities are noted [19]. Shoe fitting is difficult. Pain and instability in the hindfoot have significant impact on daily routines [60].

Johnson and Strom [61] have described three distinct stages of posterior tibial tendon dysfunction. In Stage I, the patient has pain and swelling along the course of the tendon. Because the length of the tendon is normal, the patient is able to perform single heel raise. The flatfoot deformity is minimal, the alignment of the hindfoot-forefoot complex is normal and the subtalar joint remains flexible [19,62]. In Stage II, the patient is unable to perform a single heel raise because of attenuation or disruption of the posterior tibial tendon. The tendon is enlarged and elongated and functionally incompetent. The foot has adopted a pes planovalgus position with collapse of the medial longitudinal arch, hindfoot valgus and subtalar joint eversion, and forefoot abduction through the talonavicular joint. The subtalar joint remains flexible, and the hallmark of this stage is that with the ankle in equinus, the talonavicular joint can be reduced [19,62]. Stage III disease presents with the patient unable to perform a single heel raise and a more severe flatfoot deformity. In Stage III disease, the pes planovalgus deformity is fixed and the laterally subluxed navicular cannot be reduced [19,62].

The histopathology of PTTD reveals a degenerative intratendinous process similar to that seen in Achilles tendinopathy. Mosier et al studied 15 normal cadaveric tendons and 15 surgical specimens from patients with Stage II PTTD [63]. Four types of histopathology were present in the disease samples: (1) increased mucin content, (2) hypercellularity, (3) neovascularization, and (4) chondroid metaplasia. Disruption of the normal array of collagen bundles

represented a degenerative tendinosis with a nonspecific reparative response to tissue injury [63].

Both ultrasound and magnetic resonance imaging can play a role in the diagnosis of PTTD. According to Perry et al, ultrasonography can positively identify peritendinitis and tendonitis. Tendon interruption, or inhomogeneity of tendon by MRI assessment, however, remains more sensitive than either clinical or ultrasound evaluation to evaluate for partial tears. In their study comparing clinical findings with those of magnetic resonance imaging and ultrasonography in 31 subjects, tendon dysfunction, as measured by heel raise, was not correlated with inhomogeneity by MRI [60]. They concluded that partial tears without painful sequelae may be missed by the clinician when doing tests to evaluate tendon function and resistance testing in an attempt to assess pain or dysfunction. Ultrasound to assess pain or dysfunction also may miss patients with partial tears. If ligamentous structures remain intact, they noted that there may be little change in the medial arch until the posterior tibial tendon finally tears, that pain on clinical palpation or resistance testing usually means inflammation is present, and that in these cases, the structure most likely to be affected is the tendon sheath [60].

Treatment of PTTD depends on many factors. During the time that the foot remains flexible, treatment is possible with a corrective orthosis such as the University of California Biomechanics Laboratory brace, molded ankle-foot orthosis, articulated molded ankle-foot orthosis, or Marzano brace [60]. The goal of nonoperative treatment in flexible flatfoot deformities is to control the progressive valgus of the calcaneus [62]. If a rigid deformity of the foot develops, then the orthosis should be accommodative to bony deformity and help prevent progression of the deformity. If nonoperative or conservative treatment fails, surgery is indicated because the progression of dysfunction may be rapid and disabling. When conservative treatment fails in the early stages of posterior tibial dysfunction, soft-tissue surgical procedures such as tenosynovectomy and tendon debridement may halt the progression of disease. Once flatfoot deformity develops, surgical procedures involving osteotomies and arthrodesis are necessary [60,62].

Many etiological factors have been proposed for PTTD including trauma, anatomic, mechanical, and ischemic processes [19]. None has been specifically proven to be a causative agent. Hypotheses on the association between pre-existing pes planus and PTTD suggest that the chronic stress placed on the posterior tibial tendon because of the flexible planovalgus foot and a tight heel cord could lead to an overuse injury, resulting in repetitive microtrauma and degeneration with time [19,64]. Further studies will be needed to more specifically identify risk factors and therapeutic targets in PTTD.

Peroneal tendinopathies

Peroneal tendon injuries are less common, and perhaps less commonly diagnosed, than corresponding injuries in the Achilles and posterior tibial tendons.

Accordingly, there are fewer studies in the current literature on peroneal injury and peroneal tendinopathy.

Case reports and case series document ruptures of the peroneus longus and peroneus brevis tendons in athletes and acute and chronic subluxation of peroneal tendon amenable to surgical correction [65–69]. In one series, Alanen et al reported on 38 operated cases of peroneal tendon injuries [70]. Eighty-two per cent of patients were competitive athletes. Of the 38 cases, there were 11 partial and 3 total ruptures of the peroneal brevis tendon, 2 partial and 2 total ruptures of the peroneus longus tendon, 9 cases of subluxation, 5 cases of chronic tendinitis, and 1 ganglion [70].

Peroneal injury including tendinopathy and partial tears of both the peroneus longus and brevis tendons has been linked to persistent lateral ankle pain and chronic lateral ankle instability [70–72]. Di Giovanni et al report on 61 patients who underwent a primary ankle ligament reconstruction for chronic instability [73]. Associated injuries found at surgery included 77% of patients with peroneal tenosynovitis, 54% with attenuated peroneal retinaculum, and 25% with peroneus brevis tear [73]. Based on their experiences, both Di Giovanni et al and Alanen et al suggest that peroneal tendon injuries may be an often overlooked cause of persistent lateral ankle pain and chronic ankle instability in settings of overuse and after acute trauma.

In cases of primary peroneus longus tendinopathy without antecedent acute trauma, the specific anatomy of the peroneus longus tendon as it courses through three fibro-osseus tunnels and changes directions in the hindfoot is thought to play a role in the evolution of the disease process [74]. Brandes and Smith found that there was an association, although not a statistically significant one, between location and type of injury in peroneal tendinopathy [74]. Complete ruptures of the peroneus longus were all found at the cuboid notch, whereas 89% of the partial tears involved the region of the lateral calcaneal process. Eighty-two percent of patients presenting with peroneal tendinopathy had a cavo-varus hindfoot position with arch height in the ninetieth percentile for the general population [74]. Thirty-three percent of cases had associated peroneus brevis involvement [74]. Sammarco also found associated peroneus brevis pathology in 9 of 14 cases of acute and chronic peroneus longus tears [69].

Our understanding of the mechanisms involved in peroneal tendon injury, the histopathology of peroneal tendinopathy preceding partial and acute rupture, and the specific biomechanics and extrinsic factors contributing to development of disease is in its earliest stages. As more research is done on the etiology and evolution of tendinopathies of the foot and ankle, we hope that more will come to be known about accurate diagnosis, initial treatment, and prevention.

Stress fractures

A stress fracture can be defined as a partial or complete bone fracture that results from repeated application of a stress lower than the stress required to fracture the bone in a single loading [75,76].

Stress fractures develop when bone fails to adapt adequately to the mechanical load experienced during physical activity. Ground reaction forces and muscular contraction result in bone strain. Bone normally responds to strain by increasing the rate of remodeling. In this process, lamellar bone is resorbed by osteoclasts, creating resorption cavities, which are subsequently replaced with denser bone by osteoblasts. Because there is a lag between increased activity of the osteoclasts and osteoblasts, bone is weakened during this time. If sufficient recovery time is allowed, bone mass eventually increases. If loading continues, however, microdamage may accumulate at the weakened region. Remodeling is thought to repair normally occurring microdamage. The process of microdamage accumulation and bone remodeling, both resulting from bone strain, plays an important part in the development of stress fractures. If microdamage accumulates, repetitive loading continues, and remodeling cannot maintain the integrity of the bone; a stress fracture may result [77].

Stress fractures account for 0.7% to 20% of all injuries presenting to sports medicine clinics [78]. Track and field athletes have the highest incidence of stress fractures when compared with athletes in other sports such as football, basketball, soccer, and rowing [79]. Great variation exists in the absolute percentage of stress fractures reported at each bony site, but the most common sites seem to be the tibia, followed by the metatarsals and fibula [75]. The site of stress fractures also appears to vary from sport to sport. Among track athletes, navicular stress fractures predominate; tibial stress fractures are most common in distance runners; and metatarsal stress fractures predominate in dancers [75].

Although numerous risk factors for stress fractures have been proposed, research is needed to confirm anecdotal observations. Presently, most studies in athletes are case series, and are confined to injured groups only, or are cross-sectional designs that do not allow the temporal relationship between risk factor and injury to be assessed. Results from large military epidemiological studies cannot be readily generalized to athletes, given important differences in training, fitness level, footwear, and services. These results may provide additional insight, however, especially given the deficiencies in the athletic literature [77].

One important risk factor is that of a history of a previous stress fracture. Low bone density is an identified risk factor in women, although this has not been as clearly studied in men [77,80]. Women in the military appear predisposed to stress fractures compared with their male counterparts, but this has not necessarily been confirmed in athletes. Overall training levels may influence this result [75]. Menstrual irregularity, in particular amenorrhea of longer than 6 months duration, is a risk factor [77,81]. A family history of osteoporosis is considered to be a risk factor for low bone density and osteoporosis in both females and males, but it is not clear that this necessarily predisposes to stress fractures in athletes [77,82,83].

Nutritional status, in particular low calcium intake, may contribute to stress fracture. Other dietary factors such as fiber, protein, alcohol, and caffeine intake may play a role but have not been as well studied [77].

Biomechanical factors may predispose to stress fractures by creating areas of stress concentration in bone or promoting muscle fatigue. High arches may

predispose to increased risk for femoral and tibial stress fractures, whereas pes planus may predispose to metatarsal stress fractures [77,84–86]. Leg length inequality has been postulated as a risk factor. Friberg reported a higher incidence of tibial, metatarsal, and femoral fractures in the longer leg and a higher incidence of fibular stress fractures in the shorter leg in military recruits [87]. A leg-length discrepancy has also been reported to be associated with a higher incidence of stress fractures in athletes [77,88,89]. In particular, a leg-length inequality of greater than 0.5 cm was reported in 70% of women with stress fractures, compared with only 36% of women without stress fractures [89]. Other biomechanical variables linked to increase stress fracture risk have included hip external rotation of greater than 65° [90], greater forefoot varus, restricted ankle joint dorsiflexion, narrow transverse diameter of the tibial diaphysis, and smaller calf circumference measurement [79,90,91]. No studies have reported the effects of such physiological factors of muscle mass or muscle strength on predisposition to stress fracture [77]. Additionally, no consistent relationships have been observed between body size or composition and stress fracture risk.

Anecdotal observation and clinical case series suggest that a transition in training, in particular increasing mileage, as well as a higher absolute volume of training can predispose to stress fractures in athletes, although little controlled research has examined this aspect [77]. Additionally, although no data exist on how stress fracture risk is specifically affected by training surface, it may be prudent to advise athletes to minimize the time they spend training on hard, uneven surfaces [75].

A higher incidence of stress fracture was reported in military recruits using older or worn running shoes [92]. It is unclear if this is a direct result of decreased shock absorption or perhaps decreased mechanical support [75]. Reports are conflicting about whether or not insoles can prevent stress fractures. From a practical standpoint it is important for individuals to train in shoes appropriate for their foot type. Athletes with high arched rigid feet should select cushioned shoes. Athletes with low arches should select shoes providing stability and motion control [75].

A patient with a stress fracture typically presents with a history of insidious onset of activity-related pain that progressively worsens over time. The most obvious physical examination feature is localized bony tenderness. Special clinical tests, such as the ‘‘hop test’’ for femoral stress fracture and spinal and hip extension used in diagnosis of pars stress fractures, may be helpful. Clinical reports have suggested that the application of ultrasound or a vibrating tuning fork may be helpful in the diagnosis of stress fractures by increasing pain at the fracture site; however, current literature neither supports nor refutes these commonly used clinical tools.

Commonly used imaging studies include radiographs, bone scans, and MRIs. In approximately two thirds of symptomatic patients, radiographs are initially negative and only half ever develop positive radiograph findings [93]. The most common sign in early stress fracture is a region of focal periosteal bone formation. The gray cortex sign (a cortical area of decreased density) may also be seen [94,95]. In those cases where clinically indicated, advanced imaging such as a bone scan or

MRI should be employed to confirm the diagnosis. Bone scans will confirm the diagnosis as early 2 to 8 days after the onset of symptoms [96]. MRI has shown promise in grading progressive stages of stress fractures severity. A four-stage grading system has been developed: grade 1 injuries demonstrate periosteal edema on the fat-suppressed images, grade 2 injuries demonstrate abnormal increased signal intensity on fat-suppressed T-2 weighted images, and in grade 3 injuries decreased signal intensity is seen on T-1 weighted images. In grade 4 injuries, an actual fracture line is present, typically visualized on both T-1 and T-2 weighted images [94,96].

Stress fractures can be generally classified as noncritical and critical. Noncritical stress fractures in the lower leg, foot, and ankle include the medial tibia, fibula, and metatarsals 2, 3, and 4. Treatment of these stress fractures requires relative rest. Athletes may benefit from a short period of immobilization in a walking boot (ie, 3 weeks), or in the case of metatarsal stress fractures, from a stiff sole shoe or steel insert. Return to full sport activity is generally achieved within 6 to 8 weeks. Critical stress fractures require special attention due to a higher rate of nonunion, and include the anterior tibia, medial malleolus, talus, navicular, fifth metatarsal, and sesamoids [75,77].

Medial tibia

Stress fracture of the medial tibia present with medial shin pain aggravated by exercise. Tenderness is most commonly localized in the posteromedial border of the lower third, which is the most common site. Biomechanic examination may reveal either a rigid, high-arched foot that is incapable of absorbing load, or an excessively flat foot that causes muscle fatigue. Treatment involves relative rest until the pain resolves. Athletes may benefit from a short period use of either a walking boot or pneumatic brace (an air cast), which may be removed for nonimpact cross training. The air cast brace is believed to unload the tibia by compressing the lower leg, thus redistributing forces and decreasing the amount of tibial bowing [75,97–99]. Bony tenderness typically disappears between 4 to 8 weeks, after which the athlete may begin a gradual return to activity. Although the time to return to full activity varies considerably, the average period for full release to sport activity is 8 to 12 weeks.

Anterior cortex of the tibia

Stress fractures of the anterior cortex of the midshaft of the tibia are among the critical stress fractures because they are prone to delayed union, nonunion, and complete fracture. As with other stress fractures, in the acute phase plain radiographs are often normal and diagnosis may require bone scanning or MRI. In later stages, plain radiographs may demonstrate the ‘‘dreaded black line.’’ This appearance is due to bony resorption, and indicates nonunion. At this late stage, bone scanning will often be normal and patients may only have minimal symptoms and thus may be fully participating in sports. It is believed that the mid anterior cortex of the tibia is vulnerable to nonunion due to poor vascularity and increased

tension because of morphologic bowing of the tibia [75]. Treatment programs have included prolonged periods of rest and immobilization (up to 4 to 6 months), bone stimulation, and surgery. Brukner recommends the use of a long pneumatic leg brace combined with electric stimulation for 10 hours per day for both the acute stress fracture and the established nonunion, as denoted by the presence of the dreaded black line on plain radiographs. Fracture healing is monitored both clinically and radiographically. Athletes do not return to activity until evidence exists of cortical bridging on radiography. If after 4 to 6 months there is no evidence of healing either clinically or radiologically, surgical intervention is indicated (drilling at the fracture site, bone grafting, or insertion of an intramedullary rod) [75].

Fibular stress fractures

Because the fibula has a minimal role in weight bearing, it is believed that fibular stress fractures result from muscle traction and torsional forces. Although most stress fractures are in the distal third of the fibula, proximal stress fractures have been described. Patients are treated with weight bearing rest until bony tenderness resolves (usually 4 to 6 weeks). Athletes may benefit from a short period (ie, 3 weeks) in a walking boot. Sport activity is then gradually commenced. Soft tissue tightness should be corrected, as should biomechanical abnormalities such as excessive pronation or excessive supnation [75].

Medial malleolus

Medial malleolar stress fractures generally present with a several-week history of mild discomfort followed by an acute episode that results in seeking medical attention. Although the fracture line is frequently vertical from the junction of the tibial plafond and the medial malleolus, it may run obliquely from the junction to the distal tibial metaphysis [75]. Excessive pronation and accompanying tibial rotation distributes excessive forces to the medial malleolus.

Undisplaced or minimally displaced stress fractures of the medial malleolus are treated conservatively in a pneumatic leg brace for 6 weeks. A displaced fracture or a fracture that progresses to nonunion should be treated operatively.

The talus

Talar stress fractures most commonly involve the lateral body near the junction of the body with the lateral process of the talus. Talar neck stress fractures have been reported but are considered rare. Athletes may present with prolonged pain (for several months) following an ankle sprain despite rehabilitation. Excessive subtalar pronation is felt to predispose to talar stress fractures by allowing impingement of the lateral process of the calcaneus on the concave posterolateral corner of the talus. Treatment involves non-weight–bearing rest for 6 weeks followed by rehabilitation. Orthotic control of pronation is recommended if present. Nonunion fractures respond well to surgical excision of the lateral process [75].

The calcaneus

Calcaneal stress fractures present with localized tenderness over the medial or lateral aspects of the calcaneus. The most common site is the upper posterior margin, just anterior to the apophyseal plate and at a right angle to the normal trabecular pattern. Plain radiographs may show a sclerotic appearance on lateral radiograph parallel to the posterior margin of the calcaneus. Bone scanning and MRI are more sensitive. Treatment is achieved with 6 to 8 weeks of weight-bearing rest with the use of a soft heel cushion. Joint mobilization and flexibility of the calf muscles are indicated when appropriate. Orthotics may be prescribed to control excessive pronation. Running is usually resumed after 6 weeks.

The cuboid and cuneiforms

Stress fractures of cuboid and cuneiform bones are rare. These are generally considered noncritical stress fractures that may be treated with weight-bearing rest until bony tenderness resolves, after which a gradual return to sport activity is commenced. As for other noncritical stress fractures, a short period in a walking boot may provide comfort. One report did propose a period of non-weight–bearing on crutches for 4 weeks for a cuboid stress fracture, followed by progressive weight bearing and return to sport activity at 8 weeks [75,100].

The navicular

Navicular stress fractures occur most commonly in the central third, which is believed to be susceptible to stress fracture and subsequent delayed union or nonunion because of relative avascularity and the presence of shear forces in this region. Pain is typically insidious in onset and somewhat nonspecific in location. Critical to clinical examination is palpation of the "N" spot in the proximal dorsal portion of the navicular. Plain radiography has low sensitivity and advanced imaging such as bone scan, computed tomography (CT), and MRI should be pursued early in suspected cases. Initial treatment requires 6 weeks of non-weight–bearing immobilization. If at this point clinical tenderness remains, another 2 weeks may be required. Patient should then undergo a gradual return to activity coupled with physical therapy for joint mobilization, soft tissue work, muscle strengthening, and biomechanical correction. Patients that do not respond to the conservative treatment regimen undergo surgery (screw fixation with or without bone grafting of the established nonunion) [75,77,101].

Metatarsals

Metatarsal stress fractures involving metatarsals 1, 3, 4, and the distal aspect of metatarsal 2 are usually uncomplicated and can be treated with relative rest. Patients may benefit from a walking boot or the use of a steel plate insert or counterforce arch brace. Once tenderness to palpation and pain with ambulation has resolved, a gradual return to running program is commenced. Fractures

of the base of the second metatarsal and proximal fifth metatarsal require special consideration.

Stress fractures at the base of the second metatarsal are most common among ballet dancers. Most of these fractures can be treated with weight-bearing rest but patients must refrain from training activity for 6 weeks.

Three types of proximal fifth metatarsal stress fractures have been described: (1) the tuberosity avulsion fracture, (2) the Jones fracture at the junction of the metaphysis and diaphysis, and (3) the diaphysial stress fracture [75].

The most common fracture seen is a simple avulsion fracture of the tuberosity, usually caused by the contraction of the peroneus brevis tendon as a result of an acute inversion injury or the lateral band of the plantar aponeursis. This is usually an uncomplicated type of fracture that will respond to a short period of immobilization for pain relief, followed by progressive activity. The Jones and more distal diaphyseal stress fractures are critical stress fractures prone to nonunion and require 6 to 10 weeks of non-weight–bearing rest. Athletes that fail to heal conservatively or those who require more rapid treatment undergo surgical fixation with placement of a fixation screw. Displaced fractures may require open reduction internal fixation. After screw fixation, progressive weight bearing is initiated at 2 weeks, with return to running in 7 weeks. When bone grafting is used, running activities are delayed for 12 weeks to allow for bony healing.

The sesamoids

The medial and lateral sesamoid bones at the first metatarsal phalangeal (MTP) joints act to increase mechanical advantage of the flexor hallucis brevis tendon and stabilize the first MTP joint in association with the plantar plate capsule. They also protect the flexor hallucis longus tendon and absorb weight-bearing stress on the medial forefoot. The medial sesamoid bone is more commonly affected. Radiographs are often negative and diagnosis may require advanced imaging. A bone scan or MRI is also useful in differentiating a bipartite sesamoid from a true stress fracture. Stress fractures of the sesamoid bones are prone to nonunion. Treatment involves non-weight–bearing rest for 6 weeks. Weight bearing is commenced when bony tenderness is no longer present. Padding is used to distribute weight away from the sesamoid bones in the form of a sesamoid or dancer pad. If nonunion occurs, or if the bone is splintered, excision is recommended [75].

Exertional compartment syndrome

Chronic exertional compartment syndrome is defined as reversible ischemia secondary to a noncompliant osseofascial compartment that is unresponsive to the expansion of muscle volume that occurs with exercise [102]. Most commonly seen in the lower leg, exertional compartment syndrome in athletes has also been described in the thigh and medial compartment of the foot [103–105]. It presents as recurrent episodes of leg discomfort experienced at a given distance or intensity of running. Though a characteristic history is highly suggestive of exertional

compartment syndrome, no physical examination finding can firmly establish the diagnosis [102,106]. Diagnosis based solely on clinical presentation can lead to misdiagnosis and inappropriate therapy or delay of proper therapy [107]. An exercise challenge and documentation of elevated compartment pressure in one or more of the compartments of the leg confirms the diagnosis [102].

The characteristic presenting complaint of patients with chronic exertional compartment syndrome is recurrent exercise-induced leg discomfort that occurs at a well-defined and reproducible point in the run and increases if the training persists. The quality of pain is usually described as a tight, cramplike, or squeezing ache over a specific compartment of the leg. Athletes can reliably predict at what intensity or what distances the discomfort will occur as well as how long pain will last, depending on the intensity and distance run. Relief of symptoms only occurs with discontinuation of activity [108]. Examination may or may not demonstrate fascial hernias. In some cases, the classic exertional component is not as evident, and patients complain of pain at rest or with daily activities as well. Women may be more susceptible to chronic lower leg compartment syndrome than men, and women may also, for unclear reasons, respond less well than men to operative fasciotomy [109]. Chronic compartment syndrome, left untreated, can develop into an acute syndrome [110].

Several factors are believed to contribute to an increase in intracompartmental pressure during exercise [108,111]. These are enclosure of compartmental contents in an inelastic fascial sheath, increased volume of the skeletal muscle with exertion due to blood flow and edema, muscle hypertrophy as a response to exercise, and dynamic contraction factors due to the gait cycle. It has also been proposed that myofiber damage as a result of eccentric exercise causes a release of protein bound ions and a subsequent increase in osmotic pressure within the compartment. The increase in osmotic pressure increases capillary relaxation pressure, thus decreasing the blood flow [112]. Development of symptoms may be more common at the beginning of a running season due to muscle hypertrophy, which decreases the volume in the compartment [113]. Rapid increases in muscle size due to fluid retention are also believed to play a role in the development of chronic exertional compartment syndrome in athletes taking the popular supplement creatine monohydrate [108].

A neurologic and vascular examination should also be performed with reproduction of the symptoms [102]. Understanding the distribution of nerves and functions of muscles in relation to symptoms can help identify the affected compartment in cases where the pain is not well localized to one specific compartment, or it may help determine which compartments are more severely affected in cases where more than one compartment is involved [108].

There are four major compartments in the leg. Each is bound by bone and fascia, and each contains a major nerve. The anterior compartment contains the extensor hallucis longus, extensor digitorum longus, peroneus tertius, and anterior tibialis muscles, as well as the deep peroneal nerve. The lateral compartment contains the peroneus longus and brevis as well as the superficial peroneal nerve. Posteriorly there are two compartments: the superficial posterior and the deep posterior

compartments. The superficial compartment contains the gastrocnemius and soleus muscles and the sural nerve. The deep posterior compartment contains the flexor hallucis longus, flexor digitorum longus, and posterior tibialis muscles, as well as the posterior tibial nerve. Some authors believe that the posterior tibialis should be considered a separate compartment, as it is surrounded by its own fascia [114]. Anterior compartment syndrome is most common (45%), followed by the deep posterior compartment (40%), lateral compartment (10%), and superficial posterior compartments (5%) [112].

If the anterior compartment is affected, the patient may display weakness of dorsiflexion or toe extension and paresthesias over the dorsum of the foot, numbness in the first web space, or even transient or persistent foot drop [102,113]. Paresthesias in the plantar aspect of the foot and weakness of toe flexion and foot inversion may be revealed when the deep posterior compartment is involved, whereas dorsolateral foot hypoesthesia and plantar flexion weakness may be present if the superficial posterior compartment is affected. Lateral compartment pressure elevation with compression of the superficial peroneal nerve can induce sensory changes over the anterolateral aspect of the leg and weakness of ankle eversion. An inversion as well as equinus deformity may also be present [102,115].

Several techniques have been described in the literature for measuring both static and dynamic intramuscular pressures. These techniques include the needle manometer [116], the wick catheter [117], slit catheter [118], continuous infusion [119], and a solid-state transducer intracompartmental catheter [120]. Each of these techniques offers several advantages and disadvantages. All are time-consuming, however, and require some degree of skill and experience to set up and perform [102,108].

Our preferred method for measurement of compartmental pressures is with a battery-operated, hand-held, digital fluid pressure monitor. The Stryker Intracompartmental Pressure Monitor (Stryker Corporation, Kalamazoo, Michigan) is a convenient and easy-to-use measuring device for use in the clinical setting [102,120]. This device has been found to be more accurate, versatile, convenient, and much less time-consuming to use than the needle manometer method [121]. Measurements were also found to be more reproducible among different examiners with the Stryker instrument [102].

The usefulness of pressure measurement and maintenance of patient safety with this invasive technique relies upon a thorough knowledge of the anatomy of the leg. Before attempting to measure compartment pressures, the physician should thoroughly study the anatomical structures in each compartment to avoid damaging neurovascular structures [102].

Generally accepted criteria for the diagnosis of chronic exertional compartment syndrome (CECS) are described by Pedowitz and colleagues [107]. One or more of the following pressure criteria must be met in addition to a history and physical examination that is consistent with the diagnosis of CECS: Pre-exercise pressure $\geq$15 mm Hg; 1 minute postexercise pressure $\geq$30 mm Hg; or 5 minutes postexercise pressure $\geq$ 20 mm Hg. Clinicians should also be aware that standard exercise protocols often used in the clinical setting may or may not be adequate to

raise intracompartment pressure and diagnosis may require the sport-specific activity to induce symptoms and raise intracompartment pressure [122].

Recent interest has focused on the use of noninvasive tools in the diagnosis of chronic compartment syndrome: triple phase bone scan, MRI, near-infrared spectroscopy, and technetium-99m methoxyisobutyl isonitrile (MIBI) perfusion imaging [123–131].

The dynamic bone scan may support the diagnosis based on specific tracer uptake patterns. The characteristic appearance is that of decreased radionuclide concentration in the vicinity of the area of increased pressure, with increased soft-tissue concentration both superior and inferior to the abnormality. The area of decreased uptake is believed to be due to the increased pressure and decreased blood flow to the region [127]. On MRI imaging, exercise changes are characterized by swelling within a compartment, which manifests as intramuscular diffuse high signal intensity on T2-weighted images. Failure of the edematous muscle to return to baseline normal appearance by 25 minutes after completion of exercise is diagnostic [128]. The triple-phase bone scan and MRI offer alternatives to direct intracompartmental pressure measurements in cases in which the athlete is averse to repeated needle sticks or where the results of pressure monitoring may be borderline [102,129].

Near-infrared spectroscopy measures tissue deoxygenation of skeletal muscle caused by elevated intramuscular pressure during exercise [124].

MIBI perfusion imaging is a technique that assesses the uptake of an intravenously injected radiopharmaceutical, MIBI, by peripheral muscles. The uptake of the radiopharmaceutical is largely determined by muscle perfusion, but hypoxia also inhibits uptake of the MIBI, enhancing its potential for detecting muscle ischemia. Cases have been reported where visually detectable decreases in MIBI uptake in one or more compartments were noted during exercise when compared with studies taken at rest [125].

Treatment of chronic exertional compartment syndrome can include both conservative and surgical intervention. Conservative measures include relative rest (limiting activity to the level that avoids any more than minimal symptoms), anti-inflammatories, stretching and strengthening of the involved muscles, and orthotics (particularly in cases of excessive pronation). Some athletes will simply choose to refrain from the causative activity, which is a viable option provided they remain neurovascularly intact.

In cases where symptoms persist despite 6 to 12 weeks of conservative care, or in cases of extreme pressure elevation, surgical remediation (fasciotomy of the involved compartments with or without fasciectomy) should be undertaken [132,133]. Single and double incision as well as endoscopic techniques have been described. Regardless of the technique, any fascial hernias must be included in the fascial incision.

Due to a high rate of coexistence, some authors advocate release of the lateral compartment whenever a procedure for anterior compartment syndrome is performed. Others have stated that this dual release may not be necessary if clinical evaluation and compartment pressure testing fail to demonstrate lateral compart-

ment involvement [134]. When performing a deep posterior compartment release, attention must be given to adequate decompression of the tibialis posterior [135].

Postoperatively, a compressive dressing is applied. Drains are not normally necessary. Crutches are used for comfort for a few days, but the patient begins active and passive motion immediately. Once the wound is healed, walking and bicycling are encouraged. Patients may begin a light jog in 2 weeks, and resume run training at 6 weeks. It usually takes 3 months for full rehabilitation, but patients with deep posterior compartment fasciotomies may need longer [114,136].

Shin splints

Shin splints, or medial tibial stress syndrome, can be described as a clinical entity characterized by diffuse tenderness over the posteromedial aspect of the distal third of the tibia [137]. In mild cases, pain is present only with exercise; in more severe cases rest pain is present. Shin splints have been reported to account for 12% to 18% of running injuries [43,138–140] and to occur in 4% of all military recruits in basic training [141]. Women appear more frequently affected than men [138,142,143].

Medial tibial stress syndrome is to be differentiated from stress fracture and exertional compartment syndrome [144,145]. Although different entities, they may coexist. Plain films are negative (except in cases of previous or coexistent stress fracture). Bone scans will demonstrate characteristic vertical linear increased activity along the tibial periosteum, which differs from the more focal fusiform increased radiotracer uptake exhibited by stress fractures [123].

Medial tibial stress syndrome is felt by most to represent a periostalgia or tendinopathy along the tibial attachment of the tibialis posterior or soleus muscles [43,108,146,147]. Other proposed etiologies have included posterior compartment syndrome [148,149] and fascial inflammation [147]. Detmer proposed a classification scheme for medial tibial stress syndrome based on etiology. Type 1 included local stress fractures, type 2 periostitis/periostalgia, and type 3 was due to deep posterior compartment syndrome [150].

Increased valgus forces on the rear foot and excessive pronation that result in increased eccentric contraction of the soleus and posterior tibial muscles are often contributing causes [146]. Intrinsic factors that may increase valgus forces and pronation include femoral anteversion, genu varum, tibia or forefoot varus, and an excessive Q angle [137]. Other intrinsic factors linked to medial tibial stress syndrome include excessive planus or cavus, tarsal coalition, lower extremity length inequality, and muscle imbalances [151,152]. Extrinsic risk factors include improper shoe wear, a rapid transition in training, inadequate warm-up, running on uneven or hard surfaces, running in cold weather, and low calcium intake [137,153].

Treatment of medial tibial stress syndrome includes relative rest and the correction of any recent transition in training. Hill running and running on uneven surfaces should be avoided. Proper shoe wear is essential to minimize rear foot

valgus and to correct excessive pronation, pes planus, or pes cavus. Orthotics are useful in cases that cannot be controlled by shoe wear alone.

NSAIDS and anti-inflammatory modalities (ie, iontophoresis and ultrasound) can be useful adjuncts in the rehabilitation of medial tibial stress syndrome. A strengthening and flexibility program should be initiated with the goal of correcting any muscle imbalances. Flexibility of the gastrocsoleus should be emphasized, as well as strengthening (concentric and eccentric), including the foot intrinsics, dorsiflexors, plantarflexors, invertors, evertors, and gluteals. All deficits within the kinetic chain should be corrected [108]. A compressive sleeve may provide symptomatic relief.

Operative therapy (posterior fasciotomy) has been described for the athlete with severe limitations of physical activity, frequent recurrence, or no response to available therapy [150,154]. Surgical treatment for periostalgia has not been uniformly successful and should be reserved for recalcitrant symptoms that have not responded to a well documented treatment program of at least 6 months [108].

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