NON TRAUMATIC LEG PAIN IN ATHLETES

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Introduction

Current public attitude regarding health and fitness has prompted many to try to improve cardiovascular conditioning. Therefore many persons have become involved in some type of athletic activity, performed with regularity, several days per week. Often times these programs are new and involve an unaccustomed activity for the beginning participant. As more people enter exercise programs there will be a corresponding increase in the number of injuries. Most injuries are initiated by the repetitive performance of an activity over a sustained period of time rather than as a result of a specific traumatic incident. The constant prolonged exposure causes injury as a result of repetitive microtrauma, often producing painful symptoms referred to as an "overuse injury."

Complaints concerning the lower leg comprise a category of overuse injuries that represent a fairly large group of patients seen by the sports oriented podiatric physician. This type of sports related injury will remain common as long as people continue to exercise and participate in sports.

James and associates (1) have classified the causes of musculoskeletal overuse disorders into three categories:

1. training errors,
2. anatomical factors, and
3. shoes and surfaces.

Training Errors

During the subjective part of the examination, it is important to note the nature and frequency of all athletic activities in which the patient is involved. Certainly a patient who relates running 15 miles daily is obviously a candidate for an overuse injury. Such training is extensive and does not give the body adequate time to recover. Not as obvious, however, would be the injury to the aerobic dance teacher who presents with symptoms brought on by teaching an aerobic class three days per week. However, upon questioning the physician finds that this same teacher plays racquet ball 5 times per week. Even though the nature and frequency of each of the two activities in this example may not seem excessive, the combination and frequency of the two activities possibly are excessive.

The examining physician should ascertain if there is a history of new, unaccustomed, or definite dramatic increase in the amount of activity. The zealous beginning athlete is especially prone to the overuse injury. The unaccustomed repetitive microtrauma may be taxing the musculoskeletal system that has not yet adapted to the activity. Appropriate questioning should also determine the nature and amount of warm up and flexibility exercises for pre and post activity. It is important to have the patient demonstrate such to determine if the activity is being performed properly. An athlete not adequately stretching, warming up, or using proper preparation may be predisposed to injury for any activity.

Anatomical and Biomechanical Factors

Athletes may sustain injuries from overwork or overtraining. If the athlete has associated anatomical or biomechanical pathology, the risk of overuse injury is further increased. The examination will ultimately reveal the nature and location of the injury. Therefore, it is imperative for the athlete to wear appropriate clothing conducive to a thorough examination of all lower extremity joints. A proper evaluation of leg pain cannot be performed adequately or comfortably if the young lady is attired in a mini skirt or the gentleman is wearing a three piece suit. Athletic patients should be screened by the receptionist during the initial appointment call and instructed to bring shorts and athletic shoes. A brief but thorough biomechanical examination should be performed on the initial visit for all athletic patients presenting with an overuse type injury. This examination should include:

leg length analysis,
hamstring flexibility,
hip range of motion,
knee range of motion,
determination of varus or valgus alignment of the lower leg and knee,
gastrocnemius-soleus complex flexibility,
subtalar and midtarsal range of motion,
gait and possible running analysis.

The examination will determine if tight musculature or mechanical factors are playing a role in the cause of injury. With the athlete in shorts and athletic shoes, it will allow the practitioner to observe the athlete run or jump in an attempt to reproduce the symptoms experienced during

the athletic activity. This will avoid the need for the physician to respond with the admonition to come back again "next time it hurts."

Footwear and Athletic Surfaces

The footwear of the athlete should be evaluated during the course of the examination. On many occasions the clinician will find the beginning jogger is using worn tennis shoes or that the aerobic dancer is wearing a cheap imitation of a better constructed brand of aerobic dance shoe. Often times a simple instructional session on shoe selection or shoe change is all the treatment necessary to overcome the presenting complaint. Shoes should certainly be evaluated for abnormal biomechanical wear patterns or normal excessive wear that may be causing shoe related injury. One may determine that the runner with a rigid cavus foot is inappropriately running with a shoe that is designed for an over-pronator, thereby contributing to further lack of motion in this particular foot type. If one is unfamiliar with athletic shoes, a call to the local athletic shoe store will afford ample information on shoes and shoe design from store owners or managers.

The type of surface that the patient uses for the workout should be evaluated. Running on cement or asphalt is obviously potentially more damaging to the lower extremities than is a gravel or packed dirt trail. Hill running or running on the same side of a canted road on a frequent basis can predispose the runner to surface related mechanical problems. Commonly seen is the aerobic dancer participating on the church basketball flooring which certainly can make one more prone to injury than a specially designed resilient aerobic dance floor. An attempt should be made to have the athletic patient participate on the type of surface that is least stressful to the musculoskeletal system.

Many athletes have little or no symptoms during normal ambulation but are plagued by symptoms during performance of their chosen activity. One explanation is the fact that there is a vertical force of 275% of body weight during the running foot strike as compared to 110-115% during walking. Additionally, the amount of stance phase during walking is 0.62 seconds while the running stance phase is reduced to 0.2 seconds. This marked decrease in the stance phase is one of the basic causes of overuse injuries in the running athlete, since all events, shock absorption, deceleration, transverse plane motion, foot stabilization, and acceleration, occur in approximately one-third the time in runners as they do in walkers (2).

The following will discuss the non traumatic sources of lower leg pain in the athlete.

Disorders of the Shin

According to the American Medical Association's Standard Nomenclature of Athletic Injuries (3), shin splints is defined as pain and discomfort in the leg from repetitive running on hard surfaces or forcible extensive use of the foot flexors. Diagnosis should be limited to the musculotendinous inflammations, excluding fractures or ischemic disorders. This definition does not have widespread acceptance by the sports medicine community for too many disorders may manifest with symptoms of the shin area that are not confined to the musculotendinous system. The term "shin splints" is basically a catch-all term that is used to describe the location of the leg pain as being somewhere between the tibial tubercle and the ankle joint (4).

When evaluating the running or jumping athlete for symptoms of the shin area, it is essential to attempt to identify the exact location and source of pain, i.e. bone, muscle, tendon, fascioperiosteal junction. An appropriate diagnosis based on the injured anatomical structure can then be made.

Anterior Shin Pain

During the running cycle the anterior leg musculature serves to decelerate the landing foot so as to not permit intense foot strike shock. If foot strike is made repetitively with ill cushioned footwear or on extremely unyielding surfaces, the anterior muscle group will act to decelerate this landing foot causing overwork and eventually may progress to pain and inflammation (5, 6).

Harvey (7) believes that pain in the anterior muscle group is caused by an increased training intensity in athletes who have weak anterior muscle groups as compared to strong, inflexible posterior muscle groups. These overworked anterior muscles are then prone to traction injury at their origin.

Possible mechanical factors predisposing one to an anterior muscle group overstrain would consist of a tight gastrocnemius-soleus complex or forefoot varus deformity. Both of which can result in excessive forefoot slapping during running and excessive foot strike shock.

Clinical Examination: The physician can readily palpate the origin and bellies of the anterior muscle group to determine the exact location and source of pain in this compartment of the leg. Active dorsiflexion of the foot against resistance, with palpation of the involved muscles, will reproduce the symptoms. This is especially true if the patient has worked out previous to or during the office visit. Prolonged heel walking in the hallway will reproduce the pain as well. A biomechanical examination is performed to rule out mechanical or anatomical causes. The footwear should also receive a thorough evaluation. If a possible stress fracture of the tibia is suspected a standard AP and lateral view of the lower leg should be performed.

Treatment: The athlete should be placed on a pre and post exercise gastrocnemius and Achilles tendon stretching program (Fig. 1). The patient is given exercises to improve the strength of the anterior muscle group. This is

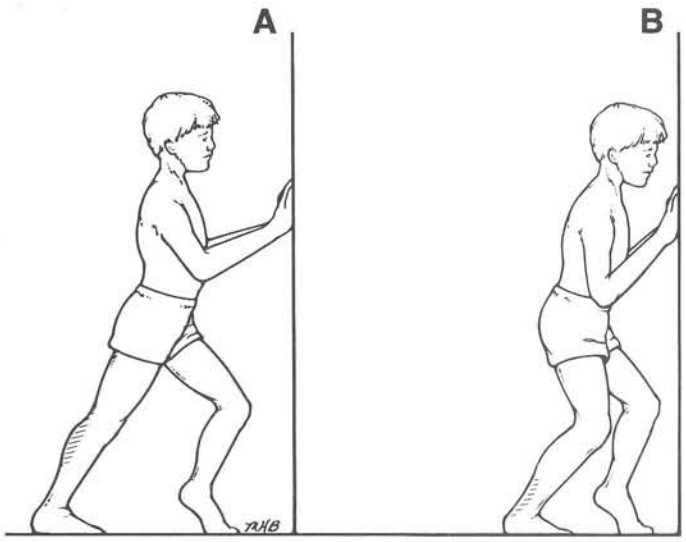


Fig. 1. A. Stretching of upper aspect of calf muscle complex. **B.** Stretching of lower aspect of calf muscle complex.

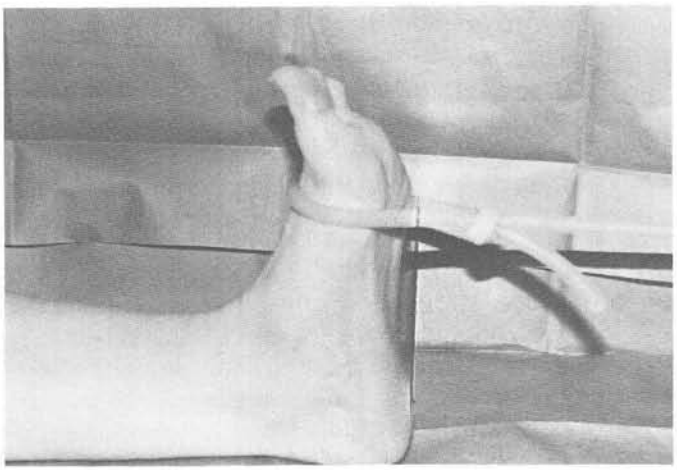


Fig. 2. With one end of surgical tubing securely anchored, patient loops other end around ball of foot. Foot is then dorsiflexed to resistance.

accomplished with the use of a 3/16 inch surgical tubing and active dorsiflexion against resistance exercises (Fig. 2). Provide the athlete patient with tangible written instructions regarding stretching and strengthening exercises. This will prove to be an incentive for the patient to adhere to the instructions more attentively. This list may additionally serve to build dialogue with athletic patients.

The patient is instructed to ice massage the anterior shin muscle for three minutes, three times daily during the first week. The same procedure is followed after each run or workout during the following week (Fig. 3). Improperly cushioned shoes are changed for better shock reducing ones. Appropriate biomechanical therapy is used if indicated. The athlete is instructed to reduce the running program to one-third the normal distance, at regular speed, for one week and is to run only on soft surfaces. Other sports patients similarly cut back on the time spent during that activity. If the patient demonstrates a decrease in symptoms after the therapy program and activity cutback is monitored for one week, he or she may increase to full activity over the next one to two weeks.

Medial Shin Pain

Medial shin pain in athletes has been documented by many authors (4, 8-11) as being a common area for shin soreness. This pain is usually encountered at the lower one-third of the posteromedial aspect of the tibia. Certain investigators (6, 12, 13) have implicated the posterior tibial muscle as the cause of medial shin pain in the running athlete. However, Michael and Holder (9) have demonstrated through cadaver dissection that the posterior tibial muscle origin is a considerable distance away from the medial border of the tibia and that its muscle belly is higher in location than the usual site of pain found with medical shin splints. During the delayed phase of a technetium 99m bone scan, a diffuse pattern was discovered corresponding more with the medial origin of the soleus muscle and its fascial attachments than it did with the posterior tibial muscle. With the insertion of the soleus muscle on the medial aspect of the calcaneous excessive stance phase pronation makes it vulnerable to elongation with resultant symptoms.

With the posterior tibial muscle being a strong supinator of the foot as well as a decelerator of excessive subtalar joint pronation (14), it appears logical that the muscle or tendon can become strained or inflamed in those athletes demonstrating increased stance phase pronation.

Detmer (8) categorized the chronic medial tibial stress syndrome type II classification as a chronic periostalgia. This occurs at the junction of the periosteum and fascia

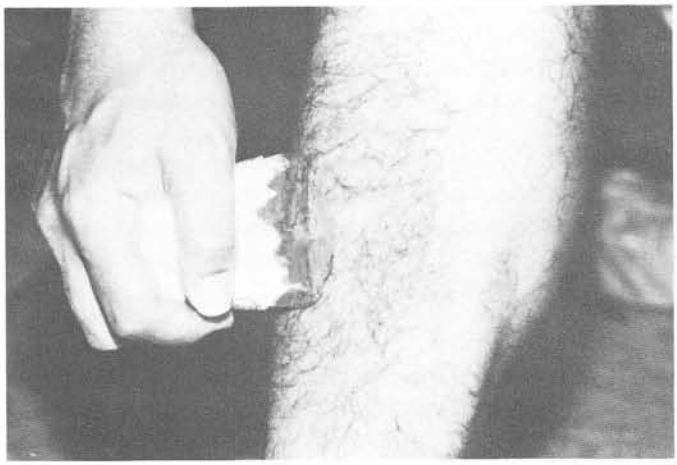


Fig. 3. Use of styrofoam coffee cup facilitates holding of ice so that it may be massaged directly to symptomatic area.

just slightly posterior to the medial border of the tibia. He encounters this disorder in ballistic sports such as sprinting, gymnastics, and basketball. The pathogenesis is the periosteum being traumatically disengaged from bone by either ballistic avulsion or by subperiosteal stress on the tibial edge resulting in hemorrhage which will lift the periosteum. He attributes this avulsion possibly to the soleus and its fascial attachments. Mubarak (15-18) has written extensively on this disorder as well and describes it as the medial tibial syndrome.

Examination: Since the medial surface of the tibia has no muscular attachments, eliciting pain on palpation at this location with no associated pain slightly posteriorly in the soft tissues, should alert the clinician for the presence of a stress fracture. If pain and swelling are noted overlying the bone with no associated muscle or tendon symptoms, then this should certainly raise suspicion as to the possibility of a stress fracture of the tibia. Appropriate radiographs should be taken.

If the clinician suspects that the soleus is responsible for medial shin complaints, pain would be found along the medial attachment of the soleus and its fascial attachments. This is accentuated by having the athlete stand on the ball of the foot during palpation of the muscle attachment medially. There would be little pain on resistance of active inversion with plantarflexion, which would isolate the tibialis posterior muscle. If the posterior tibial muscle is to be implicated, the examiner can palpate the tendon or muscle during resisted inversion with plantarflexion and elicit symptoms. It may be necessary to have the athlete exercise to the point of pain before symptoms could be elicited at either location. To determine if the pain is due to a chronic periostalgia of the medial tibia, the clinician should attempt to palpate slightly posterior to the posterior border of the medial surface of the tibia.

Treatment: The conservative treatment for symptoms of the medial aspect of the shin consists of a two week course of non steroidal anti-inflammatory drugs (NSAIDs). It may be necessary to have the athlete cease all strenuous weight-bearing activity for one to two weeks. This is especially true if the medial shin soreness is extremely intense and has progressed to night pain after activity. Ice massaging for 5 minutes, three times daily is helpful in decreasing the local soft tissue inflammation. A surgical tubing exercise program is used to build up the invertors of the ankle. Stretching of the gastrocnemius-soleus complex is extremely important because the soleus may be implicated as the cause of shin pain.

When biomechanical factors are evident, an extremely effective way to provide immediate mechanical therapy and to determine if this form of treatment is helpful is the use of a temporary orthotic device. This device is constructed in the office, taking no longer than 15 minutes, and is fabricated from Styrene/Butadiene/Rubber (SBR) (Fig. 4). If this temporary device proves effective in alleviating symptoms then a permanent sport orthotic device is used. Elevating the heel is helpful when the soleus is responsible for the shin symptoms. The athlete must also cut back on the distance or time of the workout in a similar manner as described for anterior shin pain. If all conservative therapy fails, Detmer (8) has advocated the use of a deep posterior compartment fasciotomy. The authors have performed this procedure bilaterally with good results on a 19 year old female collegiate runner who suffered with a 3 year history of Detmer's Type II chronic medial tibial stress syndrome that was resistant to all forms of conservative therapy.

Stress Fractures of the Tibia and Fibula

When evaluating the athletic patient for non traumatic leg symptoms the physician must keep a high index of suspicion for stress fractures of the tibia and fibula. This is true especially if the patient has not responded to previous care provided for apparent muscular or tendinous causes of shin pain. Stress fractures comprise as much as 10% of all sports injuries (19).

A stress fracture is a partial or incomplete fracture resulting from an inability to withstand nonviolent stress that is applied in a rhythmic, repeated subthreshold manner (20). Sports activities such as running, aerobic dancing, soccer, and racquet ball certainly afford potential exposure to constant repetitive microtrauma to the lower extremities. When the athlete is exposed to these repetitive stresses over a sustained period of time a stress fracture can result when the bone's ability to remodel itself is altered. The bone is said to be subjected to repeated strain at a vulnerable spot (21). With this repetitive microtrauma the stress distribution on the bone is increased due to muscle fatigue, thereby diminishing the muscle's shock attenuating properties around the bone.

According to Matheson and associates (22-24) the tibia is the most common site of stress fracture in athletes. Clancy (21) states that the most common location for the tibial stress fracture is the junction of the middle and distal one-third, with the proximal tibia being the second most vulnerable spot.

The incidence of fibular stress fractures, although not as commonly found as the tibial, accounts for 25% of all reported stress fractures (25). The most common site for the fibular stress fracture is the distal aspect in the area of the inferior tibio-fibular ligaments (Fig. 5), with the midfibula being the next most common location (21).

Etiological Factors: Many of the patients with stress fractures demonstrate a history of high training intensity and frequently have made a recent increase in their training routines (7). Andrews (5) believes that stress fractures are usually seen in the poorly conditioned athlete or in the

novice who continues to exert him or herself even though a persistent warning pain is being experienced. Often times there will have been a recent change of surface upon which the activity is performed, usually from a more to less resilient one. The young athlete involved in a highly structured (i.e. gymnastics) program may be a candidate for a stress fracture as the gymnast is exposed to low impact training with high frequency. Athletic footwear with inadequate

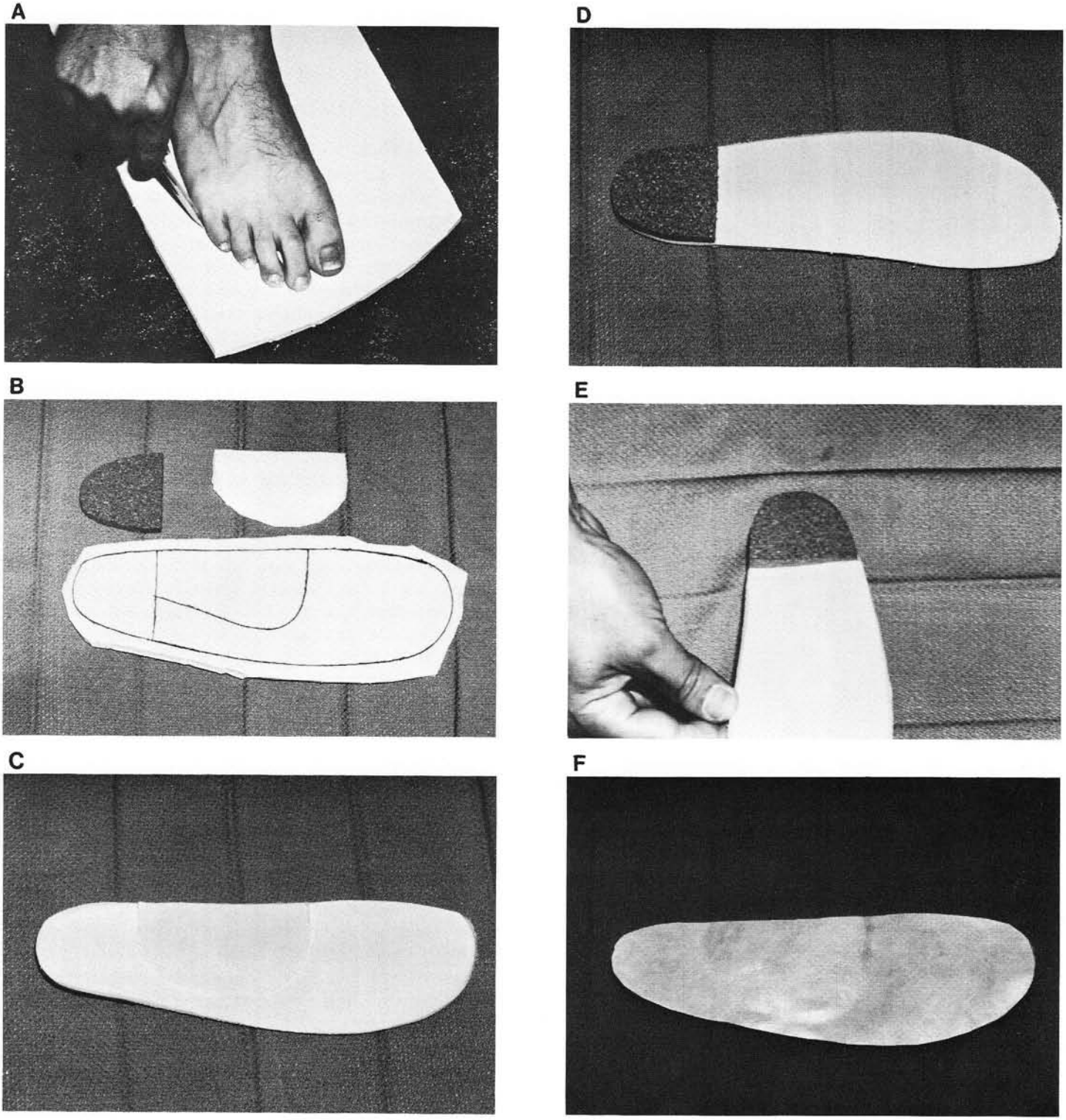


Fig. 4. A. Patient's feet are traced on 3/16 inch SBR material. **B.** Tracing is then marked to incorporate varus arch pad and heel post. **C.** Shell of device is cut out, ground, and varus arch pad applied. **D.** Heel lift, made from Korex is glued to shell and appropriately ground. **E.** Note varus grind to heel post. **F.** Naugahyde top cover is added.

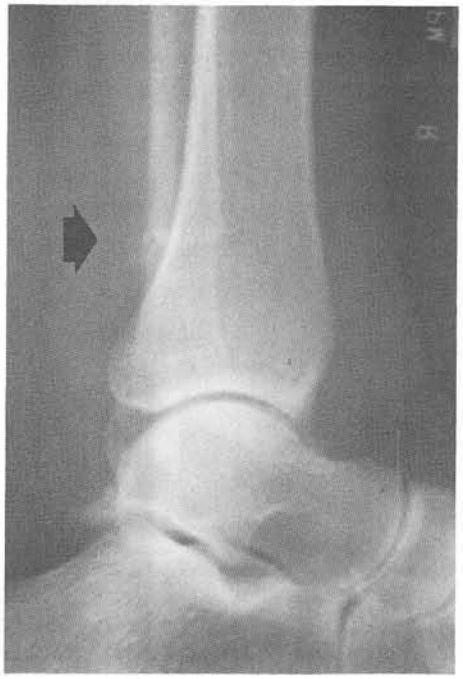


Fig. 5. Distal fibular stress fracture in 36 year old soccer player.

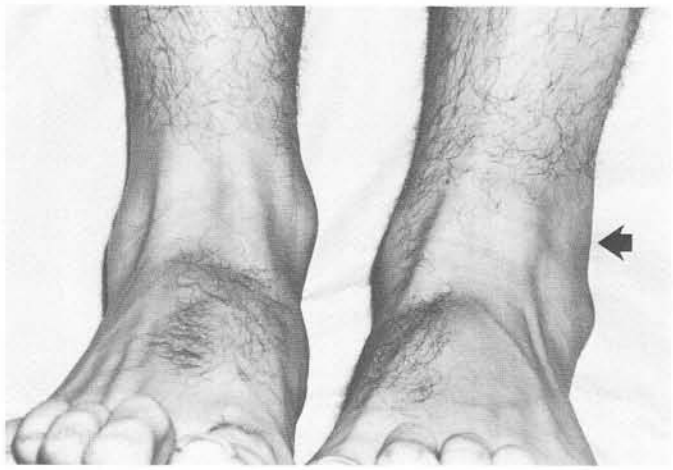


Fig. 6. Note obliteration of normal concave contour above left lateral malleolus in 29 year old runner with distal fibular stress fracture.

shock absorbing outer and midsole material may play a role in the development of a stress fracture.

Taunton and associates (23) have developed a symptoms classification that is useful for the clinician in detecting the possible presence of a stress fracture:

1. Pain initially noted after exercise and relieved by short periods of rest.
2. Pain tolerable during exercise but more marked afterwards and relieved by longer periods of rest.
3. Pain intolerable during exercise as well as after and only partially relieved by longer periods of rest.
4. Constant pain.

This progression of symptoms is commonly seen in patients who relay a history of leg pain and eventually are diagnosed as having a stress fracture. The authors have also discovered that the athlete, during the initial stages of a stress fracture, will relate more pain the morning following the activity as opposed to that during the actual activity.

Fibular Stress Fracture — Clinical Examination

With the fibula being readily accessible to clinical examination, one can palpate this bone throughout its entire length, especially the subcutaneous distal one-third. If the athlete presents with pain and swelling localized to the fibula, in the absence of acute trauma or sprain, one should suspect a fibular stress fracture. In some instances a localized periosteal reaction or new bone formation (callus) can be palpated if the fibular stress fracture has been present for several weeks. Stress fracture of the distal fibula are suspected when the normal concave contour above the lateral malleolus is obliterated due to localized edema and possible new bone formation (Fig. 6). Devas (26) has advocated a clinical test referred to as "springing the fibula," in which the suspected fibular fracture site is strained against a fulcrum to reproduce the pain (Fig. 7A).

Tibial Stress Fracture—Clinical Examination

Because of the larger muscle bulk surrounding the tibia on its posterior and lateral surfaces, this bone is more difficult to palpate than the fibula. Fortunately, most stress fractures of the tibia can be suspected on palpation of the medial subcutaneous border. A stress fracture will manifest as a discrete area of pain with possible evidence of localized swelling and localized periosteal reaction or new bone formation. Often times the pain can be reproduced by having the patient hop on the affected leg for approximately 30-60 seconds, attempting to transmit foot strike shock to the stress fracture site. "Spring the tibia" may also evoke symptoms (Fig. 7B). Nitz and Scoville (27) proposed the use of ultrasound in the diagnosis of stress fractures of the tibial plateau. With a setting of 2 to 3 watts/cm^2 an increase in pain at the stress fracture site was encountered with less than 30 seconds of ultrasound exposure. Ninety percent of their patients with pain on this examination demonstrated radiographic evidence of a stress fracture. Those that did not demonstrate pain during the application of ultrasound had no radiographic evidence of stress fractures.

Radiological Diagnosis

According to Geslien and associates (28), diagnosis on conventional radiographs is confirmed by one or more of the following signs:

1. periosteal new bone formation (Fig. 8),

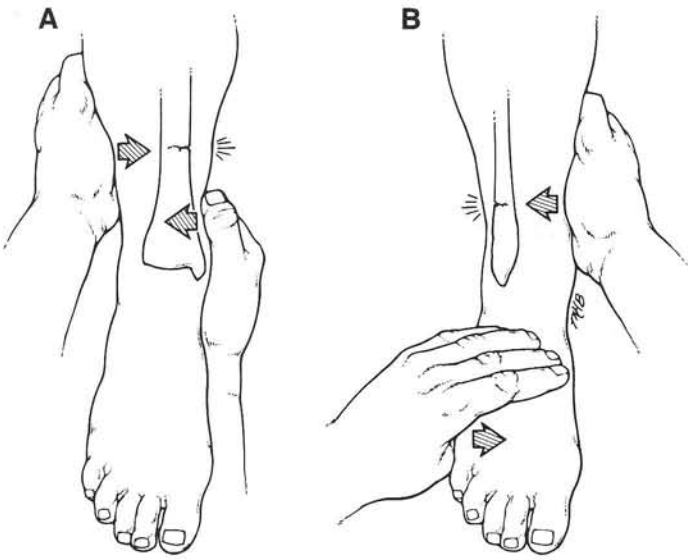


Fig. 7. A. Examiner is attempting to open up suspected tibial stress fracture site by "springing" the tibia. **B.** Examiner is attempting to open up suspected fibular stress fracture site by "springing" the fibula.

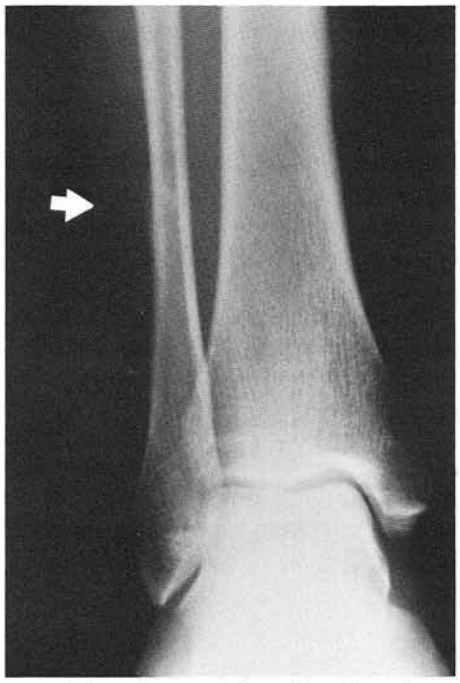


Fig. 8. Periosteal elevation is evident along lateral aspect of distal one-third of fibula in 32 year old runner.

2. endosteal thickening,
3. radiolucent line extending through at least one corex.

Pavlov and Torg (29) state that a localized periosteal reaction and cortical thickening indicate a stress reaction of bone or a healed stress fracture. A radiolucent line within an area of cortical thickening indicates a stress fracture. The shaft of the tibia and fibula will generally demonstrate a cortical break (radiolucent line) or periosteal new bone formation. The ends of these bones will usually demonstrate an area of sclerosis or transverse radiodensity (Fig. 9).

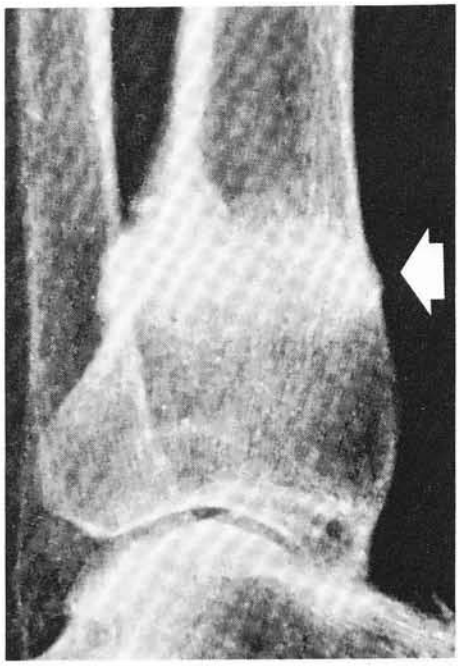


Fig. 9. Note sharp radiolucent line in 43 year old marathoner with distal tibial stress fracture.

The x-ray evidence of a stress fracture is typically delayed until adequate time has occurred for resorption of local bone and/or formation of new bone that can be detected radiographically (30). This accounts for delays in x-ray diagnosis for weeks to several months, with some never being radiographically apparent (28, 31-33).

With the highly competitive or professional athlete, the early diagnosis of a stress fracture is crucial in allowing resumed participation in a timely and safe manner. When standard radiographs do not demonstrate evidence of a stress fracture, in light of apparent clinical and subjective symptoms, the clinician must either convince the athlete to rest and repeat the x-rays in several weeks or have the patient undergo a technetium 99m bone scan. Because of its high sensitivity, the negative bone scan is considered to rule out the possibility of the pain being from a stress fracture.

Detmer (8) classifies a Type IA chronic medial tibial stress syndrome as a localized stress fracture on the medial surface of the distal one-third of the tibia and a Type IB chronic medial tibial stress syndrome as a diffuse stress reaction of the tibia. These are differentiated clinically (localized pain versus diffuse area of pain) as well as radiographically (localized uptake of technetium 99m as opposed to more diffuse uptake). A true stress fracture will demonstrate a localized uptake of radionuclide (Fig. 10), whereas a stress reaction of bone or stress microfracture will demonstrate a diffuse uptake of radionuclide. Wilcox and associates (33) stated that a normal bone scan excludes the diagnosis of a stress fracture. However, Milgrom and associates (34)

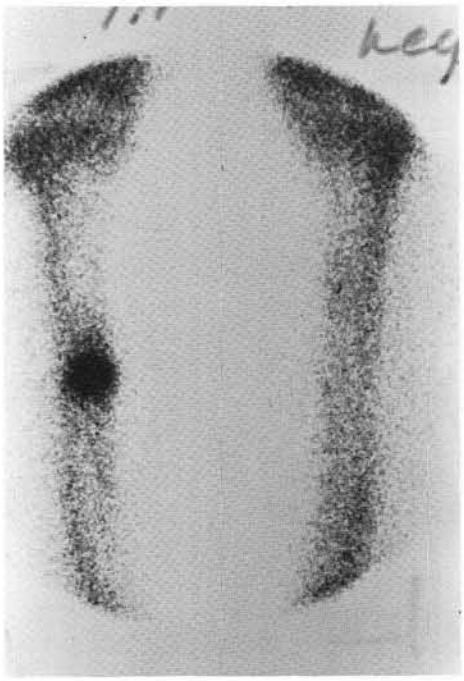


Fig. 10. Discrete uptake of radionuclide is seen in 34 year old jogger with proximal tibial stress fracture.

discovered three military recruits who presented with tibial pain on exertion and whose initial bone scans were negative. However the scans were found to be positive when they were repeated one month later.

Treatment

Rest from the inciting activity is the key to treatment of stress fractures of the tibia or fibula. In addition to allowing the repairative processes to occur, the rest will also allow the fatigued muscles to recover and improve shock absorption to the bone. With the stress fracture of the tibia the athlete should cease strenuous weight-bearing activities for 4 to 6 weeks. Bicycling should be substituted (if no symptoms are provoked) to continue with cardiovascular conditioning and maintain strength of the lower extremities. Many dedicated runners will secure a life vest and run in a swimming pool. If ceasing the activity is not reducing the symptoms, the athlete is placed on crutches until weightbearing is tolerated or may be placed in a short leg walking cast for 2-4 weeks.

Recently, the use of the Air Cast short leg training brace has allowed athletes to return to performance more rapidly after using the cast for recovery. The athlete can then maintain the use of the brace during the initial stages of return to activity. If biomechanical factors are involved as a possible cause of overuse of muscles around the tibia and fibula, appropriate biomechanical orthotic devices are utilized. The athlete is placed on a stretching and strengthening program of all lower extremity muscles during and after recovery. On rare occasions athletes with stress fractures have developed a painful nonunion, requiring currettage and bone grafting.

Compartment Syndromes of the Lower Leg

A compartment syndrome is a condition in which high pressure within a closed fascial space (muscle compartment) reduces capillary blood perfusion below a level necessary for tissue viability. These syndromes may develop in skeletal muscles enclosed by a non-compliant osseofascial boundary. A build-up of pressure is due to the inelastic nature of the muscle investing fascia. If pressure remains sufficiently high for several hours, normal function of muscles and nerves is jeopardized and myoneural necrosis eventually will result (35, 36).

There are four anatomic compartments of the lower leg, each containing muscles, nerves, and blood vessels. Garfin (37) has described the boundaries and contents of the compartments in the lower leg (Figs. 11, 12).

Recently, Davey and associates (38) have considered the posterior tibial muscle as a separate compartment, as it appears to be contained within its own fluid impermeable osseofascial compartment. They found an increase in compartment pressure within this muscle that was considerably higher than the pressure in the deep posterior compartment when the patient was exercising on a treadmill. Fasciotomy of the tibialis posterior muscle alleviated the pain and weakness of a runner that previously had a deep posterior compartment fasciotomy without good results.

Owen (39) has described clinical signs of symptoms that will assist the clinician in making the appropriate diagnosis:

1. Pain out of proportion to the problem. Pain described as deep, throbbing, and leg is under unrelenting pressure.
2. Palpably swollen, tense compartment.
3. Pain with passive stretch of the muscles involved in the compartment due to muscle ischemia.
4. Sensory deficit, initiating with paresthesia and progressing to hypoesthesia and finally, anesthesia.
5. Motor weakness.
6. Dorsalis pedis and posterior tibial pulses will usually be intact as the intracompartmental pressure is not high enough to occlude them.

Compartment syndromes can be categorized as acute or chronic. The acute form, usually following trauma, is present when the intracompartmental pressure is elevated at a level and duration such that decompression of the involved compartment is necessary to prevent necrosis and preserve limb viability (35, 36). Exercise combined with

COMPARTMENT	BOUNDARIES	CONTENTS
ANTERIOR	ANTERIOR: Crural Fascia LATERAL: Anterior Intermuscular Septum POSTERIOR: Tibia, Fibula, Interosseous Membrane	Tibialis Anterior, Extensor Digitorum Longus, Extensor Hallucis Longus, Peroneus Tertius Anterior Tibial Artery, Deep Peroneal Nerve
LATERAL	ANTERIOR & LATERAL: Crural Fascia MEDIAL: Fibula, Anterior Intermuscular Septum POSTERIOR: Posterior Intermuscular Septum	Peroneus Longus, Peroneus Brevis, Superficial Peroneal Nerve
DEEP POSTERIOR	ANTERIOR: Tibia, Fibula, Interosseous Membrane POSTERIOR: Posterior Intermuscular Septum	Tibialis Posterior, Flexor Digitorum Longus Flexor Hallucis Longus, Tibial Nerve, Posterior Tibial and Peroneal Arteries
SUPERFICIAL POSTERIOR:	ANTERIOR, MEDIAL, LATERAL AND POSTERIOR: Entirely invested by fascia.	Gastrocnemius, Soleus, Planatris, Sural Nerve Branches of Posterior Tibial and Peroneal Arteries

Fig. 11. Anatomical boundaries and contents of osseofacial compartments of lower leg.

fascial inelasticity or muscle hypertrophy, in the absence of acute trauma, can cause an acute compartment syndrome.

The chronic or exertional compartment syndrome has become increasingly prevalent in the athletic community. Martens and associates (40) reported on 29 patients having chronic leg pains due to a recurrent exertional compartment syndrome in various lower leg compartments. This condition occurs when exercise raises intracompartmental pressure sufficiently to produce ischemia, pain, and neurological deficit. This represents a mild recurrent compartment syndrome associated with exercise in which symptoms of pain and nerve loss spontaneously resolve with rest. If the athletic activity is continued, despite pain and neuromuscular deficit, a chronic compartment syndrome can progress to an acute one (35, 36).

Collagen tissue under prolonged stretch responds by aligning its fibers and increasing both its density and strength (41). A progressive increase in symptoms of an athlete with the exertional compartment syndrome is due to the increase in density and strength of the fascia against the enclosed compartment and its vital structures. Mubarak (15-18) believes that a trained athlete whose muscles have hypertrophied from repetitive training has less room in the compartment for the accommodation of swelling that may be sustained during normal exercise.

Clinical Signs and Symptoms

Anterior Compartment:

Pain and a feeling of increased pressure in the anterior leg. Weakness of dorsiflexion power to the toes, foot, or

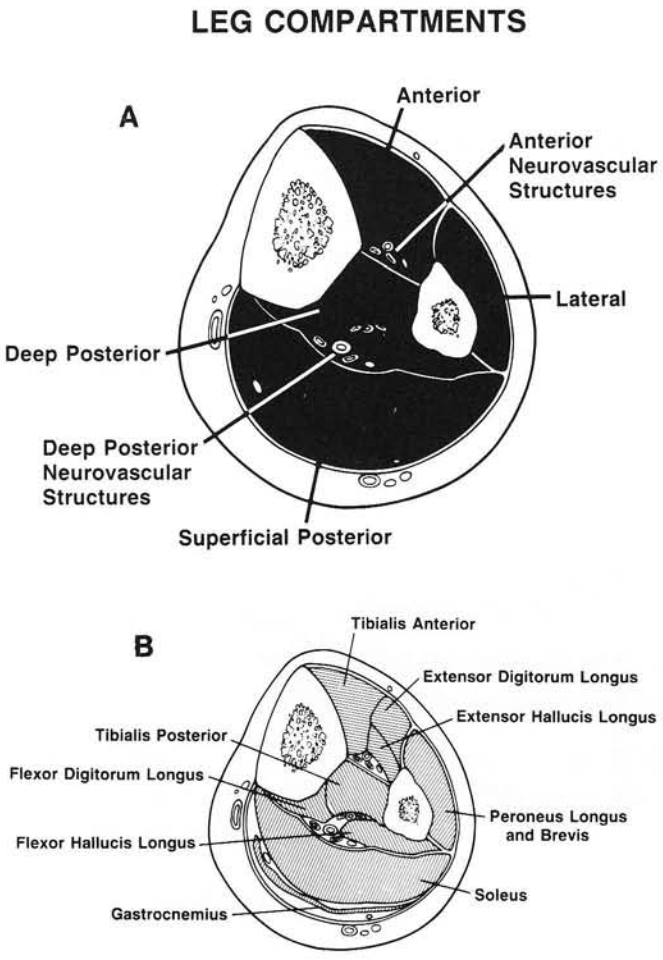


Fig. 12. A. Compartments of lower leg. **B.** Compartments of lower leg and their contents.

ankle during manual muscle testing, after exercise or trauma. Pain in the anterior muscle compartment on passive plantarflexion of the ankle, an entity that has been described as "stretch pain" (42). Obvious muscle herniations have been reported in the range of 20% to 60% (15-18, 43). A sensory loss is noted in the first web space dorsally.

Lateral Compartment:

Pain and a feeling of increased pressure along the lateral aspect of the leg. "Stretch pain" on passive inversion of the ankle. Pain and weakness during resisted eversion of the ankle. Fascial hernias have been reported in the range of 20% to 60% (15-18, 43). There is a sensory loss to the dorsum of the foot and digits.

Deep Posterior Compartment:

There is pain on passive eversion of the foot and also on active resistance to inversion with plantarflexion. Weakness of the subtalar invertors and digit plantarflexors. Decreased sensation to the plantar surface of the foot.

Superficial Posterior Compartment:

Calf pain with passive dorsiflexion or active plantarflexion against resistance. Slight weakness to toe raises may possibly be noted. Decreased sensation to the lateral border of the foot.

Laboratory Diagnosis of Compartment Syndromes

Detailed descriptions of the Wick and Slit catheters have been described in great detail (35). We have been recently introduced to the Stryker STIC pressure monitor system and were pleased with its relative ease of operation (Fig. 13). The normal resting pressure in the anterior compartment is 4 ± mm Hg. With exercise, this will rise to more than 50 mm Hg. In normal patients, pressure will decline below 30 mm Hg immediately and in 5 minutes will return to normal. In those with a chronic compartment syndrome, a resting pressure of 15 mm Hg. is noted. During exercise the pressure rises to 75 mm Hg. and at 5 minutes following exercise the pressure is over 30 mm Hg. (15-18). For patients presenting with an apparent acute compartment syndrome, a resting compartmental pressure of over 30 mg Hg., in the presence of other positive clinical findings, is diagnostic of an acute compartment syndrome (35, 36).

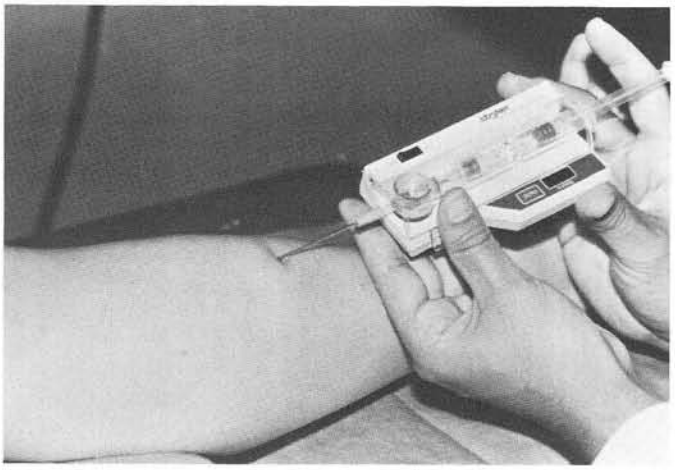


Fig. 13. Clinical measurement of anterior osteofacial compartment pressure using Stryker STIC.

Treatment

For the athletic patient with the exertional compartment syndrome, the only conservative option is to increase the rest periods between the activity or undertake several weeks of rest after the onset of the symptoms. If this fails and the athlete is unwilling to give up the activity then a fasciotomy of the involved compartment is indicated after clinical examination and pressure measurements confirm the diagnosis. In the acute compartment syndrome patient, immediate surgical decompression of the involved compartments is necessary to prevent irreversible damage to the lower leg. The surgical techniques for the decompression of the lower extremity compartments is thoroughly described by Mubarak (15-18).

Superficial Peroneal Nerve Entrapment Syndrome

Inversion sprains of the ankle have been recognized as a causative factor in the development of neuropathy to the superficial peroneal nerve (44). The mechanism is a plantarflexion-inversion injury to the ankle. The nerve is then tethered proximally at its point of origin at the fibular neck and distally by subcutaneous attachment, hence the inversion force will force the nerve taut against the fascial opening resulting in possible trauma to the nerve.

In two patients encountered in our office and in a patient evaluated by Garfin and associates (37) the entrapment of the superficial peroneal nerve was a direct result of muscular exertion from athletic activity alone, without the presence of acute trauma or sprain. The apparent cause was an exercise induced hypertrophy of the peroneal muscle compartment which then pouched through a defect in the deep fascia and compressed the nerve.

Anatomy

The superficial peroneal nerve is a branch of the common peroneal nerve. As it enters the lower one third of the leg, the nerve pierces the deep fascia through a fascial opening or foramen, then divides into the medial and intermediate dorsal cutaneous nerves. Kosinski (45) reported that the division of the superficial peroneal nerve into its component branches occurred approximately 10.5 cm above the lateral malleolus in 75% of anatomic dissection specimens in 110 extremities (Fig. 14). Continuing down from the fascial foramen, the intermediate dorsal cutane-

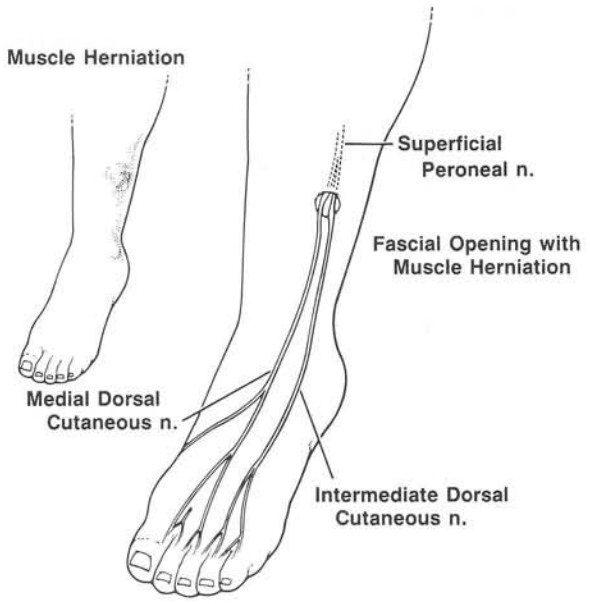


Fig. 14. Anatomy of distal portion of superficial peroneal nerve and its component branches.

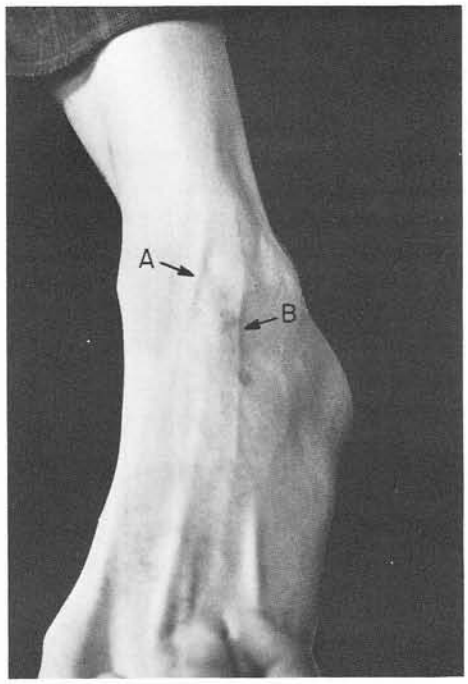


Fig. 15. Topography of medial and intermediate dorsal cutaneous nerves. Photo — Harvey Lemont, D.P.M.

ous nerve and medial dorsal cutaneous nerve can be readily identified. Lemont (46, 47) showed that the intermediate dorsal cutaneous and medial dorsal cutaneous nerves can be identified and palpated when the examiner plantarflexes and inverts the foot (Fig. 15). The intermediate dorsal cutaneous nerve continues down the distal anterolateral aspect of the leg and ankle, and supplies the lateral dorsum of the foot and contiguous sides of the third, fourth, and fifth digits dorsally. The medial dorsal cutaneous nerve continues down the anterior aspect of the ankle and courses medially to supply the skin on the medial dorsum of the foot, the medial dorsal aspect of the great toe and the contiguous sides of the second and third digits dorsally.

Symptoms

The patient will usually present with vague, non specific or non localized pain situated along the anterolateral aspects of the lowe leg and ankle. The pain may be described as burning, shooting, or sharp and a history of traveling "electric shock" sensations can be elicited as going distal towards the toes or proximal in the leg. Hypoesthesia is noted over the course of one or both branches of the nerve.

Clinical Examination

The examination will reveal pain in the area where the nerve exits from the deep fascia. There will, in most cases, be an obvious muscle herniation at the location where symptoms can be elicited (Fig. 14[inset]). This muscle herniation, if not readily seen, can be provoked by having the patient perform active dorsiflexion and eversion maneuvers of the ankle against the clinician's resistance for approximately 30 seconds to one minute. On percussion of the muscle herniation, a Tinel's sign can be elicited. Careful evaluation of the sensorium will reveal areas of hypoesthesia on the dorsum of the foot and toes. There is no loss of motor function with this disorder.

Recently, Izzo and associates (48) have demonstrated a reliable, reproducible method for obtaining antidromic conduction velocities of the sensory branches of the superficial peroneal nerve. If this disorder is suspected, a referral to a neurologist for nerve conduction studies is indicated. It is wise to accompany the patient or send a copy of the Izzo and associate's article since some neurologists are not familiar with exact anatomical placement of the distal electrodes, therefore compromising the results of the study.

The basic etiology of the disorder is a compression of the superficial peroneal nerve in an area of the fascial defect, or normal opening, with muscle herniation. Wright (49) demonstrated that there was a 20% increase in the volume of the musculature in a compartment during exercise. This increased muscle volume may cause a defect in the fascia or cause herniation through existing ones, causing pathology to the nerve.

Treatment

Conservative treatment includes the local use of mixed local anesthetic, cortisone, and vitamin B-12 injection at the trigger point of symptoms. Resting from the activity, that is increasing the underlying muscle mass, may help in the conservative management. Continued performance of

the activities, however, usually brings about a renewal of the symptoms. A more definitive approach takes the form of a fasciotomy of the involved compartment that is irritating the nerve.

The incision is centered over the fascia defect or muscle herniation so one can have good access to the fascia to incise it both proximally and distally to reduce localized muscle herniation against the nerve (Fig. 16). The nerve is then explored for a neuroma or other soft tissue pathology. The patient is then placed in a non weight-bearing posterior splint for one week. Ambulation is then initiated in a postoperative shoe until the sutures are removed. Activities can be initiated at 3 to 4 weeks following surgery in a gradual manner.

Popliteal Artery Entrapment Syndrome

In the evaluation of athletes who present with leg pain it is essential for the clinician to not only evaluate the musculoskeletal system but also the neurovascular status as well, even in the absence of acute trauma. A vascular disorder of intermittent claudication, an entity thought rare in the young athlete, has been reported with increasing frequency (50-54).

The underlying cause of this disorder is an intermittent compression or entrapment of the popliteal artery by one or both heads of the gastrocnemius muscle, usually the medial one (54). This disorder is caused by an anatomic aberration between the popliteal artery and the surrounding gastrocnemius muscle or an aberrant fibrous band that compresses the artery against the femur (52).

According to Insua and associates (55) the anatomical variations of the gastrocnemius muscle and the popliteal artery can be classified into two groups: Group 1. The artery passes medially and below the medial head of the gastrocnemius (Fig. 17). Group 2. The artery is compressed in its normal course by an aberrant insertion of the gastrocnemius or plantaris muscle.

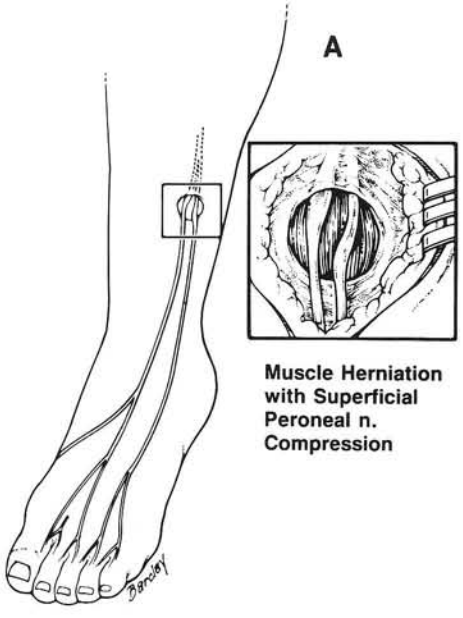
Symptoms

The major presenting symptoms include paresthesia and/or pain in the foot after running or working out (56), intermittent claudication (51, 57), burning of the foot (50), and a cool cyanotic nature to the lower leg and foot (52). The intermittent claudication is brought on by exercise and relieved by rest.

Clinical Examination

The clinical examination may reveal a cool lower leg, ankle, or foot. Additionally, pallor or a cyanotic appearance of the toes, a delayed subpapillary venous plexus fill time and an absent pulse, especially with active plantarflexion or passive dorsiflexion of the ankle with the knee extended (52) may be noted.

In the presence of diminished or absent pulses as well as other local signs and symptoms suggestive of a lack of adequate circulation, the clinician should evaluate the patient with the use of a Pulse Volume Recorder. Further positive findings would necessitate a referral to a vascular surgeon for femoral arteriography.



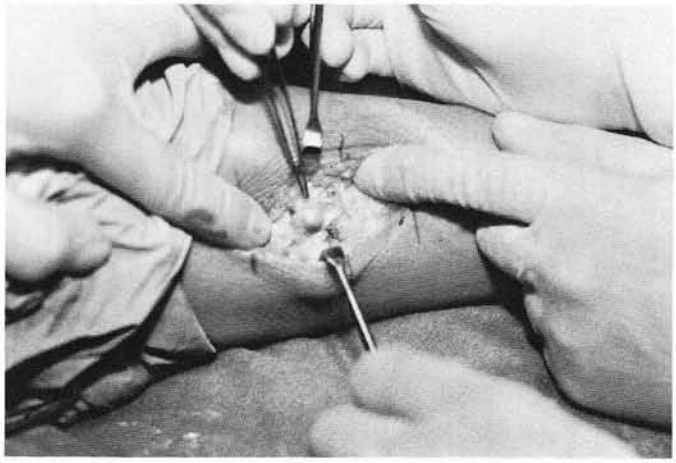


Fig. 16. A. Operative site of superficial peroneal nerve entrapment secondary to underlying muscle herniation. **B.** Note underlying peroneal muscles herniating through deep fascia and compressing superficial peroneal nerve.

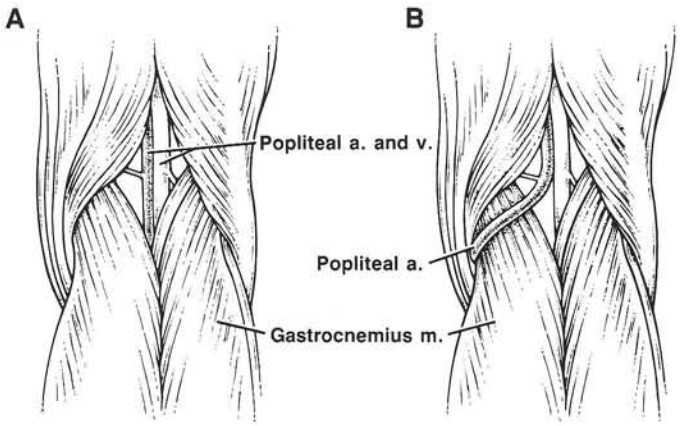


Fig. 17. A. Normal anatomy of popliteal artery in popliteal fossa. **B.** Popliteal artery is abnormally coursing medially and under medial head of gastrocnemius muscle.

The importance of early diagnosis cannot be stressed enough since progressive damage to the artery leads to thrombosis, aneurysm formation and peripheral ischemia. Patients with this problem are frequently overlooked initially because of reluctance to diagnosis vascular compromise in a young athletic patient or due to lack of knowledge of the disorder (57).

Treatment

Appropriate exploration of the popliteal area is indicated to decompress fibrous or muscular anomalies around the popliteal artery. Additionally, bypass grafts may or may not be used in conjunction with decompression or resection of constricting structures.

Ruptures of the Medial Head of the Gastrocnemius

A disorder that is commonly encountered by the sports oriented physician is a rupture or strain of the medial head or medial myotendinous junction of the gastrocnemius muscle (Fig. 18). This entity has been referred to as "tennis leg" due to its fairly high incidence in tennis players.

A partial or total rupture of the medial head of myotendinous junction of the gastrocnemius muscle typically occurs in the middle aged athlete (58, 59). The patient will generally reveal that he or she had not performed adequate stretching or warm up exercises prior to the start of a sport that requires sudden changes of direction (tennis, racquet ball, basketball). The air temperature may be slightly cool or cold, further contributing to the lack of "warm up" of the muscles.

When the athlete attempts to reach for a difficult shot during a tennis match or a poorly thrown pass in a basketball game, the gastrocnemius muscle is placed under maximum stretch in a ballistic manner. This is accentuated if the athlete has the planted foot fixed to the ground while the leg anteriorly migrates over the foot during the reaching attempt. The gastrocnemius muscle, along its medial head or medial myotendinous junction, may now be prone to partial or total rupture.

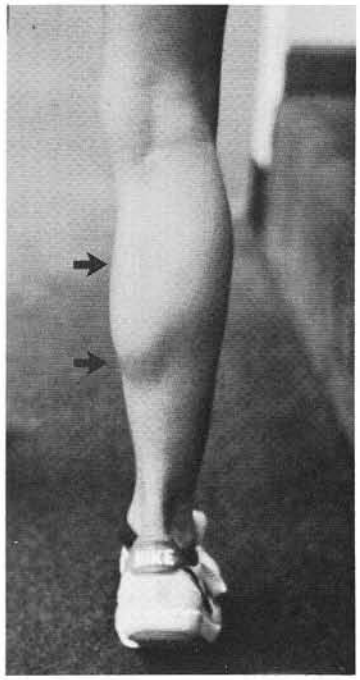


Fig. 18. Two most common locations for injury to gastrocnemius muscle involved medial head and medial myotendinous junction.

Clinical Examination

The examination will reveal that the patient will demonstrate pain at either the medial head of the gastrocnemius muscle or at the medial myotendinous junction (Fig. 19). Usually within two weeks the examiner may be able to palpate a defect at the injured site if this is not initially possible due to hemorrhage (Fig. 20) (58). Passive dorsiflexion of the foot with the knee extended will reproduce acute pain. Ecchymosis and edema may be present, but due to the effect of gravity, such ecchymosis may be evident more distally in the lower leg and possibly the ankle. The patient is generally unable to bear weight on the injured leg and cannot perform a toe raise. The Thompson's test will not be positive with this type of injury.

Although some have implicated this disorder as a tear or strain of the plantaris tendon (60), evidence gained during surgical repair of this injury has demonstrated that this injury represents a tear of the medial head of the gastrocnemius muscle (61). Arner and Lindholm (62) felt that plantarflexion weakness or significant inability to stand on the toes of the affected leg would not be associated with a rupture of the insignificant plantaris tendon.

It is generally accepted that decreased muscle and tendon blood flow may predispose the middle aged athlete to this entity (62).

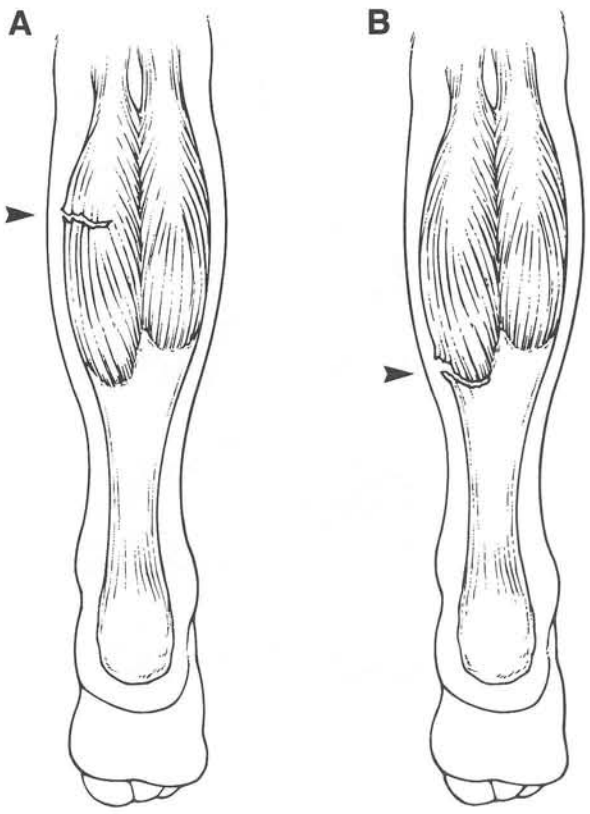


Fig. 19. A. Rupture of medial head of gastrocnemius muscle. **B.** Rupture of medial myotendinous junction of gastrocnemius muscle.

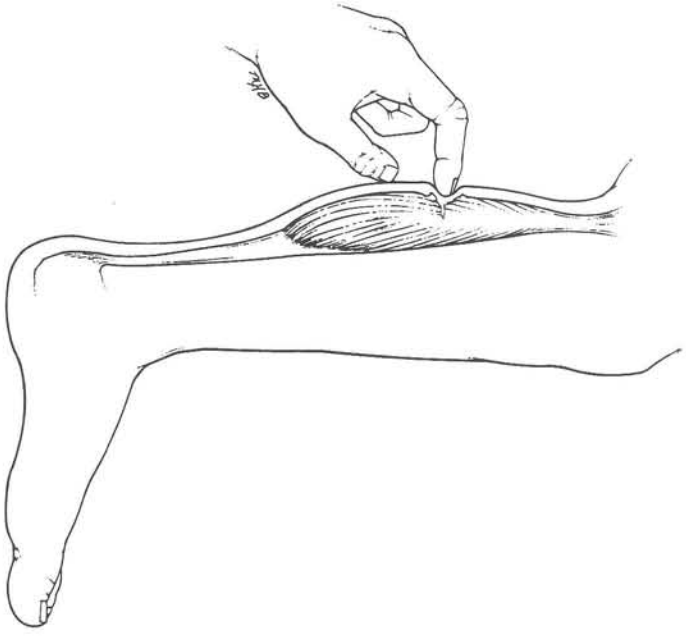


Fig. 20. Palpation of defect in ruptured medial head of gastrocnemius muscle.

Treatment

Initially the principles of rest, ice, compression, and elevation (RICE) should be utilized by the physician to decrease pain and edema. In situations where patients are in acute pain, a localized injection of a local anesthetic into the torn muscle will reduce pain as well as muscle spasm. A compression dressing is applied from the toes to the knee. The patient is placed on crutches with no weight on the affected leg for 48 hours. Ice is used continuously through the thick compression dressing for 48 hours. The patient is also placed on appropriate NSAIDs to reduce pain and inflammation. The athlete should return in 48 hours.

For partial tears of the muscle the athlete is placed in an Achilles tendon rest strapping with a one-fourth to one-half inch heel lift in a roomy shoe. Intermittent ice pack application is used for 5 minutes, three times daily. When the patient can bear weight without significant symptoms the rest strapping is discontinued and the heel lift continued. This usually takes place in one to two weeks. Light gastrocnemius stretching and strengthening is started at this time. Ultrasound therapy is also used. The activity is increased to include toe raises with weights and more intense stretching after another week or two. The athlete resumes competition with the heel lift but gradually tapers off use of the lift in the next two to four weeks.

For total ruptures of the muscle, the athlete is kept non weightbearing in a slightly plantarflexed posterior splint for 3 weeks. This progresses to strapping and heel lift in addition to physical therapy. A referral to a physical therapist will insure that the exercise prescription is followed and an accurate assessment of strength can be determined on the Cybex unit available to most therapists prior to allowing the athlete to return to activity.

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