Chronic Exercise-Induced Leg Pain in Active People

More Than Just Shin Splints

Lew C. Schon, MD

Donald E. Baxter, MD

Thomas 0. Clanton, MD

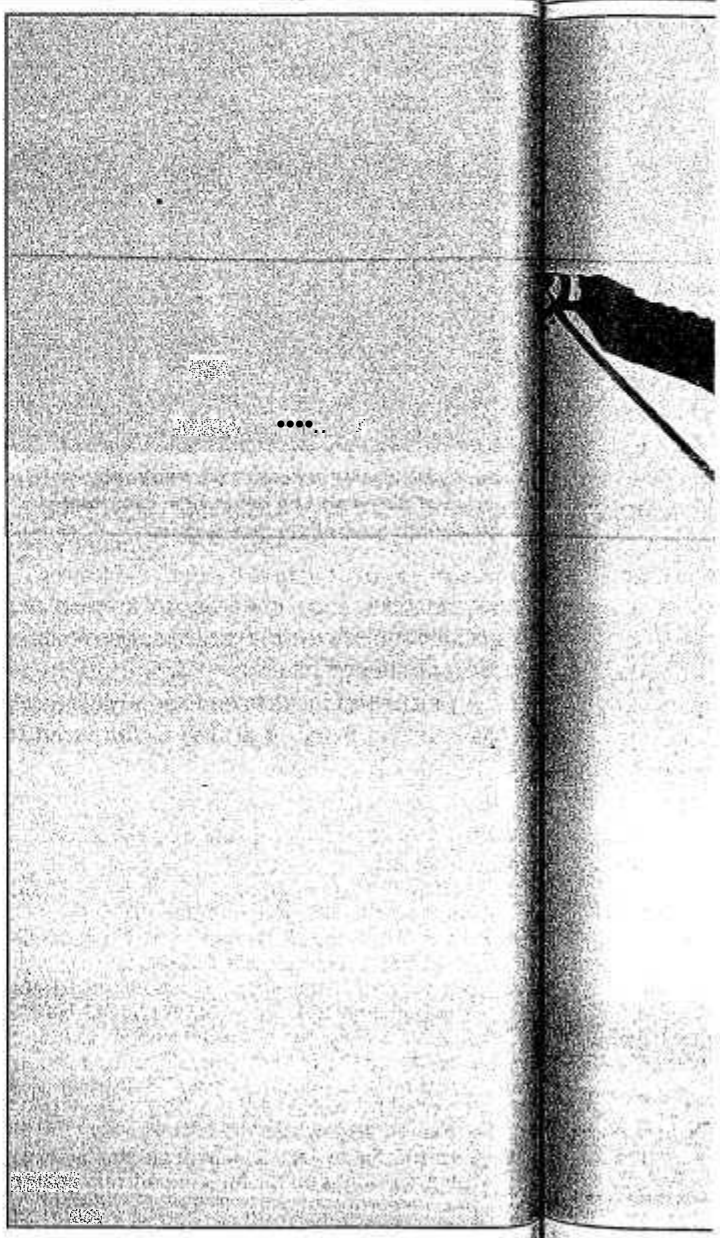
Foot and Ankle Series Editor:
G. James Sammarco, MD

In brief "Shin splints" is a catchall term for any kind of persistent exercise-related lower leg pain with no obvious cause. Such pain can originate from a number of conditions, such as medial **tibial** stress syndrome, stress fracture, compartment syndrome, vascular pathology, nerve entrapment, and others. A methodical work-up designed to detect problems in all anatomic structures from bone to skin will narrow the possibilities and lay the basis for appropriate treatment.

To most people, physicians and patients alike, persistent lower leg pain related to exercise means "shin splints." This term includes many conditions that must be considered when evaluating chronic exercise-induced leg pain in active individuals. Injuries may affect every anatomic structure in the leg: bones,

continued

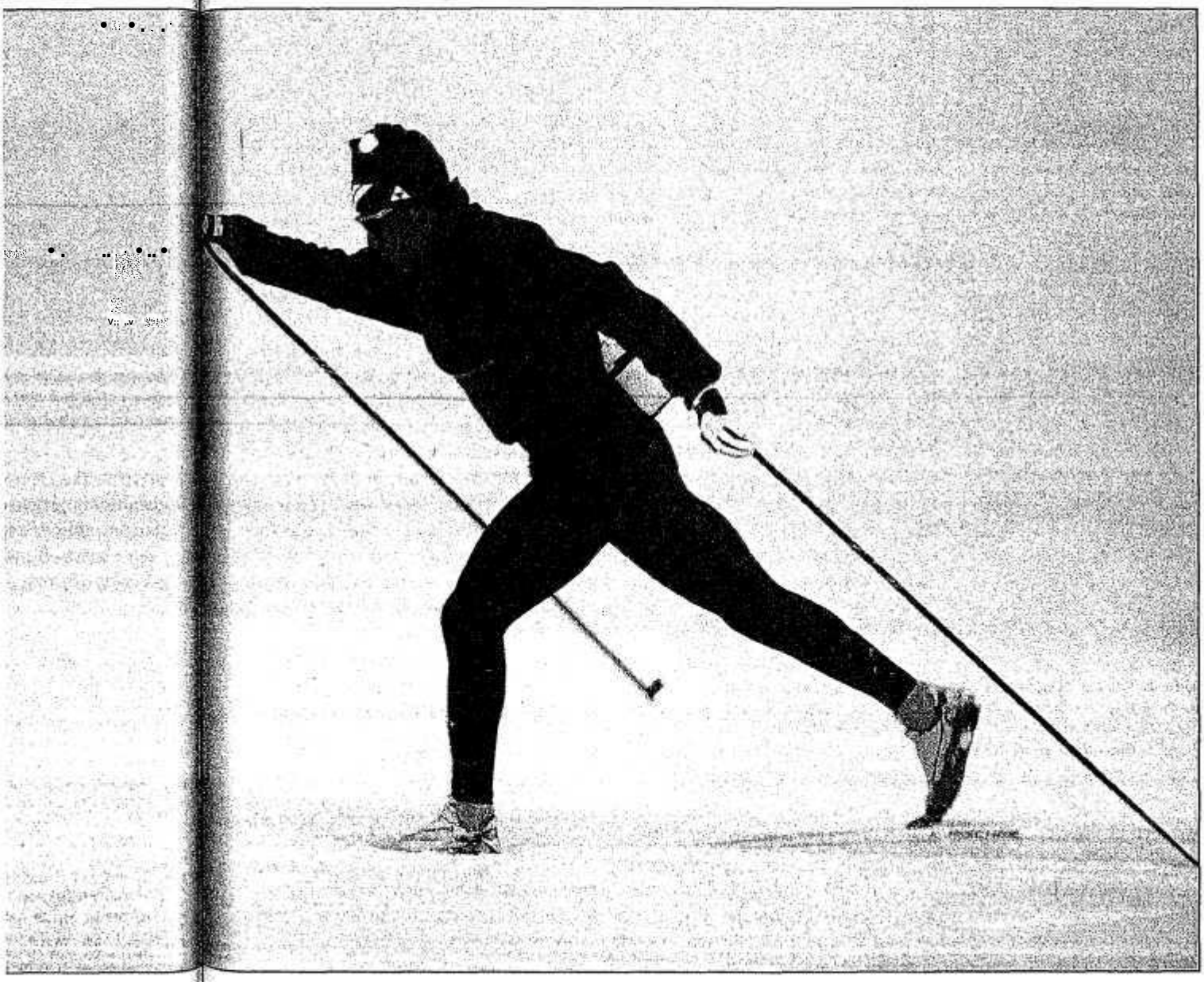
Dr Schon is an attending orthopedic surgeon at Union Memorial Hospital in Baltimore. In addition, Dr Schon is a clinical instructor of orthopedics, Dr Baxter is a clinical associate professor of orthopedics, and Dr Clanton is a clinical assistant professor of orthopedics at the University of Texas Health Science Center at Houston School of Medicine. Dr Baxter is also director of the foot and ankle fellowship at the same school, and is a clinical associate professor of orthopedics at the Baylor College of Medicine in Houston. Dr Sammarco is an orthopedist with the Center for Orthopedic Care, Inc, in Cincinnati and a consulting physician to the University of Cincinnati Department of Athletics and the Cincinnati Ballet Company. He is a member of the editorial board of THE PHYSICIAN AND SPORTSMEDICINE and a member of the American College of Sports Medicine. Drs Baxter, Clanton, and Sammarco are fellows of the American Orthopedic Foot and Ankle Society.



If repetitive impact causes leg pain, patients may need to glide into a different sport.

id Leg

Photo: © 1992. Caroline Wood/F-Stock



Photos: © 1992. Courtesy of Lawrence Holder, MD, Union Memorial Hospital, Baltimore.

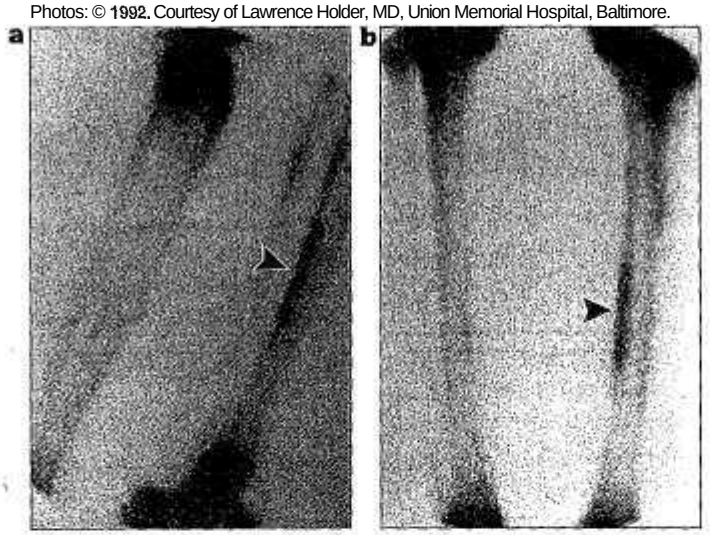


Figure 1. Medial tibial stress syndrome of a young, male adult ***runner.*** *Lateral (a) and anteroposterior (b) views of a patient by technetium bone scan show diffuse linear uptake along the posteromedial cortex of the tibia (arrows).*

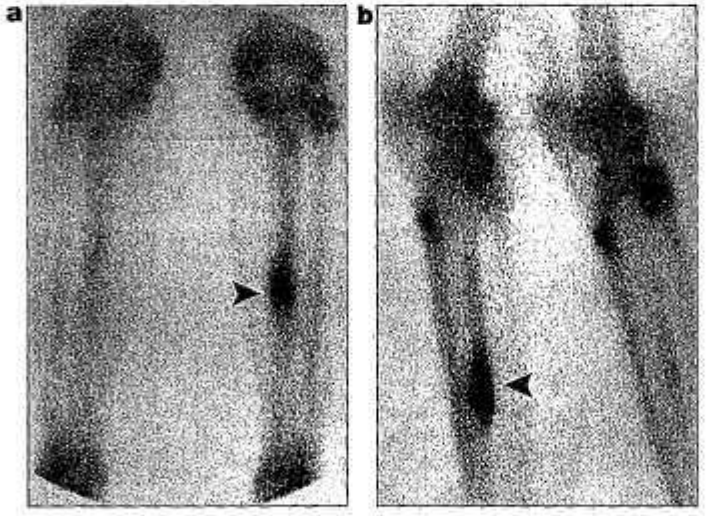


Figure 2. Medial tibial stress fracture of a young, male adult runner. Anteroposterior (a) and lateral (b) technetium bone scan views showing localized intense uptake along the posteromedial border of the midtibia (arrows).

compartments, muscles, tendons, ligaments, fascia, arteries, veins, nerves, and skin. The differential diagnosis of chronic exercise-induced leg pain, therefore, should include medial tibial stress syndrome, stress fracture, chronic compartment syndrome, muscle strain, muscle herniation, tendinitis, fasciitis, arterial insufficiency venous pathology, nerve entrapment, and radiculopathy—each, of course, with a variety of expressions.

To complicate the picture, several conditions may coexist. A history, a physical examination, and an observation of the patient performing the pain-causing activity are needed to establish an accurate diagnosis. The eight injuries most difficult to diagnose are detailed below.

Medial Tibial Stress Syndrome

The etiology of medial tibial stress syndrome is open to debate. Although some contend that the pain relates to periostitis underneath the posterior tibialis muscle, more recently others have implicated the investing fascia of the soleus as it inserts on the posterior medial tibia. Most agree that the syndrome is caused by a stress reaction of the fascia, periosteum, or bone—or a combination of these—along the posteromedial aspect of the tibia. These increased stresses on the fascia and periosteum may result from abnormal pronation, excessive subtalar motion, or both.[1,2]

A study by Clement et al[3] of 1,819 common running injuries affecting all parts of the body reported 239 cases (13.1%) of tibial stress syndrome, making it the second most common injury in the study. Detmer[4] described three causes of medial tibial stress syndrome: Type I is a stress reaction or fracture of the bone, type II indicates a chronic periosteal elevation from the bone secondary to too much pull from the soleus fascia, and type III is a posterior or isolated posterior tibialis compartment syndrome.

Patients who develop medial tibial stress syndrome report recurrent discomfort along the medial border of the middle and distal tibia. This pain is exacerbated by exercise and incompletely relieved by rest. In particular, running on banked indoor tracks or uneven tenain can induce the syndrome.

Physical examination reveals tenderness along the posteromedial border of the tibia. Occasionally, local induration is noted, but any neurologic or vascular abnormalities rule out tibial stress syndrome. Pronation and excessive subtalar range of motion are common.

Although radiographs frequently are normal, a bone scan may demonstrate diffuse linear up-

continued

take along the posteromedial border of the tibia involving one third of the bone's length (figure 1).[5] Pressure studies of the suspected compartments before and after exercise will help identify a deep posterior or posterior tibialis compartment syndrome or rule out concurrent anterior compartment syndrome.

Treatment of medial tibial stress syndrome consists of rest followed by a gradual return to activity Achilles tendon stretches, heel pads, casts, local steroid injections, and taping have been used with varying success. Athletes who overpronate or have abnormal subtalar mobility and who don't heal after relative rest, may be fitted with orthotics. If conservative modalities fail, a posteromedial fasciotomy releasing the medial soleus fascia and deep posterior compartment may be warranted.[1]

Tibial or Fibular Stress Fracture

Stress fractures develop when repetitive loads cause bones to resorb more than they form. Physicians often see stress fractures in runners. Clement et al's study[3] of common overuse injuries reported 47 (2.6%) tibial stress fractures. It was the eighth most prevalent running injury in the study. These fractures also occur regularly in basketball, soccer, dancing, and skating. Athletes who have smaller diameter bones, less cortical bone, or diminished bone mineral content are at increased risk for stress fractures. (See "Repeated Stress Fractures in an Amenorrheic Marathoner," April 1989, page 65.)

Typically, an athlete who sustains a stress fracture will report a recent change in routine, running surface, or shoes. Frequently, symptoms crescendo over the course of several weeks, but in rare cases they occur quite suddenly The pain initially may be diffuse, but with time it becomes localized. Impact activities such as jumping will exacerbate symptoms.

An athlete who has a stress fracture may have local bony tenderness associated with induration of the overlying soft tissues. Increased warmth and even erythema may also be present. Some patients may have a tender, firm mass over the fracture site. Tapping or stressing the bone may elicit pain at the fracture site.

Radiography may not show changes for 2 or more weeks and in some cases may never reveal the fracture. The first sign of a stress fracture often will be a faint localized radiopaque haze. In time, a small cortical lucency and a more established periosteal reaction may appear. In cases of longer duration, cortical lucencies may traverse the bone.

Although magnetic resonance imaging (MRI) may be more sensitive than other studies for detecting a stress fracture early, technetium bone scanning remains the diagnostic modality of choice. The characteristic finding in a stress fracture is intense local activity with a fusiform accumulation of tracer (figure 2).

Treatment involves rest and avoidance of the offending stresses. Although immobilization is rarely necessary, weight bearing is not advisable. Once the pain has diminished, the patient can resume weight-bearing activities as tolerated. During recuperation, athletes can cross-train to maintain cardiovascular and muscle conditioning.

> ***Typically, an** athlete **who** sustains a stress fracture will report a recent change in routine, running **surface,** or footwear.*

Anterior tibial stress fractures have the worst prognosis and are the most challenging to treat. If patients' x-rays show radiolucencies—and especially if diminished radionuclide uptake is also evident by technetium bone scan—there is a risk of delayed union or complete fracture (figure 3).[6] Rettig et al[7] recommend treating these fractures with pulse electromagnetic fields 10 to 12 hours a day for at least 3 to 6 months. They suggest that if the fracture has not healed by that time, a bone graft may be considered.

Based on a study in which five of six athletes went on to develop a complete fracture, Green et al[8] recommend excision and bone graft for anterior tibial stress fractures that do not heal in 4 to 6 months. These six were not initially treated with electromagnetic fields; there is considerable controversy over using pulse electromagnetic fields in these cases. Further investigation is needed to establish the fields' efficacy

Chronic Compartment Syndrome

Chronic compartment syndrome is caused by elevated intracompartmental pressures re-

continued

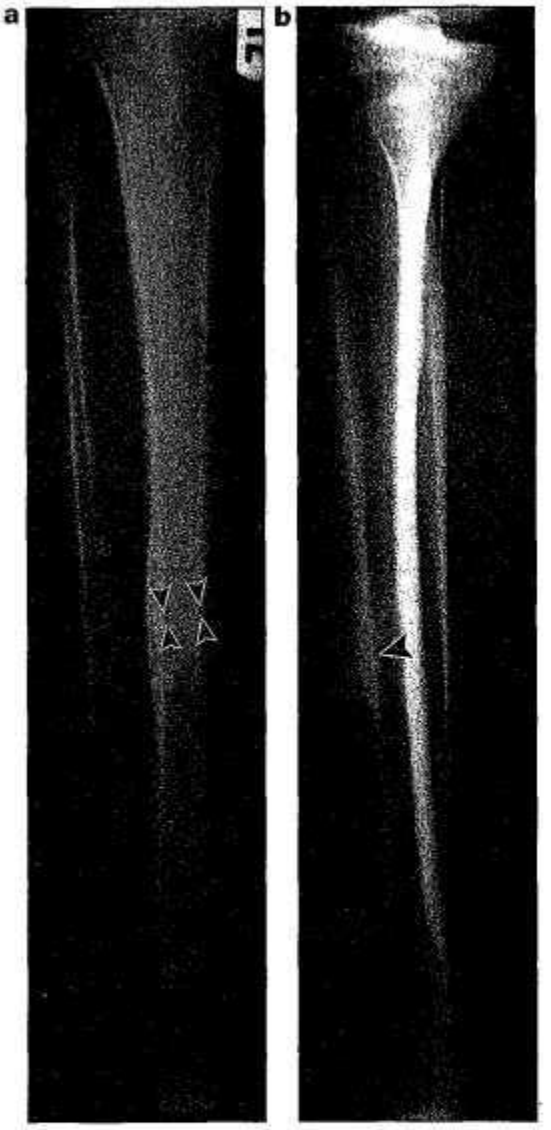


Figure 3. A male collegiate triple jumper's chronic symptomatic stress fracture of the anterior tibia (arrows): anteroposterior (a) and lateral (b) views.

suiting in ischemia of muscles and nerves. Elevated pressure may occur when exercise couples relatively rigid fascial boundaries with the increased muscle bulk caused by muscle contraction, intra- and extracellular fluid accumulation, and hemorrhaging microtears of the muscle. Venous and lymphatic compromise exacerbate the situation.

Although a physician specializing in athletic injuries to the lower extremity may see such patients with some frequency, the prevalence of this condition in the general population is low. In one study,[9] chronic compartment syndrome was diagnosed in 3 patients over the course of a year among a university population of 4,000.

Typically, athletes who have chronic compartment syndrome will have pain when they run a certain distance, speed, or duration. A few minutes to several hours of rest will usually alleviate discomfort. The pain is characterized as aching, cramping, or stabbing. The patient will often report a sensation of fullness in the leg over the involved compartments. Occasionally an athlete will report shooting pains, numbness, tingling, and burning in the leg, ankle, or foot along the nerve that runs through the compartment. Involvement of both legs is common, but simultaneous involvement is less frequent than asynchronous presentation. About 15% to 20% of patients will report a recently diagnosed stress fracture.[9]

Physical examination may be completely normal, but if a patient's leg is stressed before the evaluation, swelling in the affected compartment may be noted. Muscle hernias may be found in 20% to 60% of patients,[10,11] and neurologic abnormalities may occasionally be found. Radiographs are usually normal, but a technetium bone scan may occasionally reveal subtle changes that usually signify coexistent medial tibial stress syndrome or stress fracture.

Compartmental pressure monitoring with a wick catheter before, during, and after exercise will help confirm the diagnosis, though preexercise and postexercise pressures may be sufficient. The diagnosis is supported by compartmental pressures that do not return to baseline after 5 minutes and are greater than 15 mm Hg at rest, greater than 50 to 70 mm Hg while exercising, and greater than 30 mm Hg after exercise.[2]

Treatment begins with rest and reduction of the duration and intensity of activities. Then a gradual increase in exercise in conjunction with ice, nonsteroidal anti-inflammatory drugs (NSAIDs), and orthotic devices may work. Often, however, these measures fail to control the pain, and a fasciotomy of the involved compartment is warranted. (See "Chronic Compartment Syn-

continued

drome: Surgic
1991, page 63,
successful, s
swelling, new
persistent low
of which may

Gastrocnem

A gastrocn
in racket sport
the past, this
tails muscle r
generally acce
taris rupture
two cases in •
rupture has a
cause of its p.
players.

The injury
their knees fle
ion, and then
in jumping.[14]
tracted muscle

Athletes wh
experience su
varying degre
Typically, the
someone kick
aching pain n
severity of the
swelling are m
walk and plant
swelling and o
middle third o
ate strains. Ty
their toes beca
juries, there n
loss of gastrocr

A program
taping and in
ing, will often
stretches folio
added once the

With mode
brace may be
flexion. Some
or brace with t
tar flexed. Once
be removed. A

drome: Surgical Intervention in 12 Cases," April 1991, page 63.) Although surgical release is often successful, some athletes may experience swelling, new muscle herniations, nerve injuries, persistent low-grade pain, and recurrence—any of which may limit a patient's ability to exercise.

Gastrocnemius Rupture

A gastrocnemius rupture or strain may occur in racket sports, basketball, running, or skiing. In the past, this lesion was believed to be a plantaris muscle rupture. However, this diagnosis is generally accepted as obsolete because a plantaris rupture was documented in only one or two cases in the literature.[12,13] Gastrocnemius rupture has also been called "tennis leg" because of its prevalence in middle-aged tennis players,

The injury occurs when athletes crouch with their knees flexed and their ankles in dorsiflexion, and then suddenly extend their knees—as in jumping.[14] This eccentric loading of the contracted muscle causes the rupture.

Athletes who have a gastrocnemius rupture experience sudden, severe calf pain followed by varying degrees of swelling and ecchymosis. Typically, they report that the pain feels like someone kicked them in the leg. Cramping or aching pain may continue depending on the severity of the injury. With mild strains, pain and swelling are minimal, and the patient is able to walk and plantar flex the ankle. A fair amount of swelling and occasionally a palpable gap in the middle third of the muscle accompany moderate strains. Typically, patients cannot stand on their toes because of pain or spasm. In severe injuries, there may be a large, palpable gap and loss of gastrocnemius or soleus function.

A program of rest and icing, with or without taping and in conjunction with passive stretching, will often heal a mild strain. Standing calf stretches followed by strengthening should be added once the pain has diminished.[15]

With moderate strains, a short leg cast or brace may be applied with the ankle in plantar flexion. Some patients may need a long leg cast or brace with the knee flexed and the ankle plantar flexed. Once the pain decreases, the cast may be removed. A heel lift, with or without taping of the leg in equinus, may then be beneficial. When passive stretching is tolerable, the patient may begin calf stretches followed by strengthening exercises.

Photo: ©1992, David Madison

Skaters and other athletes who develop muscle fatigue may need to give relative rest a whirl.

For severe strains, the best course is conservative treatment with or without long leg casting, followed by reasonably aggressive physical therapy. Miller[14] advocates an operative approach with drainage of the hematoma, debridement, and apposition of the muscle. We have undertaken a retrospective review, still unpublished, that indicates that disability following moderate and severe ruptures may last several months and, occasionally, manyyears.

Muscle Overuse

Eccentric contractions during exercise frequently produce delayed-onset muscle pain, fatigue, stiffness, and tenderness. Symptoms may last several days. Treatment includes relative rest, cross-training, and avoidance of hill running. Further work-up and treatment are based

continued

Photo; © 1992, Lew C. Schon

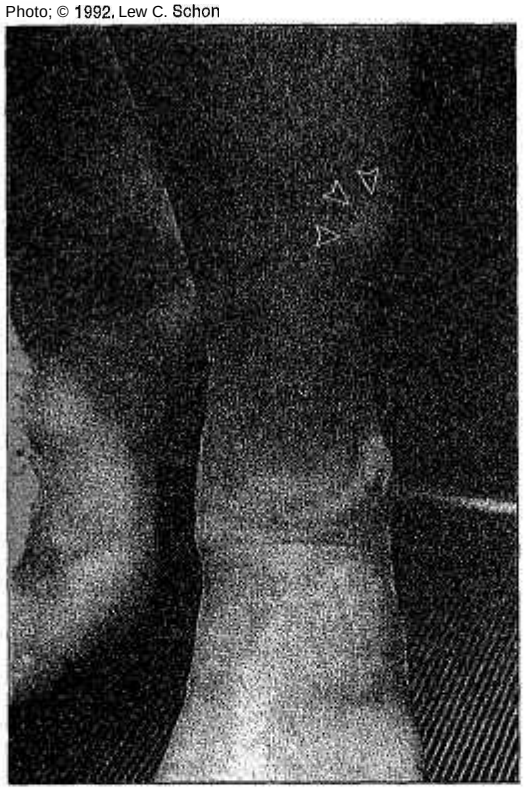


***Figure 4. A 34-year-old** female runner who has superficial peroneal nerve entrapment with fascial defect and muscle herniation (arrows) at the site of nerve penetration in the anterolateral aspect of the lower leg. The bulge is typically located 10 to 12 cm above the end*

on the clinician's suspicion of other processes being at work.[16,17]

External Iliac Arterial Occlusion

Chevalier et al[18] describe an uncommon syndrome of endofibrosis of the external iliac artery in bicycle racers. The athletes complain of a paralyzing pain in tiieir legs occurring with maximal effort. They typically have descending painful muscle contractions in the calf with subsequent diminished sensory and motor responsiveness of the extremity. A few minutes alter the cyclist returns to normal speed, the pain subsides,

Physical examination performed with the patient at rest may yield normal findings. Thigh and calf circumference may be larger in the affected extremity, but when the tiiigh is maximally flexed, a systolic bruit may be auscultated in the femoral triangle. Bicycle stress tests with Doppler ultrasound systolic pressures at the ankle may show decreased pressures when symptoms are present. Angiography may show a stenotic lesion in the external iliac artery.

Treatment depends on the athlete's dedication to cycling, because avoiding the threshold intensity will prevent recurrence. Chevalier et al[18] say competitive cyclists may need endarterectomy and shortening of the artery.

Venous Pathology

Venous insufficiency, thrombosis, or thrombophlebitis may occur as isolated conditions or in association with acute and chronic leg injuries. Risk factors include: (1) severe muscle or bone injury (eg, fracture, complete muscle rupture, contusion), (2) immobilization following a lower extremity injury, (3) prolonged inactivity (perhaps during transportation to athletic events), (4) high altitude, (5) dehydration, (6) alcohol or other drug abuse, (7) estrogen supplementation (eg, birth control pills), and (8) hemoglobinopathies (eg, sickle cell anemia).

Patients who have pain, swelling, and diffuse calf tenderness may have venous pathology and should be evaluated with noninvasive flow tests or venography. In our unpublished retrospective review, two patients with exercise-induced leg pain had severe venous sequelae. Both patients had classic histories of gastrocnemius rupture. One patient developed a superficial femoral vein thrombosis, and the other had a pulmonary embolism.

Treatment of a deep venous thrombosis usually requires anticoagulation with heparin sodium and then warfarin sodium. (See "Effort Thrombosis in a Runner," June 1990, page 76.) Superficial thrombophlebitis in the calf may be heated with elevation, compresses, rest, aspirin, and frequent observation. And compression stockings may improve venous insufficiency.

Nerve Entrapment

A final cause of exercise-induced leg pain may be nerve entrapment. In our experience, entrapment most commonly involves the superficial peroneal nerve (figure 4). Entrapment is consistently located where the nerve penetrates

continued

the fascia and sometimes results from pressure caused by a muscle hernia. Other areas of entrapment include the common peroneal nerve in the region of the fibular neck, the posterior tibial nerve in the popliteal fossa or tarsal tunnel, the saphenous nerve as it pierces Hunter's canal, and the sural nerve in the calf.[19,22]

Patients will often have vague, diffuse pain. Occasionally, they may have neuritis with burning, tingling, shooting, or radiating pain. Directed questioning will help isolate the discomfort, which often lies along the involved nerve. Localized nerve tenderness at the point of compression is characteristic. Percussion or prolonged pressure at this point usually reproduces the pain. It's also important to exclude the possibility of a coexisting chronic compartment syndrome or a more proximal nerve entrapment.

Conservative treatment consists of injections, massage, relative rest, NSAIDs, and shoe modifications. When these measures are unsuccessful, release of the nerve with a localized fasciotomy may be warranted.

Customized Care

When an active person experiences chronic exercise-induced lower leg pain, the differential diagnosis includes chronic compartment syndrome, medial tibial stress syndrome, stress fracture, gastrocnemius rupture, muscle overuse, external iliac arterial occlusion, venous pathology, and nerve entrapment. Fortunately, these injuries are minor if detected and treated properly. To make an accurate diagnosis, physicians need to know the natural history of each condition. They can then customize treatment for each patient's sport and long-term goals. KM

Address correspondence to Lew C. Schon, MD, Union Memorial Hospital, 201 E University Pkwy, Baltimore, MD 21218.

References

1. Michael RH, Holder LE: The soleus syndrome: a cause of medial tibial stress (shin splints). Am J Sports Med 1985;13(2):87-94
2. Viitasalo JT, Kvist M: Some biomechanical aspects of the foot and ankle in athletes with and without shin splints. Am J Sports Med 1983;11(3): 125-130
3. Clement DB, Taunton JE, Smart GW, et al: A survey of overuse running injuries. Phys Sportsmed 1981; 9(5):47-58
4. Detmer DE: Chronic shin splints: classification and management of medial tibial stress syndrome, Sports Med 1986;3(6):436-446
5. Holder LE, Michael RH: The specific scintigraphic pattern of "shin splints in the lower leg": concise communication. J Nucl Med 1984;25(8):865-869
6. Blank S: Transverse tibial stress fractures: a special problem. Am J Sports Med 1987;15(6):597-602
7. Rettig AC, Shelbourne KD, McCarroll JR, et al: The natural history and treatment of delayed union stress fractures of the anterior cortex of the tibia, Am J Sports Med 1988;16(3):250-255
8. Green NE, Rogers RA, Lipscomb AB: Nonunions of stress fractures of the tibia. Am J Sports Med 1985; 13(3).T71-176
9. Detmer DE, Sharpe K, Sufit RL, et al: Chronic compartment syndrome: diagnosis, management and outcomes. Am I Sports Med 1985;13(3):162-170
10. Mubarak SI, Hargens AR: Exertional compartment syndromes, in Mubarak SJ, Hargens AR (eds): Compartment Syndromes and Volkmann's Contracture. Philadelphia, WB Saunders Co, 1981
11. Reneman RS: The anterior and lateral compartmental syndrome of the leg due to intensive use of muscles. Clin Orthop 1975;Nov-Dec(ll3):69-80
12. Mennen U: Rupture of the plantaris: does it exist?, letter. J Bone Joint Surg (Am) 1983;65(7):1030
13. Severance HW Jr, Bassett FH 3rd: Rupture of the plantaris: does it exist? J Bone Joint Surg (Am) 1982; 64(9): 1387-1388
14. Miller WA: Rupture of the musculotendinous juncture of the medial head of the gastrocnemius muscle. Am J Sports Med 1977;5(5):191-193
15. Miller AP: Strains of the posterior calf musculature ("tennis leg"). Am J Sports Med 1979;7(3):172~174
16. Armstrong RB: Mechanisms of exercise-induced delayed onset muscular soreness: a brief review. Med Sci Sports Exerc 1984:16(6):529-538
17. Friden J, Sfakianos PN, Hargens AR, et al: Residual muscular swelling after repetitive eccentric contractions. J Orthop Res 1988;6(4):493-498
18. Chevalier JM, Enon B, Walder J, et al: Endofibrosis of the external iliac artery in bicycle racers: an unrecognized pathologic state. Ann Vasc Surg 1986; 1(3): 297-303
19. Mozes M, Ouaknine G, Nathan H: Saphenous nerve entrapment simulating vascular disorder. Surgery 1975;77(2):299-303
20. Schon LC, Baxter DE: Neuropathies of the foot and ankle in athletes. Clin Sports Med 1990;9(2):489-509
21. Styf J: Entrapment of the superficial peroneal nerve: diagnosis and results of decompression. J Bone Joint Surg (Br) 1989;71(1):131-135
22. Worth RM, Kettelkamp DB, Defalque RJ, et al: Saphenous nerve entrapment: a cause of medial knee pain. Am J Sports Med 1984;12(1):80-81