Medial Tibial Stress Syndrome (Tibial Fasciitis)

A Proposed Pathomechanical Model Involving Fascial Traction

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Although medial tibial stress syndrome is one of the most common lower-extremity overuse injuries, its pathomechanics remain controversial. Two popular theories have been proposed to account for this condition: tibial bending and fascial traction. This article evaluates the role of fascial traction in medial tibial stress pathomechanics. We hypothesized that with contraction of the deep leg flexors tension would be imparted to the tibial fascial attachment at the medial tibial crest. We also speculated that circumferential straps would dampen tension directed to the medial tibial crest. The amount of strain present in the tibial fascia adjacent to its distal medial tibial crest insertion during loading of the leg was investigated as a descriptive laboratory pilot study using three fresh cadaver specimens. Strain in the distal tibial fascia was measured using strain gauges placed in the fascia at its medial tibial crest insertion. As tension on the posterior tibial, flexor digitorum longus, and soleus tendons increased, strain in the tibial fascia increased in a consistent linear manner ($P < .0001$). We conclude that fascial tension may play a role in the pathomechanics of medial tibial stress syndrome. The tenting effect of the posterior tibial, flexor digitorum longus, and soleus tendons caused by muscle contraction exerts a force on the distal tibial fascia that is directed to its tibial crest insertion. Circumferential straps provided no dampening effect on tension directed to the medial tibial crest. (J Am Podiatr Med Assoc 97(1): 31-36, 2007)

The name *shin splints* persists as common terminology used by patients and health-care professionals. Although the term refers to a generalized group of leg pathologies, it continues to be used to describe a group of overuse injuries that excludes stress fracture, compartment syndrome, and fascial herniation. Thus the term *shin splints* has been limited to various overuse injuries affecting soft tissue and bone, including periostitis, fasciitis, muscle or tendon strain, tendinitis, and stress reaction of the tibia.

One specific type of shin splint limited to the medial tibial crest is the most common cause of leg pain encountered in a sports medicine practice. Although many names have been suggested, *medial tibial stress syndrome* has become the most widely accepted term. Kortebein et al[1] provided the most concise definition of this condition: "a specific overuse injury producing pain along the posteromedial aspect of the distal two-thirds of the tibia." Although the definition has been refined, controversy and uncertainty remain as to its pathology and pathomechanics.

Pathologies previously suggested for medial tibial stress syndrome include stress fracture, increased compartment pressures, and myositis or tendinitis, with periostitis, fasciitis, and, recently, stress reaction of the tibia being popular.[1-12] Historically, periostitis due to insertional traction on various muscles and tendons has been the pathology most suspected of causing medial tibial stress syndrome. However, this finding has never been validated histologically. Attachment sites of the posterior tibial,[8, 12] flexor digitorum longus,[7] and soleus muscles and tendons[2, 4, 9] and the crural fascia[1] to the posterior leg have been implicated on the basis of anatomical location. A recent anatomical study[2] concluded that the posterior tibial tendon (the most commonly implicated muscle or tendon in medial tibial stress syndrome) was not likely to be responsible because the location of the posterior tibial tendon attachment to the tibia was re-

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mote from the area of clinical symptoms. Bouché[3] suggests that the pathology of medial tibial stress syndrome represents a type of stress reaction of the tibial fascia at its medial tibial crest insertion and proposes the term *tibial fasciitis*. Numerous authors[6, 13-16] have suggested that medial tibial stress syndrome is part of a continuum of tibial bone stress, with asymptomatic stress reaction being the first stage, followed by symptomatic stress reaction and then stress fracture, with the end stage of this continuum being an overt tibial fracture.

Pathomechanically, there have been two suggested theories to account for medial tibial stress syndrome: soft-tissue traction[1, 2, 4, 7-9, 12] and tibial bending.[9, 13, 17] Soft-tissue traction has long been suggested to account for medial tibial stress syndrome, but other than anatomical studies, no scientific testing has been performed to assess the role of traction in medial tibial stress syndrome. Tibial bending, a theory initially studied by Lanyon et al[17] and popularized by Milgrom et al,[18] is now an accepted pathomechanical model that accounts for common locations of stress fractures of the tibia. Because the distal third of the tibia is a common location for stress fracture and stress reaction owing to its narrow radius, it has been speculated that medial tibial stress syndrome is a type of stress reaction that occurs there as well. The tibial bending model suggests that long-term, repetitive, weightbearing loads that induce tibial bending precipitate a stress injury continuum at the site of maximum bending.[13]

We propose the tibial fascial traction theory, which is explained as follows. Eccentrically contracting superficial and deep flexors of the leg (gastrocnemius, soleus, posterior tibial, flexor digitorum longus, and flexor hallucis longus) during stance counter midtarsal and subtalar pronatory motions of the foot. This counteracting force of the flexors results in a tenting effect that exerts tension on the tibial fascia at its insertion into the medial tibial crest. This repetitive traction force can result in a spectrum of injuries to the tibial fascia and its insertion into the medial tibial crest.

The purpose of this article is to investigate the plausibility of the tibial fascial traction theory in a cadaver model. We hypothesized that with contraction of the soleus, posterior tibial, and flexor digitorum longus tendons there would be a tenting effect exerted on the tibial fascia, with a resultant tensile force at the medial tibial fascial bone interface that could be measured. Furthermore, it was believed that this tensile force could be mitigated by means of a circumferential leg strap strategically placed on the leg. To our knowledge, this is the only study that has ever been performed to specifically evaluate the role of fascial traction as a possible pathomechanical cause of medial tibial stress syndrome.

Materials and Methods

Three fresh-frozen cadaver lower-limb specimens were thawed and disarticulated at the knee. The specimens were all from females aged 79 to 93 years (mean age, 83.7 years) at the time of death. Skin and subcutaneous tissue were removed from around the leg, preserving plantar soft tissues of the foot, deep fascia, and leg compartments 20 cm proximal to the tip of the medial malleolus. Each specimen was mounted on a custom loading frame (designed by BioConcepts Inc, Seattle, Washington, and fabricated by Advanced Biomedical Inc, Oakland, California). A central rod drilled into the intercondylar eminence applied downward load to the tibia by means of a central pneumatic actuator. Each limb was vertically aligned at initiation of the testing sequence.

The posterior tibial and flexor digitorum longus tendons and the soleus aponeurosis were attached by cables (braided Dacron; Western Filament Inc, Grand Junction, Colorado) to three additional pneumatic actuators that pulled upward, simulating muscle pull. Each tendon was attached to its pneumatic actuator by means of a cable tied with a self-tightening knot that doubled on itself to cinch tight and prevent slippage. Relative forces of pull placed on each tendon were determined using the calculation of physiologic cross-sectional area of midstance muscles presented by Brand et al.[19]

Four strain gauges (differential variable reluctance transducers) (MicroStrain Inc, Williston, Vermont) recorded strain in the tibial fascia (Fig. 1). They were positioned along the tibial crest 3, 6, 9, and 12 cm proximal to the tip of the medial malleolus (Fig. 2).

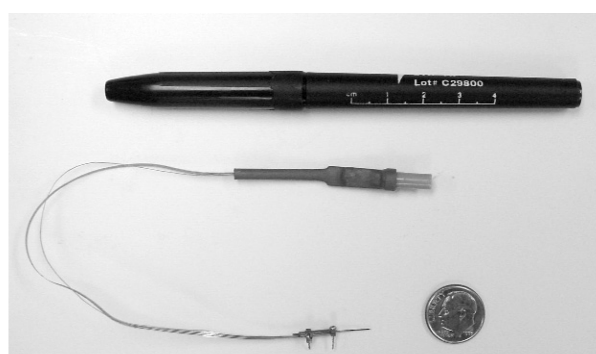

Figure 1. Sample strain gauge (differential variable reluctance transducer).

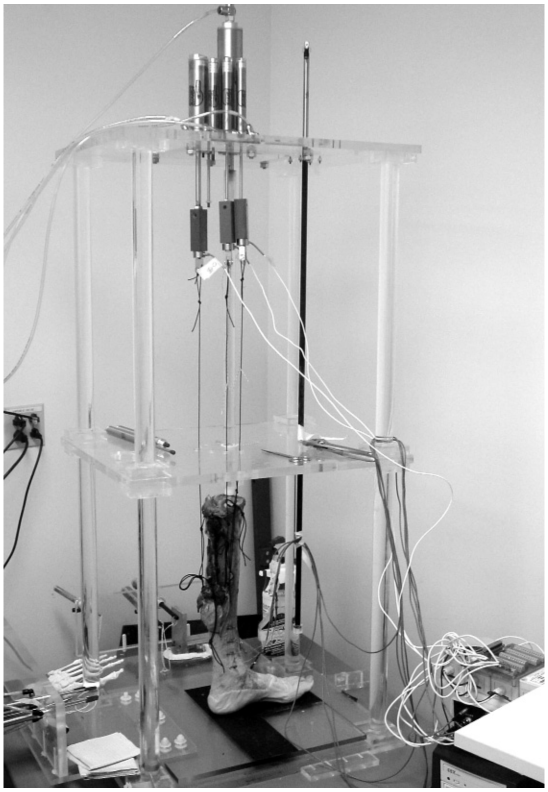

Figure 2. Cadaver specimen mounted on a load frame with a central rod applying downward load to the tibia. Tendons are attached by means of braided cords and are tensioned upward. Strain gauges are placed 3, 6, 9, and 12 cm proximal to the tip of the medial malleolus at the tibial fascial insertion.

Gauges were attached to the tibial fascia at its insertion into the posterior tibial crest, each oriented in a transverse position to monitor tension of the tibial fascia from medial to lateral. Fascial strain and tendon tension data were simultaneously collected and digitally recorded using a data-acquisition system and software (LabVIEW version 5.0; National Instruments, Austin, Texas). The stress/strain data collected were plotted on graphs of tendon tension *versus* strain measured at each strain gauge.

An increasing downward load of 0 to 600 N was applied to each specimen while data were collected from each strain gauge. Once at 600 N, the downward load was held constant. Meanwhile, the posterior tibial, flexor digitorum longus, and soleus tendons were tensioned individually at increasing loads to 150% of the predicted maximum load. In addition, on two specimens, all three tendons were tensioned simultaneously, simulating an eccentric contraction. Six trials were performed to ensure data reproducibility.

One specimen was retested with placement of three types of circumferential straps made of self-adhering compression wrap (Coban; 3M, St Paul, Minnesota): 1) a low strap, 2) a high strap, and 3) a low strap with a roll. The low strap was 2.5 cm wide and was placed between the 3- and 6-cm strain gauge locations. The high strap was approximately 6 cm wide and was placed above the proximal strain gauge, 13 cm proximal to the medial malleolar tip. The roll (1-cm-diameter cylindrical dental felt material) was oriented vertically just posterior to the medial tibial crest positioned under the low strap. The setup was observed throughout testing to ensure that the straps did not come in contact with the strain gauges. Strain gauge data were recorded during tensioning of the three tendons and downward load on the specimen as described previously here. Six trials were conducted to ensure data reproducibility.

Results

A consistent linear relationship was observed between tension on the tendon and strain measured at the tibial fascia within and across individual specimens. With increased pull on the individual posterior tibial, flexor digitorum longus, and soleus tendons, there was increased strain recorded at the strain gauges. The slopes of the strain curves were consistent at 0.0002 mm/N measured at the 3- and 6-cm locations with tension on the posterior tibial, flexor digitorum longus, and soleus tendons ($P < .0001$). A representative sample of data in graph form is shown in Figure 3. Data collected at the 9- and 12-cm locations were more variable and inconsistent with respect to tendon pull and strain measured within and

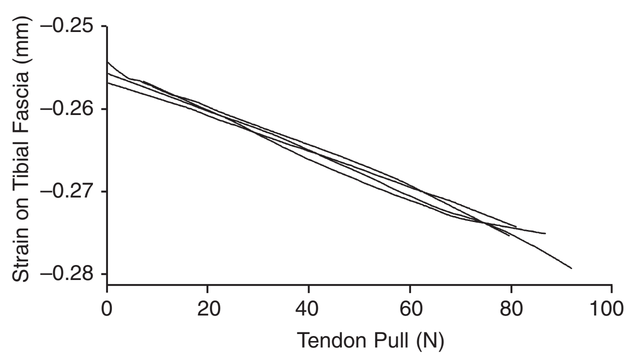

Figure 3. A representative sample of data illustrating the linear relationship of increased strain on the tibial fascia with increased tension on the posterior tibial, flexor digitorum longus, and soleus tendons and on all three combined.

across individual specimens. Visual inspection of the specimens during simulated muscle contraction revealed marked fascial tenting. These visual findings were consistent with strain measured in the tibial fascial tissues. The linear slope of the data did not change with the placement of three types of circumferential straps. There was no difference in strain measured in the tibial fascia whether there was no strap, a low strap, a low strap with a roll, or a high strap. When an increasing downward load of 0 to 600 N was applied to the tibia with no tension on the tendons, there was no change in strain in the tibial fascia at all strain gauge locations.

Discussion

When the tibia was loaded with the contraction of select leg flexors (soleus, posterior tibial, and flexor digitorum longus), a linear tension force was generated as measured by the strain gauges and visible tenting of the tibial fascia. The distal strain gauges on the leg provided the most consistent results. These findings are consistent with the hypothesis. Proximally on the leg the results were less consistent, probably because of the inability to maintain adequate anatomical integrity of the more proximal tibial fascial structure, although this is not certain.

Because there was no change in strain on the tibial fascia with an increasing downward weightbearing load (with tendons maintained at no tension), this study suggests that the force of weightbearing alone does not generate a primary traction force on the tibial fascia. However, we speculate that with increased dynamic loading *in vivo*, the superficial and deep flexor muscles and tendons would eccentrically contract as a protective mechanism, thus exerting tension on the tibial fascia.

We believe that the tibial fascial traction theory is consistent with and supported by anatomical, pathomechanical, pathologic, diagnostic, and clinical findings. Anatomically, the tibial fascia is the only structure to insert into the medial tibial crest from the ankle to the knee and is the containment structure of the leg as part of the bone-fascial system of the lower extremity.[20] As such, clinical symptoms of medial tibial stress syndrome are well localized to the medial tibial crest, more commonly affecting the distal *versus* proximal aspect of the tibial crest, as elucidated by history and physical examination, ie, careful palpation. We have encountered many cases in which symptoms are proximal and not just distal on the tibial crest. This could be accounted for by the fact that the tibial fascia attaches along the entire medial crest to knee level. No other anatomical structures insert into this specific location. Although the soleus muscle has been implicated, clinical symptoms often are proximal to the soleal line, which represents the proximal attachment point of the soleus muscle on the leg.[9] Posterior tibial and flexor digitorum longus muscle origins also are remote from the proximal medial tibial crest.

The results of this study suggest that eccentric contraction of the superficial and deep flexor tendons of the leg is the key pathomechanical factor resulting in increased tibial fascia tension directed to the medial tibial crest where the tibial fascia inserts. Patients who exercise on hard surfaces (regardless of foot type) have increased eccentric contractions of the deep flexors for shock dissipation, and patients who pronate excessively also have increased eccentric leg flexor contractions to counter pronatory moments of the midtarsal and subtalar joints.[21] Coincidentally, exercising on hard surfaces and excessive foot pronation are two suggested etiologic factors commonly associated with medial tibial stress syndrome.[22-24] We speculate that excessive tension on the tibial fascia insertion due to these eccentric contractions results in pathology.

Pathologically, the study by Johnell et al[25] demonstrated soft-tissue inflammation in the form of deep crural fasciitis with stress reaction of the bone at the medial tibial crest in a series of patients with shin splints. Our personal experience with pathology specimens obtained from the medial tibial crest at the time of fascial releases of the leg for chronic medial tibial stress syndrome are also consistent with fasciitis. Other authors[11, 26] have reported similar findings.

Diagnostic imaging studies generally correlate with these pathologic findings as well. Although radiographic findings are typically normal, bone scanning and magnetic resonance imaging (MRI) can help elucidate the area of involvement. Bone scanning, although not specific, is sensitive for bone activity and relative blood flow. In cases of isolated "fasciitis," the third-phase bone scan results may be negative, although the first phase (perfusion) and the second phase (blood pool) may reveal abnormal uptake (only in the acute phase) or be normal as well. Therefore, negative bone scan findings do not necessarily mean that the patient does not have medial tibial stress syndrome. Magnetic resonance imaging provides the most anatomical detail of the leg, and in the acute and subacute stages, soft-tissue inflammation and stress reaction of the bone can be demonstrated.[27] In the chronic stage, MRI findings in the leg will probably be normal.[28] When findings from radiographs, bone scans, and MRI are normal, diagnostic injections (with local anesthetic) to the involved medial tibial crest can confirm tibial fascia involvement and its specific location.

Proponents of the tibial bending theory believe that the end stage of persistent tibial bending is a stress or overt fracture of the tibia. In contrast, the natural history of fascial traction on the tibia is not, in our experience, a tibial stress or overt fracture. We speculate that tibial stress reaction due to fascial traction is different from tibial stress reaction due to tibial bending. An important pathologic and pathomechanical analogy of medial tibial stress syndrome would be plantar fasciitis, a stress reaction of the fascial insertion into the calcaneus, caused by fascial traction. As with tibial fasciitis, the natural history of plantar fasciitis does not result in a stress fracture (of the calcaneus) at the fascial insertion. However, stress fractures of the calcaneus typically occur at sites other than the fascial insertion owing to overuse or overload.

This study has various treatment implications. When pathology is localized to the medial tibial crest, two therapies have proved beneficial in our experience: therapeutic injections using local anesthetic and acetate steroid (providing an anti-inflammatory effect) and surgical release of the tibial fascia. Our experience with therapeutic injections has been excellent. Our experience with fascial release of the leg has been good, with the results being consistent with those reported in the literature.[29-34] Although shin splint straps are commonly used in clinical practice, the results of this study suggest that they may be ineffective. Various circumferential strap designs and locations were used, and no strap, regardless of location, provided any appreciable dampening effect on the tibial fascia.

This study and its experimental design have several limitations. This was a pilot study, and we used a small number of cadaver specimens only from female patients of advanced age. The relative tendon loads applied to each specimen were based on available physiologic muscle data.[19] Load values were estimated on the basis of a mathematical model involving cross-sectional surface area of muscle. Because of anatomical limitations, we could leave intact only the middle to distal leg fascia and were, therefore, unable to test traction in the proximal leg fascia. Furthermore, straps applied to the specimens were placed after skin and subcutaneous tissues were removed, unlike clinical placement of shin splint devices external to the skin. However, considering this, the straps still did not have an effect. In addition, only midstance function was evaluated in this model. Because of inconsistent data at the 9- and 12-cm locations, further study is needed to discern whether this is due to a study design flaw. If these data are accurate, this inconsistency would detract from the tibial fascial traction theory. Future research should also include an investigation correlating findings from diagnostic imaging studies with biopsy specimens. This would confirm the pathology of medial tibial stress syndrome and identify the best diagnostic study to validate the pathology.

Conclusion

The results of this pilot study suggest that tibial fascial traction occurs at the distal tibial fascial insertion into the medial tibial crest. These findings support the tibial fascial traction theory, which is proposed to explain the pathomechanics of medial tibial stress syndrome. As a result of this localized tension force, we speculate that the pathology of medial tibial stress syndrome is a stress reaction resulting in a tibial fasciitis, specifically, a medial tibial fasciitis. This terminology implicates the anatomical structure that is likely to be responsible for this condition, its location, and the pathology involved. Further research is needed to validate this pathomechanical model and to elucidate the role of shin straps (if any) as a treatment option for medial tibial stress syndrome.

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Conflict of Interest: None reported.

References

1. Kortebein PM, Kaufman KR, Basford JR, et al: Medial tibial stress syndrome. Med Sci Sports Exerc **32:** S27, 2000.
2. Beck BR, Osternig LR: Medial tibial stress syndrome: the location of muscles in the leg in relation to symptoms. J Bone Joint Surg Am **76:** 1057, 1994.
3. Bouché RT: "Exercise-Induced Leg Pain," in *Fundamentals of Lower Extremity Sports Medicine*, 2nd Ed, ed by S Subotnick, p 277, Churchill-Livingstone, San Francisco, 1999.
4. Detmer DE: Chronic shin splints: classification and management of medial tibial stress syndrome. Sports Med **3:** 436, 1986.
5. Devas MB: Stress fractures of the tibia in athletes or "shin soreness." J Bone Joint Surg Br **40:** 227, 1958.
6. Fredericson M, Bergman AG, Hoffman KL, et al: Tibial stress reaction in runners: correlation of clinical symptoms and scintigraphy with a new magnetic resonance

imaging grading system. Am J Sports Med **23:** 472, 1995.
7. Garth WP, Miller ST: Evaluation of claw toe deformity, weakness of the foot intrinsics, and posteromedial shin pain. Am J Sports Med **17:** 821, 1989.
8. James SL, Bates BT, Osternig LR: Injuries to runners. Am J Sports Med **6:** 40, 1978.
9. Michael RH, Holder LE: The soleus syndrome: a cause of medial tibial stress (shin splints). Am J Sports Med **13:** 87, 1985.
10. Puranen J, Alavaikko A: Intracompartmental pressure increase on exertion in patients with chronic compartment syndrome in the leg. J Bone Joint Surg Am **63:** 1304, 1981.
11. Puranen J: The medial tibial syndrome: exercise ischemia in the medial fascial compartment of the leg. J Bone Joint Surg Br **56:** 712, 1974.
12. Saxena A, O'Brien T, Bunce D: Anatomic dissection of the tibialis posterior muscle and its correlation to medial tibial stress syndrome. J Foot Surg **29:** 105, 1990.
13. Beck BR: Tibial stress injuries: an aetiological review for the purposes of guiding management. Sports Med **26:** 265, 1998.
14. Clement DB: Tibial stress syndrome in athletes. J Sports Med **2:** 81, 1974.
15. Jones BH, Harris JM, Vihn TN, et al: Exercise-induced stress fractures and stress reactions of bone: epidemiology, etiology, and classification. Exerc Sports Sci **17:** 379, 1989.
16. Roub LW, Gumerman LW, Hanley EN, et al: Bone stress: a radionuclide imaging perspective. Radiology **132:** 431, 1979.
17. Lanyon LE, Hampson WGJ, Goodship AE, et al: Bone deformation recorded in vivo from strain gauges attached to the human tibial shaft. Acta Orthop Scand **46:** 256, 1975.
18. Milgrom C, Giladi M, Simkin A, et al: The area moment of inertia of the tibia: a risk factor for stress fractures. J Biomech **22:** 1243, 1989.
19. Brand RA, Pedersen DR, Friederich JA: The sensitivity of muscle force predictions to changes in physiologic cross-sectional area. J Biomech **19:** 589, 1986.
20. Gerlach UJ, Lierse W: Functional construction of the superficial and deep fascia system of the lower limb in man. Acta Anat **139:** 11, 1990.
21. Richie DH, DeVries HA, Endo CK: Shin muscle activity and sports surfaces: an electromyographic study. JAPMA **83:** 181, 1993.
22. O'Donoghue DH: "Injuries of the Leg," in *Treatment of Injuries to Athletes*, p 686, WB Saunders, Philadelphia, 1976.
23. Sommer HM, Vallentyne SW: Effect of foot posture on the incidence of medial tibial stress syndrome. Med Sci Sports Exerc **27:** 800, 1995.
24. Yates B, White S: The incidence and risk factors in the development of medial tibial stress syndrome among naval recruits. Am J Sports Med **32:** 772, 2004.
25. Johnell O, Rausing A, Wendeberg B, et al: Morphological bone changes in shin splints. Clin Orthop Relat Res **167:** 180, 1981.
26. Mubarak ST, Gould RN, Lee YF, et al: The medial tibial stress syndrome (a cause of shin splints). Am J Sports Med **10:** 201, 1982.
27. Batt ME, Ugalde V, Anderson MW, et al: A prospective controlled study of diagnostic imaging for acute shin splints. Med Sci Sports Exerc **30:** 1564, 1998.
28. Anderson MW, Ugalde V, Batt ME, et al: MRI of shin splints: preliminary findings. Radiology **204:** 177, 1997.
29. Wallensten R: Results of fasciotomy in patients with medial tibial syndrome or chronic anterior-compartment syndrome. J Bone Joint Surg Am **65:** 1252, 1983.
30. Akermark C, Ljungdahl M, Johansson C: Long-term result of fasciotomy caused by medial tibial syndrome in athletes. Scand J Med Sci Sports **1:** 59, 1991.
31. Allen MJ, Barnes MR: Exercise pain in the lower leg. J Bone Joint Surg Br **68:** 818, 1986.
32. Holen KJ, Engebretsen L, Grontvedt T, et al: Surgical treatment of medial tibial stress syndrome (shin splint) by fasciotomy of the superficial posterior compartment of the leg. Scand J Med Sci Sports **5:** 40, 1995.
33. Jarvinnen M, Aho H, Niittymaki S: Results of the surgical treatment of the medial tibial syndrome in athletes. Int J Sports Med **10:** 55, 1989.
34. Yates B, Allen MJ, Barnes MR: Outcome of surgical treatment of medial tibial stress syndrome. J Bone Joint Surg Am **85:** 1974, 2003.