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SPORTS THERAPY



Getting a Leg Up on Shin Pain

Review of Literature

Abstract:

Shin splints is an injury of the lower leg that often afflicts athletes. Generally, symptoms include pain on the anterolateral or posteromedial surfaces of the shin. There is a great deal of research on the treatment and prevention of shin splints, and based on extensive research, several hypotheses have been proposed for its pathophysiology; however, the exact cause of shin splints remains unknown. This paper explains the anatomy of the lower leg and biomechanics of gait. It presents possible etiologies and risk factors for shin splints, and it reviews options for treatment and prevention. Its conclusion, based on extensive literature review, reveals that most cases of shin splints respond favorably to conservative care. Such care generally includes home exercise, training and equipment modifications, and treatment by a skilled practitioner. However, in more advanced cases surgery may be necessary.

Introduction and History:

Shin splints is one of the most common exercise-induced lower extremity injuries in athletes participating in activities that require prolonged or quick bursts of running and/or jumping.¹ Such activities may include track and field, soccer, basketball, volleyball, basic military training, and long-distance running.⁷⁻¹¹

During running each foot strikes the ground approximately 50-70 times per minute, or a total of 800 times per mile, with a force 2-4 times body weight.^{1,12} The muscles, tendons, and bones that support the lower extremity can usually adapt to such an increased work load if training progresses appropriately. However, the following risk factors may predispose to the development of shin splints by leading to increased ground reactive-forces acting on the lower extremity and/or a compromised musculoskeletal support structure:

training errors, foot shape/biomechanics, poor conditioning, high body mass index, poor nutrition, and degenerative changes.^{1,13-16}

Symptoms associated with shin splints may include pain over the anterolateral, or the distal two thirds of the posteromedial aspect of the shin.²⁻⁶ Usually, these symptoms are present with activity and alleviated with rest.⁷ However, if the athlete trains throughout pain and a proper treatment program is not initiated, the symptoms and severity of shin splints may progress.

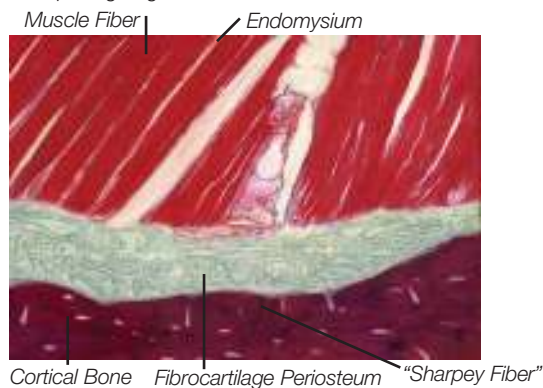
In the 1900s shin splints was initially defined as any type of pain from the hip to the ankle.¹⁴ This broad definition seemed appropriate because treatments for lower extremity injuries were rudimentary and relatively ineffective. Throughout the last four decades medical research has led to advancements in understanding the causes, treatment, and prevention of sports injuries. The exact pathophysiology of shin splints remains unknown; however several possible etiologies have been studied.^{1,17,18}

An understanding of the kinesiology, physiology, and anatomy of the lower leg, risk factors associated with shin splints, and a detailed history and physical examination may aid the practitioner in developing a proper treatment and prevention protocol.^{3,19,20} Most cases can be effectively treated with conservative care.^{1,21}

Anatomy of the Lower Leg

Long bones, such as the tibia, consist of an outer rigid layer. This external surface consists of several concentric rings of cortical bone that are surrounded by periosteum, a thin layer of dense fibrous connective tissue that is richly supplied by nerves and blood. The periosteum is anchored to the cortical bone by bundles of connective tissue "Sharpey fibers"

Fig. 1 Slide depicting origin of muscle onto bone



(figure 1). The periosteum has several functions: it protects the underlying bone, participates in the repair and remodeling process of bone, and is the site of attachment for adjacent muscles. Muscles consist of bundles of individual fibers (fasciculi) that are surrounded by endomysium, a thin delicate layer of connective tissue. The fibrocartilage strands of the periosteum intertwine with the endomysium of abutting musculature, creating an extensive origin of attachment. At these origins of attachment the underlying cortical bone is rough or thickened due to bone remodeling in response to traction exerted by muscles.^{22,23}

The tibia has three potential sites for attachments of muscles: a posterior surface, and anterolateral and medial surfaces that are separated by the anterior tibial crest. The soleus, flexor digitorum longus, and tibialis posterior musculature attach to the posterior surface of the tibia (figures 2A). The tibialis anterior musculature attaches to the anterolateral surface of the tibia (figure 2B).

Fig. 2A Posterior Leg Muscles

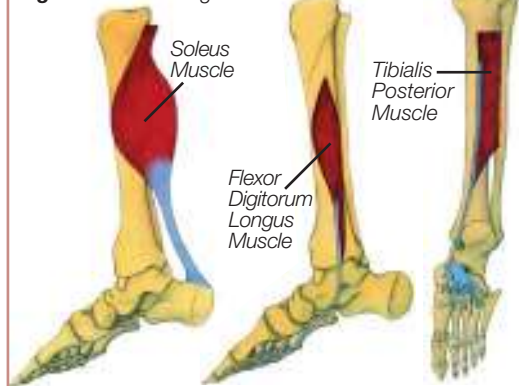
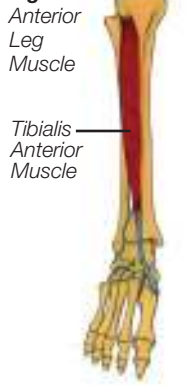
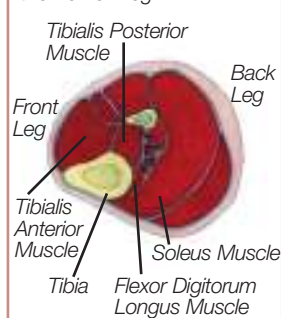


Fig. 2B Anterior Leg Muscle



These muscles are contained within the superficial posterior, the deep posterior, or the anterior compartment of the leg (figure 3). The superficial posterior compartment houses the soleus musculature. It is bounded posteriorly by the crural fascia and anteriorly by the transverse fascia. The deep posterior compartment contains the flexor digitorum longus and the tibialis posterior musculature. It is bordered posteriorly by the transverse fascia and anteriorly by the fibula, the interosseous membrane, and the tibia. The tibialis anterior muscle is contained within the anterior compartment of the leg.^{3,6,24,25} These muscles surrounding the shin aid in

Fig. 3 Cross Section of the Lower Leg



coordinating movements of the foot and ankle and in reducing ground reactive forces.

Biomechanics of the Lower Leg during Ambulation

There are two main phases to a running gait, a stance phase and a swing phase. During the stance phase the foot contacts and adapts to the ground. The swing phase begins when the stance leg lifts off the ground. Most sport-related injuries can be attributed to repetitive ground reactive forces during the stance phase of gait.²⁶ The stance phase of gait consists of the following sub-phases:

- initial contact, when the foot of the swing leg initially contacts the ground
- loading response, beginning shortly after initial contact as the foot begins to adapt to the ground.
- early midstance, when the contralateral swing leg is midline with the body and distributes the body weight over the stance leg.
- late midstance, starting when the foot of the stance leg changes from a mobile adaptor that absorbs ground reactive forces to a more rigid lever that prepares the foot for toe-off.
- terminal stance, beginning shortly after heel-lift and ending with toe-off (figure 4).^{27,28,29}

Fig. 4 The Stance Phases of Gait



Sequenced articulations of the rearfoot during the stance phases of gait can determine the function of the foot and ankle. The rearfoot is comprised of two joints, the talocrural joint and the subtalar joint. The talocrural joint (ankle mortise) consists of the articulation of the distal aspect of the tibia and the fibula with the trochlea of the talus (figure 5). The subtalar joint (STJ) consists of the articulation of the undersurface of the talus with the calcaneus (heel bone) (figures 6A, 6B).^{27,30,31}

Throughout loading response and into early midstance, the foot goes through a series of transformations coined "pronation," allowing the lower extremity to be more efficient

Fig. 5 Talocrural Joint (indicated by red demarcation)

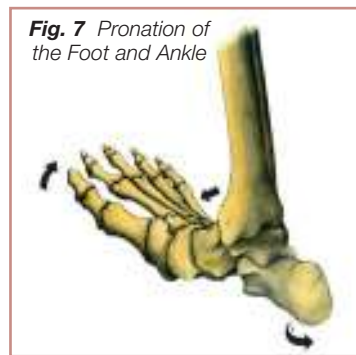


Fig. 6A & 6B Subtalar Joint Articulations (indicated by red demarcation)



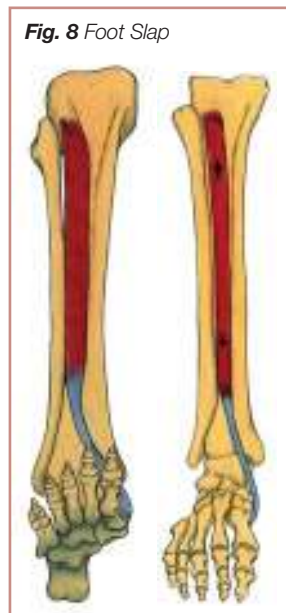
in absorbing ground-reactive forces. Pronation of the foot and ankle consists of the following movements:

1. The STJ pronates, the heel bone turns outward (everts), and the talus drops downward distally and adducts towards the midline. Simultaneously, the medial longitudinal arch (instep arch) lowers towards the ground.
2. Ground reactive dorsiflexion occurs at the talocrural joint (the tibia approximates to the toes).
3. The forefoot turns outward (abducts) (figure 7).



Normal range of pronation is four degrees of heel eversion and twenty degrees of ground reactive dorsiflexion. Pronation of the foot and ankle should end before late midstance as the foot prepares for toe-off.^{27,28,30}

Shortly after the foot strikes the ground the muscles attaching to the anterior and posterior aspect of the tibia contract eccentrically; the muscle fibers elongate as tension is produced in an attempt to decelerate particular motion. The soleus, tibialis posterior, and the flexor digitorum musculature have points of origin on the posterior aspect of the tibia. They contract eccentrically during loading response and into midstance to decelerate pronation. Shortly after initial ground contact, the tibialis anterior muscle contracts eccentrically to decelerate foot slap as the forefoot descends towards the ground (figure 8).^{24,32}



Under normal circumstances, the musculoskeletal support structures of the lower extremity can adapt to these repetitive eccentric loads. However, training errors, structural abnormalities, and other factors may predispose to excessive eccentric loads placed on the lower leg musculature, resulting in anterolateral or posteromedial shin splints. The exact pathophysiology of shin splints is unknown; however, research has come up with several hypothetical causes.

Possible Etiologies of Shin Splints

Traction Periostitis/Periostalgia

In 1964 the American Medical Association (AMA) defined shin splints as "pain and discomfort in the leg from repetitive activity on hard surfaces, or due to forcible excessive use of the foot flexors."¹¹ Current research supports the hypothesis that posteromedial shin pain in runners may be caused by excessive eccentric contractions of the superficial and deep posterior compartment muscles that originate on the tibia. Training that does not allow for proper adaptation of the musculoskeletal support structures of the lower leg, due to increased workloads, may lead to inflammation or degenerative changes to the surrounding fascia (fasciitis) or to the periosteum (periostitis/periostalgia).^{2,6,26}

Posteromedial shin pain due to traction periostitis has been given several names. In 1978 James recommended the term posterior tibial syndrome (PTS) to describe posteromedial shin pain due to a traction periostitis involving the origin of the tibialis posterior musculature. Mubarak in 1982 introduced Drez's term, medial tibial stress syndrome (MTSS). MTSS broadly includes any of the origins of the deep flexor musculature of the leg that attaches to the tibia as possible causes of traction periostitis. Later in 1985 Michael and Holder coined the term soleus syndrome. Their research utilized bone scintigraphy and biopsies on patients complaining of posteromedial leg pain. Results revealed increased bone remodeling and periostitis at the origin of the soleus muscle on the medial aspect of the tibia. However, other studies involving biopsies of patients diagnosed with posteromedial shin splints did not confirm inflammation of the periosteum.¹⁷ They led to an alternative hypothesis that shin pain may be caused by degenerative periostalgia. Currently, MTSS is the most common term to describe posteromedial shin pain thought to be caused by periostitis or periostalgia.^{1,19}

Anterolateral shin pain has been linked to repetitive eccentric contractions of the tibialis anterior musculature. Downhill running accentuates a more pronounced foot slap, predisposing to fatigue of the tibialis anterior musculature, which may result in anterolateral shin pain due to a similar mechanism of injury as MTSS.¹⁴ This paper will define this condition as "anterior tibial stress syndrome" (ATSS).

Bone Stress Reaction

Bone remodeling of the tibia in response to increased work loads commences approximately five days after stimulation.² Two types of cells are active in bone remodeling: osteoblast cells and osteoclast cells. Osteoblast cells produce new bone, while osteoclast cells react to increased stress by reabsorbing bone. Following bone reabsorption, osteoclast cells secrete osteoid, which is uncalcified bone. Osteoid calcifies into new bone after interacting with calcium and phosphate ions. These adaptations should result in a stronger more rigid skeletal support structure. However, it may take ninety days for the reabsorbed bone to be replaced by mature strong bone.¹⁴ Overtraining may lead to a higher ratio of bone reabsorption by osteoclast cells as compared to the production of new bone by osteoblast cells. This may result in a compromised porous skeletal support structure, which is susceptible to a bone stress reaction.^{1,10,11} Bone stress reactions may include microfractures, or in the more advanced stages a stress fracture. The tibia is the most common location of stress fractures in athletes,^{19,33,34} and most stress fractures occur four to five weeks after the onset of new exercise.¹²

Exercise Induced Compartment Syndrome (ECS)

Increased intracompartmental pressures due to exercise are most commonly found in the anterior and deep posterior compartments of the leg (wilder clinic sports medicine). Repetitive eccentric contractions of the muscles of the shin may cause microtrauma, leading to a release of protein ions. Results may include increased intra-compartmental swelling, decreased blood flow, ischemia, and eventually muscle dysfunction and shin pain.^{4,35}

The treatment of posteromedial or anterolateral shin splints in minor injuries is similar whether the etiology

is traction periostitis/periostalgia, bone stress reaction, or ECS.^{1,19} The initial history and physical examination should focus on discovering possible risk factors. Treatment and prevention of shin splints should include steps to eliminate or minimize these risk factors.

Risk Factors Associated with Shin Splints

Risk factors for shin splints are intrinsic (within self) and extrinsic (external to self).^{6, 36}

Intrinsic risk factors may include the following foot structure abnormalities: pes planus (a flat foot), rearfoot varus (an inverted position of the back of the heel),

forefoot varus (the forefoot is inverted in relation to the rearfoot), ankle equinus (limited dorsiflexion of the ankle most likely caused by diminished flexibility of the posterior calf musculature), a pes cavus foot structure (a high arch that is usually restricted in pronation). With the exception of the pes cavus, these foot structures may lead to prolonged or excessive pronation of the STJ, predisposing to shin splints. In the case of the pes cavus, the foot is limited in pronation and its ability to absorb ground reactive forces, leading to an increased work load on the tibia and predisposing to bone stress.^{1,2,8,15,24,37}

Table 1 lists other risk factors associated with shin splints

Table 1

Intrinsic Risk Factors	Extrinsic Risk Factors
Lower limb structural abnormalities <ul style="list-style-type: none"> • pes planus (flat foot, which may lead to excessive pronation) • pes cavus (high arch, which may limit pronation and ability to absorb shock) 	Training progression <ul style="list-style-type: none"> • inappropriate intensity, frequency, or duration of training • hill training that does not progress gradually
Poor Conditioning <ul style="list-style-type: none"> • overweight (body mass index > 30 kg/m²) • insufficient muscle endurance • insufficient muscle strength • limited flexibility 	Gear <ul style="list-style-type: none"> • unsuitable footwear
Female Triad <ul style="list-style-type: none"> • osteoporosis • amenorrhea • eating disorder 	Training surface <ul style="list-style-type: none"> • hard uneven ground
Age related changes <ul style="list-style-type: none"> • especially noticeable after 4th decade of life 	Type of sport <ul style="list-style-type: none"> • activities that involve repetitive running and/or jumping
History <ul style="list-style-type: none"> • previous stress fractures 	

1,2,4,10,11,14-16,19,22,33-36,38

A detailed history and physical examination can aid in discovering risk factors that may have contributed to the development of shin splints. Most risk factors are correctable or can be minimized, aiding in the treatment and prevention of future episodes.

Training Guidelines

Recommendations for frequency, duration and intensity of training runs:^{1,29,39,40}

Aerobic Conditioning, 70-80% maximum heart rate (Max HR=220-age):

This level of training is recommended for gradual adaptation to work loads with both cardiovascular conditioning and connective tissue adaptation. Initially, long-distance runners should establish a training base of 4 miles per run at this low intensity level. A progressive training schedule may include a frequency of 4 runs in a 7-day period, generally consisting of 3 shorter runs (each approximately 4-6 miles) during the week, and 1 longer run during the weekend.⁴¹ It is recommended that the weekly training mileage should not be increased by more than 10% per week. Based on marathon training logs and personal experience, the duration of the long

run is usually increased by an increment of 2 miles per week or every other week, and the intensity should continue at the aerobic conditioning pace. After a proper training base is established, the short duration training runs can be conducted at an increased intensity level.

Anaerobic Conditioning, 80-90% maximum heart rate:

This training is below lactate threshold, the point after which blood lactate rises rapidly, leading to increased ventilation (speaking to a training partner becomes difficult) and eventual muscle fatigue. Anaerobic conditioning training should include a 5 minute warm up, followed by a 15-20 minute duration at the anaerobic conditioning level, then a 1 mile recovery run at the aerobic conditioning pace, followed by another 15-20 minute run at the 80-90% level. Anaerobic training makes running at a submaximal lactate pace easier over a prolonged duration. Ideally,

the marathon pace should be conducted slightly below the lactate threshold. Towards the end of the race, "the last kick," the pace can be increased.

Aerobic Capacity Training (intervals), 90-95% maximum heart rate:

This training is a vigorous challenge to the athlete's aerobic and anaerobic capabilities, and stimulates slow twitch and fast twitch muscle fibers. This pace should only be maintained for 6-9 minutes, followed by a 4-5 minute recovery run at a slow pace. Aerobic capacity training runs elevate the lactate threshold and condition the body to deal more efficiently with oxygen debt and muscle fatigue.

Other Training Tips:

Add uphill training gradually. Running uphill should be conducted at a slower pace because of increased energy expenditure due to increased arm and shoulder action, and hipflexor and knee lift. Uphill running also predisposes to an increased eccentric strain on the posterior calf musculature. Downhill running should be limited because of increased risk and limited benefits.^{40,42}

Change running shoes every 300-500 miles (approximately every 3-4 months). A sneaker loses approximately 50% of its ability to absorb ground reactive forces after 300-500 miles.^{1,19,37,43,44}

Modify training. Add exercises such as swimming, bicycling, and using an elliptical machine on non-running days, or while recovering from an injury. For prevention of injury, the older athlete (after 4th decade of life) may limit running on pavement.^{1,4,11,30,45}

Implement an eccentric strength training program for the lower leg musculature. This program can aid in the treatment and prevention of injury.⁷⁶ Standing and seated calf raises strengthen the gastrocnemius, soleus, and the intrinsic musculature of the foot. A dorsiflexion-assisted resistive device, resistive tubing, or a cable machine strengthens the tibialis anterior and extensor musculature of the leg.^{1,4,30,35,43,46,47} The strength training program should address the whole lower kinetic chain.⁴ Squats eccentrically strengthen the quadriceps, hamstrings, and gluteal musculature; romanian deadlifts eccentrically strengthen the latter two muscles. Abduction, adduction, and hip extension exercises can be conducted with a cable machine or tubing.⁴⁸ Based on my training experience, this lower extremity routine should be conducted at a frequency of one time per week, usually on a Tuesday, Wednesday, or Thursday, if the long run is conducted on a Saturday or Sunday.

Stretch. Static stretching and ballistic stretching have both been shown to increase flexibility of the lower extremity.^{49,50} I have found post-workout stretching to be helpful in decreasing cramping and muscle soreness, especially after a long duration training run or bike.

Introduce pre-season conditioning. Pre-season conditioning that includes a plyometric program can jump-start the process of bone remodeling and prepare the lower extremity support structures for elevated eccentric work loads.^{2,6,14,15,26,51,76}

It should be emphasized that every athlete needs to be addressed individually. Generic marathon training schedules are helpful, however they may need to be modified for each individual to maximize gains and minimize injury. An experienced trainer may aid the athlete in achieving goals by tailoring a training program and re-assessing progress based on training logs and exertion levels.

Following the above training advice may prevent injury. However, additional treatment and training modification may be necessary for the athlete who experiences shin pain. The discouraged athlete will usually seek care when an injury makes training impossible.

Diagnosing Shin Splints

To properly diagnose the athlete's condition, the treating physician will take a detailed history and examination to discover the onset and location of pain and attributed risk factors. Plain film radiographs may be used as a baseline study to evaluate for a bone stress reaction or other pathology. However, if plain film radiographs are initially negative for pathology, and there is a question about the extent of the injury based on clinical findings, more advanced imaging techniques, such as a triple-phase bone scan (BS) or a magnetic resonance imaging study (MRI), may be useful.

The initial history should include the following:

- initial onset of injury
- current symptoms
- progression of the frequency, intensity, and duration of training
- training surface
- detailed description of hill training, if any
- age and type of shoes
- recent weight fluctuations
- past history of stress fractures
- lower extremity strength training and flexibility
- training goals^{16,35}

Palpation may reveal diffuse tenderness over the distal two thirds of the posteromedial aspect of the shin (MTSS) or the anterolateral aspect of the shin (ATSS).^{16,19,37} However, palpation that reveals the following may be a red flag for bone-stress reaction:

- focal tenderness localized to bone
- edema
- erythema
- inability to train due to severe pain at the localized site.^{14,17,19}

Vibratory irritation with a tuning fork or ultrasound may exacerbate symptoms related to more severe cases of bone stress reactions, however these findings are not typically reliable.

Provocative testing may lead to further diagnosis. These tests are meant to increase eccentric strain on the posterior calf musculature that attach to the tibia, thereby leading to or exacerbating symptoms related to MTSS. These provocative tests may include:

- one legged hops on an unstable surface
- passive dorsiflexion of the foot
- ground-reactive dorsiflexion force applied to the talocrural joint.^{6,37}

Visual observation of the medial longitudinal arch (instep) may reveal a pes planus or pes cavus foot structure. For a more detailed analysis of foot structure and compensatory movements, measurements with a goniometer can be obtained.^{27,28,30}

The initial history, palpation findings, provocative muscle testing, visual inspection, and detailed goniometer measurements can help determine risk factors and a clinical diagnosis. If the patient does not improve with conservative care or the clinical diagnosis is unclear, plain film radiographs or advanced imaging techniques, such as a BS or an MRI, can help differentiate between MTSS/ATSS, a bone stress reaction, or other pathology.

Advanced imaging techniques

Plain film radiographs are routinely taken of the leg in patients with shin pain. This baseline study may reveal a periosteal reaction, callus formation, or a radiolucent line that are common findings of a bone stress reaction or frank stress fracture. Radiographs may also rule out other pathological conditions such as an osteoid osteoma, osteosarcoma, or Ewing's sarcoma. However, bone stress reactions are usually not visualized on radiographs until the 2nd to 6th week post injury/initial complaints of symptoms.^{1,21,35,52} "The sensitivity of early fracture detection by radiography can be as low as 15%, and follow up radiographs may demonstrate diagnostic findings in only 50% cases."¹⁶ More advanced studies may be needed for further evaluation, such as a MRI or a BS. These studies are more sensitive in detecting bone pathology earlier in the stage of injury, and they aid clinical judgment about a gradual return to sport specific training. Such gradual returns may require a longer period of modified rest, immobilization, or corrective surgery for more advanced cases of bone stress reactions.^{1,10,53}

An MRI is highly sensitive to the detection of edema in musculoskeletal structures. An MRI may include two types of sequences:

- a T1-weighted sequence, which depicts anatomy and more advanced signal abnormalities in the cortical bone.
- either a short tau inversion recovery (STIR) or T2 frequency-selective fat suppression sequence, which are more sensitive in detecting edema in the muscle, periosteum, or bone marrow.^{12,21,53}

Frederickson et al came up with a graded classification of MRI findings and the severity of bone stress reactions. "Grade 1 injuries are mild and demonstrate mild periosteal edema on the T2 images only. Grade 2 injuries demonstrated more severe periosteal edema with bone marrow edema detected on T2 images only. Grade 3 injuries demonstrate moderate to severe edema of both the periosteum and bone marrow on T2 and now also on the T1 images. Grade 4 injuries demonstrate a low signal fracture line with changes of severe marrow edema on both T1 and T2 sequences." In Frederickson's study, when pain was present during training and normal ambulation, there was an 81% incidence of a Grade 3 or a Grade 4 injury depicted by an MRI study.¹⁶

A bone scan (BS) is highly sensitive in detecting osteoblast activity that occurs in bone remodeling. Usually a triple phase BS is conducted, and involves a pre-injection of an isotope (technetium-99m methylene diphosphonate). In the first phase, the blood flow phase,

images are taken over the symptomatic area for 60 seconds after the initial injection. The second phase, the blood pool phase, involves taking images immediately after the blood flow phase for 5 minutes. The third phase, the delayed skeletal phase, involves taking images 2-6 hours after the initial injection. The delayed images allow the isotope to clear from the adjacent soft tissue. Under normal circumstances, approximately 50% of the isotope is absorbed into the skeletal system; however, the amount of radiotracer absorption depends on the rate of bone remodeling/osteoblastic activity. Less severe bone stress injuries are depicted on images as ill-defined foci of increased isotope absorption and are located predominantly in the cortical region of bone. More severe bone stress injuries involve a higher rate of bone remodeling, resulting in increased isotope resorption and wide fusiform lesions that extend from the cortical bone into the medullary bone.^{4,17,54} Acute stress fractures are positive for increased isotope activity on all three phases. Soft tissue injuries indicate increased uptake in the initial two phases but not in the delayed skeletal phase. MTSS indicates linear foci of increased isotope uptake along the posterior border of the tibia in the delayed skeletal phase only.⁵⁵

MRI and BS studies can be valuable for early detection and differentiation of shin pain. They may prove useful in avoiding complications due to undetected bone stress reactions or other pathologies. However, advanced imaging findings can be vague. Previous studies utilizing MRI's and BS's have indicated false positive findings in asymptomatic patients; false negative findings have also been noted.^{3,12,16,54} To ensure proper protocol, the treating physician should correlate MRI and BS findings with the clinical examination.

The hallmark test for ECS is intra-compartmental pressure measurements with a slit catheter. Measurements consistent with the diagnosis of ECS are pre-exercise pressure ≥ 15 mm Hg, 1 minute post-exercise ≥ 30 mm Hg, or 5 minute post-exercise ≥ 20 mm Hg.¹⁹ In anterior compartment syndrome, muscle testing may reveal weakness of dorsiflexion of the toe or foot, and paresthesia on the dorsum of the foot. In deep posterior compartment syndrome, muscle testing may reveal weakness in plantar flexion and inversion, and paresthesia on the plantar aspect of the foot.¹

Treatment of shin splints

Goals of therapy include the following:

- reduce pain and promote healing.
- incorporate pain-free modified training to maintain fitness.
- correct or minimize risk factors.
- gradually re-introduce pain-free activity.
- develop realistic additional training goals.

Severity of symptoms and level of injury are generally scored on a four-grade system:

Grade 1: Pain is present at the end of the workout but is minimal.

Grade 2: Pain is present during the workout but does not affect performance.

Grade 3: Pain during the workout affects performance but dissipates when the workout ends.

Grade 4: Pain does not allow participation in sport and is present during activities of daily living.¹

Usually, the athlete will seek treatment when pain hinders performance. The initial goals of therapy are to promote healing and reduce pain and inflammation. The following initial treatments may be useful:

Inflammation reduction

Use of nonsteroidal anti-inflammatory medications per prescription, and application of a cold pack to the shin for twenty minutes on, one hour off, repeated throughout the day can reduce inflammation. Use of iontophoresis with dexamethasone may also decrease inflammation.^{1,19,56}

Ultrasound and electric muscle stimulation combination therapy

The therapy can restore normal muscle tone, aid in the healing process, and reduce pain.^{1,10,19,32,57,58}

Manual adjustments to the ankle and foot

Adjustments free-up joint motion of the talocrural, subtalar, and midtarsal joint articulations.^{57,59}

Deep tissue procedures, such as the Graston Technique (manual therapy that utilizes specially designed devices) and Active Release Technique (a patented manual therapy technique)

Procedures break up scar tissue and restore soft tissue motion (figures 9A,9B). There is considerable clinical evidence to support the effectiveness of deep tissue procedures in treatment of strain/sprain injuries.⁶⁰⁻⁶³ Myofascial techniques have been shown to stimulate fibroblast proliferation, leading to collagen synthesis that may promote healing by replacing degenerative tissue with a stronger and more functional tissue.^{45,65}

Phototherapy, such as low-level laser therapy or infrared light

Phototherapy decreases inflammation, increases the speed of tissue healing, and decreases pain.^{58,64}

The above treatments may reduce pain and inflammation, and may speed the body's normal healing response. However, time is a primary factor in recovery: Approximate return to pre-injury strength for bones, ligaments, muscles, and tendons can range from 12 weeks, 40-50 weeks, 6 weeks-6 months, and 40-50 weeks respectively.^{1,19}

During the initial treatment phase – for more severe Grade 2, Grade 3, and Grade 4 injuries – athletes can maintain cardiovascular fitness with modified training. For example, a runner should cycle or swim at a pain-free level.^{1,12,19}

There are no exact studies indicating when to re-introduce sport-specific training; however, the following may be a useful guideline: If there are no time-specific training goals, cease sport specific activity for 2 weeks, and maintain cardiovascular fitness with modified training. After 2 weeks of modified training and conservative therapy, re-introduce pain-free running on a soft track or treadmill, at approximately 50% of the pre-injury intensity and duration. Then, increase the duration of the training runs by 10% each week. It is hopeful that the pre-injury duration can be reached in 5-6 weeks. Athletes should cease running if they experience pain. A brief period of modified training

Fig. 9A Starting Position Active Release Technique Demonstrated on Tibialis Anterior Muscle



Finish Position

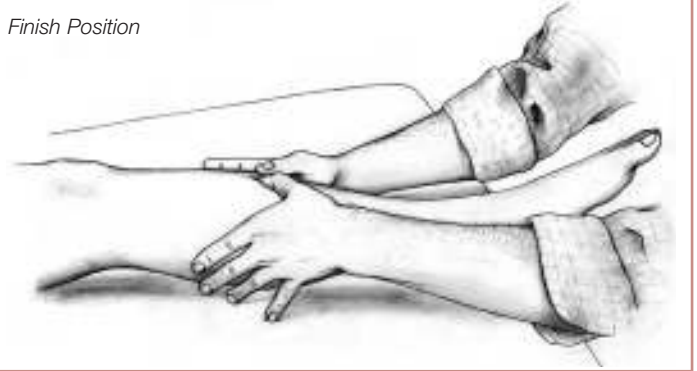
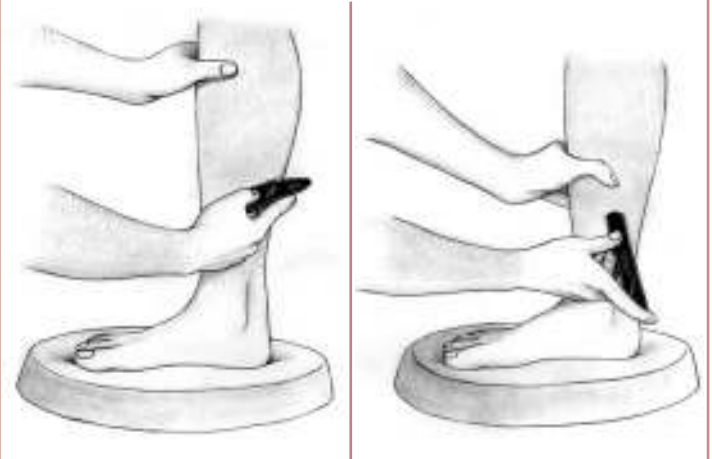


Fig. 9B Graston Technique on Posterior Leg Muscles



and resumption of pain-free running at a lower intensity and shorter duration may be necessary. The intensity of the training should progress appropriately only after the pre-injury duration is obtained.^{37,19}

Depending on the duration of the athlete's most recent pre-injury training run, my recommendations for return to activity may vary with the above guidelines. Usually the duration of the initial training run would be 2-4 miles, conducted on a treadmill, at a speed of 10 minutes/mile (very slow for most athletes). If the athlete can conduct 2-3 of these training runs without pain, we would create a program, based on the above guidelines, for achieving time-specific goals.

Before re-introducing sport-specific training, the following methods may reduce the workload placed on the musculoskeletal support structure of the lower leg that are present due to intrinsic risk factors:

- **Buy proper running shoes.**

A pes cavus foot structure may benefit from a cushioned sneaker. The sneaker liner can be removed and replaced with a cushioned insole. The rearfoot varus, pes planus valgus, and forefoot varus foot structure may benefit from a motion-control sneaker.⁶⁶

- **Use appropriate arch supports as necessary.**

A semirigid orthosis with a medial arch support, no higher than five-eighths of an inch, can limit excess or prolonged pronation.^{1,2,27,28,35,67-70}

- **Tape the foot.**

Taping can limit pronation.^{57,71,72}

- **Temporarily use a quarter-inch or three-quarter inch heel lift.**

Temporary use can limit compensatory pronation caused by ankle equinus. As range of motion of the talocrural joint in dorsiflexion improves with therapy, the heel lifts can be removed.^{1,28,68}

- **Apply a shin sleeve or strapping.**

Sleeves and strappings can add support for leg muscles.

- **Obtain nutritional advice.**

A dietitian can calculate caloric burn rate and develop a meal plan for healthy weight loss, maintenance, and proper nutrition.¹⁹

- **Obtain medical examinations.**

Primary physicians or gynecologists can rule out deficiencies common in athletes, such as low estrogen, amenorrhea, or low bone density.

- **Train for flexibility.**

Increasing flexibility can reduce compensatory pronation due to ankle equinus, and the posterior calf musculature can be stretched.^{1,35,43,73,74}

The treating physician needs to continually re-evaluate the athlete during the course of therapy. If the athlete has time-specific goals, is not responding to conservative care, or symptoms re-appear when sport-specific training is re-initiated, a BS or MRI may be needed. In addition, treatment of slow healing stress fractures may require immobilization with casting or a walking boot for approximately 3-8 weeks.^{4,10} Pain-free modified training can maintain the athlete's cardiovascular fitness, and sanity, during this period. Once union of the stress fracture is evident on repeat imaging studies, and pain is not present with ambulation, sport-specific training can be gradually re-introduced.

If non-union of the stress fracture is present on repeat imaging studies after 4-6 months of immobilization and rest, surgery may be necessary. Surgery may include intramedullary nailing, cortical drilling, or excision and bone grafting.^{14,33,75} In recalcitrant cases of MTSS, surgery may include fasciotomy of the posteromedial superficial and the deep fascia of the tibia.¹ Posteromedial fasciotomy may aid in alleviating the pull of the soleus and deep posterior calf musculature of the leg on their insertions on the tibia, as well as decreasing pain due to denervation of the periosteum.^{37,19} Post-fasciotomy studies have indicated good results in regards to reduced shin pain, however many athletes are not able to return to full sport specific training levels.^{35,75} Fasciotomy of the involved compartment would be a surgical option to reduce shin pain due to ECS that has been resistant to conservative care over a period of 6-12 weeks.^{4,35}

Conclusion:

Shin splints is one of the most common lower leg sport-related injuries. Risk factors include training errors, foot structure abnormalities, high body-mass index,

age-related degenerative changes, poor conditioning, and inadequate calcium intake or estrogen levels. Most cases of shin splints can be treated successfully with conservative care. Conservative treatment includes the following:

- reduction of pain and inflammation
- modified training to maintain cardiovascular fitness
- modifications to gear, such as obtaining new running shoes
- implementation of a strength and flexibility program
- correction of training errors
- a pain-free gradual return to sport-specific activity
- Nutritional counseling or hormonal therapy

If the injury does not respond to conservative care, or there is a time-specific training goal, a BS or MRI may be beneficial in the early detection of stress fractures or other pathologies. Other treatment options for recalcitrant shin splints may include prolonged immobilization with pain-free modified training, surgery to promote bone-union, or a fasciotomy.

REFERENCES

1. Reid DC. Sports Injury Assessment and Rehabilitation. New York: Churchill Livingstone Inc., 1992.
2. Yates B, White S. The incidence and risk factors in the development of medial tibial stress syndrome among naval recruits. *The American Journal of Sports Medicine* 2004; 32(3):772-780.
3. Hislop M, Tierney P. Anatomical variations within the deep posterior compartment of the leg and important clinical consequences. *Journal of Science and Medicine in Sport* 2004; 7(3):392-9.
4. Wilder RP, Sethi S. Overuse injuries: tendinopathies, stress fractures, compartment syndrome, and shin splints. *Clin Sports Med* 2004; 23:55-81.
5. Magnusson HI, Westlin NE, Nyquist F, Gardsell P, Seeman E, Karlsson MK. Abnormally decreased regional bone density in athletes with medial tibial stress syndrome. *American Journal of Sports Medicine* 2001; 29(6):712.
6. Herring K. A plyometric training model used to augment rehabilitation from tibial Fasciitis. *Current Sports Medicine Reports* 2006; 5(3):147-54.
7. Batt ME, Ugalde V, Anderson MW, Shelton DK. A prospective controlled study of diagnostic imaging for acute shin splints. *Medicine & Science in Sports & Exercise* 1998; 30(11):1564-1571.
8. Thacker SB, Gilchrist J, Stroup D, Dexter Kimsey C. The prevention of shin splints in sports: a systematic review of literature. *Medicine & Science in Sports & Exercise* 2002; 34(1):32-40.
9. Korpelainen R, Orava S, Karpakka J, Siira P, Hulkko A. Risk factors for recurrent stress fractures in athletes. *The American Journal of Sports Medicine* 2001; 29:304-310.
10. Jensen J. Stress fracture in the world class athlete: a case study. *Medicine & Science in Sports & Exercise* 1998; 30(6):783-787.

11. Tommasini SM, Nasser P, Schaffler MB, Jepsen KJ. Relationship between bone morphology and bone quality in male tibias: implications for stress fracture risk. *Journal of Bone and Mineral Research* 2005; 20(8):1372-1380.
12. Wall J, Feller JF. Imaging of Stress Fractures in Runners. *Clinics in Sports Medicine* 2006; 25:781-802.
13. Sommer HM, Vallentyne SW. Effect of foot posture on the incidence of medial tibial stress syndrome. *Medicine & Science in Sports & Exercise* 1995; 27(6):800-804.
14. Noakes T. *The Lore of Running* (4th Edition). Illinois: Human Kinetics, 2003; 803-816.
15. Shaffer RA, Rauh MJ, Brodine SK, Trone DW, Macera CA. Predictors of stress fracture susceptibility in young female recruits. *The American Journal of Sports Medicine* 2006; 34(1):108.
16. Spitz D, Newberg A. Imaging of stress fractures in the athlete. *Radiologic Clinics of North America* 2002; 40:313-331.
17. Bhatt R, Lauder I, Finlay DB, Allen MJ, Belton IP. Correlation of bone scintigraphy and histological findings in medial tibial syndrome. *British Journal of Sports Medicine* 2000; 34:49-53.
18. Magnusson HI, Ahlborg HG, Karlsson C, Nyquist F, Karlsson MK. Low regional tibial bone density in athletes with medial tibial stress syndrome normalizes after recovery from symptoms. *American Journal of Sports Medicine* 2003; 31(4):596.
19. Edwards PH Jr, Wright ML, Hartman JF. *American Journal of Sports Medicine* 2005; 33:1241.
20. Bennell K, Crossley K, Jayarajan J, Walton E, Warden S, Kiss SZ, Wrigley T. Ground reaction forces and bone parameters in females with tibial stress fracture. *Medicine & Science in Sports & Exercise* 2004; 36(3):397-404.
21. Aoki Y, Yasuda K, Tohyama H, Ito H, Minami A. Magnetic resonance imaging in stress fractures and shin splints. *Clinical Orthopaedics* 2004; 421:26-267.
22. Weineck J. *Functional Anatomy in Sports*, Second Edition. St. Louis: Mosby-Year Book, 1990.
23. Wheeler PR, Burkitt HG, Daniels VG. *Functional Histology*. New York: Churchill Livingstone, 1987.
24. Banks AS, Downey MS, Martin DE, Miller SJ. *Foot and Ankle Surgery*. Philadelphia: Lipincott Williams & Wilkins, 2001.
25. Clemente CD. *Anatomy: A Regional Atlas of the Human Body* (3rd Edition). Baltimore: Urban & Schwarzenberg, 1987.
26. Richie D, DeVries H, Endo C. Shin muscle activity and sports surfaces. *Journal of the American Podiatric Association* 1993; 83(4):181-190.
27. Michaud TC. *Foot orthosis and other forms of conservative foot care*. Newton MA: Thomas C Michaud, 1997.
28. Donatelli RA. *The biomechanics of the foot and ankle*, 2nd Edition. Philadelphia: F.A. Davis, 1996.
29. Norkin CC, Levangie PK. *Joint Structure and Function: A Comprehensive Analysis* (2nd Edition). F.A. Davis, Philadelphia 1992.
30. Banks AS, Downey MS, Martin DE, Miller SJ. *Foot and Ankle Surgery*. Philadelphia: Lipincott Williams & Wilkins, 2001.
31. Inman VT. *Human Locomotion*. *Can Med Assoc J*. 94:1047, 1996.
32. Chleboun GS, Busic AB, Graham KK, Stuckey HA. Fascicle length change of the human tibialis anterior and vastus lateralis during walking. *Journal of Orthopaedic & Sports Physical Therapy* 2007; 37(7):372-379.
33. Larson CM, Traina SM, Fischer DA, Arendt EA. Recurrent complete proximal tibial stress fracture in a basketball player. *The American Journal of Sports Medicine* 2005; 33(12):1914.
34. Pozderac RV. Longitudinal tibial fatigue fracture: an uncommon stress fracture with characteristic features. *Clinical Nuclear Medicine* 2002; 27(7):475-478.
35. Fredericson M, Wun C. Differential diagnosis of leg pain in the athlete. *Journal of the American Podiatric Medical Association* 2003; 93(4):321-324.
36. Reinking MF, Austin TM, Hayes AM. Exercise-related leg pain in collegiate cross-country athletes: extrinsic and intrinsic risk factors. *Journal of Orthopaedic & Sports Physical Therapy* 2007; 37(11):670-678.
37. Kortebein PM, Kaufman KR, Basford JR, Stuart MJ. Medial tibial stress syndrome. *Medicine & Science in Sports & Exercise* 2000; 32(2):S27-S33.
38. Taunton JE, Ryan MB, Clement DB, McKenzie DC, Lloyd-Smith DR, Zumbo BD. A retrospective case-control analysis of 2002 running injuries. *British Journal of Sports Medicine* 2002; 36:95-101.
39. Smurawa T. Overuse injuries curb triathlon preparation efforts. *Biomechanics* 2006; 13(5).
40. Martin DE, Coe PN. *Better Training for Distance Running* (2nd Edition). Champaign, IL: Human Kinetics, 1997.
41. Higdon H. Hal Higdon's Marathon Training Guide. www.halhigdon.com/marathon/Mar00novice.htm. Accessed March 14, 2008.
42. Armstrong RB, Ogilvie RW, Schwane JA. Eccentric exercise-induced injury to rat skeletal muscle. *Journal of Applied Physiology* 1983; 54(1):80-93.
43. Roxas M. Plantar fasciitis: diagnosis and therapeutic considerations. *Alternative Medicine Review* 2005; 10(2):83-93.
44. Messier SP, Edwards DG, Martin DF, et al. Etiology of iliotibial band friction syndrome in distance runners. *Medicine & Science in Sports & Exercise* 1995; 27(7):951-960.
45. Dyck D, Boyajian-O'Neill L. Plantar Fasciitis. *Clinical Journal of Sports Medicine* 2004; 14(5):305-309.
46. Friden J, Sfikianos PN, Hargens AR. Muscle soreness and intramuscular fluid pressure: comparison between eccentric and concentric load. *Journal of Applied Physiology* 1986; 61(6):2175-2179.

47. Allen RH, Gross MT. Toe flexors strength and passive extension range of motion of the first metatarsophalangeal joint in individuals with plantar fasciitis. *Journal of Orthopaedic & Sports Physical Therapy* 2003; 33(8):468-78.
48. reference for squats-muscle activity
49. Witvrouw E, Mahieu N, Roosen P, McNair P. The role of stretching in tendon injuries. *British Journal of Sports Medicine* 2007; 41:224-226.
50. Witvrouw E, Mahieu N, Danneels L, McNair P. Stretching and injury prevention. *Sports Med* 2004; 34(7):443-449.
51. Chmielewski TL, Myer GD, Kauffman D, Tillman SM. Plyometric exercise in the rehabilitation of athletes: physiological responses and clinical application. *Journal of Orthopaedic & Sports Physical Therapy* 2006; 36(5): 308-319.
52. Ruohola JPS, Kiuru MJ, Pihlajamaki HK. Fatigue bone injuries causing anterior lower leg pain. *Clinical Orthopaedics and Related Research* 2006; 444:216-223.
53. Gaeta M, Minutoli F, Vinci S, Salamone I, D'Andrea Letterio, Bitto L, Magauidda L, Blandino A. High-resolution CT grading of tibial stress reactions in distance runners. *American Journal of Radiology* 2006; 187:789-793.
54. Hod N, Ashkenazi I, Levi Y, Fire G, Drori M, Cohen I, Bernstine H, Horne T. Characteristics of skeletal stress fractures in female military recruits of the Israeli Defense Forces on bone scintigraphy. *Clinical Nuclear Medicine* 2006; 31(12):742-749.
55. Love C, Din AS, Tomas MB, Kalappambath TP, Palestro CJ. Radionuclide bone imaging: an illustrative review. *Radiographics* 2003; 23:341-358.
56. Pellecchia GL, Hamel H, Behnke P. Treatment of infrapatellar tendonitis: a combination of modalities and transverse friction massage versus iontophoresis. *J Sports Rehabil* 1994; 3(2):35-145.
57. Hyde T. *Conservative management of sports injury*. Baltimore: Williams & Wilkins, 1997; pp477-82.
58. Gum SL, Reddy GK, Stehno-Bittel L, Enwemeka CS. Combined ultrasound, electrical muscle stimulation, and laser promote collagen synthesis with moderate changes in tendon biomechanics. *Am J Phys Med Rehabil* 1997; 76(4):288-96.
59. Young B, Walker M, Strunce J, Boyles R. A combined treatment approach emphasizing impairment-based manual physical therapy for plantar heel pain: a case series. *The Journal of Orthopaedic & Sports Physical Therapy* 2004; 34(11):725-33.
60. Walker JM. Deep transverse frictions in ligament healing. *Journal of Orthopaedic & Sports Physical Therapy* 1984; 6(2):89-94.
61. Brosseau L, Casimiro, Milne S, et al. Deep transverse friction massage for healing tendonitis. *Cochrane Database Syst Rev* 2002; (4):CD003528.
62. Kvist M, Jarvinen M. Clinical histochemical and biomechanical features in repair of muscle and tendon injuries. *Int J Sports Med* 1982; 3 Suppl 1:12-14.
63. Roniger LR. Massage, strengthening reduce knee OA pain disability. *Biomechanics* 2007; XIV(2):17-18.
64. Roniger LR. Research focus on lower limb pain brings relief. *Biomechanics* 2008; XV(1):21.
65. Leadhetter W. Cell matrix response in tendon injury. *Clinics in Sports Medicine* 1997; 11(3):533-79.
66. Butler R, Davis I, Hamill J. Interaction of joint type and footwear on running mechanics. *The American Journal of Sports Medicine* 2006; 34(12):1998-2005.
67. Fillipou D, Kalliakmanis A, Triga A, Rizos A, Grigoriadis E. Sports related plantar fasciitis. *Current Diagnostic and Therapeutic Advances. Folia Medica* 2004; 46(3):56-60.
68. Sobel E, Levitz S, Caselli M. Orthoses in the treatment of rearfoot problems. *Journal of the American Podiatric Association* 1999; 89(5):220-33.
69. Landorf K, Keenan A, Herbert R. Effectiveness of different types of foot orthoses for the treatment of plantar fasciitis. *Journal of the American Podiatric Association* 2004; 94(6):542-49.
70. Kogler G, Veer F, Solomonidis S, Paul J. The influence of medial and lateral placement of orthotic wedges on loading of the plantar aponeurosis: an in vitro study. *Journal of Bone & Joint surgery* 1999; 81-A(1):1403-1413.
71. Landorf K, Radford J, Keenan A, Redmond A. Effectiveness of low-dye taping for the short term management of plantar fasciitis. *Journal of the American Podiatric Association* 2005; 95(6):525-30.
72. Radford J, Burns J, Buchbinder R, Landorf K, Cook C. The effect of low-dye taping on the kinematic, kinetic, and electromyographic variables. *Journal of Orthopaedic & Sports Physical Therapy* 2006; 36(4):232-41.
73. Didiovanni B, Nawoczinski D, Lintal M, Moore E, Murray J, Wilding G, Baumhauer J. Tissue-specific plantar fascia-stretching exercise enhances outcomes in patients with chronic heel pain: a prospective randomized study. *The Journal of Bone & Joint Surgery* 2003; 85-A(7):1270-77.
74. Mahieu N, McNair P, DeMuyneck M, Stevens V, Blanckaert I, Smits N, Witrouw E. Effect of static and ballistic stretching on the muscle-tendon tissue properties. *Medicine & Science in Sports & Exercise* 2007; 39(3):494-501.
75. Yates B, Allen MJ, Barnes MR. Outcome of surgical treatment of medial tibial stress syndrome. *The Journal of Bone & Joint Surgery* 2003; 85-A(10):1974-1980.
76. Woodley BL, Newsham-West RJ, Baxter GD. Chronic tendinopathy: effectiveness of eccentric exercise. *British Journal of Sports Medicine* 2007; 41:188-199.
77. Andrew JR, Harrelson GL, Wilk KE. *Physical rehabilitation of the injured athlete* (3rd Edition). Philadelphia, PA:Saunders, 2004.
78. Comfort P, Kasim P. Optimizing squat technique. *Strength and Conditioning Journal* 2007; 29(6):10-13.

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