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Mechanisms and Management of Stress Fractures in Physically Active Persons

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Objective: To describe the anatomy of bone and the physiology of bone remodeling as a basis for the proper management of stress fractures in physically active people.

Data Sources: We searched PubMed for the years 1965 through 2000 using the key words *stress fracture, bone remodeling, epidemiology,* and *rehabilitation.*

Data Synthesis: Bone undergoes a normal remodeling process in physically active persons. Increased stress leads to an acceleration of this remodeling process, a subsequent weakening of bone, and a higher susceptibility to stress fracture. When a stress fracture is suspected, appropriate management of the injury should begin immediately. Effective management includes a cyclic process of activity and rest that is based on the remodeling process of bone.

Conclusions/Recommendations: Bone continuously remodels itself to withstand the stresses involved with physical activity. Stress fractures occur as the result of increased remodeling and a subsequent weakening of the outer surface of the bone. Once a stress fracture is suspected, a cyclic management program that incorporates the physiology of bone remodeling should be initiated. The cyclic program should allow the physically active person to remove the source of the stress to the bone, maintain fitness, promote a safe return to activity, and permit the bone to heal properly.

Key Words: bone remodeling, rehabilitation, stress reaction

Stress fractures can occur in any physically active person. As a result, athletic trainers and sports therapists need to understand the injury mechanism and strategies for management. We describe the incidence, latest theories of causation, and a protocol for the management of stress fractures based on the physiology of bone remodeling. We also describe the incidence of stress fractures, distribution of forces to bone, normal and abnormal bone anatomy and remodeling, and proposed risk factors for stress fractures in a physically active population.

INCIDENCE

Stress fractures occur in several different bones. The distribution of stress fractures differs according to activity. The tibia is reported to be the most frequently injured bone in runners,[1,2] followed by the fibula, metatarsal, and pelvis (Table 1).[3] Fifteen percent of all stress fractures occur in runners,[3] accounting for 70% of all of their injuries.[4] In dancers, the metatarsal is the most common location of injury.[5] Stress fractures in the ribs have been described in golfers,[6] and stress fractures of the pars interarticularis are prevalent in racket sports and basketball players.[5]

Different study designs, populations, and classification schemes make it difficult to definitively report the incidence of stress fractures in varying populations.[7] Some trends exist in the incidence of stress fractures between the sexes and among the races. In military populations, women are more likely to sustain stress fractures.[8–10] In athletes, however, the disparity between the sexes is not as conclusive. Whereas Hickey et al[11] found differences between athletic men and women that were similar to those in military populations, others have reported that female collegiate athletes have a similar[12] or only slightly higher rate of injury than men.[13]

A disparity also exists in the incidence of stress fractures among the races. In the military, white men and women have shown a higher incidence of stress fractures than African Americans or Hispanics.[10,14] One explanation for this difference may be the lower overall bone density in whites as compared with the other 2 groups.[15]

DISTRIBUTION OF FORCES TO BONE

A stress fracture is a partial or incomplete fracture caused by the accumulation of stress to a localized area of bone.[16–20] Stress fractures are not the result of one specific insult. Instead, they arise as the result of repetitive applications of stresses that are lower than the stress required to fracture the bone in a single loading.[16–21]

Bone endures a stress whenever a force is loaded upon it. Whether the stress comes from the pull of a muscle or the shock of a weight-bearing extremity contacting the ground, it

Table 1. Percentage of Stress Fractures by Bone in Previous Studies

Study	(n)	Tibia, %	Metatarsals, %	Fibula, %	Navicular, %
Brukner et al[5]	180	20	23.3	16.6	14.4
Ha et al[2]	169	31.5	7.1	10.7	4.5
Hulkko and Orava[1]	369	49.5	19.8	12.0	2.5
Matheson et al[3]	320	49.1	8.8	6.6	25.3
Orava[4]	200	53.5	18.0	12.5	1.5

is defined as the force applied per unit area of the load-bearing bone.[7,22] Low levels of these forces cause bone to deform,[23] which is known as strain.[7] The bone's stress-strain response depends on the load's direction; the bone's geometry, microarchitecture, and density; and the influence of surrounding muscular contractions.[7] In most activities of daily living (ADLs), when the force is removed, the bone elastically rebounds to its original position. The force that a bone can endure and still rebound back to its original state without damage is within the elastic range.[17,23,24] Forces that exceed a critical level above the elastic range are in the plastic range.[20,22] Once forces reach the plastic range, a lower load causes greater deformation; it is at this level that forces summate to permanently damage the bone.[25,26]

Forces can be applied to bone through compression, tension, bending, torsion, or shear.[7] Compression forces are generally seen in cancellous bones, such as the calcaneus and femoral neck. Tension forces, however, result in bone pulling away from bone, as is common in compact bones such as the tibia and femur. As the load is applied to the bony shaft through a bend, a tension strain is placed upon the convex surface of the shaft[27] and compressive forces act on the concave side (Figure 1).[24]

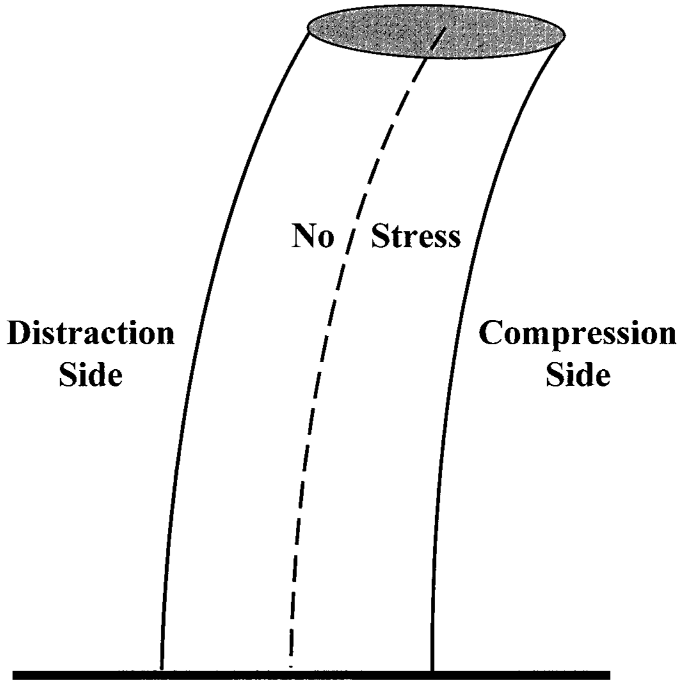

Figure 1. Types of forces applied to a bone include compression and distraction.

The muscles attached to the surface of compact bones can help to increase or decrease the intensity of a load.[7] The muscular attachments on the surface of compact bones can produce a tension force that acts circumferentially[28,29] or acts as a shock absorber by controlling bone strain.[30,31] In cases of excessive muscular pull, a stress fracture may develop near the bone-tendon junction. This mechanism is common in non-weight-bearing bones such as the ribs and fibula.[5,6] Conversely, weakness or fatigue in the shock-absorbing muscles may allow for an increased load to be translated to the bone, making it more susceptible to stress fracture.[3]

Anatomy

Bone has both cortical and cancellous components. Cortical bone is dense and highly organized and withstands stress in compression better than in tension.[7] Cancellous (trabecular) bone is an irregularly shaped meshwork[7] and withstands stress according to the alignment of the fiber matrix.[32] The outer shafts of long bones (eg, tibia, humerus) are mainly cortical, with a large percentage of cancellous bone making up the ends of the bone and the central portion of the shaft.[16] Short and flat bones such as the tarsals and pelvis have a higher content of cancellous bone.

The fundamental unit of cortical bone is the osteon. In the osteon, concentric layers of lamellar bone surround small channels called haversian canals. These canals house nerves and blood vessels. On the outside of the lamellae are small cavities, known as lacunae. Each lacuna contains a single bone cell, or osteocyte. Canaliculi form a transport system between the lacunae and the haversian canals that is responsible for the nutrition and metabolic transport system within the bone.[7,33]

Surrounding the outer surface of long bones is a highly vascular outer coating called the periosteum. The periosteum is responsible for providing nutrition to the outer portion of the cortex and enlarges during remodeling to provide support to the cortex. On the inner portion of the cortex, medullary canals allow the vascular passage for nutrients and blood vessels to the inner two thirds of the cortex (Figure 2).[34]

Remodeling

Bone constantly remodels itself to more efficiently endure external forces.[35,36] According to column law, the magnitude of stress is greatest on the surface of a column and decreases to zero at the center. Accordingly, most of the remodeling in long bones takes place in the outer cortex.[37] Remodeling involves the resorption of existing bone by osteoclasts and the formation of new bone cells by osteoblasts.[22,23,38–41] Participating in regular activity promotes bone strength through proper perfusion of nutrients to the osteocytes and normal bone remodeling. Conversely, a sedentary lifestyle contributes to bony atrophy.[35,36,42–44]

In order to begin remodeling, osteoclastic cells need to be activated. The piezoelectric effect is one mechanism implicated in the activation of bone remodeling.[45,46] Tension forces create a relative electropositivity on the convex, or tension side, of the bone. This increase in positive charge is conducive to osteoclastic resorption.[29,45–47] Thus, as torque or bending produces repeated distraction forces at a focal point of a bone, the electropositive charge may stimulate osteoclastic absorption.

The streaming effect is the movement of extracellular fluids in the haversian canals and canaliculi during deformation. If the surface charge on the haversian canal or canaliculi walls

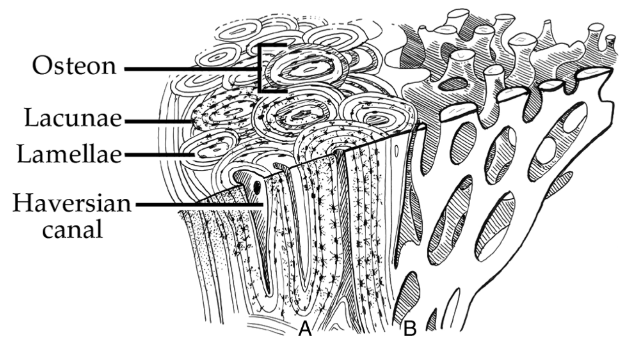

Figure 2. The anatomy of bone. A, Cortical bone is made up of functional units called osteons. Osteons include haversian canals surrounded by concentric lamellae and lacunae. B, Trabecular bone is in an irregular mesh-like matrix. (Adapted with permission from Matin P. Basic principles of nuclear medicine techniques for detection and evaluation of trauma and sports medicine injuries. *Semin Nucl Med.* 1988;18:90–112.)

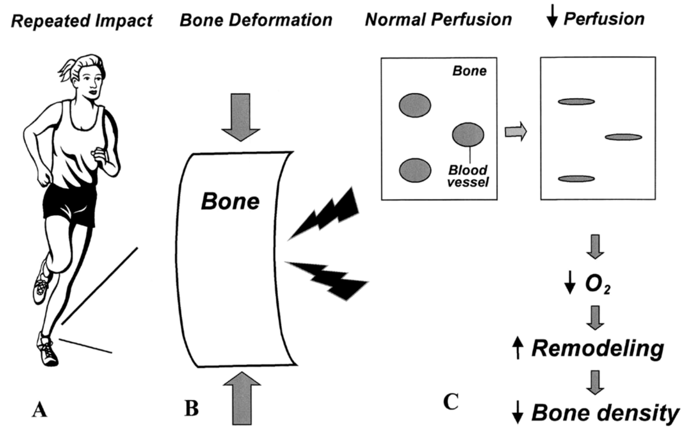

Figure 3. Ischemic mechanisms of stress fracture formation. Blood normally flows through vessels within the bone. As forces are applied (A), bone deforms (B), and the bone blood flow is temporarily restricted (C). This restriction in blood flow causes a decrease in oxygen perfusion and an increase in osteoclast resorption that results in increased remodeling and decreased bone density.

is positive, negative ions in the fluid are attracted to the outside of the fluid stream, creating a positively charged current in the middle. As bone is bent, the positive stream is forced toward the bone's open, or distracted, surface. The electropositive stream may, in turn, stimulate osteoclastic activity.[41] Other possible activators are bone ''sensors'' that recognize increased and decreased mechanical strains,[48] hormones,[41] decreased venous flow,[49] and decreased oxygen.[42]

Upon activation, osteoclastic cells form a cone and begin to secrete proteolytic enzymes to cut longitudinal tunnels through the bone. These new haversian canals are aligned with the stresses placed on the bone. Each osteoclast cone can resorb nearly 3 times its volume in burrowing a canal from 3 to 10 mm deep.[50] The new haversian canals are filled with osteoblasts that create a mineralized matrix that supports the walls of the new channel.[23,51] The remaining space of the channel is then filled with immature lamellar bone.

Haversian canal formation and osteoblast support with lamellar bone begins 10 to 14 days after the onset of remodeling.[52] The conversion of lamellar bone into mature osteocytes cells lags behind resorption by about a week[23] and may continue for as long as 20 to 90 days.[23,50] The result is a temporarily weakened bone due to the new, hollow haversian canals. The inflammation of periosteum is designed to bolster the weakened area of bone until it can mature.[52] However, the periosteum does not mature until about 20 days after the remodeling process begins. This 6- to 10-day lag between the deposit of immature lamellar bone and periosteal maturity leaves the bone temporarily weakened at the point of stress during the third week of remodeling.[22,52] Continued stress applied to remodeling bone during the ''weak third week'' may lead to an accelerated breakdown of the cortex. It is at this time that a stress fracture is most likely to develop.[3,22,53]

STRESS FRACTURES

Bone's response to stress has been confused in the literature by several different names and classification schemes. The terms *shin splints*,[54,55] *medial tibial stress syndrome*,[56–58] and *medial tibial syndrome*[59] are often used interchangeably to describe the symptoms and radiologic findings commonly associated with advanced bone remodeling and tibial stress fractures. Currently, bone's response to stress is evaluated on a dynamic continuum between early remodeling and periostitis to a cortical stress fracture.[3,60,61] It is important to note that the changes associated with bone's reaction to stress (eg, stress reaction) reflect a wide spectrum of physical findings and radiographic presentations.[60–62]

A true stress fracture is a visible cortical fracture. Stress fractures have traditionally been classified into 2 types: fatigue and insufficiency. The fatigue fracture is caused by an abnormal stress to a normally elastic bone.[19] Fatigue fractures are thought to occur in different sites depending on the age, sex, and activity of the athlete. Insufficiency fractures arise from the application of a normal stress on a bone that is mineral deficient or abnormally inelastic.[19] Insufficiency fractures are most prevalent in nutrient-deficient (osteomalacia) and older populations in whom osteoporosis and rheumatoid arthritis are more common.[17,19]

The fatigue fracture is more common in the physically active population.[19] The abnormal forces that cause a deterioration of healthy bone may result from increased training intensity, hard training surfaces, worn or inappropriate shoes, or poor anatomical alignment of the feet.[63] Muscular and aerobic capacity improve within the first week of an exercise regimen.[17,19] The result is an increase in exercise duration and pull of stronger muscles on bones that are still in a weakened phase of remodeling.[17]

Until recently, the cause of stress fractures was thought to be due to the breakdown of bone after repetitive loading. It has been estimated that, at normal physiologic levels of strain, it would require 10^8 cycles of loading to produce failure of a weight-bearing bone such as the tibia.[64] This level of loading is not easily attained, and stress fractures commonly occur soon after the onset of a stressful activity.[53,65,66] Greaney et al[53] found that 64% of the stress fractures in a military population began within the first 7 days of training. The rapid onset of symptoms and bone remodeling consistent with stress fracture suggest that mechanical stress cannot be the only cause.

Otter et al[67] proposed that the perfusion and reperfusion of bone after a repetitive load causes a temporary oxygen debt to the area of bone being stressed. This ischemia, in turn, facilitates bone remodeling and subsequent bone weakness and stress fracture. When a bone is loaded to normal physiologic levels, the small blood vessels that supply the cortex are squeezed.[43] In most cases, this pressure is necessary for proper movement of the blood.[42] When the load is higher, the blood flow may be temporarily cut off. The result is a brief period of ischemia in the cells that would normally be perfused by the compressed medullary vessels. Repeated loads over a prolonged period of an activity, such as a long run, cut off the oxygen during that period as well. This decrease in oxygen to the bone is believed to trigger the remodeling process.[42] In fact, Kelly and Bronk[49] found that restricting venous flow without any mechanical loading was enough to stimulate bone remodeling. In the above scenario, blood flow and oxygen perfusion are both restricted. This restriction is believed to signal the bone to remodel and cause the osteocytes to channel into the bone. The result is a weakened bone that is less able to withstand subsequent loads (Figure 3).[41]

The temporary lack of oxygen is not the only cause of ischemia. Repeated pressure to the capillaries is also believed to cause microdamage to the vessels. As neutrophils respond to plug the damaged capillaries, the blood flow through the vessels is further restricted.[68] In addition, small leaks in the vessels allow fluid flow into the surrounding tissue, further restricting the perfusion of oxygen into the cells. This leaking increases with subsequent bouts of loading, worsening ischemia and triggering a further increase in remodeling.[67] The repetition of this cycle causes an increase in remodeling, a breakdown in the cortex, a weakening of the bone, and potentially a stress fracture (Figures 4 and 5).

Ischemic mechanisms of tissue damage are common in other athletic injuries. For example, ice and compression are routinely used after an ankle sprain to limit effusion and secondary hypoxic injury. In this case, fluids from the damaged blood vessels in the anterior talofibular ligament allow leakage into the surrounding tissue. This excess fluid decreases oxygen tension and restricts oxygen perfusion to the adjacent cells. The result is damage to the ligament from the initial injury and damage to the tissue adjacent to the ligament from a lack of oxygen.

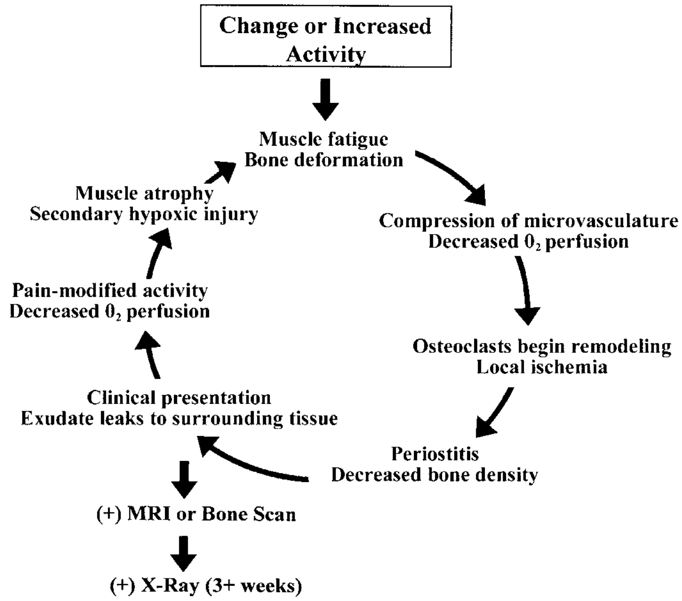

Figure 4. Cyclic etiology of stress fracture formation. Changes or increases in activity intensity cause muscle fatigue, bone deformation, and compression of the bone's microvasculature. A decrease in oxygen perfusion causes local ischemia and signals the beginning of osteoclast remodeling, decreased bone density, and periostitis. It is at this point that clinical signs of a stress fracture may be evident. Exudate from damaged blood vessels reduces oxygen perfusion to surrounding tissue and results in secondary hypoxic injury to surrounding bone cells. Pain leads to a modification or restriction of activity and muscular atrophy.

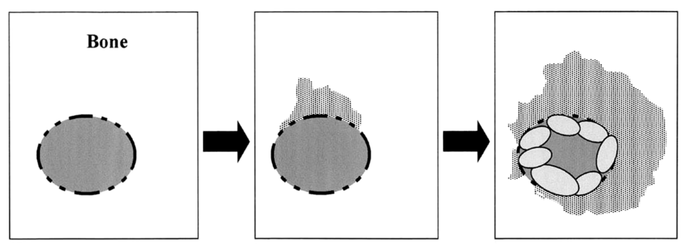

Figure 5. A, Microdamage to blood vessels allows fluid to leak into surrounding tissue. B, C, Neutrophil and macrophage proliferation partially restricts blood flow to the injured area, resulting in decreased oxygen perfusion around the damaged tissue.

Risk Factors

Several risk factors exist for insufficiency and fatigue stress fractures. Because weakened bone is susceptible to insufficiency stress fractures, populations with mineral-deficient conditions such as rickets or osteomalacia may also have bones that are unable to withstand normal forces. Moreover, normally strong bones may be weakened by cysts or surgical or medical procedures, such as screw fixation, tendon transfer, joint arthroplasty, bunionectomy, or radiation treatment.[19]

The unique nutritional demands of women place them at a higher risk for insufficiency stress fractures than men. Fredericson et al[60] found that stress fractures occurred more often in women, while Ha et al[2] found that the highest incidence of stress fractures was in teenage girls. One explanation for this difference may be the female athlete's susceptibility to the female athlete triad of eating disorders, amenorrhea,[69] and osteoporosis.[18] These findings are supported by a 12-month, prospective study of 53 female and 58 male track athletes: lower bone density, less lean body mass in the lower limb, a low-fat diet, and a history of menstrual disturbance in the female athletes were significant risk factors for stress fractures.[70]

Several authors[17,63] suggested that increased pronation is common among athletes with stress fractures of the lower extremity. Similarly, rigid cavus feet are a common predisposing factor to tarsal and femoral stress fractures.[3] Hard surfaces or inappropriate shoes may exaggerate these conditions.

Even though poor foot alignment or muscle imbalances may contribute to the onset of a stress fracture, some type of change is the common ingredient in most diagnoses.[20,24,37,40,55,71] This change may be an increase in the intensity or type of exercise or a change in playing surfaces or footwear. Any of these changes may create an increase in stress to the bone and a subsequent increase in the rate of remodeling. Goldberg and Pecora[13] found that 67% of 58 stress fractures in college varsity athletes were in freshmen who may have been experiencing changes in training intensity at the collegiate level.

R	Removal of the abnormal stress
E	Exercise to maintain cardiovascular fitness and prevent atrophy
S	Safe, pain-free return to previous level of activity
T	Time for bone maturity to catch up with increased remodeling

Figure 6. R.E.S.T. acronym for the goals of stress fracture management.

MANAGEMENT

Prompt identification of an abnormal reaction to stress, such as a stress fracture, is essential. Once diagnosed, the injury can be managed with a cyclic management protocol based on the physiology of bone remodeling and a strategy for prevention.

Diagnosis

Prompt diagnosis of stress fractures is important, as continuing the aggravating activity may delay management and increase morbidity.[72] Very often, symptoms resembling those of a stress fracture are actually due to advanced bone remodeling resulting from the bone's reaction to stress. This stress reaction may only be a point along the continuum of remodeling before the development of a true stress fracture. The clinician often intervenes at this stage of the continuum to prevent the progression of the injury to a true stress fracture. In patients with a true stress fracture, prompt intervention is important to minimize the risk of a displaced fracture.[20,72] This intervention may include casting, splinting, or surgical fixation.[73]

Diagnosing stress fractures can be difficult as their symptoms are comparable with other injuries. Common diagnostic techniques include clinical examination,[19,20,58,69] x-ray films,[17,69,74] bone scan,[18,72,75] magnetic resonance imaging,[60,76–78] and ultrasound.[79–82] Differential diagnoses include shin splints,[83,84] osteomyelitis,[71] compartment syndrome,[55] and tumor.[16,71,84,85]

Management

Management begins immediately after an abnormal reaction to stress or a stress fracture is suspected. Since an x-ray film may not be positive for 10–21 days after the onset of symptoms, a delay in intervention may allow the accelerated remodeling to progress to a true stress fracture, thus risking a full fracture of the bone. The first priority is a period of rest from the stress or activity that is causing the symptoms. Zelko and DePalma[20] described the rest as ''active,'' allowing the athlete to exercise in a pain-free manner and prevent muscle atrophy.[20] Pain should be used as a guideline to treatment intensity, as pain during an activity may indicate exacerbation at the injury site. The goals during active rest are described by the acronym R.E.S.T (Figure 6).

Management of a stress reaction or stress fracture should include a 3-phase process that takes advantage of the physiologic healing process of the bone. Phase I should allow time for the maturing of the periosteum, healing of damaged blood vessels to prevent ischemic injury to bone, and maturing of osteocytes.[20,86] Phase II should include general conditioning and strengthening specific to the injured extremity. Functional weight bearing in phase III should allow for gradual remodeling of the bone and a return to the original level of activity. This 3-phase process differs from other 2-phase protocols that call for a removal of the stress and a gradual increase in activity.[7,17–19,58,69,87] In the 3-phase protocol, gradually increased stress in phase III is alternated with periods of rest to let new osteocytes and periosteum mature during periods of remodeling, when the bone is weakest (Table 2).

Several factors affect the management progression. The location, type, and age of the lesion make some exercises easier than others. It is important that the patient progress on the basis of symptoms and physiology rather than on a predetermined schedule. The exercises described within the 3 phases are not exclusive from one phase to the next. Instead, they are expected to overlap and serve as a guideline for the management progression. Because the clinician is often intervening before a true stress fracture develops, the condition that is being treated is usually a stress reaction. This term will be used throughout the discussion of the management.

Phase I. Phase I of the management process focuses on removing the stress from the injured area, controlling pain, and preventing deconditioning. It is during this phase that the haversian canals are forming, the osteoblasts are laying down new cells, and the periosteum is maturing to buttress the weakened area of bone.[50,52] This phase usually lasts for 1 to 3 weeks or until acute symptoms no longer occur with normal activities. Casting may be indicated when the physically active individual cannot or will not avoid the antagonistic stressor or a true stress fracture is present. However, casting should not be used regularly as it may contribute to a further weakening of the bone and deconditioning of the surrounding soft tissue. Crutch walking is a preferable alternative to casting, as it allows for nonstressful exercise and weight bearing. The use of pneumatic splints may reduce abnormal tibial loading, provide

Table 2. Bone Remodeling Activity and Rehabilitation Goals Based Within Each Phase of Cyclic Rehabilitation Protocol

Phase	Days	Remodeling Activity	Goals of Rehabilitation
I	1–10	Haversian canal formation	Control inflammation, modify or remove abnormal stress, maintain cardiovascular fitness
II	11–24	Periostitis, osteocyte maturation	Begin ADLs* pain free, transition to functional rehabilitation, maintain cardiovascular fitness
III Functional	1–14	Haversian canal formation	Allow stress to facilitate normal bone remodeling, increase activity level
III Rest	15–21	Periostitis, osteocyte maturation	Allow healing and osteocyte maturity during ''weak 3rd week'' of bone remodeling

*ADLs indicates activities of daily living.

support around the fracture site, and reduce the length of the rehabilitation process.[88,89] If poor foot alignments are present, orthotics should be instituted at this juncture to correct them.[20,69,90]

A typical phase I protocol for an involved lower extremity should include daily ice massages or contrast baths to decrease swelling. Transcutaneous electric stimulation (TENS) and high-volt electric stimulation (HVES) are also excellent modalities for reducing swelling and pain and may be augmented by nonsteroidal anti-inflammatory medications.[20,58,69] These modalities may be especially useful in light of new findings regarding the potential role of inflammation in an ischemic mechanism of stress reactions. Further research is needed to determine the efficacy of anti-inflammatory modalities, including ultrasound, electric stimulation, and ice, in decreasing the inflammation that accompanies bone remodeling.

Ambulation should progress from crutch walking to full weight bearing as soon as it can be tolerated without pain. Conditioning of the involved lower extremity begins daily with towel toe curls, ankle isometrics, and sitting range of motion on a wobble board.[20,58,69] As long as the patient remains free of pain, exercises can be progressed by adding weight to the towel curls and allowing active-range strengthening with rubber tubing. Strength training for the upper extremity and well-leg conditioning should continue 3 times a week while cardiovascular fitness can be maintained by using the upper body ergometer or stationary bicycle or treading water in the deep tank of the pool.

Phase II. Phase II of the management program begins when phase I exercise or ADLs can be performed without inflammation or symptoms. In many cases, pain is an indication of overload to the bone,[16,60] but this is not always the case.[3,79] As a result, patients must be instructed to keep their activity within a pain-free intensity and report any recurrence of pain to their therapist. Caution in using modalities must be exercised in this stage, as they can mask the pain that signals a potentially harmful stress to the injured area. Ice is continued, but ice, TENS, and HVES should be used only after exercise to avoid masking any pain the treatments might be causing.

Pool training that progresses from treading water in the deep tank to jogging in chest-deep water should be added to the swimming workouts. Wobble-board exercises should begin to include weight bearing and balancing, and rubber tubing exercises should progress to bilateral- and eventually single-leg toe raises. Pain-free walking during ADLs must continue (otherwise the patient should return to phase I), and the patient should eventually walk without pain for 30 consecutive minutes, 3 times a week.

Phase III. After 2 weeks of pain-free exercise in phase II, the running and functional activities of phase III are introduced. The efficacy of a cyclic training program to prevent stress fractures in military recruits has been documented.[22] By limiting the number of repetitive, high skeletal stresses in the first 2 weeks of basic training and modifying activity in the third week to exclude running, jumping, and double-time exercises, the fracture rate was significantly reduced from 4.8% to 1.6%. Scully and Besterman[22] hypothesized that the initial 2 weeks of training promoted the formation of osteonized new bone, whereas rest in the third week allowed for the formation of periosteal new bone. In the same way that Scully and Besterman[22] used a cyclic training process to strengthen bone and prevent stress fractures, Zelko and DePalma[20] described a cyclic management strategy to facilitate normal bone remodeling in preparation for the person's return to activity after a stress fracture.

Phase III of the management process depends on the physically active person's completion of the activities in a pain-free manner. The patient must be asymptomatic in the previous phases of treatment and cleared by the physician before initiating this functional phase of the program. Running and functional activity start out slowly and should be based on the individual's goals for return to function. A good guideline is to increase activity no more than 15% to 20% per week. A ''walk-jog'' in which the injured person jogs the straightaways and walks the curves of a track for 0.80 km (0.5 mile), followed by a day of rest, is a good starting point for a person who hopes to return to a running, field, or court sport. Once that distance is completed without pain, the injured person can begin walk-jogs 3 times per week. Distance is added in 0.80-km (0.5-mile) increments per week until the athlete can complete 3.22 km (2 miles). At this point, jogging begins for 1.61 km (1 mile) and increases by 0.80 km (0.5 mile) per week until 4.83 km (3 miles) or a goal distance commensurate with the person's activity is reached. During the functional phase of the program, the athlete continues the phase II exercises and progresses to mobility and jumping activities in the pool and on land. Once the athlete can squat 1½ times body weight, higher-level plyometric training may begin. The pool is an excellent trainer for jumping and cutting. These and all functional activities should be implemented in the pool before their initiation on dry land. This progression enables the remodeling bone to begin adapting to the stresses of jumping and cutting in a less stressful environment (Figure 7).

An important point for clinicians is that not all athletes will be able to begin their functional progression with running. Some may need to start with a 0.80-km (0.5-mile) walk-jog, and others may be able to move more quickly. The key point is that pain is the only guide that the athletic trainer and injured person have, and it should be used as a guide to all activity.

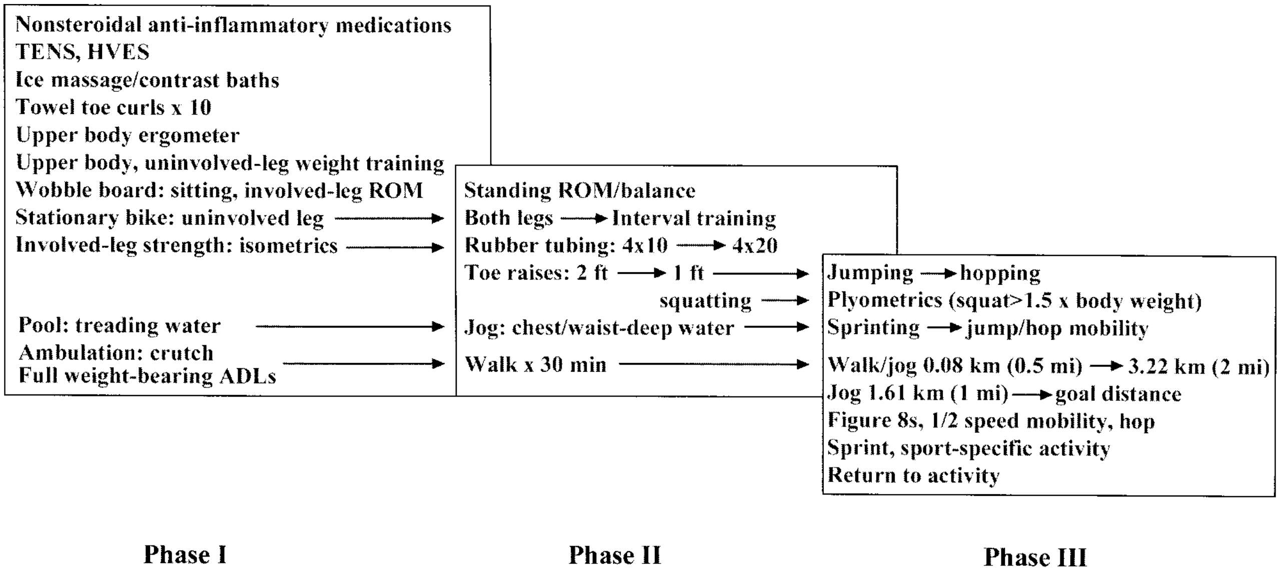

Figure 7. An example of a 3-phase progression of stress fracture rehabilitation. Activities between phases I and II and between phases II and III overlap to form a continuum of exercise and functional return to activity. TENS indicates transcutaneous electric stimulation; HVES, high-voltage electric stimulation; ROM, range of motion; ADLs, activities of daily living.

The running portion of phase III is completed in a cyclic fashion that mimics bone growth. As bone is being resorbed in the first 2 weeks of activity, running is encouraged to promote the formation of trabecular channels (functional phase). In the third week, when the newly formed osteocytes and periosteum are maturing, running activity is decreased (rest phase). During the first cycle of phase III, functional activity is reduced to the phase II level. In each successive cycle, the activity intensity in the rest phase is reduced to the functional level of the previous cycle. The cycle of 2 weeks on, 1 week off continues through the duration of the rehabilitation process, usually from 3 to 6 weeks. As the running program progresses to sprinting and sport-specific activities, the rest days between functional activities decrease, and the athlete is gradually prepared for the return to competition (Figure 8).

The injured person may note an increase in pain during the management process. If the increase in pain occurs during phase I or phase II, the offending activity should be discontinued or modified. Those who notice pain during ADLs or treatment should not be progressed to the next phase of the protocol until the activity can be completed pain free. During phase III, pain is usually an indication that the level of activity is too high, and functional activity should resume at the last level that was completed pain free within that 3-week cycle. If pain persists even at a reduced level, the activity intensity should be scaled back to the level from the previous 3-week cycle. Individuals who have persistent pain should be referred back to their physician. In these cases, resuming treatment at the phase I or phase II level may be indicated.

Compliance with the management program is critical for a timely return to activity. This is most difficult during the rest phase of phase III. Because the treated person has been predominantly pain free up to this point, stopping a pain-free functional activity is difficult to accept. Satterfield et al[91] went so far as to recommend referring patients to behavior-modification specialists in some cases. In any event, the rehabilitation of a stress fracture is a team effort involving the injured person, coach, physician, athletic trainer, and sport psychologist. Only by working together can the proper diagnosis, goal setting, education, rehabilitation, and successful return to sport be accomplished.

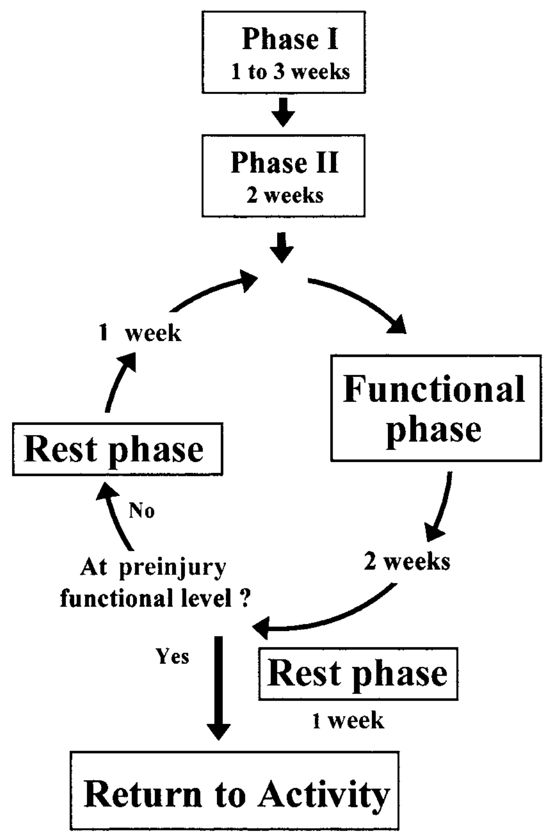

Figure 8. Three-phase cyclic functional model for stress fracture rehabilitation. Phase III includes a 2-week functional phase followed by a 1-week rest phase.

Prevention

Awareness of the causes of stress fractures can lead to appropriate preventive interventions. Bone is the weakest in the third week after the initiation of a stressful activity. By altering training intensity during the third week of workouts,[22] osteoblastic filling of absorptive areas and bone maturity can occur. For example, a change from plyometrics to a lower-impact aerobic activity during the third week of practice may reduce the stressors associated with stress fractures. In a military population participating in basic training exercises, the incidence of stress fracture in a cyclic training group was reduced to one third that of a noncyclic training group.[22] Another effective strategy in prevention is identifying and minimizing changes in shoes or surfaces. Limiting activity to one playing surface or pair of shoes can reduce the likelihood of the surface and shoes becoming stressors and contributing to the formation of a stress reaction or ultimately a stress fracture.[20,90]

CONCLUSIONS

Stress fractures can occur to just about any bone in a physically active person. They are at the endpoint of a continuum of a bone's reaction to stress that ranges from early remodeling to a cortical fracture. Normal levels of stress facilitate normal bone remodeling. When activity levels change or increase, the level of bone remodeling also increases. A gradual decrease in bone density follows this higher level of remodeling and places the bone at risk for a stress fracture. Stress fracture risk may be highest during the third week after the onset of the new or increased activity. Proper management of stress fractures should begin immediately. A 3-phase management process has been described based on the physiology of bone remodeling. It is important for the athlete, coach, and athletic therapist to understand the causes and cyclic formation of bone remodeling and management strategies for stress reactions and true stress fractures so that the physically active person can return to competition quickly and safely.

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