Chapter 11

COMPREHENSIVE EVALUATION AND MANAGEMENT OF STRESS FRACTURES IN MILITARY TRAINEES

MICHAEL D. ROSENTHAL, PT, DSC^{*}; AND DANNY J. McMILLIAN, PT, DSC[†]

INTRODUCTION

ANATOMY

Functional Anatomy Pathogenesis and Recovery Incidence and Etiology Risk Factors Prediction and Prevention

CLINICAL EVALUATION AND MANAGEMENT Prescreening History Physical Examination Plain-Film Radiographs Bone Scintigraphy (Scan) Magnetic Resonance Imaging Computed Tomography Scanning Laboratory Testing

TREATMENT

Role of Recovery Physical Training Phased Approach to Rehabilitation Use of Pharmaceuticals Bracing Casting Surgery Supplemental Modalities Ice and Heat Nutritional Supplementation Anatomical Considerations Failure of Conservative Measures

SUMMARY

*Lieutenant Commander, Medical Service Corps, US Navy; Naval Special Warfare Center, San Diego, California 92155 [†]Major, Medical Specialist Corps, US Army; Mannheim Health Clinic, Unit 29920, APO AE 09086

INTRODUCTION

Overuse musculoskeletal injuries are responsible for the greatest loss of training time in military trainees.^{1,2} Included in the wide spectrum of overuse injuries are stress fractures, which also prevent completion of military training. Although stress fractures are most commonly noted in the lower extremities, these injuries also occur in the upper extremities, ribs, spine, and pelvis.

The earliest report of stress fractures is attributed to Dr Breithaupt, a Prussian army physician, who in 1855 described significant dorsal foot swelling and pain in soldiers as a result of prolonged marching.³ Using conventional radiography,⁴ these signs and symptoms were first reported as stress fractures in 1897.

Clinical evaluation of stress fractures includes a thorough history, a physical examination, and imaging studies. The recent advancement of musculoskeletal imaging techniques has aided the evaluation and diagnosis of stress-related injuries. Standard management of stress fractures consists of pain-free crutch ambulation for lower extremity or pelvic stress fractures followed by a gradual return to weight-bearing activity culminating in running. Although this long-standing, conservative treatment regimen suffices for military personnel or athletes, it does not optimize the recovery period for reentry to training and competition. Without addressing potential contributory factors for the stress fracture injury, it is not unusual for the recruit to incur a subsequent stress fracture.

The incidence of stress fractures is high in military training programs, basic training posts,⁵ military training academies,^{6,7} and specialty training units.^{8,9} Given the military-wide directive to achieve a fit force and given the athletic nature of many active duty members, the possibility of stress fractures continues to exist after completion of entry-level training and should be considered when evaluating patients with exercise-related musculoskeletal complaints.

This chapter provides a framework for comprehensive management of stress-related musculoskeletal injuries. Although accurate diagnosis and rest periods normally result in recovery from stress fractures, this course of management does little to prevent recurrence or to advance the physical condition of an individual. Thus, treatment programs must be available for those patients who push the boundaries of prevention and recuperation to ensure that there are ample opportunities for optimal recovery. See also Appendix 1, Bone Scan Imaging of Stress Injuries in the Recruit.

ANATOMY

Bone is composed of dense outer cortical bone surrounding a spongy inner core, called cancellous bone. Although it has been recognized for years that bone responds to the stress placed on it, research has shown that bone also responds to the type of stress it encounters.¹⁰⁻¹⁶

Functional Anatomy

Bone is structured to absorb compression forces. During functional activity, however, physical stresses are produced simultaneously in multiple planes. Therefore, tension, compression, and shearing stresses are applied to the bone in physical training activities.

Stress placed on bone causes changes in cellular activity. Bone responds optimally to intermittent stresses. Research indicates that a few cycles of short duration loading is adequate for maintenance of bone strength. Desensitization of bone cells occurs in the presence of sustained loading.¹⁶ Sustained stressing (prolonged loading) of bone causes reduced mechanosensitivity, reduced cellular activity, and impaired healing.¹⁷ Complete removal of bone stress results in a decrease in bone strength.¹⁰

Dynamic intermittent loading produces fluid activity in the medullary canal that stimulates cellular activity and facilitates bony adaptation (strengthens bones).^{14,16}High-impact exercises cause a greater push of fluids through the bony architecture. Hsieh and Turner¹⁴ reported that increasing the frequency of loading also increases the osteogenic effect and reduces the magnitude of strain necessary to enhance osteogenesis. Review of these methods in humans has been limited to retrospective studies. Milgrom et al¹³ reported that military trainees accustomed to playing basketball for a 2-year period before commencing military training were significantly less likely to sustain a stress fractures than individuals accustomed to running. Conversely, prolonged static loading of bone impedes osteoblastic activity and is more likely to produce osteoclastic activity, which increases the risk of stress fractures.

Bone fatigue occurs in response to training load. Training load is determined by volume, duration, and intensity of training, as well as by the method of loading. Walking imparts a load of 1.5 to 2.75 times the body weight to the lower extremity, running conveys a load of 3 to 5 times the body weight, and jumping produces a load up to 10 times the body weight. The ability of muscle to shield bones and joints from these stresses is based on the conditioning level of the individual (eg, fatigability and strength).

Muscular forces on bones also play an important role in both protection from and development of stress fractures. Research indicates that muscle fatigue results in a dramatic increase in stress absorbed by the bony architecture.¹⁸ Although muscle fatigue, as described in the literature, was related to running duration (eg, after a 2-mile run), muscle fatigue in military trainees spans a wide range of activities based on individual conditioning levels. Muscle fatigue results in an increase in strain rate, strain magnitude, and strain distribution. A 20% to 35% increase in bone strain has been reported with muscle fatigue following as little as a 2-mile run. Although well-designed studies on the effect of lower extremity strength training and stress fracture incidence are needed, studies have reported that increased calf girth was associated with reduced stress fracture occurrence.^{19,20}

Pathogenesis and Recovery

In general, stress fractures occur as a result of abnormal stress to normal bone (typical athlete stress fracture) or normal stress to abnormal bone (from osteoporosis). Although abnormal stress to normal bone incorporates most physical training-related stress fractures, the precise mechanism is a source of debate.²¹ Stress fractures occur as a result of microdamage from the physical stresses on the bone, as well as remodeling, which is a natural adaptation to these stresses. Physical stress to the bone is caused by tensile stress from the attachment of muscles or more direct stress, compression, and torsional strain, resulting from impaired muscle function (fatigue) and the inability of the muscles to effectively attenuate bone stress. Microdamage is produced by high strain rates, increased loading frequency, and high stress magnitude. Lower bone density, increasing age, and impaired remodeling further amplify the effects of microdamage. Remodeling, although generally a protective mechanism to strengthen bone, also contributes to development of a stress fracture. Accelerated remodeling further weakens bones. Genetic, hormonal, and dietary factors affect remodeling.

Stress fractures can heal by two methods: (1) remodeling and (2) adaptation.²² Bone remodeling results from organized cellular units, called basic multicellular units, that detect and remove microcracks. Adaptation (or strengthening of bony structure) occurs from bone deposition. Remodeling is the primary means of early recovery, because the adaptation process occurs more slowly and takes longer than the typical healing time frame for return to activity after a stress fracture.²³ Dynamic loading incites a greater osteogenic effect than static loading.¹²

Incidence and Etiology

Numerous studies have described the incidence of stress fractures in military trainees. Most US studies show that 1% to 9% of military trainees will sustain a stress fracture.^{5,8,24} Reports on the Israeli Defense Forces place incidence rates as high as 31%. The high degree of variability in these studies is attributed to surveillance of these injuries. Most US studies are based on passive surveillance, in which diagnosis of a stress fracture relies on individuals seeking medical care for the related injury. The Israeli Defense Forces used active surveillance and bone scintigraphy for diagnosing stress-related injuries.^{25,26}

Risk Factors

The relationship of various contributing factors to the incidence of stress fractures has been widely studied. Multiple variables have been reported as affecting the onset of bone-related stress injuries. The variables are most commonly divided into intrinsic risk factors and extrinsic risk factors. Intrinsic risk factors are variables commonly associated with morphological measurements, some of which can be affected by training (Exhibit 11-1). Examples of intrinsic risk factors include gender, age, ethnicity, body mass index, bone characteristics, muscle strength, pretraining fitness level, lower extremity morphology, nutritional factors, and genetics. Extrinsic risk factors, although less numerous, are commonly derived from external forces. They are modifiable by the awareness and education of individuals involved with the training programs. Examples of extrinsic risk factors include training errors, exercise / training surfaces, training footwear, and prediction and prevention.

Intrinsic Risk Factors

Gender. Multiple studies have indicated that female recruits are at greater risk for stress fractures than their male counterparts.^{67,27} Special consideration should be given to females presenting with stress fractures, including clinical evaluation of the female athlete triad (amenorrhea, disordered eating, and osteoporosis).^{28,29} Disordered eating extends from caloric restriction and food group avoidance to the more severe eating disorders of anorexia nervosa and bulimia. Thorough discussion of the female athlete triad is beyond the scope of this chapter and review of the 1997 position

EXHIBIT 11-1

INTRINSIC AND EXTRINSIC RISK FACTORS IN THE CAUSATION OF STRESS FRACTURES

Intrinsic Risk Factors

- Gender
- Age
- Ethnicity
- Body mass index
- Bone characteristics
- Muscle strength
- Pretraining fitness level
- Lower extremity morphology
- Nutrition factors
- Genetics
- Menstrual dysfunctions
- Muscle fatigue
- Flexibility
- · Previous injury and inadequate rehabilitation

Extrinsic Risk Factors

- Training errors
- Training surfaces
- Worn-out/inappropriate footwear
- Excessive training intensity
- Environment

statement published by the American College of Sports Medicine is recommended.²⁹ Late-onset menarche has also been associated with an increased likelihood of developing stress fractures.¹⁹ Although oral contraceptive pills may help maintain bone mineral density and reduce the risk of stress fracture development,³⁰⁻³² other studies suggest no benefit,^{19,33} or potentially negative effects.^{34,35}

Increasing age is associated with greater risk for stress fractures; however, published reports vary. Although the majority of these studies indicated that increasing age results in greater occurrence of stress fractures,^{36,37} Milgrom et al³⁸ reported an inverse association.

Ethnicity. Differences in fracture risk between racial and ethnic groups have been noted. White recruits have shown an increased incidence of stress fractures in comparison with African American, Hispanic, and Ethiopian recruits.³⁶⁻³⁸

Body Mass Index. Body mass index has been both directly and inversely associated with stress fracture rates.³⁹ Discrepancies in the literature occur in part because of the operational definition of body mass

index and its application. In studies in which a high body mass index has been linked with an increased risk for stress fractures it is tied to poor physical conditioning.²⁷ In contrast, Drinkwater et al⁴⁰ reported that weight gain—and a resultant increase in body mass index—increases bone mineral density and resumption of menses.

Bone Characteristics. Investigators have determined that bone structure indicates a predisposition to stress fractures. Osteoporosis (low bone mineral density) is commonly associated with a high risk for stress fractures. Typical assessment of this condition uses dual-energy x-ray absorptiometry scan testing. Turner and Robling¹⁶ found that small changes in bone mass, which are structurally significant, might not be detected by the dual-energy x-ray absorptiometry scan. Utility of this test in assessing stress fracture risk in a military training population is questionable because most young, active individuals have scores within acceptable age group norms. Other measurable bone characteristics include bone mineral content, bone width, medullary canal width, cortical bone thickness, and bone geometry.

Bone mineral density, bone mineral content, and bone shape and size have all been studied in response to exercise, ultimate bone strength, and loading. In long bones (eg, tibia, femur), strength is proportional to the size (width) of the bone.⁴¹ Giladi et al^{41,42} found that narrow tibial bone width was associated with a greater risk for both tibial and femoral stress fractures. Conversely, Bennell et al⁴³ did not find an association between tibial bone width and tibial stress fractures in female athletes.

Individuals with lower bone mineral density have an increased incidence of stress fractures.¹⁹ Conflicting reports did not identify a relationship between lower bone mineral density and stress fractures.⁴³ Bone mineral density and bone mineral content have increased in response to loading (exercise).⁴⁴ Robling et al¹⁵ reported that, in rats, small increases in bone mineral density and bone mineral content have a dramatic effect on bone strength. The influence of prolonged loading duration had a detrimental effect on bone adaptation and bone mineral density.¹²

Bone geometry (shape and size) is an accurate indicator of bone strength.¹⁶ Periosteal bone width is also a key indicator of ultimate bone strength.¹⁶ However, periosteal bone growth occurs primarily in childhood and adolescence. Although numerous studies have suggested some value in each of these measurements, conflicting reports indicate that further research is warranted.

Muscle Strength. Skeletal muscle strength and mass have been associated positively with bone mineral

density. Skeletal muscle activity has an osteogenic role. Bennell et al¹⁹ described that women with less lean mass in the lower leg and smaller calf girth were more likely to incur stress fractures. In male military trainees,⁴⁵ poor lower body strength was linked with an increased risk of lower extremity stress fractures. Muscle fatigue results in increased stress on skeletal structures. Fatigue of the anterior tibialis and triceps surae muscles (gastrocnemius and soleus) dramatically increases calcaneal and metatarsal stresses.⁴⁶

Pretraining Fitness Level. Although most individuals believe that a higher level of physical fitness before initiation of military training is protective for stress fractures, the available research in this area is less definitive. Consideration should be given to the type of exercise used before military induction rather than to the aerobic fitness level. For example, Milgrom et al¹³ noted that trainees who swam had an increased stress fracture risk versus trainees who played basketball, who had a decreased stress fracture risk. Multiple studies reported no correlation between level of prior participation in sports or aerobic fitness levels and the occurrence of stress fractures.^{41,47} Although Hoffman et al⁴⁵ found no statistically significant difference between stress fracture incidence and prior physical activity levels, the highest incidence of stress fracture was reported in those who did not participate in a conditioning program before military training. Shwayhat et al⁹ noted a significant increase in lower extremity overuse injuries in Navy SEAL (Sea, Air, and Land) recruits who ran fewer miles and shorter duration runs before the formal training period. Similar findings have been found in other studies.⁷ Physical training (for a period of 1–2 years before military training)-which induces greater shear, tension, and compression stresses than standard running—reduces the incidence of stress fractures.¹³

Lower Extremity Morphology. Various lower extremity morphological measurements have been reported in relation to lower extremity overuse injuries. Available research provides conflicting accounts of most variables; therefore, caution is recommended when interpreting individual measurements as key factors in the development of stress fractures.

Pes cavus (high arch) and pes planus (flatfoot) have both been reported to predispose individuals to lower extremity stress fractures.⁸ Pes planus has been linked with an increased risk for tibial and tarsal bone stress fractures.⁴⁸ Individuals with cavus feet have an increased association with metatarsal stress fractures.⁴⁸

Genu valgum (knock-knees) and genu varum (bowlegs) are associated with an increased the risk for incurring a stress fracture. Cowan et al⁴⁹ found that, in US Army basic infantry trainees, those trainees with the most valgus knees were more likely to sustain an overuse injury of the lower extremity. A wider pelvis and genu valgum in women have also been linked with a greater incidence of stress fracture.⁵⁰

Unequal leg length has been associated with an increased risk for lower extremity overuse injuries.¹⁹ However, other research has not implicated leg-length differences with lower extremity injuries.⁴⁹ External rotation of the hip was reported by Giladi et al^{41,51} as being predictive of increased risk of lower extremity stress fractures in Israeli soldiers. Conversely, Bennell et al¹⁹ did not find a similar association in track and field athletes with lower extremity stress fractures.

Nutritional Factors. Adequate caloric intake to meet the demands of physical training is necessary to maintain and increase bone mineral density.³⁹ Short-term energy restriction (eg, restricted caloric intake) during periods of physical activity has been shown to impair collagen synthesis and reduce the availability of insulin-like growth factor 1, which aids in bone formation.⁵² Armstrong et al⁵³ reported that weight loss in military trainees during periods of daily physical training was associated with a greater risk for developing stress-fracture injury.

Deficient dietary intake, in particular calcium deficiency, makes trainees more susceptible to stress fractures.⁵⁴ Large amounts of calcium are lost in sweat during intense exercise, and supplementation with calcium citrate and vitamin D increases bone mineral content in athletes.⁵⁵ Myburgh et al⁵⁶ found that dietary calcium intake at levels below the recommended daily allowance was associated with stress fracture occurrence. A recent report on female military trainees did not find a difference in dietary calcium intake between the stress-fracture and the non-stress-fracture groups.⁵⁷ Consumption of high levels of sodium, phosphorus (soft drinks), fiber, protein, caffeine, and alcohol negatively impact calcium balance. However, Bennell et al¹⁹ did not find an association between dietary intake of those substances and the incurrence of stress fractures. Because calcium is a threshold nutrient (ie, nutritional intakes above a certain level do not result in further benefit to bone), supplementation is unlikely to influence stress fracture incidence in patients whose dietary intake meets the recommended daily allowance.58 Consideration should be given to Matkovic's⁵⁹ research proposing that the recommended daily allowance for calcium be increased to promote adequate calcium intake for skeletally maturing individuals. Consideration of vitamin D supplementation was recommended by Givon et al.⁶⁰ Nattiv and Armsey⁶¹ noted the potential complications of hypercalcemia and hypercalciuria. Trainees should be made aware of this data and limit intake of the foods and nutrients that might adversely affect calcium balance.

Genetics. The exact role that genetic makeup plays in the incidence of stress fractures remains to be investigated. Givon et al⁶⁰ reported that a high percentage of trainees with stress fractures had a first-generation relative who had also sustained a stress fracture. Genetic markers might be eventually proven to be an effective screening tool to identify trainees likely to incur a stress fracture. Nattiv and Armsey stated that "attainment of peak bone mass is largely determined by genetics, however a significant impact is made through optimizing lifestyle patterns."^{61(p215)} Although genetic testing is not used to restrict military training selection, such information might support specific pretraining intervention programs to reduce the incidence of stress fractures.

Extrinsic Risk Factors

Training Errors. Training errors are a frequent cause of stress fractures. They are typically associated with training volume that is increased too rapidly (eg, mileage, frequency) and hill running.^{5,48}

Exercise/Training Surfaces. Various theories exist regarding the role of training surfaces on stress fracture incidence.⁶¹ Training on hard surfaces increases the mechanical shock to the bone and potentially increases the incidence of stress fractures. Running on soft surfaces requires greater muscular activity, induces early muscle fatigue, and contributes to stress fractures.

Training Footwear. Although footwear is believed to contribute to stress fractures, the available research is equivocal.^{62,63} Because foot structure (eg, size of the first metatarsal, plantar arch), biomechanical factors, and stability vary greatly among military trainees, the common requirement that all trainees wear the same type of training shoe might be less than ideal. However, there are no studies to support this hypothesis.

Footwear modifications and insole use have gained widespread consideration. Milgrom et al⁶⁴ noted a reduction in stress fractures with the use of a shock-absorbing orthosis. In contrast, Gardner et al³⁷ found that the incorporation of an insole with good shock absorption prop-

CLINICAL EVALUATION AND MANAGEMENT

Prescreening

Although not commonly performed on a large scale in military trainees, prescreening to identify those susceptible to musculoskeletal injuries (including stress fractures) may lead to injury prevention. Areas for possible pretraining assessment include lower extremity biomechanics, bone mineral density, lower extremity strength, cardiovascular fitness, and erties did not reduce stress fracture incidence in military recruits. A recent Cochrane Database report suggests that insoles might reduce stress fracture rates.⁶⁵ However, the effectiveness of custom orthoses versus a generic over-thecounter product is yet to be clearly determined. The use of Zohar boots (manufactured in Tel Aviv, Israel) by the Israeli Defense Forces reduced tibial strain contributing to stress fractures.^{62,63} In US military training, running in boots is commonplace. Boot manufacturers, in response, have modified components to produce a boot that is more shock absorbent, lightweight, and running-shoe–like.

Prediction and Prevention

Optimal management of stress fractures centers on preventing injury occurrence. Consideration of all the intrinsic and extrinsic risk factors is invaluable in the evaluation and management of stress fractures in the training environment. A recent review of the stress fracture prevention literature emphasized "how little we know about what works to prevent one of the most common and potentially serious sports and exerciserelated overuse injuries."^{66(p243)}

Attempts to reduce the incidence of stress fractures have included modification of training programs, modification of footwear, and the use of insoles. Scully and Besterman⁶⁷ reported that altering the third week of training—by introducing nonrunning training activities—significantly reduced stress fracture rates in US Army basic trainees. This efficacy of 1-week periods of restricted running in the early phases of military training to reduce stress fracture incidence has been questioned in more recent literature.⁶⁸

Jump training (playing basketball) in the 1- to 2year period leading up to military training has been associated with a reduction in the incidence of stress fractures.¹³ Although jump training has proved beneficial, it is not practical to implement with an effective time line (1–2 years needed for bone adaptation) and can also result in secondary injuries (eg, joint and ligamentous sprains) that could negate the positive effects of bone strengthening.

footwear selection. Intrinsic and extrinsic risk factors should be addressed when performing a pretraining analysis of potential contributing factors to injury.

History

As with any musculoskeletal injury, a thorough history is essential in guiding the physical examination and using ancillary testing. In the clinical setting, it is not uncommon to focus on the location of symptoms and conservative management while overlooking potential causes of stress fractures. Key areas to be investigated include training history, menstrual pattern, prior history of overuse musculoskeletal injuries, dietary pattern (eg, pattern of caloric restriction), changes in training, changes in footwear, and pattern of symptoms (eg, after activity, during activity, or constant ache). Nonmusculoskeletal pathology (eg, tumors), which may masquerade as a stress fracture, should be considered when evaluating a patient who has not experienced improvement following reduced physical training. Patient report of night pain, night sweats, weight loss, local swelling, enlarged lymph nodes, or pain unchanged with mechanical stress (movement) should raise the clinician's level of suspicion for nonmusculoskeletal conditions.

Physical Examination

A physical examination should include a focused examination of the region of interest, as well as a comprehensive assessment to identify potential contributory factors. A thorough examination should include gait observation, joint range of motion of involved joints proximal and distal to the symptomatic region, strength assessment, palpation, and use of special tests.

Palpatory skills help localize the lesion to the bony surface or to adjacent soft-tissue components. Matheson et al⁴⁸ found that 66% of athletes with a positive bone scan had localized tenderness on palpation. Palpation of the symptomatic region should always be compared with the contralateral extremity, with particular awareness of applied pressure.

Use of special tests has been widely reported in the literature; however, sensitivity has not been unequivocally noted. The percussion test (percussing the heel with the palm of the hand) can potentially induce pain at the fracture site (eg, tibia).^{50,69,70} Utilization of the fulcrum test (applying a valgus or posterior-toanterior stress to long bones) has also been proven clinically useful. Various studies have recommended this test for the diagnosis of femoral shaft stress fractures.^{71,72} This test is also useful for the assessment of tibial and metatarsal shaft stress fractures. Application of a vibrating tuning fork can also aid in the process of differential diagnosis. Using a 128-Hz tuning fork, Lesho⁷³ reported 75% sensitivity and 67% specificity with the tuning fork test (which percusses the bone and elicits pain not reproduced with soft-tissue injury). However, given the negative likelihood ratio of 0.34, a negative tuning fork test is not adequate for ruling out a tibial stress fracture. Other researchers have found

less effectiveness using the tuning fork test. Several studies have suggested that applying ultrasound to the bone surface (eg, tibia) might also be beneficial in evaluating potential stress fractures.74-77 Conversely, other studies have reported that ultrasound examination is not a reliable tool in the diagnosis of tibial stress fractures.^{78,79}A hop test (single leg hopping) has also been indicated as a useful test for tibial and femoral stress fractures;^{70,80,81} however, the hop test is not recommended as a diagnostic tool because excessive weight bearing can cause an incomplete stress fracture to become complete. Spinal and hip extensions, which increase loading of the pars interarticularis region of the lumbar spine, have been suggested for evaluating potential pars stress fractures.^{82,83} Clinicians should always perform these special tests on both extremities for comparison. Awareness of the applied force should also be considered when comparing involved and uninvolved extremities.

Plain-Film Radiographs

When clinical suspicion of a stress fracture exists, the first line of defense of ancillary testing is the plainfilm radiograph. Radiographic changes might not be evident for a least 2 to 3 weeks after the onset of the patients' symptoms.⁸³ Sensitivity of radiographs is low.^{80,84} Radiographs should be assessed for evidence of periosteal reaction, fracture lines, endosteal callous, or focal sclerosis. Typically, periosteal reaction (Figure 11-1) is the first plain-film radiographic sign of a stress fracture.⁸⁵ Although sensitivity of radiographic findings in patients with suspected stress fractures does improve with time, two thirds of standard radiographs are negative in the early phases, and only one half of the patients developed radiographic evidence of a stress fracture.^{83,86-88} Although not very sensitive, plainfilm radiographs provide a high degree of specificity for diagnosing stress fractures.^{87,89} Despite the low yield for early plain-film radiographs, this remains the usual first line of imaging in military trainees. Often, trainees are unable to recall the duration of symptoms. Thus, the use of plain-film radiographs is warranted because actual bone stress and subclinical symptoms may be of many weeks' duration.

Bone Scintigraphy (Scan)

The long-standing standard for imaging of stress fractures has been the technetium bone scan. This test might be positive (Figure 11-2) as early as 2 to 3 days after onset of a patient's symptoms.^{53,90} Although grading systems (Table 11-1) to define the severity of bone stress injuries have been studied by various authors,



Fig. 11-1. Periosteal reaction at the distal medial tibial diaphysis.

their usefulness in directing clinical management is not widely accepted.⁹¹⁻⁹⁴ Bone scans provide a high degree of sensitivity for an underlying bony lesion with less specificity than MRI for defining the severity of the lesion. When faced with a positive bone scan, consideration should also be given to other conditions, such as osteoid osteoma, infection, and malignancy; thus, plain-film radiographs are recommended as well.⁹⁵A triple-phase bone scan provides more specific information on the presence of a well-localized stress fracture and reduces the likelihood of a false-negative result.⁸⁷ The three phases are the angiogram, the blood pool, and the delayed (bone) image.⁸⁹ In the case of a stress fracture, all three phases are positive; whereas, in the case of shin-splints, only the delayed image phase is positive.⁸⁵ Increased uptake from non-stress-fracture entities (eg, shin-splints, iliotibial band syndrome) is from a disruption of Sharpey's fibers.^{85,96} Application of ice to a symptomatic area before undergoing a bone scan should be avoided because it might adversely affect test results.⁹⁷ It is not uncommon for a bone scan to expose additional asymptomatic areas of radionucleotide uptake, and this situation can pose a diagnostic dilemma. These areas of increased uptake indicate subclinical locations of bone remodeling and should be addressed if the provider has a high level of clinical suspicion. If patients are placed on crutches to

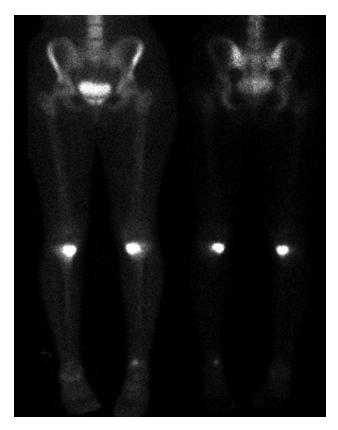


Fig. 11-2. Bone scan demonstrating increased uptake in the medial tibial plateaus, bilaterally.

promote early healing of the stress fracture, the contralateral lower extremity may become symptomatic in previously subclinical areas.

A negative bone scan does not always eliminate the possibility of a stress fracture. Milgrom et al⁹⁸ reported cases in which an initial bone scan was negative, but after continued symptoms and physical training, repeat bone scans 1 month later showed a stress fracture. Other studies have documented false-negative bone scans when evaluating femoral neck and sacral stress fractures.^{87,99,100}

In patients with suspected shin splints, positive bone scans are not uncommon.⁷⁰ This finding might represent a continuum of injury, and patients should be monitored carefully for worsening symptoms. In addition, positive bone scans are not uncommon in clinically asymptomatic patients.⁷⁰ The severity of findings has not been positively associated with recovery time in tibial stress fractures.¹⁰¹ Furthermore, the presence of bilateral tibial stress fractures has not been associated with prolonged recovery time, compared with trainees having unilateral involvement.¹⁰¹

Bone scans are not usually recommended for assessing fracture healing. However, some studies suggest

TAE	BLE	11	-1
-----	-----	----	----

GRADING OF S	FRESS FRACTURES
---------------------	------------------------

Grade	Radiograph	Bone Scan	MRI
Ι	Normal	Poorly defined area	Positive STIR im- age
II	Normal	More intense area	Positive STIR and positive T2- weighted
III	Discrete line	Sharper uptake area	Positive T1- and T2-weighted, but no definite corti- cal break
IV	Fracture	More intense, localized transcortical uptake area	Positive T1- and T2-weighted fracture lines

MRI: magnetic resonance imaging

STIR: short-tau inversion recovery

Data sources: (1) Arendt EA, Griffiths HJ. The use of MR imaging in the assessment and clinical management of stress reactions of bone in high-performance athletes. *Clin Sports Med.* 1997;16(2):291-306. (2) Fredericson M, Bergman AG, Hoffman KL, Dillingham MS. Tibial stress reaction in runners. Correlation of clinical symptoms and scintigraphy with a new magnetic resonance imaging grading system. *Am J Sports Med.* 1995;23(4):472-481.

their usefulness in this area.^{94,95,102} MRIs taken after falsepositive bone scans in endurance athletes with hip pain have revealed non–stress-fracture lesions of tendonitis, synovial pit, bone cyst, and avascular necrosis.¹⁰³

Magnetic Resonance Imaging

MRI is gradually supplanting bone scintigraphy as the preferred study for stress-related bone injuries.¹⁰³⁻¹⁰⁶ Greater specificity supplied by MRI provides more detail about the severity of these injuries. MRI grading scales of stress-related bone injuries have been reported by Fredericson et al⁶⁹ and Arendt and Griffiths.⁹² Although this information can assist rehabilitation specialists in designing return-to-activity programs, Arendt and Griffiths⁹² advocate caution. The use of MRI in suspected stress-related bone injuries is not without limitation. Early tumors (eg, myeloma), osteomyelitis, and bone bruises also produce stress-fracture–like findings, further amplifying the importance of a corroborating clinical examination.⁹²

MRI has been recommended for imaging of acute hip (Figure 11-3) and pelvis injuries in which immediate diagnosis is necessary (avoiding a 2- to 3-day lag time to prevent false-negative findings on bone scanning¹⁰³). In addition, the greater specificity provided by MRI in the evaluation of high-risk bone stress injuries is helpful in determining if surgical intervention is warranted. Although MRI is in high demand and not readily accessible at many military treatment facilities, the development of dedicated extremity scanners might increase availability. Further benefits include less time required for completion of the test and the absence of radiation exposure.

Some studies have indicated the potential for oversensitivity in MRI, however. Positive findings have been reported in asymptomatic patients.^{70,107} Dutton et al¹⁰¹ noted that in military trainees, there was poor correlation between degree of stress fracture severity on MRI and bone scan to clinical outcome. Individuals with grade I and II findings on scintigraphy were less likely to resume military training than those with grade III and IV stress fractures.¹⁰¹ However, Fredericson et al⁶⁹ noted a more consistent recovery period in direct correlation with severity of MRI findings.

Research comparing findings of the MRI examination with those of bone scans is conflicting. Batt et al⁷⁰ reported poor agreement between the grading systems used for bone scans and MRI. Contradicting those findings is research from Ishibashi et al⁸¹ who described correlation between the two grading systems and the degree of clinical symptoms.

Computed Tomography Scanning

Computed tomography scanning is not normally used in the assessment of stress fractures, but has been recommended as an adjunct in the assessment of

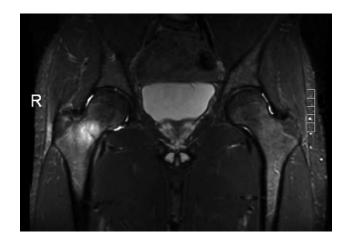


Fig. 11-3. Magnetic resonance imaging demonstrating bone edema and an incomplete stress fracture on the compression side of the right femoral neck. Anterior view. R: right

tarsal navicular injuries and known stress fractures.⁹² McFarland and Giangarra¹⁰⁸ recommended computed tomography to evaluate sacral stress fractures.

Laboratory Testing

Although serum analysis has been investigated in a few studies,^{60,109-111} this data is not incorporated rou-

TREATMENT

When clinical suspicion is high that stress fractures exist, the prudent course of treatment is cessation of the exacerbating activity until proven unnecessary by serial clinical examination and supporting imaging studies. Medical practitioners should be aware that the highly motivated patient might not fully grasp the importance of accurately reporting persistent or worsening symptoms. Unfortunately, some military trainees have pushed themselves to the point of developing a complete fracture.

A team approach to managing trainees with stress fractures reduces delays in return to training. Thorough education of patients, training instructors, and medical personnel regarding the stress injury is crucial to an uncomplicated return to activity.

Role of Recovery

Rest is essential to a timely recovery from stress fractures. Sleep deprivation, not uncommon in military training environments, might also contribute to the development of stress fractures.³⁰ Adequate rest periods between training cycles are also important to facilitate cellular activity, which promotes bone strengthening.¹¹²

The optimal rest period between training cycles depends on multiple variables (eg, muscle fatigability, training volume) and is difficult to control in a large group of military trainees with varying recovery needs. Robling et al,¹⁷ in a study using rats, reported that 8 hours of recovery time between loading activity was helpful in restoring bone mechanosensitivity and maximizing the effects of exercise on bone strength.

Physical Training

Although traditional management of stress fractures incorporates extended periods of nonweightbearing activity, the duration of rest varies. Return to full activity following stress fractures varies both in individuals with the same region of injury and between the various anatomical locations (eg, femur vs tarsal navicular).⁵³ It is difficult to predict the time course for return to full activity because of the interplay of previously described factors. Multiple studies have reported on the negative effects of complete non-weight-bearing activity.^{10,113} Therefore, we only recommend strict non-weight-bearing status for individuals with highrisk stress fractures. Continued loading of low-grade (per bone scan or MRI findings) stress injuries does not always progress to a more severe lesion⁹² and likely prevents bone strength reduction secondary to disuse.¹⁰ Arendt and Griffiths⁹² reported that grade I and II stress injuries can continue to heal, while still allowing graduating levels of physical activity. Burr et al¹¹⁴ described continued healing in an animal model, wherein cyclical stress was applied to the hind limbs of rabbits with stress fractures. Stress fractures in highrisk locations (ie, femoral neck, medial malleolus, talus, patella, great toe sesamoids, anterior tibia, base of the fifth metatarsal, and tarsal navicular) should be treated with extreme caution.¹¹⁵

tinely in clinical evaluation and management of stress

fractures. In soldiers with stress fractures, Givon et

al⁶⁰ found high levels of osteocalcin and bone-specific alkaline phosphatase, along with lower levels of 25-

hydroxyvitamin D. Further research on biochemical markers specific to individuals with stress fractures

may enable use of these tests as a clinical adjunct when

imaging studies are not available.

A phased approach to rehabilitation promotes a systematic progression of activities. Progression is based not on time, but on response to current exercises. This program uses a variety of methods for loading bone to incite shear, compression, and tension stresses. Rubin and Lanyon¹⁰ recommended a varied training program to produce a greater osteogenic response (eg, cross training vs emphasis on progression of running). Turner and Robling¹⁶ introduced the concept of an osteogenic index to measure the effectiveness of exercise programs. Subsequently, an equation was developed that allowed insight into the osteogenic potential of various exercise programs. Components of the equation included intensity of loading (peak load of the exercise being performed), volume (number of repetitions or loads on the bone during the training session), and time between sessions. This equation demonstrated that short, intense exercise sessionswith adequate rest between sessions—have a greater osteogenic index than lengthening the duration of training sessions.

Because the measure of success for most military trainees is a return to training, it is important to educate

patients about varying their exercise regimen (intensity and volume) and include adequate rest between training sessions. Sustained loading activities (eg, walking, running) without adequate rest intervals can impair healing by reducing the mechanosensitivity to loading.^{16,112} Clinicians must actively monitor the patient's response to each exercise session. Increasing levels of pain will necessitate a return to less stressful training. In addition to pain, clinicians should be aware of aberrant movement patterns that signal favoring of the injured segment.

Phased Approach to Rehabilitation

A three-phased approach to rehabilitation emphasizes a systematic progression of activities based on response to current exercises: (1) protection; (2) normalizing movement; and (3) return to running and jump skills training.

Phase I—Protection

Bone stress is minimized until weight-bearing becomes pain-free. To avoid atrophy of major muscle groups, resistance exercises should be performed. Bone stress of the injured segment should be minimal with early resistance training. Although crutches are often prescribed to offload the bone and reduce pain, complete non-weight-bearing crutch ambulation is not normally used and in some instances has been associated with prolonged recovery time.¹¹³ Although upper body and core resistance training are emphasized in phase I, lower body resistance exercises in the open kinetic chain (foot off the ground and no axial loading through the injured extremity) are usually well tolerated. If only one lower extremity is injured, single-leg stance exercises performed on the uninjured leg are indicated. From this single-leg stance, the injured lower extremity can perform open kinetic chain exercises that maintain pelvic stability (eg, resistance in all planes using a sports cord).

Although options for aerobic conditioning are limited in phase I, reduced weight-bearing options should be pursued to minimize deconditioning. Swimming, aqua jogging, and biking are the most common choices. Because other training options are limited during the protection phase, it is an ideal time to address flexibility concerns discovered during the evaluation.

Criteria to advance to phase II: Pain-free ambulation of 100 meters.



Fig. 11-4. Split squat.



Fig. 11-5. Single leg squat.

Phase II-Normalizing Movement

There are two primary goals of normalizing movement: (1) weight-bearing is systematically increased so that brief running is well tolerated; and (2) optimal, total body movement patterns are restored.

Weight-bearing activities include body-weight resistance exercises, such as squats, lunges, and aerobic conditioning. Weight-bearing resistance should begin with symmetrical weight distribution, and progress to staggered-stance activities (eg, split squats [Figure 11-4]) and end with single-leg stance exercises (eg, squat [Figure 11-5], step-down, multiplanar straight leg raises of the uninjured leg against sports cord resistance).

The weight-bearing associated with aerobic conditioning should be carefully monitored during this phase. Elliptical and step machines can be initiated at this time, with low intensity and duration. The non–weight-bearing modalities used in phase I can be continued at a greater intensity and for longer durations.

Restoration of optimal movement patterns should be a focus of training during this phase. The patient should be exposed to a variety of drills that challenge balance and coordination without exposing the injured segment to excessive stress. Examples of such drills include lateral shuffle (Figure 11-6) and crossover step (Figure 11-7) agility drills at a walking pace, single-leg stance catch/throw (eg, chest pass, overhead pass, rotational pass), warrior/sun-salutation poses from yoga (Figure 11-8), balance and reach (eg, stand on injured leg, then reach forward/backward/diagonally/laterally as far as possible with the uninjured leg [Figure 11-9]).

Criteria to advance to phase III: Pain-free running for 3 minutes and bilateral symmetry in measurements of balance and strength.

Phase III-Running and Jumping Progression

The goals of this final phase of rehabilitation are to ensure optimal attenuation of ground reaction forces during jumping and landing, and to instill proper body mechanics during powerful movements. Concurrently, running is systematically progressed toward the patient's end-stage running goal (Table 11-2).¹¹⁶ Because running requirements vary among recruit training environments, the clinician should assess the point at which the recruit meets a particular requirement. The decision to return to full duty must not be made solely on the recruit's ability to pass a fitness test. Take



Fig. 11-6. Lateral shuffle.



Fig. 11-7. Crossover step.

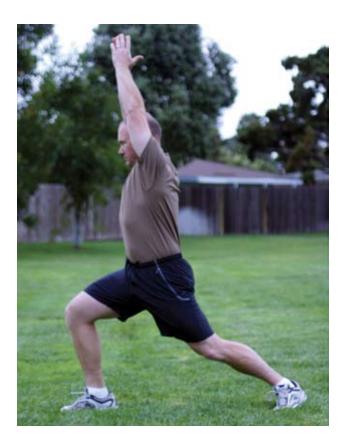




Fig. 11-8. Warrior pose.

Fig. 11-9. Balance and reach.

into consideration the cumulative physical demands on the injured bone of the trainee. Before returning to full activity, trainees should be able to train for 2 to 3 weeks at a level (ie, total amount of running each day and cumulative amount for the week) equal to what they will experience upon reentry to training.

Previous drills to restore optimal movement skills are progressed in this phase, with greater emphasis on agility. Drills such as the lateral shuffle and crossover step are performed at a quicker pace and change of direction is added. Balance and reach exercises from phase II might be replaced by lunges (Figure 11-10) performed in multiple directions (eg, forward, backward, diagonally, and laterally). Progressively increasing external resistance can be added to squatting and other strength development exercises.

Jump training should begin with mastery of jumping and landing mechanics. Good squatting technique is the basis for good jumping and landing skills. Ensure that, at the depth of the squat, the feet and knees are aligned in both the frontal and sagittal planes. The hips should be well to the rear, with the trunk straight but tilted forward for counterbalance. Once the basic squat technique is mastered, add speed to the squat repetitions to prepare for the explosiveness of jumping and landing. Begin jump training with relatively low-level drills such as submaximal vertical and broad jumps with double-leg landings. The patient should be encouraged to soften impact by landing from toe to heel while adequately bending at the hips and knees. The clinician should cue the patient to listen to the landing and encourage quiet repetitions. Count the number of jumps in a given training session in order to progress systematically.

If the initial jump training is well tolerated, progress to higher intensity jumps such as drop jumps (stepping off of a 20–25 cm platform and landing in the squat position). Gradually progress to drop jumps, followed immediately upon landing by a vertical or broad jump. Over several sessions, the height of the platform can be slowly and incrementally raised if the patient demonstrates sufficient strength as evidenced by good landing technique, and if the jumps are well tolerated. Further progression might incorporate single leg jumps and other drills that simulate the jumping and landing requirements of the recruit. Jump training should be used in combination with other conditioning modalities; therefore, consideration of the total

TABLE 11-2

THE WALK-TO-RUN PROGRAM*

Phase Activity

- I Walk 2 miles at your own pace
- II Progress to walking 2 miles in 35 minutes
- III Walk 1/4, Run 1/4, Walk 1/4, Run 1/4
- IV Walk 1/4, Run 1/4, Walk 1/4, Run 1/4, Walk 1/4, Run 1/4, Walk 1/4, Run 1/4
- V Walk 1/4, Run 1/2, Walk 1/4, Run 1/2, Walk 1/4, Run 1/2
- VI Walk 1/4, Run 3/4, Walk 1/4, Run 3/4
- VII Walk 1/4, Run 1, Walk 1/4, Run 1
- VIII Walk 1/4, Run 1, Walk 1/4, Run 1, Walk 1/4, Run 1

*These guidelines apply to use of this walk-to-run program:

- use brand name running shoes, not court or cross-trainers;
- begin at an easy pace on level surfaces with no hills until 3 to 5 weeks after phase VIII;
- stop if increased pain, swelling, or stiffness is noted, especially while running and if symptoms are present by the next morning;
- do not run more than three times per week and do not run daily until 3 to 5 weeks after phase VIII;
- try each phase at least twice before advancing to the next phase—do not progress if pain, swelling, or stiffness is noted;
- after phase VIII, gradually increase running without walking;
- all increments for walk-to-run progression are based on miles (ie, walk 1/4 mile, run 1/4 mile, walk 1/4 mile, run 1/4 mile for Phase III); and
- after phase VIII, do not increase distance or pace by more than 10% per week.

musculoskeletal load is important. Generally, jump training should be performed two or three times per week on nonconsecutive days and completed either 8 hours before or after additional training activities.

Use of Pharmaceuticals

The role of nonsteroidal antiinflammatory medications (NSAIDs) in the management of stress injuries is equivocal. NSAIDs are commonly recommended^{117,118}; they are prescribed to provide pain control and to aid in the resolution of inflammation. There is concern about the potential adverse effects of NSAIDs on bone healing as a result of cyclooxygenase 2 inhibition in fracture healing.¹¹⁹ Most published research (in animal models) has demonstrated inhibition of fracture healing with administration of both nonspecific NSAIDs and cyclooxygenase 2 selective NSAIDs.^{120,121}



Fig. 11-10. Lateral lunge.

Giannoudis et al¹²² reported delayed fracture healing in patients with femoral fractures who had taken NSAIDs. Although the pathophysiology of fracture healing in humans probably mirrors that of animal models, further research is needed to confirm this adverse impact of NSAIDs on fracture healing in humans.¹²³ Given the detrimental effects noted in animal models, Gerstenfeld and Einhorn¹²⁴ recommended short-term use of NSAIDs or other drugs (eg, acetaminophen) in patient management. A review by Wheeler and Batt¹²⁵ emphasized the lack of evidence regarding the use of NSAIDs in patients with stress fractures and called for further research in this area.

Bracing

Aircast, Inc (Summit, New Jersey) produces a pneumatic leg brace for tibial and fibular stress fractures. The most recently published study reports no additional benefit of bracing for military trainees¹²⁶; however, previous studies noted that use of this brace results in a more rapid return to pain-free activity and resumption of preinjury activity levels.¹²⁷⁻¹²⁹ The ability of the brace to act like a venous tourniquet is the mechanism by which this brace facilitates healing. Air cells within the brace combine with muscular activity during ambulation to produce a shift in interstitial fluids and to enhance osteoblastic activity.¹²⁹ Clinicians should be aware that the pneumatic leg brace for tibial stress fractures is different (a longer length) than the pneumatic brace normally used for ankle sprains (Figure 11-11).

In addition to using the Aircast brace for tibial stress fractures of the mid to distal regions of the tibia, we also used a standard knee range-of-motion brace (Figure 11-12) for proximal tibia stress fractures. This brace limits valgus forces to the knee region that typically incite pain in patients presenting with such injuries. This is an effective brace that promotes early, pain-free weightbearing, as advocated by Kimball and Savastano¹¹³ for treating proximal tibia stress fractures.

Casting

Tibial fractures that do not heal with bracing or activity limitation can be considered for a "walking" cast. Casting modalities are effective for noncompliant individuals. Casting should be for a period of at least 4 weeks or until the patient is pain free.

Surgery

Documented tibia fractures that are refractory to all conservative means (ie activity modification, bracing,



Fig. 11-11. Right leg: standard ankle Aircast (Aircast, Inc [Summit, NJ]) brace. Left leg: stress fracture Aircast brace.

or casting) after an appropriate treatment period of 90 days may be considered for surgical intervention after an evaluation by an orthopaedic surgeon or podiatrist.

Supplemental Modalities

The use of electrical stimulators and ultrasound to accelerate bone healing has been extensively reported in the literature.¹³⁰⁻¹³⁵ The vast majority of studies have used these modalities in fresh, nonunion, or delayed union fractures. The exact details regarding the acceleration of osteogenesis have been debated, but the underlying mechanism appears to be an increase in intracellular calcium that increases osteoblastic activity.^{131,132} Products marketed and researched for



Fig. 11-12. Knee range-of-motion brace.

bone healing normally use specific electrical or ultrasound parameters not common in standard electrical stimulation and ultrasound devices found in most physical therapy clinics. Although some manufacturers of therapeutic electrical stimulation units include a bone stimulation protocol in the device handbook, the parameters have not been reported in the peerreviewed literature.

Electrical stimulation has been advocated in the treatment of stress fractures.^{136,137} This modality has been used in patients with delayed union and nonunion fractures.^{130,131,133} Based on personal clinical experience, use of interferential electrical stimulation can produce localized increased pain following treatment of patients with tibial stress fractures. Careful consideration should be given to the parameters used with electrical stimulation application, and patients should report any increase in pain during or following treatment.

Ultrasound produces a mechanical effect to stimulate fracture healing.¹³⁰ The Exogen device (Smith & Nephew BV, Hoofddorp, the Netherlands) has improved the healing rates of fresh fractures and nonunion fractures at a dosage of 20 minutes per day.^{130,135} Fredericson et al⁶⁹ reported that ultrasound reduces periosteal inflammation in athletes with tibial stress reaction; however, no specifics were provided about particular treatment parameters. Only one study to date has reported on the use of ultrasound in the treatment of stress fractures. Brand et al¹³⁸ explained that ultrasound application (the Exogen device) in athletes with tibial stress fractures resulted in a more rapid return to pain-free activity and the ability to continue in sports. Participants in this study wore a pneumatic tibial brace, which should be considered a confounding variable to the results, especially given the previously described efficacy of this brace.

The effectiveness of both ultrasound and electrical stimulation in individuals with stress fractures remains to be thoroughly studied in well-controlled clinical trials. Although there are no apparent adverse effects of either modality, the machines specifically manufactured and marketed for bone healing are expensive (>\$1,500 per unit), have limited battery life (requiring manufacturer refurbishment), and are marketed for use by individual patients (ie, one patient per unit).

Ice and Heat

These two modalities are commonly used for their analgesic effects. Ice treatment helps minimize swelling, whereas heat treatment helps to relax and loosen tissues. The efficacy of thermal modalities in the treatment of stress fractures lacks supporting research.

Nutritional Supplementation

The role of supplementation in prevention of or recovery from stress fractures is yet to be established. The National Institutes of Health has established guidelines for calcium needs based on age group. It is possible that the calcium requirements in military trainees and athletes exceed that of the general population and that supplementation is beneficial.⁵⁵ However, Schwellnus and Jordan¹³⁹ did not find a beneficial effect of calcium supplementation. Caution has been recommended with vitamin D supplementation.⁶¹

Bisphosphonate supplementation has been advocated in osteoporotic women and has been associated with an increase in bone mass. However, prophylactic use in military trainees has not demonstrated a reduction in stress fracture incidence.¹⁴⁰ Mashiba et al¹⁴¹ found microdamage accumulation in animals treated with bisphosphonates. A small case series on female intercollegiate athletes reported that 80% of athletes who began intravenous pamidronate treatment were able to continue unrestricted training and competition.¹⁴² Unwanted effects of microdamage accumulation in military trainees suggest against utilization of these supplements until further research is accomplished.

Anatomical Considerations

Stress fractures that can have serious consequences, such as nonunion fractures or those that would need surgical intervention if mismanaged, are considered high-risk. High-risk stress fracture locations include the femoral neck, patella, anterior tibial shaft, medial malleolus, talus, navicular, base of the fifth metatarsal, and the sesamoids. Clinical suspicion of a stress fracture in any of these locations should be treated with extreme caution (eg, strict nonweightbearing crutch ambulation) until bone stress injury is ruled out. Stress fractures in other locations are less likely to advance to nonunion or require surgical intervention; these are considered low-risk.

Tibia

Stress fractures of the tibia present the most common anatomical region of involvement in military trainees and athletes.^{7,48} The location of the stress fracture can include any of the following: tibial plateau, proximal tibia, midtibia, distal tibia, or medial malleolus.^{113,143-145} Differential diagnosis of tibia pain encompasses the spectrum of shin splints, periostitis, stress reaction, stress fracture, and nonmusculoskeletal causes. Additional consideration should be given to exertional (transient) compartment syndrome, soleus muscle strain, and flexor digitorum longus strain.⁷⁰ Batt et al⁷⁰ reported that the findings on bone scan and MRI suggest a likely continuum of injury from shin-splints to stress fracture and that diffuse uptake on bone scan should not be simply considered as a self-limiting lesion, but rather treated with a rest period.

Midshaft (diaphyseal) stress fractures are encountered most commonly. This region of involvement had the poorest outcome regarding resumption of military training.¹⁰¹ Pain production along the medial aspect of the tibia can also occur from the origins of the tibialis posterior, soleus, or flexor digitorum longus muscles.^{69,146} The previously described clinical tests, in conjunction with a thorough history, will aid in developing the level of concern for a stress fracture of this region.

Proximal tibia stress fractures have also been widely reported and appear to have good potential for return to military training.¹⁰¹ Differential diagnosis of pain in this region should include pes anserine tendonitis or bursitis, medial collateral ligament sprains, medial meniscus tears, and muscle strains. Based on clinical experience, individuals with stress injuries to this area can present with a sudden onset of symptoms, pain with valgus stress to the knee joint, and edema around the pes anserine region.

Conflicting evidence regarding the correlation of findings on imaging studies to the time course for return to activity has been reported previously.^{69,101} This evidence underscores the importance of close clinical management by the team of medical personnel to ensure an uneventful return to training.

Metatarsals

Stress fractures of the second and third metatarsals are the most common. Less common, but with greater potential for adverse outcome, are stress fractures of the base of the fifth metatarsal. Key components of the physical examination in patients with metatarsal pain are axial loading, fulcrum testing, and a good palpatory examination. Differential diagnosis includes extensor or fibularis (peroneal) tendon strains or tendonitis, Morton's neuroma, Frieberg's infarction, metatarsalgia, and cuboid syndrome.⁵⁰

Tarsals

Tarsal stress fractures of the calcaneus, talus, and navicular^{89,147,148} have been reported. Matheson et al⁴⁸ noted a longer recovery time in athletes with tarsal stress fractures than other lower extremity stress

fractures. The prolonged recovery period required for these stress fractures was attributed to a delay in accurate diagnosis, thus amplifying the need to be aware of such conditions. Differential diagnosis of calcaneal stress fractures should include retrocalcaneal bursitis, Achilles tendonitis, plantar nerve entrapment, radiculopathy, and posterior ankle impingement.^{89,148} Stress fractures of the talus, although typically located in the talar neck, may occur in various locations of the bone and have been associated with excessive subtalar joint pronation.^{102,149} Navicular stress fractures also present a diagnostic challenge. Trainees typically complain of a vague dorsal midfoot pain. Differential diagnosis should include extensor hallucis longus tendonitis, anterior tibialis tendonitis, and a symptomatic accessory navicular bone.

Femur

Stress fractures of the femur can involve the femoral neck, femoral shaft, or condylar region.^{28,30,103} Differential diagnosis of hip, thigh, or knee pain in the military trainee should include stress fractures, musculotendinous strains, tendonitis, synovitis, intra-articular pathology, avascular necrosis, and malignancy. Significant findings on clinical examination might include the hop test, the log roll, the active straight leg raise, the fulcrum test, and percussion.^{71,72}

Typical presenting symptoms in patients with femoral neck stress fractures include groin or inguinal pain with radiation of symptoms to the anterior or medial thigh. Less common, but noted from personal clinical experience, are gluteal pain and vague sacroiliac-region pain. Patients often complain of pain with straight leg raise. Patients complaining of lateral hip pain with tenderness to palpation should also be evaluated for greater trochanteric bursitis or gluteus medius tendonopathy. According to the work of Shin et al,¹⁰³ the MRI should be the preferred source of imaging in a trainee with acute or gradual onset hip pain indicative of a femoral neck stress fracture.

The early signs and symptoms of femoral shaft stress fractures are often diagnosed as quadriceps muscle strains. The most useful clinical test has been the fulcrum test.⁷² The location of femoral shaft stress fractures is typically on the medial (compression) side of the femur. This location is the site of attachments for the adductor and vastus medialis musculature, which might be causative agents for stress fractures in this location. Patients with femoral shaft stress fractures have recovered faster than other lower extremity sites.⁴⁸

Although stress fractures of the condylar region of the femur are less frequent, this lesion should be considered in the differential diagnosis of trainees with persistent or worsening distal thigh and knee joint pain. Femoral condyle stress fractures have occurred in both the medial and lateral condyles.^{28,30,150} Differential diagnosis of this region is made difficult by common overuse problems around the knee, such as muscle strains, ligamentous sprains, iliotibial band syndrome, and patellofemoral pain syndrome.

Fibula

Although the fibula is not a major weight-bearing bone, stress fractures are not uncommon and must be considered in the differential diagnosis of a patient with lateral lower leg pain. Stress fractures of the fibula are attributed to muscle traction and torsional stresses.⁸² Other considerations in the differential diagnosis should include muscle strains, tibiofibular syndesmotic sprain, and exertional compartment syndrome. Stress fractures of the fibula have occurred in the proximal, middle, or distal thirds of the shaft.¹⁵¹

Sacrum

Sacral stress fractures, although not among the more commonly encountered injuries, should be considered in the military trainee with persistent lower back pain. Case reports in the literature have noted that sacral stress fractures occur predominantly in long distance runners^{108,152,153} and are caused by the transmission of forces from the lower extremities and spine to the sacrum.¹⁵⁴ Differential diagnosis of these patients should include lumbar strain or sprain, sacroiliac joint dysfunction, lumbar disc involvement, or congenital anomaly. The most reliable finding on physical examination is localized tenderness over the sacrum and sacroiliac joint.¹⁰⁸ Less consistent findings on physical examination are positive Patrick's test and leg-length inequality.^{155,156}

Spine (Pars Interarticularis)

Stress injuries to the pars interarticularis region, normally in the lower lumbar region of the spine, should be considered in the patient involved in activities requiring repeated extension movements of the lumbar spine. Key components of the physical examination include reproduction of symptoms with lumbar extension or hip-pelvic girdle extension. Neurological sequelae (eg, radiating pain into the lower extremity) might or might not be present in conjunction with a pars stress fracture. Radionuclide examination can be useful in the early assessment of such injuries, and triple-phase bone scans can assist in the determination of lesion chronicity.⁸⁵

Pelvis

Pelvic stress fractures have been found in female military trainees and long distance runners. Injuries in female trainees have been linked to shorter physical stature and marching in the rear of formations, which resulted in greater than normal stride lengths in an attempt to maintain pace with the group.^{157,158} Alternatively, consideration should be given to the possibility of osteitis pubis, athletes pubalgia/hernia, and adductor muscle strains. The key finding on physical examination is well-localized bony tenderness over the inferior or superior pubic rami.

Sesamoids

Stress fractures of the sesamoid bones, with the medial sesamoid more commonly affected, are prone to nonunion.⁸² These bones play an important role in the strength and stability of the first metatarsal phalangeal joint. Given the demands on the joint in running, this should be considered a possible diagnosis in trainees presenting with plantar first metatarsal phalangeal joint pain.

Patella

Overuse injuries around the knee constitute a high number of training-induced visits to military treatment facilities. Stress fractures of the patella,¹⁵⁹ although not widely reported, can occur and clinicians should be aware of this possibility.

Upper Extremity/Thorax

Stress fractures of the upper extremity and thorax have been reported in the literature.¹⁶⁰⁻¹⁶⁴ Rib stress fractures have been found in rowers and golfers.^{147,163} These types of stress fractures are typically related to specific activities that are not common in military training.

Failure of Conservative Measures

If the patient's symptoms have not improved after an appropriate treatment period, usually 90 days, he or she should be referred to an orthopaedic surgeon or podiatrist for evaluation. The patient should be considered for surgical intervention or fitness for duty evaluations.

SUMMARY

Knowledge of bone function, response to physical training, and the interplay of multiple intrinsic and extrinsic risk factors have advanced over the past 30 years. The significant adverse impact of musculoskeletal injuries—stress fractures in particular—on the throughput of military trainees in all environments (basic trainees, service academies, and advanced military training settings) necessitates vigilance from healthcare providers. Clinical awareness and skill in formulating a differential diagnosis for musculoskeletal injuries are needed to prevent adverse sequelae that might result from unwarranted continuation of physical training in the presence of a stress fracture. Effective management of patients with stress fractures requires a team approach, a comprehensive and closely monitored rehabilitation program, and education of the trainee and the personnel providing instruction to military training programs.

REFERENCES

- 1. Jordaan G, Schwellnus MP. The incidence of overuse injuries in military recruits during basic military training. *Mil Med.* 1994;159:421–426.
- 2. Ross J. A review of lower limb overuse injuries during basic military training. Part 1: Types of overuse injuries. *Mil Med.* 1993;158:410–415.
- 3. Briethaupt MD. Zur pathologie des menschlichen fusses. Med Zeitung. 1855;24:169.
- 4. Stechow AW. Fussoedem und roentgenstrahlen. Dtsch Mil-Aerztl Zeitg. 1897;26:465–471.
- 5. Almeida SA, Williams KM, Shaffer RA, Brodine SK. Epidemiological patterns of musculoskeletal injuries and physical training. *Med Sci Sports Exerc.* 1999;31:1176–1182.
- Protzman RR. Physiologic performance of women compared to men. Observations of cadets at the United States Military Academy. *Am J Sports Med.* 1979;7:191–194.
- 7. Bijur PE, Horodyski M, Egerton W, Kurzon M, Lifrak S, Friedman S. Comparison of injury during cadet basic training by gender. *Arch Pediatr Adolesc Med.* 1997;151:456–461.
- 8. Kaufman KR, Brodine SK, Shaffer RA, Johnson CW, Cullison TR. The effect of foot structure and range of motion on musculoskeletal overuse injuries. *Am J Sports Med.* 1999;27:585–593.
- Shwayhat AF, Linenger JM, Hofherr LK, Slymen DJ, Johnson CW. Profiles of exercise history and overuse injuries among United States Navy Sea, Air, and Land (SEAL) recruits. *Am J Sports Med.* 1994;22:835–840.
- 10. Rubin CT, Lanyon LE. Regulation of bone formation by applied dynamic loads. J Bone Joint Surg Am. 1984;66:397–402.
- 11. Ekenman I, Halvorsen K, Westblad P, Fellander-Tsai L, Rolf C. Local bone deformation at two predominant sites for stress fractures of the tibia: An in vivo study. *Foot Ankle Int*. 1998;19:479–484.
- 12. Turner CH. Three rules for bone adaptation to mechanical stimuli. Bone. 1998;23:399–407.
- 13. Milgrom C, Simkin A, Eldad A, Nyska M, Finestone A. Using bone's adaptation ability to lower the incidence of stress fractures. *Am J Sports Med.* 2000;28:245–251.
- 14. Hsieh YF, Turner CH. Effects of loading frequency on mechanically induced bone formation. J Bone Miner Res. 2001;16:918–924.
- 15. Robling AG, Hinant FM, Burr DB, Turner CH. Improved bone structure and strength after long-term mechanical loading is greatest if loading is separated into short bouts. *J Bone Miner Res.* 2002;17:1545–1554.
- 16. Turner CH, Robling AG. Designing exercise regimens to increase bone strength. Exerc Sport Sci Rev. 2003;31:45–50.

- 17. Robling AG, Burr DB, Turner CH. Recovery periods restore mechanosensitivity to dynamically loaded bone. *J Exp Biol.* 2001;204:3389–3399.
- 18. Yoshikawa T, Mori S, Santiesteban AJ, et al. The effects of muscle fatigue on bone strain. *J Exp Biol*. 1994;188:217–233.
- 19. Bennell KL, Malcolm SA, Thomas SA, et al. Risk factors for stress fractures in track and field athletes. A twelve-month prospective study. *Am J Sports Med.* 1996;24:810–818.
- 20. Milgrom C. The Israeli elite infantry recruit: a model for understanding the biomechanics of stress fractures. *J R Coll Surg Edinb*. 1989;34(suppl 6):S18–S22.
- 21. Bennell KL, Malcolm SA, Wark JD, Brukner PD. Models for the pathogenesis of stress fractures in athletes. *Br J Sports Med.* 1996;30:200–204.
- 22. Taylor D, Kuiper JH. The prediction of stress fractures using a 'stressed volume' concept. J Orthop Res. 2001; 19:919–926.
- Taylor D, Casolari E, Bignardi C. Predicting stress fractures using a probabilistic model of damage, repair and adaptation. J Orthop Res. 2004;22:487–494.
- Linenger JM, West LA. Epidemiology of soft-tissue/musculoskeletal injury among US Marine recruits undergoing basic training. *Mil Med.* 1992;157:491–493.
- Milgrom C, Giladi M, Stein M, et al. Stress fracture in military recruits. A prospective study showing an unusually high incidence. J Bone Joint Surg Br. 1985;67:732–735.
- 26. Giladi M, Ahronson Z, Stein M, Danon Y, Milgrom C. Unusual distribution and onset of stress fractures in soldiers. *Clin Ortho Rel Res.* 1985;192:142–146.
- 27. Jones BH, Bovee MW, Harris JM, Cowan DN. Intrinsic risk factors for exercise-related injuries among male and female army trainees. *Am J Sports Med.* 1993;21:705–710.
- 28. Glorioso JE, Leadbetter WB. Femoral supracondylar stress fractures. Phys Sportsmed. 2002;30:25–28.
- 29. Otis CL, Drinkwater B, Johnson M, Loucks A, Wilmore J. American College of Sports Medicine position stand. The female athlete triad. *Med Sci Sports Exerc.* 1997;29:i–ix.
- 30. Boden BP, Speer KP. Femoral stress fractures. Clin Sports Med. 1997;16:307–317.
- 31. Barrow GW, Saha S. Menstrual irregularity and stress fractures in collegiate female distance runners. *Am J Sports Med*. 1988;16:209–216.
- Lloyd T, Triantafyllou SJ, Baker ER, et al. Women athletes with menstrual irregularity have increased musculoskeletal injuries. *Med Sci Sports Exerc.* 1986;18:374–379.
- 33. Winfield AC, Moore J, Bracker M, Johnson CW. Risk factors associated with stress reactions in female Marines. *Mil Med.* 1997;162:698–702.
- 34. Polatti F, Perotti F, Filippa N, Gallina D, Nappi RE. Bone mass and long-term monophasic oral contraceptive treatment in young women. *Contraception*. 1995;51:221–224.
- 35. Register TC, Jayo MJ, Jerome CP. Oral contraceptive treatment inhibits the normal acquisition of bone mineral in skeletally immature young adult female monkeys. *Osteoporos Int.* 1997;7:348–353.
- Brudvig TJ, Gudger TD, Obermeyer L. Stress fractures in 295 trainees: A one-year study of incidence as related to age, sex, and race. *Mil Med.* 1983;148:666–667.

- Gardner LI Jr, Dziados JE, Jones BH, et al. Prevention of lower extremity stress fractures: A controlled trial of a shock absorbent insole. *Am J Public Health*. 1988;78:1563–1567.
- Milgrom C, Finestone A, Shlamkovitch N, et al. Youth is a risk factor for stress fracture: A study of 783 infantry recruits. *J Bone Joint Surg Br.* 1994;76:20–22.
- Zanker CL, Cooke CB. Energy balance, bone turnover, and skeletal health in physically active individuals. *Med Sci Sports Exerc.* 2004;36:1372–1381.
- Drinkwater BL, Nilson K, Ott S, Chesnut CH 3rd. Bone mineral density after resumption of menses in amenorrheic athletes. JAMA. 1986;256:380–382.
- 41. Giladi M, Milgrom C, Simkin A, Danon Y. Stress fractures. Identifiable risk factors. Am J Sports Med. 1991;19:647–652.
- 42. Giladi M, Milgrom C, Simkin A, et al. Stress fractures and tibial bone width. A risk factor. *J Bone Joint Surg Br*. 1987;69: 326–329.
- 43. Bennell K, Crossley K, Jayarajan J, et al. Ground reaction forces and bone parameters in females with tibial stress fracture. *Med Sci Sports Exerc.* 2004;36:397–404.
- 44. Snow CM, Williams DP, LaRiviere J, Fuchs RK, Robinson TL. Bone gains and losses follow seasonal training and detraining in gymnasts. *Calcif Tissue Int*. 2001;69:7–12.
- 45. Hoffman JR, Chapnik L, Shamis A, Givon U, Davidson B. The effect of leg strength on the incidence of lower extremity overuse injuries during military training. *Mil Med.* 1999;164:153–156.
- 46. Gefen A. Biomechanical analysis of fatigue-related foot injury mechanisms in athletes and recruits during intensive marching. *Med Biol Eng Comput.* 2002;40:302–310.
- 47. Swissa A, Milgrom C, Giladi M, et al. The effect of pretraining sports activity on the incidence of stress fractures among military recruits. A prospective study. *Clin Orthop.* 1989;245:256–260.
- Matheson GO, Clement DB, McKenzie DC, Taunton JE, Lloyd-Smith DR, MacIntyre JG. Stress fractures in athletes. A study of 320 cases. Am J Sports Med. 1987;15:46–58.
- 49. Cowan DN, Jones BH, Frykman PN, et al. Lower limb morphology and risk of overuse injury among male infantry trainees. *Med Sci Sports Exerc.* 1996;28:945–952.
- Maitra RS, Johnson DL. Stress fractures. Clinical history and physical examination. Clin Sports Med. 1997;16:259– 274.
- 51. Giladi M, Milgrom C, Stein M, et al. External rotation of the hip. A predictor of risk for stress fractures. *Clin Orthop*. 1987;216:131–134.
- 52. Zanker CL, Swaine IL. Responses of bone turnover markers to repeated endurance running in humans under conditions of energy balance or energy restriction. *Eur J Appl Physiol*. 2000;83:434–440.
- 53. Armstrong DW 3rd, Rue JP, Wilckens JH, Frassica FJ. Stress fracture injury in young military men and women. *Bone*. 2004;35:806–816.
- 54. Brukner P, Bennell K, Matheson G. Stress Fractures. Victoria, Australia: Human Kinetics; 1999.
- 55. Klesges RC, Ward KD, Shelton ML, et al. Changes in bone mineral content in male athletes. Mechanisms of action and intervention effects. *JAMA*. 1996;276:226–230.
- Myburgh KH, Hutchins J, Fataar AB, Hough SF, Noakes TD. Low bone density is an etiologic factor for stress fractures in athletes. *Ann Intern Med.* 1990;113:754–759.

- 57. Cline AD, Jansen GR, Melby CL. Stress fractures in female army recruits: implications of bone density, calcium intake, and exercise. *J Am Coll Nutr*. 1998;17:128–135.
- 58. Matkovic V, Heaney RP. Calcium balance during human growth: Evidence for threshold behavior. *Am J Clin Nutr*. 1992;55:992–996.
- 59. Matkovic V. Calcium and peak bone mass. J Intern Med. 1992;231:151-160.
- 60. Givon U, Friedman E, Reiner A, Vered I, Finestone A, Shemer J. Stress fractures in the Israeli defense forces from 1995 to 1996. *Clin Orthop.* 2000;373:227–232.
- 61. Nattiv A, Armsey TD Jr. Stress injury to bone in the female athlete. Clin Sports Med. 1997;16:197–224.
- 62. Milgrom C, Burr D, Fyhrie D, et al. A comparison of the effect of shoes on human tibial axial strains recorded during dynamic loading. *Foot Ankle Int*. 1998;19:85–90.
- 63. Milgrom C, Burr D, Fyhrie D, et al. The effect of shoe gear on human tibial strains recorded during dynamic loading: A pilot study. *Foot Ankle Int.* 1996;17:667–671.
- 64. Milgrom C, Giladi M, Kashtan H, et al. A prospective study of the effect of a shock-absorbing orthotic device on the incidence of stress fractures in military recruits. *Foot Ankle*. 1985;6:101–104.
- 65. Gillespie WJ, Grant I. Interventions for preventing and treating stress fractures and stress reactions of bone of the lower limbs in young adults. *Cochrane Database of Systematic Reviews*. 2004;1.
- 66. Jones BH, Thacker SB, Gilchrist J, Kimsey CD, Sosin DM. Prevention of lower extremity stress fractures in athletes and soldiers: A systematic review. *Epidemiol Rev.* 2002;24:228–247.
- 67. Scully TJ, Besterman G. Stress fracture—a preventable training injury. Mil Med. 1982;147:285–287.
- 68. Popovich RM, Gardner JW, Potter R, Knapik JJ, Jones BH. Effect of rest from running on overuse injuries in army basic training. *Am J Prev Med*. 2000;18(suppl 3):147–155.
- 69. Fredericson M, Bergman AG, Hoffman KL, Dillingham MS. Tibial stress reaction in runners. Correlation of clinical symptoms and scintigraphy with a new magnetic resonance imaging grading system. *Am J Sports Med.* 1995;23: 472–481.
- 70. Batt ME, Ugalde V, Anderson MW, Shelton DK. A prospective controlled study of diagnostic imaging for acute shin splints. *Med Sci Sports Exerc.* 1998;30:1564–1571.
- 71. Casterline M, Osowski S, Ulrich G. Femoral stress fracture. J Athl Train. 1996;31:53–56.
- 72. Johnson AW, Weiss CB Jr, Wheeler DL. Stress fractures of the femoral shaft in athletes—more common than expected. A new clinical test. *Am J Sports Med.* 1994;22:248–256.
- 73. Lesho EP. Can tuning forks replace bone scans for identification of tibial stress fractures? Mil Med. 1997;162:802–803.
- Devereaux MD, Parr GR, Lachmann SM, Page-Thomas P, Hazleman BL. The diagnosis of stress fractures in athletes. JAMA. 1984;252:531–533.
- 75. Giladi M, Nili E, Ziv Y, Danon YL, Aharonson Z. Comparison between radiography, bone scan, and ultrasound in the diagnosis of stress fractures. *Mil Med.* 1984;149:459–461.
- 76. Moss A, Mowat AG. Ultrasonic assessment of stress fractures. Br Med J (Clin Res Ed). 1983;286:1479–1480.
- 77. Nitz AJ, Scoville CR. Use of ultrasound in early detection of stress fractures of the medial tibial plateau. *Mil Med*. 1980;145:844–846.

- Boam WD, Miser WF, Yuill SC, Delaplain CB, Gayle EL, MacDonald DC. Comparison of ultrasound examination with bone scintiscan in the diagnosis of stress fractures. J Am Board Fam Pract. 1996;9:414–417.
- 79. Romani WA, Perrin DH, Dussault RG, Ball DW, Kahler DM. Identification of tibial stress fractures using therapeutic continuous ultrasound. *J Orthop Sports Phys Ther*. 2000;30:444–452.
- Matheson GO, Clement DB, McKenzie DC, Taunton JE, Lloyd-Smith DR, Macintyre JG. Scintigraphic uptake of 99mTc at non-painful sites in athletes with stress fractures. The concept of bone strain. *Sports Med.* 1987;4:65–75.
- Ishibashi Y, Okamura Y, Otsuka H, Nishizawa K, Sasaki T, Toh S. Comparison of scintigraphy and magnetic resonance imaging for stress injuries of bone. *Clin J Sport Med.* 2002;12:79–84.
- Wilder RP, Sethi S. Overuse injuries: Tendinopathies, stress fractures, compartment syndrome, and shin splints. *Clin* Sports Med. 2004;23:55–81.
- 83. Savoca CJ. Stress fractures. A classification of the earliest radiographic signs. Radiology. 1971;100:519–524.
- Prather JL, Nusynowitz ML, Snowdy HA, Hughes AD, McCartney WH, Bagg RJ. Scintigraphic findings in stress fractures. J Bone Joint Surg Am. 1977;59:869–874.
- 85. Deutsch AL, Coel MN, Mink JH. Imaging of stress injuries to bone. Radiography, scintigraphy, and MR imaging. *Clin Sports Med.* 1997;16:275–290.
- 86. Daffner RH, Pavlov H. Stress fractures: Current concepts. AJR Am J Roentgenol. 1992;159:245–252.
- 87. Kiuru MJ, Pihlajamaki HK, Hietanen HJ, Ahovuo JA. MR imaging, bone scintigraphy, and radiography in bone stress injuries of the pelvis and the lower extremity. *Acta Radiol*. 2002;43:207–212.
- 88. Knapp TP, Garrett WE Jr. Stress fractures: General concepts. Clin Sports Med. 1997;16:339–356.
- 89. Hershman EB, Mailly T. Stress fractures. Clin Sports Med. 1990;9:183-214.
- 90. Matin P. The appearance of bone scans following fractures, including immediate and long-term studies. *J Nucl Med*. 1979;20:1227–1231.
- Chisin R, Milgrom C, Giladi M, Stein M, Margulies J, Kashtan H. Clinical significance of nonfocal scintigraphic findings in suspected tibial stress fractures. *Clin Orthop*. 1987;220:200–205.
- Arendt EA, Griffiths HJ. The use of MR imaging in the assessment and clinical management of stress reactions of bone in high-performance athletes. *Clin Sports Med.* 1997;16:291–306.
- Zwas ST, Elkanovitch R, Frank G. Interpretation and classification of bone scintigraphic findings in stress fractures. J Nucl Med. 1987;28:452–457.
- Roub LW, Gumerman LW, Hanley EN Jr, Clark MW, Goodman M, Herbert DL. Bone stress: A radionuclide imaging perspective. *Radiology*. 1979;132:431–438.
- Nielsen MB, Hansen K, Holmer P, Dyrbye M. Tibial periosteal reactions in soldiers. A scintigraphic study of 29 cases of lower leg pain. Acta Orthop Scand. 1991;62:531–534.
- 96. Holder LE, Michael RH. The specific scintigraphic pattern of "shin splints in the lower leg": Concise communication. *J Nucl Med.* 1984;25:865–869.
- 97. Ho SS, Coel MN, Kagawa R, Richardson AB. The effects of ice on blood flow and bone metabolism in knees. *Am J Sports Med.* 1994;22:537–540.
- 98. Milgrom C, Chisin R, Giladi M, et al. Negative bone scans in impending tibial stress fractures. A report of three cases. *Am J Sports Med.* 1984;12:488–491.

- 99. Keene JS, Lash EG. Negative bone scan in a femoral neck stress fracture. A case report. *Am J Sports Med.* 1992;20: 234–236.
- 100. Sterling JC, Webb RF Jr, Meyers MC, Calvo RD. False negative bone scan in a female runner. *Med Sci Sports Exerc.* 1993;25:179–185.
- 101. Dutton J, Bromhead SE, Speed CA, Menzies AR, Peters AM. Clinical value of grading the scintigraphic appearances of tibial stress fractures in military recruits. *Clin Nucl Med.* 2002;27:18–21.
- 102. Rupani HD, Holder LE, Espinola DA, Engin SI. Three-phase radionuclide bone imaging in sports medicine. *Radiology*. 1985;156:187–196.
- 103. Shin AY, Morin WD, Gorman JD, Jones SB, Lapinsky AS. The superiority of magnetic resonance imaging in differentiating the cause of hip pain in endurance athletes. *Am J Sports Med.* 1996;24:168–176.
- 104. Kiuru MJ, Pihlajamaki HK, Ahovuo JA. Fatigue stress injuries of the pelvic bones and proximal femur: Evaluation with MR imaging. *Eur Radiol*. 2003;13:605–611.
- 105. Gaeta M, Minutoli F, Scribano E, et al. CT and MR imaging findings in athletes with early tibial stress injuries: Comparison with bone scintigraphy findings and emphasis on cortical abnormalities. *Radiology*. 2005;235:553–561.
- 106. Aoki Y, Yasuda K, Tohyama H, Ito H, Minami A. Magnetic resonance imaging in stress fractures and shin splints. *Clin Orthop*. 2004;421:260–267.
- 107. Kiuru MJ, Niva M, Reponen A, Pihlajamaki HK. Bone stress injuries in asymptomatic elite recruits. *Am J Sports Med*. 2005;33:272–276.
- 108. McFarland EG, Giangarra C. Sacral stress fractures in athletes. Clin Orthop. 1996;329:240-243.
- 109. Whipple TJ, Le BH, Demers LM, et al. Acute effects of moderate intensity resistance exercise on bone cell activity. *Int J Sports Med.* 2004;25:496–501.
- 110. Murguia MJ, Vailas A, Mandelbaum B, et al. Elevated plasma hydroxyproline. A possible risk factor associated with connective tissue injuries during overuse. *Am J Sports Med.* 1988;16:660–664.
- 111. Bennell KL, Malcolm SA, Brukner PD, et al. A 12-month prospective study of the relationship between stress fractures and bone turnover in athletes. *Calcif Tissue Int*. 1998;63:80–85.
- 112. Robling AG, Burr DB, Turner CH. Partitioning a daily mechanical stimulus into discrete loading bouts improves the osteogenic response to loading. *J Bone Miner Res.* 2000;15:1596–1602.
- 113. Kimball PR, Savastano AA. Fatigue fractures of the proximal tibia. Clin Orthop. 1970;70:170–173.
- 114. Burr DB, Milgrom C, Boyd RD, Higgins WL, Robin G, Radin EL. Experimental stress fractures of the tibia. Biological and mechanical aetiology in rabbits. *J Bone Joint Surg Br*. 1990;72:370–375.
- 115. Boden BP, Osbahr DC. High-risk stress fractures: Evaluation and treatment. J Am Acad Orthop Surg. 2000;8:344–353.
- 116. Moore JH, Ernst GP. Therapeutic exercise. In: O'Connor FG, Wilder RP, eds. *Textbook on Running Medicine*. New York: McGraw-Hill; 2001: 567–578.
- 117. Sanderlin BW, Raspa RF. Common stress fractures. Am Fam Phys. 2003;68:1527–1532.
- 118. Tuan K, Wu S, Sennett B. Stress fractures in athletes: Risk factors, diagnosis, and management. *Orthopedics*. 2004;27: 583–591.
- 119. Simon AM, Manigrasso MB, O'Connor JP. Cyclo-oxygenase 2 function is essential for bone fracture healing. *J Bone Miner Res*. 2002;17:963–976.

- 120. Goodman S, Ma T, Trindade M, et al. COX-2 selective NSAID decreases bone ingrowth in vivo. *J Orthop Res.* 2002;20: 1164–1169.
- 121. Endo K, Sairyo K, Komatsubara S, et al. Cyclooxygenase-2 inhibitor inhibits the fracture healing. J Physiol Anthropol Appl Human Sci. 2002;21:235–238.
- 122. Giannoudis PV, MacDonald DA, Matthews SJ, Smith RM, Furlong AJ, De Boer P. Nonunion of the femoral diaphysis. The influence of reaming and non-steroidal anti-inflammatory drugs. *J Bone Joint Surg Br.* 2000;82:655–658.
- 123. Seidenberg AB, An YH. Is there an inhibitory effect of COX-2 inhibitors on bone healing? *Pharmacol Res.* 2004;50: 151–156.
- 124. Gerstenfeld LC, Einhorn TA. COX inhibitors and their effects on bone healing. Expert Opin Drug Saf. 2004;3:131–136.
- 125. Wheeler P, Batt ME. Do non-steroidal anti-inflammatory drugs adversely affect stress fracture healing? A short review. *Br J Sports Med.* 2005;39:65–69.
- Allen CS, Flynn TW, Kardouni JR, et al. The use of pneumatic leg brace in soldiers with tibial stress fractures—a randomized clinical trial. *Mil Med*. 2004;169:880–884.
- 127. Dickson TB Jr, Kichline PD. Functional management of stress fractures in female athletes using a pneumatic leg brace. *Am J Sports Med.* 1987;15:86–89.
- 128. Swenson EJ Jr, DeHaven KE, Sebastianelli WJ, Hanks G, Kalenak A, Lynch JM. The effect of a pneumatic leg brace on return to play in athletes with tibial stress fractures. *Am J Sports Med.* 1997;25:322–328.
- 129. Whitelaw GP, Wetzler MJ, Levy AS, Segal D, Bissonnette K. A pneumatic leg brace for the treatment of tibial stress fractures. *Clin Orthop.* 1991;270:301–305.
- 130. Anglen J. The clinical use of bone stimulators. J South Orthop Assoc. 2003;12:46-54.
- 131. Nelson FR, Brighton CT, Ryaby J, et al. Use of physical forces in bone healing. J Am Acad Orthop Surg. 2003;11:344–354.
- 132. Pilla AA. Low-intensity electromagnetic and mechanical modulation of bone growth and repair: Are they equivalent? *J Orthop Sci.* 2002;7:420–428.
- 133. Uhl RL. The use of electricity in bone healing. Orthop Rev. 1989;18:1045–1050.
- 134. Yonemori K, Matsunaga S, Ishidou Y, Maeda S, Yoshida H. Early effects of electrical stimulation on osteogenesis. *Bone*. 1996;19:173–180.
- 135. Zorlu U, Tercan M, Ozyazgan I, et al. Comparative study of the effect of ultrasound and electrostimulation on bone healing in rats. *Am J Phys Med Rehabil*. 1998;77:427–432.
- 136. Rettig AC, Shelbourne KD, McCarroll JR, Bisesi M, Watts J. The natural history and treatment of delayed union stress fractures of the anterior cortex of the tibia. *Am J Sports Med.* 1988;16:250–255.
- 137. Benazzo F, Mosconi M, Beccarisi G, Galli U. Use of capacitive coupled electric fields in stress fractures in athletes. *Clin Orthop*. 1995;310:145–149.
- 138. Brand JC Jr, Brindle T, Nyland J, Caborn DN, Johnson DL. Does pulsed low intensity ultrasound allow early return to normal activities when treating stress fractures? A review of one tarsal navicular and eight tibial stress fractures. *Iowa Orthop J.* 1999;19:26–30.
- 139. Schwellnus MP, Jordaan G. Does calcium supplementation prevent bone stress injuries? A clinical trial. *Int J Sport Nutr.* 1992;2:165–174.

- 140. Milgrom C, Finestone A, Novack V, et al. The effect of prophylactic treatment with risedronate on stress fracture incidence among infantry recruits. *Bone*. 2004;35:418–424.
- 141. Mashiba T, Hirano T, Turner CH, Forwood MR, Johnston CC, Burr DB. Suppressed bone turnover by bisphosphonates increases microdamage accumulation and reduces some biomechanical properties in dog rib. *J Bone Miner Res.* 2000;15:613–620.
- 142. Stewart GW, Brunet ME, Manning MR, Davis FA. Treatment of stress fractures in athletes with intravenous pamidronate. *Clin J Sport Med.* 2005;15:92–94.
- 143. Engber WD. Stress fractures of the medial tibial plateau. J Bone Joint Surg Am. 1977;59:767–769.
- 144. Vossinakis IC, Tasker TP. Stress fracture of the medial tibial condyle. Knee. 2000;7:187–190.
- 145. Shelbourne KD, Fisher DA, Rettig AC, McCarroll JR. Stress fractures of the medial malleolus. *Am J Sports Med.* 1988;16:60–63.
- 146. Michael RH, Holder LE. The soleus syndrome. A cause of medial tibial stress (shin splints). *Am J Sports Med*. 1985;13: 87–94.
- 147. Bennell KL, Brukner PD. Epidemiology and site specificity of stress fractures. Clin Sports Med. 1997;16:179–196.
- 148. Giladi M, Alcalay J. Stress fracture of the calcaneus—still an enigma in the Israeli Army. JAMA. 1984;252:3128–3129.
- 149. Umans H, Pavlov H. Insufficiency fracture of the talus. Diagnosis with MR imaging. Radiology. 1995;197:439–442.
- 150. Milgrom C, Chisin R, Margulies J, et al. Stress fractures of the medial femoral condyle. J Trauma. 1986;26:199–200.
- 151. Blivin SJ, Martire JR, McFarland EG. Bilateral midfibular stress fractures in a collegiate football player. *Clin J Sport Med.* 1999;9:95–97.
- 152. Boissonnault WG, Thein-Nissenbaum JM. Differential diagnosis of a sacral stress fracture. *J Orthop Sports Phys Ther*. 2002;32:613–621.
- 153. Klossner D. Sacral stress fracture in a female collegiate distance runner: A case report. J Athl Train. 2000;35:453–457.
- 154. Holtzhausen LM, Noakes TD. Stress fracture of the sacrum in two distance runners. Clin J Sport Med. 1992;2:139–142.
- 155. Shah MK, Stewart GW. Sacral stress fractures: an unusual cause of low back pain in an athlete. *Spine*. 2002;27: E104–E108.
- 156. Atwell EA, Jackson DW. Stress fractures of the sacrum in runners. Two case reports. Am J Sports Med. 1991;19:531–533.
- 157. Pope RP. Prevention of pelvic stress fractures in female army recruits. Mil Med. 1999;164:370–373.
- 158. Kelly EW, Jonson SR, Cohen ME, Shaffer R. Stress fractures of the pelvis in female Navy recruits: An analysis of possible mechanisms of injury. *Mil Med.* 2000;165:142–145.
- 159. Teitz CC, Harrington RM. Patellar stress fracture. Am J Sports Med. 1992;20:761–765.
- 160. Brukner P. Stress fractures of the upper limb. Sports Med. 1998;26:415–424.
- 161. Rettig AC, Beltz HF. Stress fracture in the humerus in an adolescent tennis tournament player. *Am J Sports Med.* 1985;13:55–58.
- 162. Marymount JV, Coupe KJ, Clanton TO. Sports-related spontaneous fractures of the humerus. *Orthop Rev.* 1989;18: 957–960.

- 163. Sinha AD, Kaeding CC, Wadley GM. Upper extremity stress fractures in athletes: Clinical features of 44 cases. *Clin J Sport Med*. 1999;9:199–202.
- 164. Boyer DW Jr. Trapshooter's shoulder: Stress fracture of the coracoid process. Case report. J Bone Joint Surg Am. 1975;57:862.