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The Pathophysiology of Stress Fractures

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As athletes continue to be more competitive in their desire to be the best, they will train exceptionally hard, sometimes to the point of injury. For society generally in the United States, there is a desire to have a better fitness level and the initial zeal of running or getting in shape will occur in such a rapid fashion that injuries will sometimes happen. A common type of injury seen in competitive as well as recreational athletes is the stress fracture. This type of fracture can arise when the stresses on the bone are greater than the capacity of the bone to withstand and heal from the stresses. An understanding of the science and adaptations that occur with human bones when they undergo stress activity is necessary when looking at the causes and pathophysiology of stress fractures.

STRESS FRACTURES

Bone Basic Science

Injury to bone encompasses an array of defects of bone architecture, including bone strains, stress reactions, and nondisplaced and displaced stress fractures. These injuries occur essentially when bone fails to remodel adequately with the application of repetitive subthreshold stress. Because running and jogging involve ground reaction forces that are three to eight times greater than walking, distance runners and track athletes are particularly prone to developing stress fractures.

An understanding of bone basic science is needed to illuminate the causes and treatment principles for stress fractures. Bone is a highly organized and dynamic living tissue, with both metabolic and structural components. These components are interdependent and responsive to each other. The metabolic component involves mineral homeostasis and bone remodeling, and the structural component involves maintaining skeletal integrity and bone remodeling.

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At the microscopic level, bone has two forms, woven and lamellar. Woven bone is an immature type found in the embryo and newborn; lamellar bone is a more mature bone and, through remodeling, replaces woven bone by 4 years of age [1]. Lamellar bone is more highly organized, containing stress-oriented collagen, which makes it anisotropic (mechanical properties differ depending on the direction of applied force) [2].

Normal lamellar bone is structurally organized into cortical (compact) bone or cancellous (trabecular) bone. Cortical bone makes up 80% of the skeleton and is composed of tightly packed osteons or a haversian system. Osteons usually are oriented in the long axis of the bone and are connected by haversian canals [3]. Cortical bone is found principally at the diaphysis of long bones and the "shell" of cuboid-like bones such as vertebral bodies and tarsal or carpal bones. Cortical bone is characterized by a metabolic turnover rate that is eight times slower than cancellous bone and four times greater in mass. The majority of stress fractures in runners occur in cortical bone.

Cancellous (trabecular) bone is found principally at the metaphysis and epiphysis of long bones and in cuboid-like bones. It is less dense and undergoes more stress remodeling. Clinically, bone mineral density (BMD) studies measure areas containing mostly cancellous bone (vertebral bodies, femoral trochanter, and sacrum) because of its earlier and higher rate of bone turnover and its greater likelihood of demonstrating changes in BMD.

There are three major types of bone cells: osteoblasts, osteocytes, and osteoclasts. Osteoblasts are derived from undifferentiated mesenchymal cells. They line the surface of bones and function primarily to produce bone matrix (type I collagen and osteocalcin). Osteoblasts have receptors for parathyroid hormone (PTH) and 1,25-dihydroxyvitamin D [1,4]. Generally, these hormones function systemically through an osteoblastic mediator. PTH directly inhibits osteoblastic formation of osteocalcin, whereas 1,25-dihydroxyvitamin D stimulates osteocalcin formation. Locally, an osteoblast is stimulated by several growth factors, including transforming growth factor β -1, -2, and -3; bone morphogenic proteins 1 through 7; insulin-like growth factors I and II; and acidic and basic fibroblast growth factor [1,4].

Osteocytes are former osteoblasts that have become surrounded with bone mineral matrix (calcified bone). Osteocytes function to maintain bone and control extracellular concentrations of calcium and phosphorus. The final cell type, osteoclasts, is derived from hematopoietic precursors and functions to resorb bone. Osteoclasts bind to the bone surface and resorb an isolated area of bone by dissolving the hydroxyapatite crystals and digesting the collagen. Osteoclasts have specific receptors for calcitonin and, when bound, calcitonin directly inhibits bone resorption. Osteoclasts do not have receptors for PTH or 1,25-dihydroxyvitamin D and are therefore stimulated indirectly by these hormones through an osteoblast-mediated mechanism to increase bone resorption [1].

The macroscopic composition of bone differs depending on anatomic site, age, diet, and disease. Generally, the mineral, or inorganic, phase accounts for 60% of the tissue, the organic phase accounts for 35%, and water accounts for the

remaining 5%. The mineral or inorganic phase consists of crystalline calcium hydroxyapatite ($\text{Ca}_{10} [\text{PO}_4]_6 [\text{OH}]_2$). It is responsible for the compressive strength of bone. The organic phase consists of 90% type I collagen and is responsible for the tensile strength of bone. The remainder of the organic phase consists of proteoglycans, matrix proteins, growth factors, and cytokines.

Generally, the expected age range of peak bone mass accrual is between 25 and 30 years [4]. After this age period, both men and women gradually lose bone mass. Women appear to acquire most of their bone mass at an earlier age than boys do (age 11–14 years compared with 13–17 years) [4,5]. Women who are postmenopausal or are hypoestrogenic for other reasons experience accelerated bone loss caused by increased bone resorption compared with formation. Therefore, female athletes who are hypoestrogenic during adolescence initially may accrue a lower peak bone mass, which may be an irreversible problem after a certain age [4,6,7]. In studies that used bone turnover markers, it has been observed that amenorrheic runners have a reduced bone turnover, especially in bone formation, than eumenorrheic runners do. This reduced turnover is believed to be linked to various endocrine abnormalities (including hypoestrogenemia), low body mass index, and low energy intake relative to expenditure [8]. Recent studies suggest that it may be the altered energy balance that ultimately causes this imbalance in bone homeostasis. Both chronic undernutrition and acute dietary energy restrictions have been found to be accompanied by reduced bone formation. The latter also has been associated with depressed levels of IGF-I, a hormone that has been shown to stimulate the production of type I collagen [9–11]. The uncoupling of bone formation and resorption can be seen with restricted energy availability of as little as 30 kcal/kg of lean body mass/d [12].

Bone Remodeling

According to Wolff's law, bone has a cellular and molecular remodeling response to applied mechanical stress. The bony adaptation is a function of the number of loading cycles, cycle frequency, and the amount of strain, strain rate, and strain duration per cycle [13]. Both cortical and cancellous bone remodel continuously by osteoclastic and osteoblastic activity. This remodeling occurs throughout life and is affected by multiple factors including metabolic state, nutritional status, menstrual patterns, age, gender, level of fitness, and ethnicity. Bone also responds to piezoelectric changes, such that tensile forces create electropositivity and thereby stimulate osteoclastic activity [13], whereas compressive forces create electronegativity and thereby stimulate osteoblastic activity. Most cortical stresses in nature are tensional. Torsion or twisting provides tension circumferentially, whereas bending produces tension on the convex side and compression on the concave side. Tensional forces have been shown to result in microfracture and debonding at cement lines [14].

Stress Injury

A stress injury occurs on a continuum from normal bone remodeling and repair to frank cortical fracture. Overall bone health depends on mechanical, hor-

BOX 1: INITIATION OF STRESS FRACTURE

1. Accumulation of microtrauma from repetitive loading of bone (fatigue failure)
2. Fatigue failure persists, leading to crack initiation
3. If initial failure is inadequately repaired, it can lead to more loading and crack propagation

monal, nutritional, and genetic factors. The susceptibility of bone to fracture under fluctuating stresses is related to the crystalline structure and collagen orientation of the osteon. Fatigue load under certain strain rates can cause a progressive accumulation of microdamage (crack initiation) (Box 1) (Fig. 1) [15]. When such a process is prolonged, bone eventually may fail through crack propagation. Bone simultaneously repairs these cracks by new bone formation at their tips, thereby decreasing the chance for propagation.

A stress injury of bone is the result of either excessive bone strain with the accumulation of microdamage and the inability to keep up with appropriate skeletal repair (fatigue reaction or fracture) or depressed bony remodeling in response to normal strain (insufficiency reaction or fracture). The former situation most likely occurs in athletes and military recruits. The latter most likely occurs with the female athlete triad, metabolic bone disease, and osteoporosis. Sacral insufficiency stress fractures have been found to occur in female runners and often mimic the presentation of sacroiliitis. There may also be a component of reperfusion injury following prolonged strenuous exercise that results in bone tissue ischemia. This may help explain how some stress fractures occur in cortical bone areas of lower strain and when intracortical osteopenia precedes evidence of microscopic cracks [16].

An additional consideration in the athletic population is the relationship between training regimens and stress injury. Muscles exert a protective effect

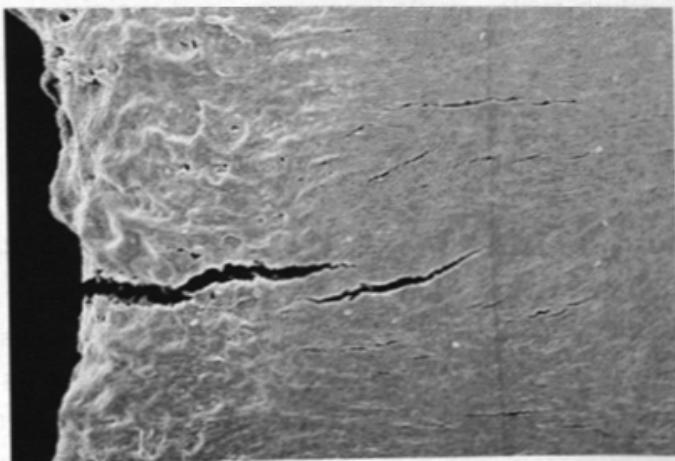


Fig. 1. Example of crack initiation in bone. (From Nalla RK, Kinney JH, Ritchie RO. Letters: mechanistic fracture criteria for the failure of human cortical bone. *Nat Mater* 2003;2:164–8; with permission.)

on cortical bone by acting as the major shock absorbers. With muscle contraction, cortical bone surface bending strains are reduced [17,18]. In most weight-bearing bones, it is believed that, with muscle fatigue, the shock-absorbing effect is lessened and more force is transmitted directly to bone, thereby increasing the likelihood of microdamage accumulation. In nonweight-bearing and some weight-bearing bones, repetitive contraction of muscle at its insertion may generate enough force to cause stress-induced injury [19].

EXTRINSIC AND INTRINSIC FACTORS LEADING TO STRESS FRACTURES

There are numerous factors contributing to the risk of stress fractures. However, many of these proposed factors remain unproven. Additionally, many risk factors for stress fractures are interrelated and are methodologically difficult to analyze independently.

The cause of stress fractures is multifactorial, and individual athletes vary in their susceptibility to stress injury. These factors are divided into extrinsic factors (characteristics of the environment in which the athlete trains or competes) and intrinsic factors (characteristics of the athlete him- or herself).

Extrinsic Factors

Training regimen

A high training volume is a major risk factor in stress fracture development. Multiple studies in runners have demonstrated that higher weekly running mileage correlates with an increased incidence of stress fractures [20] and overall running injuries [21–23]. Ballet dancers who train for more than 5 hours per day have a significantly higher risk for stress fractures than those who train for less than 5 hours daily [24].

Abrupt or rapid changes in duration, frequency, or intensity of training programs also increase an athlete's risk of stress fracture. Reducing the intensity or frequency of the training program leads to fewer stress fractures in female and male military recruits [25–27]. However, this intervention has not been studied in athletes.

Footwear

Athletic footwear is designed to reduce the impact on ground contact and to provide stability by controlling foot and ankle motion [28]. Shoe age has been shown to be a better indicator of shock-absorbing quality than shoe cost. Gardner and colleagues [29] have shown that training in shoes older than 6 months increases the risk for stress fracture. However, there has been no association between shoe cost and stress fracture risk [29]. A woman's foot has a wider forefoot-to-hindfoot ratio, which can result in poor shoe fit, leaving the hindfoot relatively unsupported. Custom-made biomechanical shoe orthoses that place the foot in a neutral subtalar position as well as absorb shock have been shown to lower the overall incidence of stress fractures in infantry recruits [30,31]. However, this may not be applicable to running athletes [32].

Training surface

The surface on which an athlete trains may also contribute to the risk of stress fracture. Theoretically, training on uneven surfaces could increase the risk of stress fracture by causing increased muscle fatigue and redistributing the load to bone. Hard or less compliant surfaces, such as cement, could also increase stress fracture risk through higher mechanical forces being transmitted to bone during impact. It is difficult to control for and quantify training surface in observational or prospective studies; however, a correlation has been demonstrated in some studies [21,33], whereas other studies have shown no effect [20,23]. One small study found that treadmill runners were at a lower risk for developing tibial stress fracture but were also less likely to achieve tibial bone strengthening than overground runners [33].

Type of sport

An Australian study by Goldberg and Pecora [34] in 1994 quantified the rate of stress fracture in men and women in different sports. In this study, the percentage of athletes per season who had stress fractures were: softball 6.3%, track 3.7%, basketball 2.9%, tennis 2.8%, gymnastics 2.8%, lacrosse 2.7%, baseball 2.6%, volleyball 2.4%, crew 2.2%, and field hockey 2.2%. Sprinters, hurdlers, and jumpers tend to have more foot fractures, whereas middle and long-distance runners have more long bone and pelvic fractures [35]. Rowers and golfers have been found to have increased rates of rib stress fractures [36,37].

INTRINSIC FACTORS

Demographic Factors

Gender

Most studies have found that women have a higher incidence of stress fractures compared with men. This higher incidence probably is multifactorial, secondary at least in part to gender-associated risk factors such as dietary deficiencies, menstrual irregularities, lower BMD, and narrower bone width. Gender differences in muscle physiology, especially neuromuscular control, also may be to blame because several studies have shown that women have a slower rate of force development in the muscle than men do [38–40].

In the United States military, the risk of stress fractures in female recruits undergoing the same training program as men is up to 10 times higher [4]. This increased risk also has been observed in athletic populations [34,41,42]. Bennell and colleagues [35] have reported no overall difference between stress fracture incidence in male and female athletes. However, the data appeared to show a trend for a higher risk of stress fractures in females when the amount of training hours were taken into account. Women, however, appear to have more femoral neck, metatarsal, and pelvic stress fractures than men do [43]. Further research is needed to determine whether the apparently higher incidence of stress fractures in women is independent of other known risk factors.

Age

The role of age as a risk for stress fractures in female athletes is not established. Studies in military recruits have been inconsistent, with some studies finding an increased risk of stress fractures with increasing age [44,45] and others finding a decreased risk [29,46–48] or no effect [49,50]. This lack of agreement is most likely caused by confounding factors such as previous physical activity level, hormonal status, BMD, and training level. Most studies in athletic populations have not found a correlation between age and stress fracture risk, although rigorous studies controlling for other possibly confounding variables are presently lacking.

Race

The incidence of stress fractures is significantly higher in white and Asian women than in African American women [29,44–46]. This is believed to be related to differences in bone turnover and peak bone density and not to race independently. It also appears that both ethnic differences in bone mineralization and bone integrity in athletes are mediated by heritable differences in titratable acid, sodium, and calcium excretion [51].

Aerobic fitness, muscle strength, and flexibility

Previously inactive or less active military recruits have a higher incidence of stress fractures compared with those who are active before beginning basic training [29,48,49]. There are several possible factors contributing to this, including decreased aerobic fitness, decreased muscle strength, lower endurance, and poor flexibility. A study of military recruits found no association between aerobic fitness (predicted $\text{VO}_{2\text{max}}$) and stress fracture risk [52]. It is unlikely that aerobic fitness alone accounts for the difference [53,54]. The role of flexibility on stress fractures has yet to be well defined [35,47,55].

BIOMECHANICAL FACTORS**Bone Mineral Density**

Lower BMD, especially of the femoral neck, has been shown to be associated with an increased risk of stress fractures in the female athlete [56]. Although there are case-control studies that both support [57] and refute [49] this finding, Bennell and colleagues [53] were the first to examine this condition prospectively. They found that lower BMD in the lumbar spine and foot were significant predictors of later stress fracture development in female track and field athletes. Of note, an athlete with an apparently normal BMD (caused by the increased bone loading of sport) may be at an increased risk of stress fracture if she falls below the mean among female athletes. Also, as observed by dual x-ray absorptiometry scanning in athletic females, cancellous bone stress fractures correlate with early onset osteopenia much more than cortical bone stress fractures do [58]. This indicates the necessity of bone density evaluation in any young female with cancellous stress fracture. Menstrual disturbance and lower BMD most likely are not risk factors independent of each other but are interrelated, with amenorrheic athletes having both lower BMD and higher stress fracture incidence [59].

Bone Geometry

The amount of force a bone can withstand is proportional to its cross-sectional area and moment of inertia (a measure of bone resistance to bending). Studies of military personnel have found these parameters to be significantly lower among those who develop stress fractures [60–62]. These studies also have found that of those who sustained femoral, tibial, or foot stress fractures, 31% had narrowed tibial width compared with those without fracture [63]. This narrowed tibial width may be an indicator of a biomechanically weaker skeletal structure. It is hypothesized that women are likely to have overall narrower bones than men [64], and this is a possible factor contributing to a higher incidence of stress fractures in female athletes.

ANATOMIC FACTORS

Foot Morphology

The structure of the foot helps to determine how much ground contact force is absorbed in the foot and how much is transferred to the bones of the leg and thigh. A rigid, high-arched foot (*pes cavus*) absorbs less stress and transmits greater force to the tibia and femur. A flexible, low-arched foot (*pes planus*) absorbs more force in the foot itself and transmits less to the tibia, fibula, and femur. One military study evaluating foot morphology found persons with the highest arches sustained 3.9 times as many stress fractures as those with the lowest arches [65]. Other studies [65,66] suggest that individuals with *pes cavus* seem to be at an increased risk of tibial and femoral stress fractures, whereas those with *pes planus* may sustain more metatarsal stress fractures. However, other studies have not found a significant correlation between foot structure and stress fracture risk [20,53]. It is possible that both *pes planus* and *pes cavus* foot structures may increase the risk of stress fracture at various sites, but this has not yet been adequately evaluated or proven.

Leg Length Discrepancy

Leg length discrepancy also has been associated with an increased risk of stress fractures in female athletes [20,53]. The degree of leg length difference may correlate with increasing stress fracture risk [67]. However, one study of male military recruits did not confirm this relationship [68]. It is reasonable to evaluate and correct significant leg length discrepancy in runners, especially those with other stress fracture risks.

Knee Alignment

Valgus knee alignment and a quadriceps angle greater than 15° may also increase the risk for tibial stress fracture [68,69].

HORMONAL FACTORS

Delayed Menarche

Female athletes generally reach menarche at a later age than female nonathletes do, particularly those in certain sports such as ballet, running, and gymnastics [70,71]. Delayed menarche may cause lower peak bone mass attainment or may

be a marker for other possible influences on stress fracture risk, such as low body fat, low body weight, future menstrual disturbance, or excessive training. The effect of this delay on bone health and the risk of stress fractures have not been well studied; however, some studies suggest osteopenia, stress fracture, and scoliosis may be potential complications of delayed menarche [53,71]. Scoliosis, in particular, has been observed in female ballet dancers with delayed menarche [71]. This may also lead to pelvic obliquity and relative leg length discrepancies and the potential for increasing the risk for stress fracture.

Menstrual Disturbance

Multiple studies have demonstrated that stress fractures occur more commonly in women with amenorrhea or oligomenorrhea than in eumenorrheic women [17,46,48,53,57,71]. Athletes with menstrual disturbances have low basal estrogen concentrations [72] and a lower BMD than eumenorrheic athletes do [73]. It has been hypothesized that estrogen deprivation increases the physiologic set point for bone modeling and remodeling, making it more difficult to activate the cellular response necessary to induce bone adaptation to stress [4,74] and increasing the risk of stress fractures. Health care providers, athletes, coaches, and parents need to be aware that menstrual disturbance is not simply a normal product of training and that such disturbances can have devastating consequences. Menstrual disturbances also are seen in association with disordered eating and endothelial cell dysfunction (as in the female athlete triad, discussed later). Therefore, athletes with menstrual irregularity should be further evaluated accordingly.

Contraception

Some studies have shown that oral contraceptive pills (OCP) seem to have a protective effect in preventing stress fractures in female athletes [17,75]. It appears that exogenous estrogen may help curb further bone loss in the hypoestrogenic amenorrheic athlete; however, it may not be sufficient to stimulate bone growth [71,76–79]. Several small studies of amenorrheic women or those with anorexia have found BMD at the lumbar spine or hip to be higher for women taking OCP compared with those who were not [76,77,80], whereas other studies show no significant change [71,78]. It has also been theorized that OCP may act through another mechanism, such as improving bone microarchitecture and quality without significantly affecting BMD [4]. However, to add to the controversy, a recent study by Hartard and colleagues [81] has shown that OCP use is associated with decreased BMD of the spine (7.9%) and the femoral neck (8.8%) in female endurance athletes compared with non-OCP users. The authors [81] also found that early age at initiation of OCP was an important risk factor for low peak bone mass in young women. Based on the conflicting results from research and the lack of well-controlled studies, it is difficult to assess the effects of OCP on skeletal health in normally menstruating women. In women who have menstrual disturbances, the use of OCP or another hormonal replacement therapy may be effective in preventing further bone loss; however, the resumption of menses may mask an underlying nutritional

disorder and provide a false sense of security. Recent evidence also suggests that depo-medroxyprogesterone may contribute to impaired bone accretion and low bone mineral density, and it should be avoided in young women [82].

NUTRITIONAL FACTORS

Low Calcium and Vitamin D Intake

Low calcium intake is associated with low BMD [83] and therefore may contribute to the development of stress fractures. Myburgh and colleagues [57] observed an association between decreased calcium intake and increased stress fracture risk. However, other studies found no association between calcium intake and stress fracture risk, with both stress fracture and nonstress fracture groups having normal calcium intake [13,84,85]. Athletes whose calcium intake is below the daily recommended value are likely to be at risk for stress fractures, but for those with normal dietary calcium intake, other factors play a larger role.

Vitamin D is also essential to bone health, and its functions include stimulating calcium transport, osteoblastic stimulation, and decreasing parathyroid hormone. Recent studies have focused on the role of the vitamin D receptor allele in predicting bone density. More research is necessary to determine the clinical applications of its use in screening [86–88].

Disordered Eating

Inadequate caloric intake relative to the energy expenditure required for exercise appears to be the primary mechanism by which female athletes are predisposed to menstrual dysfunction and detrimental effects on bone. Anorexia nervosa has been associated with a significantly decreased BMD [7,89]. Nearly 75% of adolescent girls with anorexia have a BMD more than two standard deviations below the normal value [90]. Not surprisingly, women with anorexia nervosa are at an increased risk for stress fracture development [91,92]. Interestingly, disordered eating is associated with low BMD in the absence of menstrual irregularities [59].

The Female Athlete Triad

The female athlete triad refers to an interrelated problem consisting of disordered eating, amenorrhea, and osteoporosis. Hoch and colleagues [93] have also found that amenorrheic athletes have reduced brachial artery endothelium-dependent flow-mediated vasodilation compared with oligomenorrheic and eumenorrheic athletes. Furthermore, in a 2-year follow-up study, the original amenorrheic athletes were found to have a significant improvement in BMD with different combinations of estrogen and progesterone or the return of menses naturally. The female athlete triad is a potentially lethal combination of medical disorders reported in some female athletes [4,42]. The athletes at greatest risk appear to be those who feel significant pressure to excel in sports for which leanness and a low body weight are considered advantageous, such

as gymnastics, figure skating, ballet, and distance running [94]. Also, athletes participating in individual sports are at higher risk than those in team sports.

The problem usually begins with disordered eating, which includes a spectrum of abnormal and harmful eating patterns such as binging and purging, restrictive eating, fasting, and the use of diet pills or laxatives. The preoccupation with food, a distorted body image, and intense fear of becoming fat are often present as well. Some athletes will meet the Diagnostic and Statistical Manual of Mental Disorders, revised edition IV [95], criteria for anorexia nervosa or bulimia, whereas other athletes may display similar behaviors without meeting full diagnostic criteria. A new classification of eating disorder not otherwise specified (ie, ED-NOS) allows for the identification of women who do not meet other classification criteria. This classification has been helpful in this population because the athlete's weight may seem adequate as a result of increased lean tissue mass; however they are not consuming enough calories to meet their energy needs.

Abnormal eating patterns may lead to athletic-associated amenorrhea. Athletic amenorrhea is a complex multifactorial condition with serious associated comorbidities. Extreme caloric restriction, excessive exercise, physical and emotional stress associated with exercise and competition, percentage of body fat, and genetics contribute to the condition. There is, however, increasing evidence that suggests nutritional restrictions and the resulting endocrine and metabolic changes are a critical initiator of hypothalamic-induced athletic amenorrhea and osteoporosis [96].

Disordered eating, estrogen deficiency, and menstrual dysfunction predispose women to the third component of the triad, osteoporosis [89]. Reduced BMD in premenopausal women appears to be irreversible, despite weight gain, resumption of menses, or estrogen replacement [6,7]. One study found that, with the resumption of menses, there was a significant increase in vertebral BMD. However after 2 years of normal menses, BMD remained below the age-normative level. Slemenda and colleagues [97] have shown that the low estrogenic state associated with amenorrhea has a more profound effect on cancellous bone than on cortical bone. Cancellous bone is found in a higher percentage in the pelvis, sacrum, and femoral neck areas where female athletes tend to have a higher occurrence of stress fractures. These factors put the female athlete who is experiencing the triad complex at a significant risk for stress fractures. Although some investigators have found that weight-bearing exercise has a skeletally protective effect and may attenuate the bone loss usually seen in anorexics [89], the use of excessive training to control weight could also contribute to the increased risk of stress fractures associated with the female athlete triad.

Several other factors are known to increase the risk for osteoporosis, but these factors have not been thoroughly investigated as possible risks for stress fractures in female athletes. These other factors include smoking, caffeine consumption, and certain medications such as thyroid hormone and corticosteroids. In a study of female army recruits, it was found that current or past smoking, alcoholic consumption of ≥ 10 drinks per week, corticosteroid use, use of

depo-medroxyprogesterone acetate, lower adult weight, and no history of regular exercise increased the likelihood of stress fracture [45,82].

SUMMARY

Stress fractures can occur in any active individual, from the weekend warrior to the elite athlete. As these injuries occur, it is important to understand how bones respond to the stresses placed on them. The understanding of potential intrinsic and extrinsic causes is important in treatment of these injuries. The proper identification and prevention of these stress injuries allows for athletes to return to activity expeditiously.

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