

## Stress Fractures and Knee Injuries in Runners

Anne Z. Hoch, DO, PT<sup>a,\*</sup>, Michelle Pepper, MD<sup>b</sup>,  
Venu Akuthota, MD<sup>c</sup>

<sup>a</sup>*Department of Orthopaedic Surgery, Medical College of Wisconsin, Froedtert East Clinics,  
Fifth Floor, 9200 West Wisconsin Avenue, Milwaukee, WI 53226, USA*

<sup>b</sup>*Medical College of Wisconsin, 9200 West Wisconsin Avenue, Milwaukee, WI 53226, USA*

<sup>c</sup>*Department of Physical Medicine and Rehabilitation University of Colorado School of  
Medicine, P.O. Box 6510, Mail Stop F721, Aurora, CO 80045, USA*

The increase of athletics in the lives of women has been muted by their rate of lower limb musculoskeletal injury. Stress fractures and knee overuse injuries, in particular, have become epidemic in female running athletes. The putative “female athlete triad” and subsequent bone demineralization has led to higher rates of stress fractures among women. Knee injuries, including patellofemoral pain and iliotibial band syndrome, are extremely common in sports medicine and occur with a much higher incidence in female runners. These entities are discussed in detail in this following article.

### Stress fractures

#### *Bone basic science*

Injury to bone encompasses an array of defects of bone architecture from bone strain to stress reaction to nondisplaced stress fracture to displaced fracture. Essentially, these injuries occur 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 etiologies and treatment principles for stress fractures. Bone is a highly organized and dynamic living tissue with metabolic and structural

---

\* Corresponding author.

E-mail address: [azeni@mcw.edu](mailto:azeni@mcw.edu) (A.Z. Hoch).

components. These components are interdependent and responsive to each other. The metabolic component involves mineral homeostasis and bone remodeling, whereas the structural component involves maintaining skeletal integrity and bone remodeling.

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

Normal lamellar bone is organized structurally into cortical (compact) bone or cancellous (trabecular) bone. Cortical bone makes up 80% of the skeleton and is composed of tightly packed osteons or 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 cuboidlike bones, such as vertebral bodies or tarsal and carpal bones. Cortical bone is characterized by eight times slower metabolic turnover compared with cancellous bone and four times greater mass. Most stress fractures in runners occur in cortical bone (Box 1).

Cancellous (trabecular) bone is found principally at the metaphysis and epiphysis of long bones and in cuboidlike bones. It is less dense and undergoes more stress remodeling. Clinically, bone mineral density (BMD) studies measure areas that contain mostly cancellous bone (vertebral bodies, femoral trochanter, and sacrum) because of its earlier/greater rate of bone turnover and its greater likelihood of demonstrating a change 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]. In general, these hormones function systemically by way of 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- $\beta$ -1, -2, and -3; bone morphogenic proteins 1-7; insulin-like growth factors I and II; and acidic and basic fibroblast growth factor [1,4].

**Box 1. Frequent sites of stress fractures in runners (by order of incidence)**

Tibia  
Metatarsal  
Fibula  
Navicular

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 function to resorb bone. They 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 therefore, are stimulated indirectly by these hormones through an osteoblast-mediated mechanism to increase bone resorption [1].

The macroscopic composition of bone differs depending on site, age, diet, and disease. In general, 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.

In general, the expected age of peak bone mass accrual is between 25 and 30 years [4]. After this age, men and women gradually lose bone mass. Girls seem to acquire most of their bone mass at an earlier age than boys (age 11–14 years compared with 13–17 years) [4,5]. Women who are postmenopausal or hypoestrogenic for other reasons have accelerated bone loss that is due to increased bone resorption compared with formation. Therefore, female athletes who are hypoestrogenic during adolescence can accrue a lower peak bone mass, which might be an irreversible problem after a certain age [4,6,7]. Using bone turnover markers, it was observed that amenorrheic runners have a reduced bone turnover, especially in bone formation, compared with eumenorrheic runners. This is believed to be linked to the various endocrine abnormalities (including hypoestrogenemia), low body mass index, and low energy intake relative to expenditure [8]. Recent studies suggest that the altered energy balance ultimately may cause this imbalance in bone homeostasis. Chronic undernutrition and acute dietary energy restriction are accompanied by reduced bone formation. The latter also has been associated with depressed levels of insulin-like growth factor 1, a hormone that was shown to stimulate the production of type I collagen [9–11]. Uncoupling of bone formation and resorption can be seen with restricted energy availability as little as 126 kJ/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, amount of strain, strain rate,

and strain duration per cycle [13]. 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, sex, level of fitness, and ethnicity. Bone also responds to piezoelectric changes. Tensile forces create electropositivity, and thereby, stimulate osteoclastic activity [13]. Compressive forces create electronegativity, and thereby, stimulate osteoblastic activity. Most cortical stresses in nature are tension. Torsion or twisting provides tension circumferentially, whereas bending produces tension on the convex side and compression on the concave side. Tension forces were shown to result in microfracture and debonding at cement lines [14].

Clinically, this concept becomes important in the treatment of femoral neck stress fractures. Compression-side injuries (on the medial inferior cortex of the femoral neck) heal much better than traction-side stress injuries (on the lateral superior cortex of the femoral neck). Because of their robust ability to heal, nondisplaced compression-type stress fractures typically are treated with nonweight bearing until the patient becomes pain-free, which may take several days to weeks. Tension-type femoral neck stress fractures may fail to heal adequately with serious sequela, such as avascular necrosis, malunion or nonunion, or varus deformity. Thus, tension- side fractures often require internal fixation.

### *Stress injury*

Stress injury occurs on a continuum from normal bone remodeling and repair to frank cortical fracture. Overall bone health depends on mechanical, hormonal, nutritional, and genetic factors. The susceptibility of bone to fracture under fluctuating stresses is related to the crystal structure and collagen orientation of the osteon. Fatigue load under certain strain rates can cause progressive accumulation of microdamage [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, and thereby, decreases the chance for propagation.

Stress injury of bone is the result of excessive bone strain with accumulation of microdamage and inability to keep up with appropriate skeletal repair (fatigue reaction/fracture), or depressed bony remodeling in response to normal strain (insufficiency reaction/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 also may be a component of reperfusion injury following prolonged strenuous exercise that results in bone tissue ischemia. This may help to 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 training regimens and stress injury. Muscles exert a protective effect on cortical bone by acting as the major shock absorber. 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 which increases the likelihood of microdamage accumulation. In nonweight-bearing and some weight-bearing bones, repetitive contraction of the muscle at its insertion may generate enough force to cause stress-induced injury [19].

### *Etiology of stress fractures*

Numerous factors contribute to the risk of stress fractures in runners, particularly female athletes; however, many of these proposed etiologies remain unproven. Much of the research that investigates stress fracture risks has been done in male subjects and military recruits; therefore, findings may not necessarily be generalized to female athletic populations. Furthermore, many risk factors for stress fractures are interrelated and methodologically are difficult to analyze independently.

The etiology of stress fractures is multifactorial; individual athletes vary in their susceptibility to stress injury. Risk factors (Box 2) are divided into extrinsic (characteristics of the environment in which the athlete trains or competes) and intrinsic (characteristics of the athlete herself) types.

### *Extrinsic risk factors*

*Training regimen.* High training volume is a major risk factor in stress fracture development. Multiple studies in runners demonstrated that higher weekly running mileage correlates with 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 stress fracture risk 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 led 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 impact on ground contact and provide stability by controlling foot and ankle motion [28]. Shoe age is a better indicator of shock-absorbing quality than shoe cost. Gardner et al [29] showed that training in shoes that are 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 greater forefoot to hindfoot ratio, which can result in poor shoe fit and leave the hindfoot less

**Box 2. Risk factors for stress fractures***Extrinsic*

Training regimen  
Footwear  
Training surface  
Type of sport

*Intrinsic*

## Demographic factors

Gender  
Age  
Race  
Aerobic fitness  
Muscle strength  
Flexibility

## Biomechanical factors

Bone mineral density  
Bone geometry

## Anatomic factors

Foot morphology  
Leg length discrepancy  
Knee alignment

## Hormonal factors

Delayed menarche  
Menstrual disturbance  
Contraception

## Nutritional factors

Low calcium and vitamin D intake  
Disordered eating

supported. Custom made biomechanical shoe orthoses that place the foot in neutral subtalar position and absorb shock decreased 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 also may contribute to her risk of stress fracture. Theoretically, training on uneven surfaces could increase the risk of stress fracture by causing increased muscle fatigue and redistributing load to bone. Hard or less compliant surfaces, such as cement, also could 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 was demonstrated in some studies [21,33], whereas

others showed no effect [20,23]. One small study found treadmill runners to be at lower risk for developing tibial stress fracture, but also less likely to achieve tibial bone strengthening, than overground runners [33].

*Type of sport.* An Australian study 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 as follows: 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% [34]. Sprinters, hurdlers, and jumpers tended to have more foot fractures, whereas middle and long-distance runners had more long bone and pelvic fractures [35]. Rowers and golfers have increased rates of rib stress fractures [36,37].

### *Intrinsic risk factors*

*Demographic factors.* Most studies have found that women have a higher incidence of stress fractures. It is probably multifactorial, and 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. Several studies have shown that women have a slower rate of force development in the muscle [38–40].

In the U.S. military, the risk of stress fractures in female recruits who undergo 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 et al [35] reported no difference in the overall stress fracture incidence between male and female athletes; however, the data seem to show a trend for a higher risk of stress fractures in women when the amount of training hours were taken into account. Women, however, seem to have more femoral neck, metatarsal, and pelvic stress fractures than men [43]. Further research is needed to determine whether the apparent higher incidence of stress fractures in women is independent of other known risk factors.

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 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 due to 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 that controlled for other possible confounding variables are lacking.

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 seems that ethnic differences in bone mineralization and bone integrity in athletes are mediated by heritable differences in titratable acid, sodium, and calcium excretion [51].

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]. Several possible factors include 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 not been well-defined [35,47,55].

*Biomechanical factors.* Lower BMD, especially of the femoral neck, was associated with an increased risk of stress fractures in the female athlete [56]. Although there are published case-control studies that support [57] and refute [49] this finding, Bennell and colleagues [53] were the first to examine this 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 (due to the increased bone loading of sport) may be at increased risk of stress fracture if she falls below the mean among female athletes. It also was observed in athletic females that cancellous stress fractures correlate with early onset osteopenia by Dual Energy Xray Absorptiometry scan much more so than cortical bone stress fractures [58]. This indicates the necessity of bone density evaluation in any young woman who has a cancellous stress fracture. Menstrual disturbance and lower BMD most likely are not risk factors that are independent of each other, but are interrelated; amenorrheic athletes have lower BMD and higher stress fracture incidence [59].

The amount of force that a bone can withstand is proportional to its cross-sectional area and cross-sectional moment of inertia (a measure of bone resistance to bending). Studies of military personnel found these parameters to be significantly lower among those who develop stress fractures [60–62]. They also found that of persons 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 biomechanically weaker skeletal structures. It is hypothesized that women are likely to have overall narrower bones than men [64]; this is a possible contributing factor to the higher incidence of stress fractures in women.

*Anatomic factors.* 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 that evaluated foot morphology found that persons who had the highest arches sustained 3.9 times as many stress fractures as those who had the lowest arches [65]. Other studies [65,66] suggest that individuals who have pes cavus seem to be at increased risk of tibial and femoral stress fractures, whereas those who have pes planus may sustain more metatarsal stress fractures. Other investigators have not found a significant correlation between foot structure and stress fracture risk [20,53]. It is possible that pes planus and pes cavus foot structure may increase the risk of stress fracture at various sites, but this has not been evaluated adequately or proven.

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.

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

*Hormonal factors.* Female athletes, in general, reach menarche at a later age than nonathletes, 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 risk of stress fractures is not well-studied; however, some studies suggest that 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 also may lead to pelvic obliquity and relative leg length discrepancies and the potential for increasing the risk for stress fracture.

Multiple studies have demonstrated that stress fractures occur more commonly in women who have amenorrhea or oligomenorrhea than in eumenorrheic women [17,46,48,53,57,71]. Athletes who have menstrual disturbances have low basal estrogen concentrations [72] and a lower BMD than eumenorrheic athletes [73]. It was hypothesized that estrogen deprivation increases the physiologic set point for bone modeling and remodeling and makes it more difficult to activate the cellular response that is necessary to induce bone adaptation to stress [4,74] and increases the risk of stress fractures. Health care providers, athletes, coaches, and parents need to be aware that menstrual disturbance is not 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 (see “The female athlete Triad,” below). Therefore, athletes who have menstrual irregularity should be evaluated further.

Some studies have shown that oral contraceptive pills (OCPs) have a protective effect in preventing stress fractures in female athletes [17,75]. It seems that exogenous estrogen may help to 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 among amenorrheic women or those who had anorexia found that BMD at the lumbar spine or hip was higher for those who were taking OCPs compared with those who were not [76,77,80], whereas others showed no significant change [71,78]. It also was theorized that OCPs may act through another mechanism, such as improving bone microarchitecture and quality without significantly affecting BMD [4]. To add to the controversy, a recent German study by Hartard et al [81] showed 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. They also found that early age at initiation of OCPs 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 OCPs on skeletal health in normally menstruating women. In those who have menstrual disturbances, OCPs or other hormonal replacement therapy may be effective in preventing further bone loss; however, resumption of menses may mask an underlying nutritional disorder and provide a false sense of security. Recent evidence also suggests that depomedroxyprogesterone may contribute to impaired bone accretion and low BMD, and it should be avoided in young women [82].

*Nutritional factors.* 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. Other studies found no association between calcium intake and stress fracture risk; groups that did and did not have stress fractures had normal calcium intake [13,84,85]. Athletes whose calcium intake is less than the daily-recommended value are likely to be at risk for stress fractures, but for those who have a normal dietary calcium intake, other factors play a larger role.

Vitamin D also is essential to bone health and functions, including stimulating calcium transport, osteoblastic stimulation, and decreasing parathyroid hormone. Recent studies focus 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].

Inadequate caloric intake relative to energy expenditure that is required for seems 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 who had anorexia had a BMD that was more than two standard deviations below the normal value [90]. Not

surprisingly, women who have anorexia nervosa are at increased risk for stress fracture development [91,92]. 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 that consists of disordered eating, amenorrhea, and osteoporosis. Hoch et al [93] also found that amenorrheic athletes had reduced brachial artery endothelium-dependent flow-mediated vasodilation when compared with oligomenorrheic and eumenorrheic athletes. Furthermore, in a 2-year follow-up study, the original amenorrheic athletes had a significant improvement in BMD with different combinations of estrogen and progesterone or return of menses naturally. This female athlete triad is a potentially lethal combination of medical disorders that is reported in some female athletes [4,42]. Athletes who are at greatest risk seem 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 who participate in individual sports are at higher risk than those who participate in team sports.

The problem usually begins with disordered eating; this includes a spectrum of abnormal and harmful eating patterns, such as bingeing and purging, restrictive eating, fasting, and the use of diet pills or laxatives. 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, Fourth Edition* criteria for anorexia nervosa or bulimia, whereas others may display similar behaviors without meeting full diagnostic criteria. A new classification—eating disorder, not otherwise specified—allows for identification of women who do not meet other classification criteria. This has been helpful in this population because the athlete's weight may seem to be adequate because 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 that are associated with exercise/competition, percentage of body fat, and genetics contribute to the condition. There is increasing evidence, however, that nutritional restrictions and the resulting endocrine and metabolic changes are a critical initiator of hypothalamic-induced athletic amenorrhea and osteoporosis [95].

Disordered eating, estrogen deficiency, and menstrual dysfunction predispose women to the third component of the triad, osteoporosis [89]. Reduced BMD in premenopausal women seems 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 age-normative. Slemenda et al [96] showed that the low estrogenic state that is 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 females tend to have a higher occurrence of stress fractures. These factors put the woman who suffers from the female athlete triad at significant risk for stress fractures. Although some investigators found that weight-bearing exercise has a skeletal protective effect and may attenuate the bone loss that usually is seen in anorexics [89], the use of excessive training to control weight also could contribute to the increased risk of stress fractures that is associated with the female athlete triad.

Several other factors are known to increase the risk for osteoporosis but have not been investigated thoroughly as possible risks for stress fractures in female athletes. These include smoking, caffeine consumption, and certain medications, such as thyroid hormone and corticosteroids. In a study of female army recruits, current or past smoking, consumption of more than 10 alcoholic drinks/week, corticosteroid use, use of depomedroxyprogesterone acetate, lower adult weight, and no history of regular exercise increased the likelihood of stress fracture [45,82].

### *Screening and prevention of stress injury in female runners*

Ideally, stress fractures and the female athlete triad are treated best through prevention. With many of the risk factors for stress fracture now known, screening and prevention are much easier. Evaluation of all components of the triad should be performed on every female runner. A thorough history is essential and should include assessments of nutrition; amount of, and changes in, physical activity; and menstrual history. A history of stress fractures or overuse injuries as well as signs or symptoms of an eating disorder justify further evaluation. Characteristics of anorexia nervosa include cachexia, bradycardia, hypotension, lanugo, hypothermia, cold intolerance, yellow skin (hypercarotenemia), dry hair and skin, alopecia, and pruritus. Signs and symptoms of bulimia include fatigue, abdominal pain, chest pain, swollen parotid glands, sore throat, eroded tooth enamel, and knuckle calluses. Screening with bone density testing should be considered in athletes who have known risk factors. The diagnosis of osteoporosis in premenopausal women should not be made on the basis of densitometric criteria alone. It also is not appropriate to use T-scores when peak bone mass has not been reached; therefore, athletes who are 20 years old and younger should be evaluated with Z-scores [97]. Treatment of the triad often requires a team approach. A sports physician, dietitian, and psychologist may be needed to work with the athlete in addition to her coach, parents, and close friends. Treatment for underlying disordered

eating, correction of an energy deficit, and restoration of menses are essential to stimulate bone accretion. Nutritional supplementation with calcium and vitamin D and reduced training may be recommended. Early intervention speeds recovery.

Reductions in bone density and an increased risk of stress fracture are noted when there is an uncoupling of bone formation and resorption rates as seen when energy availability is too low [59]. Several metabolic hormones that influence bone formation (IGF-1, T<sub>3</sub>, leptin), as well as bone formation markers (serum type I procollagen carboxyl-terminal propeptide, osteocalcin) and bone resorption markers (urine N-telopeptide, serum C-telopeptide), can be followed to form an impression of the overall bone turnover status and assess energy status indirectly. Uncoupling of bone resorption and formation is seen with restricted energy availability as little as 126 kJ/kg of lean body mass/d [12]. Therefore, increasing caloric intake to offset the high energy demand may help to restore menses and stimulate bone accretion. Calcium intakes of 1200 mg/d to 1500 mg/d and 400 IU to 800 IU of vitamin D also help to minimize bone loss. Antiresorptive therapies, such as bisphosphonates and calcitonin, have not been tested and are not approved by the U.S. Food and Drug Administration in younger patients who have reduced BMD/osteoporosis. Their use in this population is controversial; they are teratogenic and have a half-life in bone of greater than 10 years.

Treatment with exogenous estrogens has been evaluated in numerous studies with varying results. It seems that this may help to reduce further bone loss in hypoestrogenic amenorrheic athletes; however, it may be insufficient to stimulate bone growth [79]. Another concern is that with the restoration of menses, estrogen replacement may mask the underlying disorder. Recent evidence suggests that depomedroxyprogesterone acetate contributes to impaired bone accretion and loss in mean BMD and should be avoided in amenorrheic athletes [82]. Other preventative measures include not smoking and the minimal use of alcohol [45].

Because training errors frequently are the cause of stress fractures, any abrupt increase in training should be avoided. Cyclical training—a limited period of training followed by relative rest during the third week (the interval of greatest skeletal vulnerability)—is recommended over progressive training [25]. For runners, mileage increases should be gradual, with some investigators recommending that mileage should not be increased by more than 3.2 total km/wk [98]. Using shock-absorbing insoles and changing shoes every 6 months or before 800 km also is recommended.

### *Diagnosis of stress fractures*

The treatment of stress fractures starts with a specific and accurate diagnosis. Diagnosis of stress fractures often can be difficult and requires a high degree of suspicion. A thorough history and physical examination often diagnoses most stress fractures successfully. The onset of pain with an

abrupt change in training, such as increasing mileage or intensity, should raise suspicion of a stress fracture. Pain typically occurs at the end of, or after, a run. Point tenderness on physical examination is the hallmark. For example, the so-called “N-spot” on the dorsum of the navicular can be a site of exquisite tenderness in runners who have navicular stress fractures. Swelling and redness may be present, but deformity usually is not. Percussion tapping of bone, vibrating tuning fork, single leg hop, femoral fulcrum, and lumbar extension tests (for pars stress fracture) are nonspecific physical examination maneuvers that help in the identification of stress fractures. Application of stress to the area or passive stretching also can reproduce pain [99]. If multiple or recurrent stress fractures are present, evaluation for sources of metabolic bone disease and the female athlete triad is warranted. Some stress fractures are considered to be at high risk (**Box 3**) for nonunion and should be treated aggressively, often with a period of nonweight bearing, and perhaps, surgery [106]. These high-risk fractures include ones at the femoral neck, the mid shaft of the tibia, and the navicular.

The most common location of stress fractures in runners is the tibia; however, tibial stress fractures may be difficult to discern from so-called “shin splints,” more properly termed medial tibial stress syndrome (MTSS). Typically, pain with stress fractures worsens as the run goes on. Occasionally with MTSS, pain may diminish as the runner warms up. Physical examination reveals focal tenderness with tibial stress fractures, whereas MTSS presents with diffuse tenderness (with maximal tenderness at the junction between the middle and distal third of the medial tibia). Percussion tenderness, a tuning fork test, or hop tests are more likely to be

**Box 3. High-risk stress fractures that require aggressive treatment**

Femoral neck  
Anterior cortex or midshaft of tibia  
Navicular  
Medial malleolus  
Talus fracture extending to subtalar joint  
Proximal second metatarsal  
Proximal fifth metatarsal diaphysis (Jones fracture)  
Sesamoid  
Pars interarticularis

---

*Data from* Fredericson M, Bergman AG, Matheson GO. [Stress fractures in athletes] [German]. *Orthopade* 1997;26(11):961.

positive with tibial stress fractures than with MTSS. Posterior cortex tibial stress fractures can present with calf pain rather than shin pain.

A definitive diagnosis of a stress fracture often requires imaging. Plain radiographs have poor sensitivity but are highly specific and are recommended as an initial step in diagnosis. The “gray cortex” sign may be evident on the radiograph if the cortex has decreased density with associated hyperemia, edema, and early resorption [106]. The “dreaded black line” also can be seen on radiograph in individuals who have a midshaft tibia fracture that has failed to heal and developed a nonunion [106]. Many of these will be falsely negative as callus formation can take up to 3 months to appear. If suspicion is high with negative radiographs, conservative treatment should be initiated followed by repeat evaluation and radiographs in 2 weeks. If the injury is in the season of competition, it is reasonable to perform further imaging before implementing the 2-week rest.

MRI and bone scintigraphy (triple phase bone scan) have comparable sensitivity and either is acceptable. MRI is preferred because it is less invasive; avoids radiation exposure (an important consideration in this young female population); has better specificity (ie, can differentiate fracture from tumor); and shows more diagnostic information, such as fracture line and periosteal edema. MRI can show stress changes, or edema, within the bone, even before a fracture develops. Fat-suppressed T2-weighted images also are recommended to help identify any bone marrow edema. Areas of increased activity by scintigraphy seem to be consistent with grades of MRI [99,100]. The main drawback of MRI, aside from cost, is that it does not image cortical bone as well as CT. CT is useful in differentiating conditions that may mimic stress fracture on bone scan, such as osteoid osteoma, osteomyelitis with Brodie’s abscess, and various malignancies. It is also helpful in detecting fracture lines as evidence of stress fractures and often can differentiate between stress fracture and stress reaction [101]. This is particularly important in the elite athlete as it may affect their rehabilitation program and upcoming competitions considerably. Ultrasound is not reliable in diagnosing stress fractures and is not recommended [102].

### *Conservative treatment for stress fractures*

Conservative treatment for stress fractures is a three-part protocol using pain as a guide to rehabilitation. Phase 1 includes cessation of painful activity, ice, analgesics, maintaining fitness by cross-training, modification of risk factors, and possible bracing or electrical stimulation [101]. It is important that the runner who has a stress fracture be able to maintain aerobic fitness and strength during the healing period. The most common ways to unweight the fractured extremity and maintain cardiovascular fitness are cycling, water running, and swimming. Cross-training should be as sport specific as possible in duration and intensity. Extrinsic factors, especially training errors, need to be addressed because these are the most

common cause of stress fractures. Particularly important to female runners are intrinsic conditions, such as disordered eating, amenorrhea, and premature osteoporosis. Moreover, certain stress fractures occur in areas of hypovascularity and are at risk for nonunion or avascular necrosis; in these cases, surgery should be considered.

Adjunctive therapies, such as pneumatic braces or capacitative coupling, also may be instituted. The use of pneumatic braces in the rehabilitation of tibial stress fractures seems to reduce the time to recommencing training in athletes and military personnel; it allowed resumption of light activity in 7 days (median) and full, unrestricted activity in  $21 \pm 2$  days versus 21 days (median) and  $77 \pm 7$  days with rest alone [31,103]. Capacitative coupling also has been used to shorten healing time. Stress fractures typically take 8 to 12 weeks to heal; however, one study found that healing time with capacitative coupling was shortened to 7 weeks on average [104]. Weight-bearing trials should be performed every other day. After 3 to 5 days free from pain, phase 2 begins.

Phase 2 consists of light-weighted exercises and nonimpact-loading activities, such as walking, using a stair stepper, elliptical, or cross-country skiing machine. Activity should resume slowly and increase by 5 to 10 minutes per day up to 45 minutes. If any bony pain occurs, activity should stop for 1 to 2 days and restart at a level below which the pain occurred [101,105]. Recovery of strength that is lost during phase 1 also should be addressed. Sport-specific muscle rehabilitation also may be started.

Phase 3 is gradual re-entry into the athlete's sport-specific activity, starting every other day and gradually progressing to normal activity. This program may take from 3 to 18 weeks, depending on the extent of the injury [105]. All athletes also should have a biomechanical and gait evaluation to assess for leg length abnormalities, excessive pronation or supination, pes planus, pes cavus, and other structural abnormalities following a stress fracture.

## **Knee injuries**

The knee joint is an innocent bystander in the lower limb kinetic chain (ie, injuries at the knee usually are the result of biomechanical problems above or below the knee). The differential diagnosis of knee pain in the runner is described in [Table 1](#).

### *Patellofemoral pain syndrome*

Patellofemoral pain syndrome (PFPS) is the most common diagnosis for knee pain in athletic and nonathletic populations; the incidence may be twice as high in female athletes compared with their male counterparts [107,108]. Adolescent females seem to be at particular risk because their accelerated bony changes have not been accommodated by muscle changes

Table 1  
Differential diagnosis of knee pain

Medial	Lateral	Anterior	Posterior
PFPS	PFPS	PFPS	Baker's cyst
Plica	ITBS	Quadriceps tend.	Hamstring tend.
MMT	Popliteus	Patellar tend.	Popliteus
MCL sprain	LCL sprain	Patellar instability	
Intra-articular	LMT	Hoffa's syndrome	
Osteoarthritis	Intra-articular	Patellar OCD	
MMT	Osteoarthritis	Osteoarthritis	
		Bursitis	

*Abbreviations:* ITBS, iliotibial band syndrome; LCL, lateral collateral ligament; LMT, lateral meniscus tear; MCL, medial collateral ligament; MMT, medial meniscus tear; OCD, osteochondral defect; PFPS, patellofemoral pain syndrome; tend., tendinopathy.

[107]. The PFPS pain syndrome is an amalgam of diagnoses and generally can be classified into patellar instability, PFPS with malalignment, and PFPS without malalignment.

#### *Patellofemoral pain syndrome pain generators*

Traditional thinking has assumed that the patellar cartilage is the primary pain generator in the PFPS. Accordingly, the term “chondromalacia patellae” often is used for nebulous anterior knee pain; however, there are no nerve endings in the articular cartilage [109]. Arthroscopy studies also have failed to correlate the degree of patellar chondral and subchondral injury with the severity of anterior knee pain [109]. The current theory of pathogenesis is injury to the subchondral bone from patellar maltracking and increased patellofemoral joint reaction forces. Dye et al [110] proposed that the actual pain generator is the loading of nerve endings (ie, a degenerative neuroma) in the patellar retinaculum which causes a resultant synovitis. Other pain generators may exist in PFPS, such as irritated infrapatellar fat pad, plica, bursa, tendons, and apophyses.

#### *Predisposing factors for patellofemoral pain syndrome*

Biomechanical structural problems may contribute to patellofemoral pain [109]. For instance, miserable malalignment syndrome in women predisposes to PFPS. Muscle imbalances may exacerbate knee pain. Weak quadriceps musculature, particularly the vastus medialis obliquus (VMO) portion, has been implicated in patellofemoral pain. When the VMO is weak or inhibited, lateral vector forces that are created by the vastus lateralis, iliotibial band (ITB), and lateral retinaculum become dominant. Thus, the patella becomes displaced laterally. Tight lateral structures (ITB and lateral retinaculum) exacerbate this phenomena. Specifically in runners, a tight ITB and tensor fascia lata muscle, coupled with weak gluteus medius, creates excessive internal rotation of the femur and a lateral pelvic tilt. Tight hamstrings and gastrocnemius musculature also may exacerbate PFPS by

increasing knee flexion and creating greater patellofemoral joint reaction forces. In addition, hyperpronation seems to predispose runners to PFPS.

### *Clinical presentation*

The classic presentation of PFPS is an insidious onset of progressively severe diffuse anterior knee pain, especially with loading activities that involve repetitive flexion and extension, such as running. It may manifest as an ill-defined, usually bilateral, ache which is aggravated by hill training or stair climbing. Knee buckling can occur occasionally as a result of a painful reflex inhibition of the knee extensor mechanism. Other complaints may include crepitus or a “catching” sensation that is experienced with knee joint flexion and extension; however, crepitus may be common, even in asymptomatic individuals. Prolonged sitting with knee flexion, such as on an airplane flight or in a theater, may aggravate patellofemoral pain and lead individuals to extend their knee and legs into the aisle, also known as the “movie theater sign.”

In runners, patellar or quadriceps tendinopathy can occur, which is manifested as pinpoint pain at the inferior and superior pole of the patella, respectively. Occasionally, patellar instability symptoms can occur with descriptions of the “knee cap slipping out.” Infrapatellar fat pad impingement, occurs with forceful knee extension in individuals who have a prominent fat pad and posterior tilting of the patella. In adolescent runners, apophysitis of the inferior pole of the patella (Sinding-Larsen-Johansen syndrome) or at the tibial tuberosity (Osgood-Schlatter syndrome) should be suspected [111].

### *Diagnosis*

The history and physical evaluation of the runner who has knee pain should include analysis of the kinetic chain. Plain radiographs, particularly lateral and “sunrise” (or “skyline”) views, may be helpful in evaluating the patellofemoral joint [112]. Sunrise radiographs are axial views that are taken with the knee flexed 30° or 45°. If the patellofemoral joints shows joint space narrowing, osteophytes, subchondral sclerosis, and cysts, patellofemoral osteoarthritis should be suspected. Osteochondral lesions sometimes can be seen with these views. The lateral radiograph may demonstrate patella alta, rotational malalignment, or trochlear dysplasia. More commonly, particularly in the younger female runner who has PFPS, these radiographs are normal. Occasionally, MRI is useful in identifying the quality of the patellar cartilage pathology and ruling out other knee pathologies.

### *Treatment*

In general, the management for PFPS should be individualized and aimed at symptom relief acutely, and then focus on correcting the etiologic factors that contribute to the pain. The patient should be reminded that successful alleviation and prevention of further discomfort with PFPS might take

several weeks to months. In the authors' experience, patellar tendinopathy cases may take even longer with a longer period of relative unloading.

*Acute phase.* Relative rest is often helpful in alleviating patellofemoral pain. Runners often benefit from switching to low-impact aerobic activities for a defined period of time, such as swimming, aqua aerobics, "elliptical" training, or biking. Biking may exacerbate PFPS if the seat is too high or too low or if increased resistance is maintained. Runners who have PFPS should avoid step aerobics or using the stair-climbing apparatus because these can cause tremendous repetitive compressive loads on the patellofemoral joint. Patellar tendinopathy cases often need 2 to 3 weeks of relative unloading of the tendon.

In the acutely swollen knee, ice may provide some relief from the acute discomfort. Although anti-inflammatory medications have not been shown conclusively to benefit chronic PFPS, nonsteroidal anti-inflammatory drugs or acetaminophen may have a role in acute pain. Glucosamine/chondroitin also has been suggested to provide relief in PFPS, particularly if osteoarthritis is demonstrated on imaging [113].

*Rehabilitation phase.* Physical therapy was shown to be efficacious in relieving pain in PFS in uncontrolled, and, more recently, controlled trials [114]. Crossley et al's [114] program outlines elements of physical therapy intervention that can be used on all individuals (Box 4). It stands to reason that a more individualized program that attacks specific biomechanical deficits may be even more effective. Closed kinetic chain exercises serve as the cornerstone of the muscle strengthening program in PFPS. Studies showed that although open and closed kinetic chain exercises seem to improve PFPS symptoms, closed chain exercises also seem to improve functional performance. In particular, the literature has emphasized VMO strengthening in the open and closed kinetic chain positions [109]. Isokinetic open chain exercises also have been used for lower limb strengthening; however, they require specialized equipment and are not functional. For patients who are in acute pain, isometric exercises may be painless and just as beneficial as isokinetic exercise. A stretching program, as outlined by Crossley et al [114], also has a sound theoretic basis in eliminating biomechanical deficits.

Debate exists about whether VMO strengthening is important and whether VMO can be strengthened preferentially [115,116]. Some studies suggest that VMO firing is delayed in patients who have patellofemoral pain [115]; however, a robust relationship has yet to be established. A recent study revealed that nine common exercises that are given for preferential VMO strengthening showed equal firing of all of the different portions of the quadriceps [117]. Based on that study, some investigators have advocated for a generalized quadriceps strengthening program, rather than a selective VMO strengthening program. Other researchers showed that patients who

#### **Box 4. Elements of physical therapy intervention**

##### *Stretches*

- Medial/lateral glides and mobilization of the patella
- Deep friction massage to lateral soft tissues
- Hamstring stretch
- Anterior hip stretch

##### *Patellar taping*

##### *Strengthening*

- Isometric VMO strengthening with knees at 90°
- Partial squats with isometric gluteal contraction
- Isometric hip abduction against wall while standing
- Step-downs with pelvis parallel to floor
- Isometric hip abduction while standing away from wall
- Lunges

*Home exercise program: two times per day*

##### *Surface EMG*

---

*Adapted from* Crossley K, Bennell K, Green S, Cowan S, McConnell J. Physical therapy for patellofemoral pain. *Am J Sports Med* 2002;30(6):860.

have PFPS can learn the motor skill of selectively firing the VMO with patellar taping or EMG biofeedback [118,119]. These patients may have a better short-term outcome [119].

*Patellar taping, braces, and foot orthoses.* Some investigators believe that taping the patella may improve patellar alignment, tracking, tilt, or glide. Although numerous studies have been performed on this intervention for the treatment of PFPS, there are variable results [109,113,120]; however, in the authors' clinical practice, runners who perform a single leg squat and have improvement of pain with manual force on the patella may benefit from patellar taping. Patellar taping is continued only if a 50% reduction of pain is achieved [120]. Patellar taping likely works by reducing pain and improving proprioception rather than by way of a true improvement of patellar orientation. Taping may be especially beneficial in infrapatellar pad impingement by unloading the inflamed fat pad.

Elastic knee sleeves that have an anterior cut-out over the patella may provide some comfort, and because of their low cost, may be a useful strategy in some cases; however, the ability of the sleeve to alter patellar biomechanics is not known, and if used, should be an adjunct with therapeutic exercises. More expensive, more elaborate knee braces have not been shown to be particularly effective in patients who have PFPS [121].

Foot orthoses have been theorized to improve the biomechanics in the lower extremity, including the improvement of patellar tracking [122]. In

a study of 20 adolescent girls who had PFPS and exhibited excessive forefoot varus or hindfoot valgus, individuals who were treated with foot orthoses and an exercise regimen had a significant decrease in the level of pain over an 8-week period compared with the control group who underwent exercises alone [123]. It is more economical to try off-the-shelf orthoses first to determine whether the patient derives any benefit initially, and progress to custom orthoses if increased support is required.

*Surgery.* Nonsurgical options in the treatment plan for PFPS should be attempted first; however, more than 100 surgical options have been described for persistent patellofemoral pain that is nonresponsive to conservative treatment. Lateral retinacular release may be the most common procedure that is performed. It works best in individuals who have isolated lateral patellar tilt as the primary cause of patellar malalignment. Lateral retinacular release does not correct a more global malpositioning of the patella nor a large dynamic Q-angle. Inappropriate lateral release can lead to medial patellar subluxation, which can be a debilitating problem. Other more extensive realignment procedures can be performed experimentally to correct Q-angles and patellar orientation. These procedures typically do not allow athletes to return to running activity [120].

Articular cartilage procedures may include open or arthroscopically-performed debridement or shaving of patellar cartilage to achieve a smoother patellofemoral articulation, local excision of defects with drilling of the subchondral bone, or facetectomy and transplantation of autologous chondrocytes [124]. Local excision of diseased cartilage and subchondral drilling has been used commonly with satisfactory results, especially in patients who are younger than 25 years [124]. The long-term results of these articular cartilage procedures are not known, and the relative effectiveness of one procedure compared with the others also is unknown. Their role, although commonplace, is still experimental.

### *Iliotibial band friction syndrome*

In runners, the next most common knee injury is ITB friction syndrome (ITBS). The ITB is dense fascia that connects the gluteal muscles to the anterolateral tibia. An anatomic pouch can be found underlying the posterior ITB at the level of the lateral femoral epicondyle. The ITB passes over the lateral femoral epicondyle with knee flexion and extension. Maximum friction occurs when the posterior fibers of the ITB pass over the lateral femoral epicondyle at 20° to 30° of knee flexion—the putative “impingement zone.” Repeated knee flexion and extension, particularly with increased mileage per week, was shown to predispose to lateral knee pain. Although not studied extensively, poor neuromuscular control seems to be an important modifiable risk factor for ITBS. Specifically, the neuromuscular system is needed to control the valgus/internal rotation

vectors at the knee after heel strike. If appropriate control is not available, the ITB may have an abrupt increase in tension at its insertion site. Strengthening the gluteus medius and tensor fascia lata, which are decelerators of the valgus/internal rotation vectors at the knee, was shown to ameliorate ITBS.

### *Clinical presentation*

Symptoms of ITBS can emanate at three typical sites—proximal lateral hip, over the lateral femoral epicondyle, or at Gerdy's tubercle. Runners often note more pain with downhill running because of the increased time that is spent in the impingement zone. Paradoxically, runners state that faster running and sprinting often does not produce pain. Fast running allows the athlete to spend more time in knee angles that are greater than 30°.

### *Physical examination*

The modified Thomas and Ober tests are used to assess flexibility of the ITB and its attached muscles. Knee tenderness is noted at the lateral femoral epicondyle (above the lateral joint line) or at Gerdy's tubercle. Pain also can be elicited frequently by the Noble compression test which is performed in nonweight-bearing and standing positions; the knee is flexed and extended through the impingement zone (20°–30° of knee flexion) while the examiner applies pressure over the lateral femoral epicondyle.

### *Rehabilitation*

Successful treatment of ITBS can be achieved by incorporating a comprehensive, kinetic chain-oriented approach. Rehabilitation includes proper stretching of the ITB and associated muscles. A standing ITB stretch with the affected leg crossed over, lateral trunk side-bending to the unaffected side, and overhead arm extension to the unaffected side may be the most effective stretch [125] (Fig. 1). Some muscle groups do not respond to stretch unless myofascial and joint restrictions are addressed concomitantly. A qualified physical therapist or massage therapist can release trigger points and fascial adhesions that are identified on physical examination. Strengthening of weak or inhibited muscles can be started in conjunction with a flexibility program [126]. Weak hip abductors are seen often in patients who have ITBS. Hip abductor strengthening, with single leg squats and step-downs, is an efficacious treatment for runners who have ITBS [127]. The core strengthening concepts that were described previously play an empiric role in prevention and rehabilitation. The final stages of rehabilitation focus on sports-specific activity. Correction of form flaws can be invaluable to a runner. Frequently, runners have form deviations that lead to a *sine qua non* of uncontrolled valgus/internal rotation of the knee.



Fig. 1. An effective iliotibial band stretch.

Common abnormalities include excessive pronation, inability to shock attenuate at the knee, and Trendelenburg frontal plane gait at the pelvis. Changing to shock-absorbing or motion-control shoes can accommodate supination and overpronation, respectively. Occasionally, foot orthoses may be helpful in runners who have foot types that exacerbate ITBS.

### *Injections and surgery*

Injections that are directed to the anatomic pouch underneath the ITB at the lateral femoral epicondyle is a simple procedure that is used for patients who have persistent pain and swelling. A mixture of anesthetic (eg, 1 mL of 1% lidocaine) and long-acting steroid (eg, 1 mL of betamethasone) is instilled to the affected site [126]. Surgical treatment for ITBS rarely is needed. Surgery involves excising the posterior half of the ITB where it passes over the lateral femoral epicondyle or removing the underlying putative bursa. These surgical procedures give mixed results and should be contemplated only for patients in whom all other options have been exhausted, including a comprehensive rehabilitation program as outlined above.

## Summary

Running often can cause injuries to the knee as a result of kinetic chain dysfunctions. Addressing these dysfunctions in rehabilitation can prevent future injury. Stress fractures often occur in runners who engage in training errors. Female runners are particularly susceptible to stress fractures, especially in the setting of the female athlete triad. Proper identification and prevention of these injuries allows for athletes to return to running expeditiously.

## References

- [1] Kaplan FS, Hayes WC, Keaveny TM, et al. Form and function of bone. In: Simon SR, editor. Orthopaedic basic science. Rosemont (IL): American Academy of Orthopaedic Surgeons; 1994. p. 127–94.
- [2] Monteleone GP Jr. Stress fractures in the athlete. *Orthop Clin N Am* 1995;26(3):423–32.
- [3] Markey KL. Stress fractures. *Clin Sports Med* 1987;6(2):405–25.
- [4] Nattiv A, Armsey TD Jr. Stress injury to bone in the female athlete. *Clin Sports Med* 1997; 16(2):197–224.
- [5] Theintz G, Buchs B, Rizzoli R, et al. Longitudinal monitoring of bone mass accumulation in healthy adolescents: evidence for a marked reduction after 16 years of age at the levels of lumbar spine and femoral neck in female subjects. *J Clin Endocrinol Metab* 1992;75(4): 1060–5.
- [6] Drinkwater BL, Nilson K, Ott S, et al. Bone mineral density after resumption of menses in amenorrheic athletes. *JAMA* 1986;256(3):380–2.
- [7] Rigotti NA, Neer RM, Skates SJ, et al. The clinical course of osteoporosis in anorexia nervosa. A longitudinal study of cortical bone mass. *JAMA* 1991;265(9):1133–8.
- [8] Zanker CL, Swaine IL. Relation between bone turnover, estradiol, and energy balance in women distance runners. *Br J Sports Med* 1998;32(2):167–71.
- [9] 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(4–5):434–40.
- [10] Ammann P, Rizzoli R, Muller K, et al. IGF-I and pamidronate increase bone mineral density in ovariectomized adult rats. *Am J Physiol* 1993;265(5 Pt 1):E770–6.
- [11] Chevalley T, Rizzoli R, Manen D, et al. Arginine increases insulin-like growth factor-I production and collagen synthesis in osteoblast-like cells. *Bone* 1998;23(2):103–9.
- [12] Ihle R, Loucks AB. Dose-response relationships between energy availability and bone turnover in young exercising women. *J Bone Miner Res* 2004;19(8):1231–40.
- [13] Carter DR, Caler WE. A cumulative damage model for bone fracture. *J Orthop Res* 1985; 3(1):84–90.
- [14] Carter DR, Hayes WC. Compact bone fatigue damage: a microscopic examination. *Clin Orthop* 1977;127:265–74.
- [15] Fyhrie DP, Milgrom C, Hoshaw SJ, et al. Effect of fatiguing exercise on longitudinal bone strain as related to stress fracture in humans. *Ann Biomed Eng* 1998;26(4):660–5.
- [16] Otter MW, Qin YX, Rubin CT, et al. Does bone perfusion/reperfusion initiate bone remodeling and the stress fracture syndrome? *Med Hypotheses* 1999;53(5):363–8.
- [17] Barrow GW, Saha S. Menstrual irregularity and stress fractures in collegiate female distance runners. *Am J Sports Med* 1988;16(3):209–16.
- [18] Egol KA, Koval KJ, Kummer F, et al. Stress fractures of the femoral neck. *Clin Orthop* 1998;348:72–8.

- [19] Stanitski CL, McMaster JH, Scranton PE. On the nature of stress fractures. *Am J Sports Med* 1978;6(6):391–6.
- [20] Brunet ME, Cook SD, Brinker MR, et al. A survey of running injuries in 1505 competitive and recreational runners. *J Sports Med Phys Fitness* 1990;30(3):307–15.
- [21] Macera CA, Pate RR, Powell KE, et al. Predicting lower-extremity injuries among habitual runners. *Arch Intern Med* 1989;149(11):2565–8.
- [22] Marti B, Vader JP, Minder CE, et al. On the epidemiology of running injuries. The 1984 Bern Grand-Prix Study. *Am J Sports Med* 1988;16(3):285–94.
- [23] Walter SD, Hart LE, McIntosh JM, et al. The Ontario Cohort Study of Running-Related Injuries. *Arch Intern Med* 1989;149(11):2561–4.
- [24] Kadel NJ, Teitz CC, Kronmal RA. Stress fractures in ballet dancers. *Am J Sports Med* 1992;20(4):445–9.
- [25] Scully TJ, Besterman G. Stress fracture—a preventable training injury. *Mil Med* 1982; 147(4):285–7.
- [26] Rudzki SJ. Injuries in Australian Army recruits. Part I: decreased incidence and severity of injury seen with reduced running distance. *Mil Med* 1997;162(7):472–6.
- [27] Popovich RM, Gardner JW, Potter R, et al. Effect of rest from running on overuse injuries in army basic training. *Am J Prev Med* 2000;18(3 Suppl):147–55.
- [28] Frey C. Footwear and stress fractures. *Clin Sports Med* 1997;16(2):249–57.
- [29] 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(12): 1563–7.
- [30] Finestone A, Giladi M, Elad H, et al. Prevention of stress fractures using custom biomechanical shoe orthoses. *Clin Orthop* 1999;360:182–90.
- [31] Gillespie WJ, Grant I. Interventions for preventing and treating stress fractures and stress reactions of bone of the lower limbs in young adults (Cochrane Review). The Cochrane Library, edition 4. Chichester (UK): John Wiley & Sons, LTD; 2004.
- [32] Ekenman I, Milgrom C, Finestone A, et al. The role of biomechanical shoe orthoses in tibial stress fracture prevention. *Am J Sports Med* 2002;30(6):866–70.
- [33] Milgrom C, Finestone A, Segev S, et al. Are overground or treadmill runners more likely to sustain tibial stress fracture? *Br J Sports Med* 2003;37(2):160–3.
- [34] Goldberg B, Pecora C. Stress fractures: a risk of increased training in freshman. *Phys Sportsmed* 1994;22(3):68–78.
- [35] Bennell KL, Malcolm SA, Thomas SA, et al. The incidence and distribution of stress fractures in competitive track and field athletes. A twelve-month prospective study. *Am J Sports Med* 1996;24(2):211–7.
- [36] Lord MJ, Ha KI, Song KS. Stress fractures of the ribs in golfers. *Am J Sports Med* 1996; 24(1):118–22.
- [37] Hickey GJ, Fricker PA, McDonald WA. Injuries to elite rowers over a 10-yr period. *Med Sci Sports Exerc* 1997;29(12):1567–72.
- [38] Bell DG, Jacobs I. Electro-mechanical response times and rate of force development in males and females. *Med Sci Sports Exerc* 1986;18(1):31–6.
- [39] Hakkinen K. Force production characteristics of leg extensor, trunk flexor and extensor muscles in male and female basketball players. *J Sports Med Phys Fitness* 1991;31(3): 325–31.
- [40] Winter EM, Brookes FB. Electromechanical response times and muscle elasticity in men and women. *Eur J Appl Physiol Occup Physiol* 1991;63(2):124–8.
- [41] 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(2):248–56.
- [42] Zernicke RF, McNitt-Gray J, Otis C, et al. Stress fracture risk assessment among elite collegiate women runners. *J Biomech* 1994;27:854.
- [43] Lombardo SJ, Benson DW. Stress fractures of the femur in runners. *Am J Sports Med* 1982; 10(4):219–27.

- [44] 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(8):666–7.
- [45] Lappe JM, Stegman MR, Recker RR. The impact of lifestyle factors on stress fractures in female Army recruits. *Osteoporos Int* 2001;12(1):35–42.
- [46] Friedl KE, Nuovo JA, Patience TH, et al. Factors associated with stress fracture in young army women: indications for further research. *Mil Med* 1992;157(7):334–8.
- [47] 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(1):20–2.
- [48] Winfield AC, Moore J, Bracker M, et al. Risk factors associated with stress reactions in female Marines. *Mil Med* 1997;162(10):698–702.
- [49] 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(2):128–35.
- [50] Reinker KA, Ozburne S. A comparison of male and female orthopaedic pathology in basic training. *Mil Med* 1979;144(8):532–6.
- [51] Vaitkevicius H, Witt R, Maasdam M, et al. Ethnic differences in titratable acid excretion and bone mineralization. *Med Sci Sports Exerc* 2002;34(2):295–302.
- [52] 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):256–60.
- [53] 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(6):810–8.
- [54] Beck TJ, Ruff CB, Shaffer RA, et al. Stress fracture in military recruits: gender differences in muscle and bone susceptibility factors. *Bone* 2000;27(3):437–44.
- [55] Giladi M, Milgrom C, Stein M, et al. External rotation of the hip. A predictor of risk for stress fractures. *Clin Orthop* 1987;(216):131–4.
- [56] Lauder TD, Dixit S, Pezzin LE, et al. The relation between stress fractures and bone mineral density: evidence from active-duty Army women. *Arch Phys Med Rehabil* 2000;81(1):73–9.
- [57] Myburgh KH, Hutchins J, Fataar AB, et al. Low bone density is an etiologic factor for stress fractures in athletes. *Ann Intern Med* 1990;113(10):754–9.
- [58] Marx RG, Saint-Phard D, Callahan LR, et al. Stress fracture sites related to underlying bone health in athletic females. *Clin J Sport Med* 2001;11(2):73–6.
- [59] Cobb KL, Bachrach LK, Greendale G, et al. Disordered eating, menstrual irregularity, and bone mineral density in female runners. *Med Sci Sports Exerc* 2003;35(5):711–9.
- [60] Beck TJ, Ruff CB, Mourrada FA, et al. Dual-energy X-ray absorptiometry derived structural geometry for stress fracture prediction in male US Marine Corps recruits. *J Bone Miner Res* 1996;11(5):645–53.
- [61] Milgrom C, Giladi M, Simkin A, et al. An analysis of the biomechanical mechanism of tibial stress fractures among Israeli infantry recruits. A prospective study. *Clin Orthop* 1988;(231):216–21.
- [62] Milgrom C, Giladi M, Simkin A, et al. The area moment of inertia of the tibia: a risk factor for stress fractures. *J Biomech* 1989;22(11–12):1243–8.
- [63] Giladi M, Milgrom C, Simkin A, et al. Stress fractures and tibial bone width. A risk factor. *J Bone Joint Surg Br* 1987;69(2):326–9.
- [64] Miller GJ, Purkey WW Jr. The geometric properties of paired human tibiae. *J Biomech* 1980;13(1):1–8.
- [65] Giladi M, Milgrom C, Stein M. The low arch, a protective factor in stress fractures. A prospective study of 295 military recruits. *Orthop Rev* 1985;(14):709–12.
- [66] Simkin A, Leichter I, Giladi M, et al. Combined effect of foot arch structure and an orthotic device on stress fractures. *Foot Ankle* 1989;10(1):25–9.
- [67] Friberg O. Leg length asymmetry in stress fractures. A clinical and radiological study. *J Sports Med Phys Fitness* 1982;22(4):485–8.
- [68] 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(8):945–52.

- [69] Finestone A, Shlamkovitch N, Eldad A, et al. Risk factors for stress fractures among Israeli infantry recruits. *Mil Med* 1991;156(10):528–30.
- [70] Stager JM, Hatler LK. Menarche in athletes: the influence of genetics and prepubertal training. *Med Sci Sports Exerc* 1988;20(4):369–73.
- [71] Warren MP, Brooks-Gunn J, Hamilton LH, et al. Scoliosis and fractures in young ballet dancers. Relation to delayed menarche and secondary amenorrhea. *N Engl J Med* 1986;314(21):1348–53.
- [72] Loucks AB, Horvath SM. Athletic amenorrhea: a review. *Med Sci Sports Exerc* 1985;17(1):56–72.
- [73] Drinkwater BL, Nilson K, Chesnut CH, et al. Bone mineral content of amenorrheic and eumenorrheic athletes. *N Engl J Med* 1984;311(5):277–81.
- [74] Frost HM. A new direction for osteoporosis research: a review and proposal. *Bone* 1991;12(6):429–37.
- [75] Bennell KL, Malcolm SA, Thomas SA, et al. Risk factors for stress fractures in female track-and-field athletes: a retrospective analysis. *Clin J Sport Med* 1995;5(4):229–35.
- [76] Hergenroeder AC. Bone mineralization, hypothalamic amenorrhea, and sex steroid therapy in female adolescents and young adults. *J Pediatr* 1995;126(5)(Pt 1):683–9.
- [77] Cumming DC, Wall SR, Galbraith MA, et al. Reproductive hormone responses to resistance exercise. *Med Sci Sports Exerc* 1987;19(3):234–8.
- [78] Klibanski A, Biller BM, Schoenfeld DA, et al. The effects of estrogen administration on trabecular bone loss in young women with anorexia nervosa. *J Clin Endocrinol Metab* 1995;80(3):898–904.
- [79] Warren MP, Perlroth NE. The effects of intense exercise on the female reproductive system. *J Endocrinol* 2001;170(1):3–11.
- [80] Seeman E, Szmukler GI, Formica C, et al. Osteoporosis in anorexia nervosa: the influence of peak bone density, bone loss, oral contraceptive use, and exercise. *J Bone Miner Res* 1992;7(12):1467–74.
- [81] Hartard M, Kleinmond C, Kirchbichler A, et al. Age at first oral contraceptive use as a major determinant of vertebral bone mass in female endurance athletes. *Bone* 2004;35(4):836–41.
- [82] Berenson AB, Radecki CM, Grady JJ, et al. A prospective, controlled study of the effects of hormonal contraception on bone mineral density. *Obstet Gynecol* 2001;98(4):576–82.
- [83] Specker BL. Evidence for an interaction between calcium intake and physical activity on changes in bone mineral density. *J Bone Miner Res* 1996;11(10):1539–44.
- [84] Bennell K, Matheson G, Meeuwisse W, et al. Risk factors for stress fractures. *Sports Med* 1999;28(2):91–122.
- [85] Cooper KL, Beabout JW, Swee RG. Insufficiency fractures of the sacrum. *Radiology* 1985;156(1):15–20.
- [86] Eisman JA. Vitamin D receptor gene alleles and osteoporosis: an affirmative view. *J Bone Miner Res* 1995;10(9):1289–93.
- [87] Fleet JC, Harris SS, Wood RJ, et al. The BsmI vitamin D receptor restriction fragment length polymorphism (BB) predicts low bone density in premenopausal black and white women. *J Bone Miner Res* 1995;10(6):985–90.
- [88] Morrison NA, Qi JC, Tokita A, et al. Prediction of bone density from vitamin D receptor alleles. *Nature* 1994;367(6460):284–7.
- [89] Rigotti NA, Nussbaum SR, Herzog DB, et al. Osteoporosis in women with anorexia nervosa. *N Engl J Med* 1984;311(25):1601–6.
- [90] Bachrach LK, Guido D, Katzman D, et al. Decreased bone density in adolescent girls with anorexia nervosa. *Pediatrics* 1990;86(3):440–7.
- [91] Frusztajer NT, Dhuper S, Warren MP, et al. Nutrition and the incidence of stress fractures in ballet dancers. *Am J Clin Nutr* 1990;51(5):779–83.
- [92] Nattiv A, Puffer JC, Green GA. Lifestyles and health risks of collegiate athletes: a multi-center study. *Clin J Sport Med* 1997;7(4):262–72.

- [93] Hoch AZ, Dempsey RL, Carrera GF, et al. Is there an association between athletic amenorrhea and endothelial cell dysfunction? *Med Sci Sports Exerc* 2003;35(3):377–83.
- [94] Nattiv A, Agostini R, Drinkwater B, et al. The female athlete triad. The inter-relatedness of disordered eating, amenorrhea, and osteoporosis. *Clin Sports Med* 1994;13(2):405–18.
- [95] Loucks AB. Energy availability, not body fatness, regulates reproductive function in women. *Exerc Sport Sci Rev* 2003;31(3):144–8.
- [96] Slemenda CW, Reister TK, Hui SL, et al. Influences on skeletal mineralization in children and adolescents: evidence for varying effects of sexual maturation and physical activity. *J Pediatr* 1994;125(2):201–7.
- [97] Writing Group for the ISCD Position Development Conference. Diagnosis of osteoporosis in men, premenopausal women, and children. *J Clin Densitom* 2004;7(1):17–26.
- [98] Sullivan D, Warren RF, Pavlov H, et al. Stress fractures in 51 runners. *Clin Orthop* 1984;(187):188–92.
- [99] Miller C, Major N, Toth A. Pelvic stress injuries in the athlete: management and prevention. *Sports Med* 2003;33(13):1003–12.
- [100] Ishibashi Y, Okamura Y, Otsuka H, et al. Comparison of scintigraphy and magnetic resonance imaging for stress injuries of bone. *Clin J Sport Med* 2002;12(2):79–84.
- [101] Brukner P. Exercise-related lower leg pain: bone. *Med Sci Sports Exerc* 2000;32(3 Suppl): S15–26.
- [102] Boam WD, Miser WF, Yuill SC, et al. Comparison of ultrasound examination with bone scintiscan in the diagnosis of stress fractures. *J Am Board Fam Pract* 1996;9(6):414–7.
- [103] Swenson EJ Jr, DeHaven KE, Sebastianelli WJ, et al. The effect of a pneumatic leg brace on return to play in athletes with tibial stress fractures. *Am J Sports Med* 1997;25(3):322–8.
- [104] Benazzo F, Mosconi M, Beccarisi G, et al. Use of capacitive coupled electric fields in stress fractures in athletes. *Clin Orthop* 1995;(310):145–9.
- [105] Verma RB, Sherman O. Athletic stress fractures: part I. History, epidemiology, physiology, risk factors, radiography, diagnosis, and treatment. *Am J Orthop* 2001;30(11):798–806.
- [106] Fredericson M, Bergman AG, Matheson GO. [Stress fractures in athletes]. *Orthopade* 1997;26:961–71 [in German].
- [107] Arendt E, Griffin LY. Musculoskeletal injuries. In: Drinkwater BL, editor. *Women in sport*. Oxford (UK): Blackwell Science; 2000. p. 208–40.
- [108] Almeida SA, Trone DW, Leone DM, et al. Gender differences in musculoskeletal injury rates: a function of symptom reporting? *Med Sci Sports Exerc* 1999;31(12):1807–12.
- [109] Heintjes E, Berger MY, Bierma-Zeinstra SM, et al. Exercise therapy for patellofemoral pain syndrome. *Cochrane Database Syst Rev* 2003;4:CD003472.
- [110] Dye SF, Vaupel GL, Dye CC. Conscious neurosensory mapping of the internal structures of the human knee without intraarticular anesthesia. *Am J Sports Med* 1998;26(6):773–7.
- [111] Duri ZA, Patel DV, Aichroth PM. The immature athlete. *Clin Sports Med* 2002;21(3): 461–82.
- [112] Fredericson M. Common injuries in runners. Diagnosis, rehabilitation and prevention. *Sports Med* 1996;21(1):49–72.
- [113] Heintjes E, Berger MY, Bierma-Zeinstra SM, et al. Pharmacotherapy for patellofemoral pain syndrome. *Cochrane Database Syst Rev* 2004;CD003470.
- [114] Crossley K, Bennell K, Green S, et al. Physical therapy for patellofemoral pain. *Am J Sports Med* 2002;30(6):857–65.
- [115] Cowan SM, Bennell KL, Hodges PW, et al. Delayed onset of electromyographic activity of vastus medialis obliquus relative to vastus lateralis in subjects with patellofemoral pain syndrome. *Arch Phys Med Rehabil* 2001;82(2):183–9.
- [116] Cerny K. Vastus medialis oblique/vastus lateralis muscle activity ratios for selected exercises in persons with and without patellofemoral pain syndrome. *Phys Ther* 1995;75(8): 672–83.
- [117] Powers CM. Rehabilitation of patellofemoral joint disorders: a critical review. *J Orthop Sports Phys Ther* 1998;28(5):345–54.

- [118] Cowan SM, Bennell K, Hodges PW. Therapeutic patellar taping changes the timing of vasti muscle activation in people with patellofemoral pain syndrome. *Clin J Sport Med* 2002;12: 339–47.
- [119] Cowan SM, Bennell KL, Crossley KM, et al. Physical therapy for patellofemoral pain: a randomized, double-blinded, placebo-controlled trial. *Med Sci Sports Exerc* 2002;34(12): 1879–85.
- [120] Fredericson M, Powers CM. Practical management of patellofemoral pain. *Clin J Sport Med* 2002;12(1):36–8.
- [121] D'Hondt NE, Struijs PA, Kerkhoffs GM, et al. Orthotic devices for treating patellofemoral pain syndrome. *Cochrane Database Syst Rev* 2002(2):CD002267.
- [122] Hertel J, Sloss BR, Earl JE. Effect of foot orthotics on quadriceps and gluteus medius electromyographic activity during selected exercises. *Arch Phys Med Rehabil* 2005;86(1): 26–30.
- [123] Gross MT, Foxworth JL. The role of foot orthoses as an intervention for patellofemoral pain. *J Orthop Sports Phys Ther* 2003;33(11):661–70.
- [124] Fulkerson JP. Diagnosis and treatment of patients with patellofemoral pain. *Am J Sports Med* 2002;30(3):447–56.
- [125] Fredericson M, White JJ, Macmahon JM, et al. Quantitative analysis of the relative effectiveness of 3 iliotibial band stretches. *Arch Phys Med Rehabil* 2002;83(5):589–92.
- [126] Akuthota V, Stilp S, Lento P. Iliotibial band syndrome. In: Frontera W, Silver J, editors. *Essentials of physical medicine and rehabilitation*. Philadelphia: Hanley and Belfus; 2002. p. 328–33.
- [127] Fredericson M, Cookingham CL, Chaudhari AM, et al. Hip abductor weakness in distance runners with iliotibial band syndrome. *Clin J Sport Med* 2000;10(3):169–75.