Exercise-Induced Loss of Bone Density in Athletes

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Abstract

In athletes, the rarely identified malady of osteoporosis differs from other chronic effects of exercise. The most obvious difference is that hormonal imbalance leads to compensatory mechanisms that in turn lead to osteoporosis and increased incidence of fracture. Most research on this subject has dealt with women, because hormonal imbalances in women are easier to detect than those in men. Endurance athletes are known to have decreased levels of sex hormones, which can cause physiologic changes that lead to bone loss. This may result in relative osteoporosis despite the loading of the bone during exercise, which would normally increase bone mineral density. Premature osteoporosis may be irreversible, causing young athletes to become osteoporotic at an earlier age and have an increased risk of fracture later in life.

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The special demands that athletes place on their bodies entail some heretofore poorly understood endocrinologic consequences. The ramifications of certain hormonal imbalances include a greater prevalence of osteoporosis (in male as well as female athletes) and an increased risk of fracture due to exercise-induced bone loss. Current research indicates that vigilance for these problems is essential when providing orthopaedic care to the high-performance athlete.

Osteoporosis

Bone adapts to mechanical stresses, hormonal changes, and nutritional states. Remodeling of bone—the balance between bone formation and bone resorption—constantly adjusts to these factors so as to maintain homeostasis in the amount of bone and bone mineral in the skeleton. Throughout childhood and adolescence, the balance is tipped toward formation. After peak bone mass is achieved in young adulthood, the balance changes, leaving deficits in the bone at a rate of about 1% loss per year. These small deficits accumulate, accounting for osteoporosis associated with age.

Osteoporosis is defined as low density of bone relative to norms for age and sex.[1] It can be definitively diagnosed only on the basis of histologic examination but is suggested by dual-energy x-ray absorptiometry (DEXA) values 2 SD from the norm. Osteoporosis can occur at any age when the bone mineral density (BMD) reaches abnormally low levels. If the BMD (measured as grams of hydroxyapatite per unit of bone area or volume) falls below a critical threshold, the patient is at increased risk for fractures. In younger persons, osteoporosis is defined as premature bone loss and/or inadequate bone formation, which leads to low bone mass, increased skeletal fragility, and increased risk of fracture (Fig. 1).[1]

Regardless of whether homeostatic mechanisms are increasing or decreasing bone density, the same remodeling process occurs. First, the bone resorbs trabeculae at a stressed area; then new trabeculae form along the lines of stress. Since the two phases are out of synchronization, there is a period of vulnerability when resorption has occurred but formation lags behind. If small repetitive stresses continue at an increased rate, microfractures may occur. It is theorized that these microfractures may then aggregate, leading to an overt fracture. This scenario must be considered when evaluating athletes for return to competition.

During the remodeling process, most activity occurs in the trabecular bone, which has a higher proportion of osteoclasts and osteoblasts. In a period of increased bone turnover, as the trabeculae

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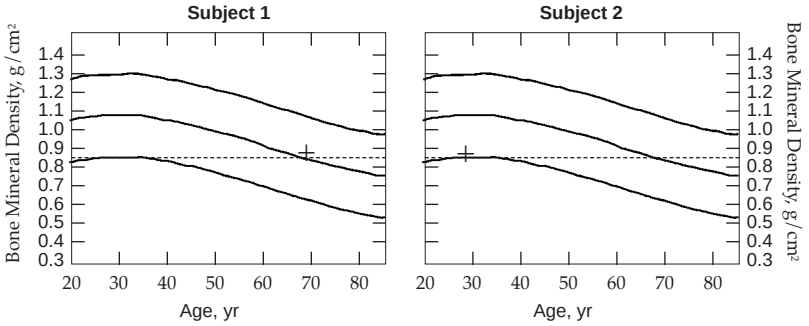

Fig. 1 Lumbar spine bone mineral density (BMD) values of two women (the curves on both graphs represent the BMD norm for age ± 2 SDs). The graph on the left is that of a normal 69-year-old woman who had never received estrogen replacement therapy. The graph on the right is that of a eumenorrheic 28-year-old runner with an 8-year history of exercise-induced amenorrhea. Her BMD level is very near the fracture threshold for bone (dashed line). (Reproduced with permission from Snow-Harter CM: Bone health and prevention of osteoporosis in active and athletic women. *Clin Sports Med* 1994;13:389-404.)

are replaced, less of the compressive load can be borne by the trabecular bone, and more must therefore be borne by the cortical bone. The cortex cannot resist compressive loads as well, and stress fractures develop as it tries to remodel itself.[2]

Influence of Sex Hormones on Bone Mass

The bone-remodeling process is affected by many factors that can tip the balance toward formation or resorption. Some of the factors are well known, but their mechanism of action may not be defined, as is the case with the effects of estrogen and testosterone.

Estrogen is found in both sexes but at higher concentrations in women. The physiologic effects of estrogen are many and varied. For example, lack of estrogen leads to increased loss of urinary calcium.[3] It also causes decreased intestinal calcium absorption.[1] Both of these processes decrease the serum calcium available for bone formation. Most important, estrogen controls the speed of the remodeling process; high concentrations of estrogen slow the remodeling process, and relative estrogen deficiency speeds up the process.

Both men and women have a steady decline in BMD after achieving peak density sometime between the ages of 20 and 30 years. The peak bone mass and its time of occurrence are determined by genetic factors, nutrition, exercise, and hormonal levels.[4] Dietary calcium influences the peak; a high intake is associated with a higher bone mass. Exercise places mechanical demands on the skeleton and also increases bone mass. Hormonal levels, especially in women, are probably among the more important factors in determining bone mass.[4]

After peak bone mass has been achieved, both men and women lose bone with each cycle of remodeling. In women, bone loss is accelerated in early menopause. After 5 to 8 years of accelerated loss, the rate slows to near the usual 1% loss per year, but menopausal loss places women at higher risk for fracture compared with men of the same age.[5] This same process occurs in young women who have undergone an oophorectomy or are prematurely amenorrheic for other physiologic reasons. If these women are treated with estrogen, they will have rates of bone loss similar to those in normal individuals; left untreated, they will lose bone at a rate more than 80% higher than average.[6]

Bone Remodeling

Remodeling (and therefore osteoporosis) occurs primarily in areas where fatty marrow is in contact with trabecular bone or the inner surface of cortical bone, suggesting that cellular messengers known as cytokines may be involved. One of these cytokines, interleukin-6 (IL-6), promotes osteoclast and osteoclast-precursor development.[7,8] The formation of IL-6 is inhibited by sex hormones, with estrogen being a much more effective inhibitor of IL-6 than testosterone.[7] Therefore, the sex hormones may decrease the number of osteoclasts produced, which will decrease the rate of bone resorption and remodeling.

Estrogen also causes changes in the number and composition of the cells involved in the remodeling process. In oophorectomized mice, remodeling is accelerated, and estrogen given to the mice will decrease the number and size of osteoclasts in contact with bone while increasing the size and number of osteoblasts.[9] If estrogen is withheld from these same mice, there is an increase in the size and number of osteoclasts, leading to a 50% to 60% decrease in secondary spongiosa. In seeming contrast, the number of osteoblasts also increases, as does the amount of osteoid produced when estrogen is withheld. Although this may seem to run against expectations, it should be kept in mind that estrogen does not have a direct effect on the formation of bone, but rather has an effect on the speed of remodeling of bone, which is slightly unbalanced after skeletal maturity.

More evidence for cytokine control of remodeling has been found in women within 2 weeks after oophorectomy. They have increased serum levels of bone-resorption indicators, such as IL-1, tumor necrosis factor-α, and osteocalcin, along with elevations of the urinary hydroxyproline-creatinine and calcium-creatinine ratios, which are nonspecific indices of bone resorption. These changes are reversed within 2 weeks after the institution of estrogen therapy.[10] The urinary hydroxyproline-creatinine and calcium-creatinine ratios are being replaced by commercially available tests for determining the deoxypyridinoline-creatinine and pyridinoline-creatinine ratios, which are more specific for bone loss; these indices measure cross-links of collagen from bone.[11] Researchers have recently recommended a 3-day collection period to ensure accuracy when measuring these breakdown products of bone.[12]

Testosterone may have the same effect in men that estrogen has in women, but this has not been as extensively studied due to the relative difficulty of screening men for hormone deficiency. It is known, however, that men with hypogonadism have osteoporosis associated with increased bone resorption and decreased mineralization; both of these effects are reversed with testosterone supplementation.[13]

In boys during puberty, a close relationship has been found between testosterone level, osteoblast activity, and bone mineralization. In one study,[14] peak increases in serum testosterone concentration were followed by peak increases in bone mineral content 4.7 months later (Fig. 2).

Sex Hormone Levels in Athletes

Endurance athletes generally have abnormally low sex hormone levels. Strength-training athletes typically have higher levels, although even they may have levels lower than those of sedentary control subjects. Therefore, it appears that sex hormone levels in athletes are related to the amount and type of exercise performed.

In men, testosterone decreases skeletal muscle breakdown during endurance training, but during periods of prolonged activity, testosterone release is suppressed. Testosterone levels can drop by as much as 25% within 48 hours of strenuous training, but will return to normal after a period of rest.[15] Endurance training also inhibits the reproductive axis subclinically in men, but its effects are less obvious than in women. For example, in one study,[16] testosterone levels in endurance-trained men running at least 64 km per week were much lower than those in sedentary control subjects (Fig. 3), which may have been due to a decrease in hormone production.

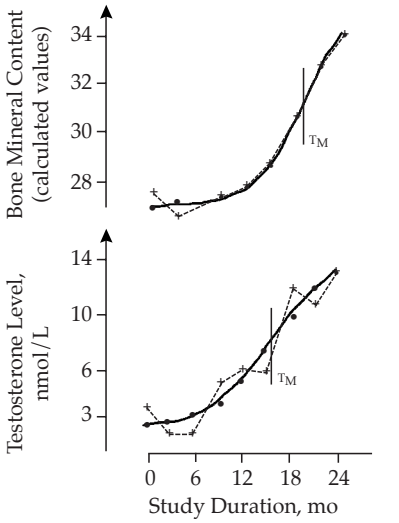

Fig. 2 In a study of 20 adolescent boys, Krabbe et al[14] found that as serum levels of testosterone increase, BMD also increases, with a 6-month lag between the peak testosterone level and the increase in BMD. These graphs show the findings in one subject. The two lines represent calculated values (filled circles) and observed values (crosses); T_M indicates calculated time of maximal increase. (Reproduced with permission from Krabbe S, Hummer L, Christiansen C: Longitudinal study of calcium metabolism in male puberty: II. Relationship between mineralization and serum testosterone. *Acta Paediatr Scand* 1984;73:750-755.)

Hypothalamic gonadotropin-releasing hormone, important in the reproductive axis, is known to be decreased in male marathon runners who are training by running 125 to 200 miles per week.[17] These low levels have been successfully treated by decreasing mileage by 70%, but this has not been found to increase the runner's serum testosterone concentration from such a low baseline.

In contrast, male gymnasts and weight lifters may have slightly lower testosterone levels when compared with sedentary control subjects (although their testosterone levels will rise if they pursue a lighter training schedule).[18] However, in one study,[19] it was found that testosterone levels in 120 runners were not significantly lower than those in control subjects. Further study in this area is warranted, but research is difficult because men are not as dependent as women on cyclic endocrine function, and small alterations in reproductive hormone levels may have only a small effect on gametogenesis.[16]

Since passage of Title IX legislation in 1972, there has been an increase in the number of female athletes participating and competing in sports. Although adolescent girls are typically not well conditioned, when they join the military or enter collegiate sports, they are usually trained in a fashion similar to that used for men. One of the consequences of excessive or incorrect training is athletic amenorrhea.[1] Primary amenorrhea is the lack of menses by the age of 16. Secondary amenorrhea is the absence of three to six consecutive

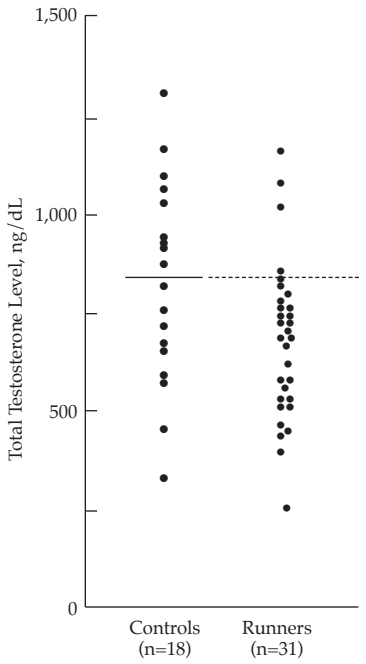

Fig. 3 In one study of 31 male runners and 18 control subjects, serum levels of testosterone in runners were statistically lower than those in control subjects. (Reproduced with permission from Wheeler GD, Wall SR, Belcastro AN, Cumming DC: Reduced serum testosterone and prolactin levels in male distance runners. *JAMA* 1984;252:514-516.)

menses after the cycle has been established. Oligomenorrhea is characterized by menstrual cycles longer than 36 days. It must always be kept in mind that athletic amenorrhea is a diagnosis of exclusion, with pregnancy being the most common cause of amenorrhea in the athletic population. Pregnancy must be ruled out before ovarian, thyroid, and pituitary abnormalities are sought as causes of amenorrhea.

A higher risk of amenorrhea has been noted in women who begin training before menarche, train the most intensively, consume the fewest calories, and have low body weights.[1] Those in individual sports that emphasize low body weight, such as distance running, gymnastics, skating,[20] and cycling, are at an even higher risk.

One theory for the cause of athletic amenorrhea is that caloric intake may be too low for needed energy expenditure. The resultant energy drain may lead to a decrease in the basal metabolic rate in order to conserve the body's energy reserve.[21] Frisch and McArthur[21] theorized that a critical level of body fat is needed to maintain menstrual function; however, other researchers have found very low body fat percentages in eumenorrheic athletes. Amenorrheic athletes average caloric intakes 25% below normal,[22] which may help substantiate the concept that some bodily energy conservation occurs with cessation of menses. A concurrent factor may be the presence of eating disorders, which have been reported in 15% to 66% of female athletes.[21] Such disorders are much more common in female athletes than in male athletes (although sports like wrestling may be an exception).

Irregular menses, whether amenorrhea or oligomenorrhea, occurs in 2% to 66% of athletes, compared with 2% to 5% of nonathletes.[1,23] In one study,[22] irregular menses affected 25% of noncompetitive runners but 50% of competitive runners, especially if they began competition or intensive training at an age closer to menarche. Feicht et al[23] found that runners who trained by running 10 miles per week had a 6% incidence of amenorrhea, while those who ran 80 miles per week had a 43% incidence.

Amenorrheic athletic women may have a subtype of hypothalamic amenorrhea, with the disruption occurring in the ovary-pituitary axis.[20] Another theory is that pulsatile release of gonadotropin-releasing hormone (GnRH) from the hypothalamus is deficient or absent in female athletes, which results in low estrogen levels and cessation of menses. Other theories maintain that neurohormones, such as melatonin, dopamine, and β-endorphins, which are involved in the "runner's high," may suppress GnRH pulsatile secretion.[24] Furthermore, opioid antagonists, such as naltrexone and naloxone, have been used to restore gonadotropin pulses and even ovulation and menses in selected cases.[25]

Bone Mineral Density in Athletes

In males, prolonged testosterone deficiency is associated with decreased bone mass. Males with a history of delayed puberty have lower cortical and trabecular BMD and may be at increased risk for osteoporotic fracture later in life.[3] Bone loss in aging men has been found to be greater in trabecular bone than in cortical bone, just as it is in women.[3]

Male runners have decreased bone mass and evidence of high bone turnover, suggesting accelerated bone loss[19] due to decreased testosterone level, in much the same way that menstrual dysfunction in women leads to premature osteoporosis. Male runners who train by running 15 to 20 miles per week have increased BMD in their lower legs; however, those who train by running 60 to 75 miles per week have decreased BMD.[26] Weekly running distance is negatively correlated with BMD, especially in areas with a high content of trabecular bone, such as the spine. Also, bone turnover is 20% to 30% greater in elite runners, in accordance with their higher rate of bone metabolism.[26]

The highest BMD values are found in strength- and power-training athletes; endurance athletes have lower bone densities. Both of these groups have higher BMDs than sedentary control subjects; therefore, it appears that exercise may partially block the effects

of hormone deficiencies in endurance athletes. In one study,[19] male long-distance runners had lower BMD values in the lumbar spine than control subjects, although tibial values were the same. This suggests accelerated trabecular bone loss in the spine due to the decrease in hormones, but the effects of exercise help maintain bone density in the lower extremities. In another study,[26] bone density was lower in male triathletes than in rowers but was similar to that in sedentary control subjects. Although the BMD in triathletes might seem to be acceptable, in that it is the same as the BMD in sedentary control subjects, this is actually a disconcerting finding because the effects of exercise should increase bone mass. In yet another study,[27] serum testosterone in runners was lower than that in rowers or sedentary control subjects, suggesting that low testosterone may negate the positive effects exercise can have on bone density.

In female athletes, delay in onset of menses is associated with delay of physeal closure and bone maturation. Because 48% of skeletal mass is attained during adolescence, delayed menarche negatively influences skeletal development by decreasing the amount of bone produced during adolescence and thereby decreasing bone mass.[28]

Several studies have focused on the incidence of low BMD in college-age amenorrheic athletes. It has been found that amenorrheic athletes have lower BMD than eumenorrheic athletes and sedentary control subjects (Fig. 4) but higher BMD than nonactive amenorrheic women.[5,29] Vertebral BMD is 15% to 20% lower in amenorrheic athletes than in eumenorrheic athletes and 25% to 30% lower than in sedentary eumenorrheic women, despite the effects of exercise.[5] Loss from the spine is approximately five times greater than that from the peripheral skeleton, with the greatest decrease occurring within 6 months after cessation of ovarian function.[30] The lowest BMDs are associated with the lowest estradiol levels; therefore, as the estrogen decreases, so does the BMD.

Although amenorrhea is associated with decreased BMD, the amount of cortical bone in the peripheral skeleton in the amenorrheic athlete has been found to be similar to that in sedentary control subjects. This may be due to the fact that exercise maintains bone density in the limbs only at normal levels. The expected increase in BMD in stressed bone does not occur in these women.

High-intensity exercise may increase BMD in specific sites in rowers,[31] figure skaters, and gymnasts, even though they may be amenorrheic.[1] Gymnasts have the same incidence of menstrual irregularity as runners, but their BMD is above normal. This may be due to their extremely high mechanical stresses, which would increase their BMD. In some instances, this may be enough to overcome the negative influence of low hormone levels.[1] The BMD in the lumbar spine is higher in amenorrheic rowers than in amenorrheic runners. In amenorrheic dancers, higher BMDs can be found in the legs.

One way to explain this phenomenon is by the "mechanostat theory," which maintains that there is a set point for the bone-remodeling rate. The set point is influenced by estrogen and mechanical stimuli: high mechanical loads create a low set point for remodeling, causing a net increase in bone; lack of estrogen increases the set point for remodeling, leading to a net loss of bone.[32] This means that the positive effects of exercise may overcome the negative effects of low levels of estrogen in certain situations. However, exercise may not make up for the influence of hormonal changes in all instances. Although the BMD in a female long-distance runner may be greater than that in a sedentary control subject, the question is whether the increase is enough to withstand the repetitive loads placed on the bones over a period of training.

Myburgh et al[33] assessed injuries in athletes and found that menstrual dysfunction was associated with low BMD and injury in female athletes and that oral contraceptives protect women against stress fractures. They also found that women who had to alter their running schedule because of bone or soft-tissue injuries were more likely to be amenorrheic. Furthermore, they examined cortical bone densities in the lower extremities of male and female runners after noting that in most other studies of runners the measurements were not obtained in bones that were maximally stressed.

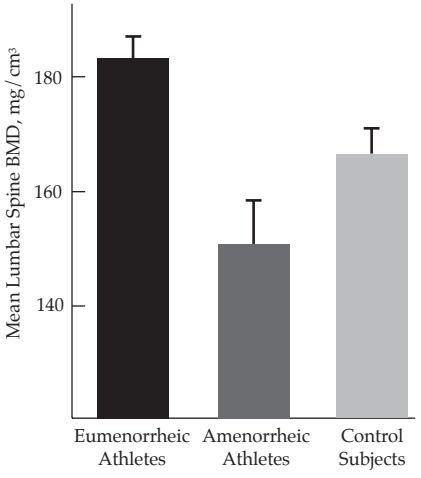

Fig. 4 Average lumbar spine BMD in a study group of 6 eumenorrheic athletes, 11 amenorrheic athletes, and 17 female control subjects. Eumenorrheic athletes have the highest BMD, and amenorrheic athletes have the lowest despite the positive effects of exercise on bone density. (Reproduced with permission from Snow-Harter CM: Bone health and prevention of osteoporosis in active and athletic women. *Clin Sports Med* 1994;13:389-404.)

They also noticed that the density of trabecular bone rather than cortical bone (where stress fractures more often occur) was evaluated in those other studies. Myburgh et al found that injured male and female athletes have low BMDs even in areas of cortical bone.

Overt fractures in athletes are not as common as stress fractures, especially among endurance athletes. Stress fractures are considered to be due to cyclic stresses that are below the failure level of the bone but are repeated over a short period of time with inadequate bone remodeling. It is theorized that microtrauma to the bone may accumulate to cause an overt fracture if the insulting force is allowed to continue (Fig. 5). Stress fractures associated with menstrual irregularities, and presumably an increase in bone remodeling, usually occur in long bones despite the fact that exercise has been shown to increase bone mass in long bones. In one study,[29] amenorrheic runners had a 49% incidence of stress fractures, compared with 0% for eumenorrheic runners over the same time period and with the same mileage.[29] Radiographically documented fractures occurred in 24% of amenorrheic athletes, compared with 9% of eumenorrheic athletes.[29]

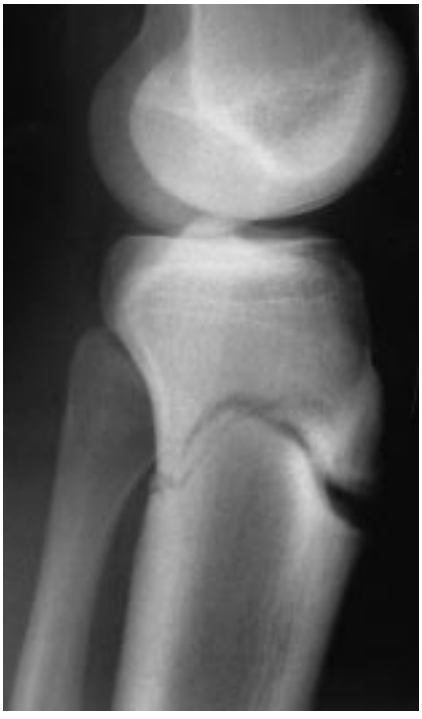

Fig. 5 Lateral radiograph of the tibia of a 22-year-old male triple jumper with a history of proximal tibia pain and radiologic evidence of a stress fracture. The patient failed to return for follow-up and continued to train until he suffered a displaced fracture of the tibia, which required operative repair.

Evaluation

The medical evaluation of an athlete with suspected bone loss must be thorough and multifactorial to arrive at the correct diagnosis. The nutritional history is essential to the evaluation. Calcium intake is obviously important, but the caloric and protein intake must be evaluated as well. Eating disorders, such as bulimia and anorexia nervosa, are more common in young women and should be aggressively investigated. Signs of anorexia include hair loss, lanugo, loose skin from rapid weight loss, and brittle nails. Dental caries and fingernail erosions are found in bulimia. Male and female athletes are much more likely than nonathletes to have disordered, nutritionally unhealthy eating patterns, but such irregularities are often difficult to uncover. Adequate amounts of carbohydrates, fats, and proteins must be consumed to support the athlete's level of activity and prevent a metabolic drain.

Questions regarding specific training regimens should be aimed at finding a recent change in intensity or length of training and the inclusion of high-impact or high-stress exercises (e.g., plyometrics) in the training regimen. An athlete's perception of stress related to competition itself and its impact on home, work, and school should also be assessed. Female athletes who associate a high degree of stress with competition are more likely to be amenorrheic.[34] A complete medical workup is necessary for anyone over 16 years old with primary amenorrhea regardless of probable cause; a woman with an established menstrual history may need a more focused examination. The serum estrogen level may not be helpful unless it is determined after a progestin challenge; otherwise, the value may appear to be normal despite being low enough to cause amenorrhea.

If an increased remodeling rate is suspected in a mature male or female athelete, the serum level of bone Gla protein (BGP) should be determined. The concentration of this substance, a bone-specific noncollagenous protein made by osteoblasts, is indicative of bone turnover; the serum concentration has been found to correlate with the rate of bone loss in the forearm and lumbar spine. A twofold increase in BGP level occurs in oophorectomized women within 6 weeks after surgery and lasts for up to 24 months, indicating an increase in bone turnover or remodeling. The concentration returns to normal with estrogen therapy.[35]

Bone mineral density should be measured in every patient found to have athletic amenorrhea. If an abnormal value is found initially or the athlete refuses treatment, follow-up measurements should be performed every 1 to 2 years.[5] The most commonly used method of determining BMD is DEXA. This study involves less than 5 mrem of radiation per scan, compared with 20 to 50 mrem for a chest radiograph.[1] The density of bone is determined in a specific area (usually the femoral neck, lumbar spine, or distal radius), and then computer

analysis is used to compare the BMD with established norms. Normal BMD is defined as an average for a given age. For example, the BMD should be higher in the young than in the elderly and should be higher in areas of predominantly cortical bone than in trabecular bone. Total body scans are becoming more available, allowing study of specific areas, such as the tibial shaft. One of the shortcomings of DEXA is that control values for young adults are based on small populations and may, therefore, be inaccurate. Scans of young athletes still need further study, and results should be considered only one part of the workup and not the definitive test for low BMD. However, recent advances in techniques may make DEXA measurements more accurate and more specific for bone loss in certain areas.[36,37]

Treatment

Maximum bone loss occurs in the early phase of amenorrhea. Therefore, treatment should begin immediately after the diagnosis of osteoporosis. Patients should be informed of the potential problems associated with low BMD, especially the increased risk of fractures as they become middle-aged and elderly, which may be permanently disabling.

Calcium intake should be increased to at least 1,500 mg per day for any athlete. Intake greater than 120% of the recommended dietary allowance has been found to protect male and female athletes against stress fractures (Table 1).[33] Despite calcium supplementation for 1 to 2 years, there may be no change in the BMD in the femur or spine in athletes, but there can be an increase in tibial BMD, suggesting a site-specific effect that may protect those bones withstanding the most stress.

Increasing the number of menstrual cycles by even one or two per year might improve the skeletal health of a female athlete.[1] Lindberg et al[38] found that in runners who decreased their mileage by 43%, increased their body weight by 5%, and took calcium supplements, menses resumed, estradiol levels rose, and BMD increased by 6.7%. In contrast, women who did not change their training regimen over the same time period had no change in BMD despite supplemental calcium. A similar experiment by Drinkwater et al[39] demonstrated that decreasing mileage alone increased vertebral bone mass by 6.4% and allowed the resumption of menses. Subjects who did not decrease their mileage lost 3.4% of their BMD over the same time period, leading to a nearly 10% difference in bone mass over a short interval.

It cannot be emphasized enough that persuading an athlete to decrease his or her training regimen can be very difficult. Education about long-term sequelae is extremely important. Counseling about changing regimens, such as cross-training or moderating the current program, may be necessary to effect the changes needed.

It can take months to years for normal menstrual function to resume, in contrast to the quick onset of amenorrhea. To help hasten the return to a normal estrogen level, replacement with birth control pills or estrogen alone can be used. The goal of estrogen replacement is to maintain BMD, especially in amenorrheic adolescents with stress fractures. To date, there are no controlled studies comparing the use of birth control pills with estrogen replacement therapy. Estrogen can cause a 0.2% to 2.9% increase in BMD per year in amenorrheic athletes, with the lumbar spine and proximal femur being affected most.[40] In one study,[41] young oophorectomized women treated with estrogen had a 4% incidence of minor trabecular fractures, compared with 38% in those not treated. In another study,[24] estrogen in combination with calcium worked even better, with a 4% increase in BMD over the course of 1 year; this may have been due to the effect of estrogen in increasing the ability of the renal and digestive systems to absorb and resorb calcium.

Despite supplemental calcium, estrogen replacement, or resumption of menses, premature osteoporosis secondary to long-term amenorrhea in the young female athlete may be irreversible. If amenorrhea lasts more than 3 years (nearly equivalent to the time course of menopause in middle-aged women), decreased BMD is not reversible with calcium supplements or estrogen replacement.[1] Even if the rate of bone turnover can be decreased, these athletes are still at increased risk of fracture because their BMD continues to be

Table 1
Daily Calcium Requirements

Age and Sex	Recommended Dietary Allowance, mg/day*
General	
1-5 yr	1,000
6-11 yr	1,200
12-24 yr	1,200-1,500
Women	
Premenopausal	1,000
Postmenopausal	1,500
Athlete	1,500
Men	
25-64 yr	1,000
>65 yr	1,500
Athlete	1,500

* As an example, 1 cup (8 oz) of milk contains 300 mg of calcium.

lower than that of age-matched normal individuals.[5,26,29]

For men, testosterone, bisphosphonates, and calcitonin may help, but clinical trials have yet to prove this.[13] There are no short- or long-term studies of any treatment for men with low bone density; therefore, we can only recommend empiric treatment, including calcium supplementation and decreased training. Any treatment involving testosterone should be done under the guidance of an endocrinologist. Inasmuch as men are not subject to a sudden decrease in testosterone at middle age, their risk of fracture does not increase as much as that of age-matched women with similarly decreased BMD.

Summary

Athletes involved in endurance activities are prone to having low levels of sex hormones due to poor diet and overtraining. The resultant low BMD places them at increased risk for stress fractures and overt fractures. A concern for orthopaedists is the relatively young age at which these patients will need treatment, possibly even fixation, of fractures. It is imperative to thoroughly question patients who are athletes if stress fractures are suspected and consider metabolic workups for patients in the high-risk category.

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