The Biomechanics of Human Bone Growth and Development

After completing this chapter, you will be able to:

- Explain how the material constituents and structural organization of bone affect its ability to withstand mechanical loads.
- Describe the processes involved in the normal growth and maturation of bone.
- Describe the effects of exercise and of weightlessness on bone mineralization.
- Explain the significance of osteoporosis and discuss current theories on its prevention.
- Explain the relationship between different forms of mechanical loading and common bone injuries.

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What determines when a bone stops growing? How are stress fractures caused? Why does space travel cause reduced bone mineral density in astronauts? What is osteoporosis and how can it be prevented?

The word bone typically conjures up a mental image of a dead bone—a dry, brittle chunk of mineral that a dog would enjoy chewing. Given this picture, it is difficult to realize that living bone is an extremely dynamic tissue that is continually modeled and remodeled by the forces acting on it. Bone fulfills two important mechanical functions for human beings: (a) It provides a rigid skeletal framework that supports and protects other body tissues, and (b) it forms a system of rigid levers that can be moved by forces from the attaching muscles (see Chapter 12). This chapter discusses the biomechanical aspects of bone composition and structure, bone growth and development, bone response to stress, osteoporosis, and common bone injuries.

COMPOSITION AND STRUCTURE OF BONE TISSUE

The material constituents and structural organization of bone influence the ways in which bone responds to mechanical loading. The composition and structure of bone yield a material that is strong for its relatively light weight.

Material Constituents

The major building blocks of bone are calcium carbonate, calcium phosphate, collagen, and water. The relative percentages of these materials vary with the age and health of the bone. Calcium carbonate and calcium phosphate generally constitute approximately 60–70% of dry bone weight. These minerals give bone its stiffness and are the primary determiners of its compressive strength. Other minerals, including magnesium, sodium, and fluoride, also have vital structural and metabolic roles in bone growth and development. Collagen is a protein that provides bone with flexibility and contributes to its tensile strength.

The water content of bone makes up approximately 25–30% of total bone weight. The water present in bone tissue is an important contributor to bone strength. For this reason, scientists and engineers studying the material properties of different types of bone tissue must ensure that the bone specimens they are testing do not become dehydrated. The flow of water through bones also carries nutrients to and waste products away from the living bone cells within the mineralized matrix. In addition, water transports mineral ions to and from bone for storage and subsequent use by the body tissues when needed.

Structural Organization

The relative percentage of bone mineralization varies not only with the age of the individual but also with the specific bone in the body. Some bones are more porous than others. The more porous the bone, the smaller the proportion of calcium phosphate and calcium carbonate, and the greater the proportion of nonmineralized tissue. Bone tissue has been classified into two categories based on porosity (Figure 4-1). If the porosity is low, with 5–30% of bone volume occupied by nonmineralized tissue, the tissue is termed cortical bone. Bone tissue with a relatively high porosity, with 30% to greater than 90% of bone volume occupied by nonmineralized tissue, is known as spongy, cancellous, or
**trabecular bone.** Trabecular bone has a honeycomb structure with mineralized vertical and horizontal bars, called *trabeculae*, forming cells filled with marrow and fat.

The porosity of bone is of interest because it directly affects the mechanical characteristics of the tissue. With its higher mineral content, cortical bone is stiffer, so that it can withstand greater stress, but less **strain** or relative deformation, than trabecular bone. Because trabecular bone is spongyer than cortical bone, it can undergo more strain before fracturing.

The function of a given bone determines its structure. The shafts of the long bones are composed of strong cortical bone. The relatively high

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(A) In the femur, trabecular bone is encased by a thin layer of cortical bone.
(B) In the skull, trabecular bone is sandwiched between plates of cortical bone.

Because cortical bone is stiffer than trabecular bone, it can withstand greater stress but less strain. Anisotropic exhibiting different mechanical properties in response to loads from different directions. Bone is strongest in resisting compression and weakest in resisting shear.

Axial skeleton the skull, vertebrae, sternum, and ribs
Appendicular skeleton bones composing the body appendages
Short bones small, cubical skeletal structures, including the carpals and tarsals
Flat bones skeletal structures that are largely flat in shape—for example, the scapula
Irregular bones skeletal structures of irregular shape—for example, the sacrum
Long bones skeletal structures consisting of a long shaft with bulbous ends—for example, the femur
Articular cartilage protective layer of firm, flexible connective tissue over the articulating ends of long bones

Trabecular bone content of the vertebrae contributes to their shock-absorbing capability.

Both cortical and trabecular bone are anisotropic; that is, they exhibit different strength and stiffness in response to forces applied from different directions. Bone is strongest in resisting compressive stress and weakest in resisting shear stress (Figure 4-2).

Types of Bones

The structures and shapes of the 206 bones of the human body enable them to fulfill specific functions. The skeletal system is nominally subdivided into the central or axial skeleton and the peripheral or appendicular skeleton (Figure 4-3). The axial skeleton includes the bones that form the axis of the body, which are the skull, the vertebrae, the sternum, and the ribs. The other bones form the body appendages, or the appendicular skeleton. Bones are also categorized according to their general shapes and functions.

Short bones, which are approximately cubical, include only the carpals and the tarsals (Figure 4-4). These bones provide limited gliding motions and serve as shock absorbers.

Flat bones are also described by their name (Figure 4-4). These bones protect underlying organs and soft tissues and also provide large areas for muscle and ligament attachments. The flat bones include the scapulae, sternum, ribs, patellae, and some of the bones of the skull.

Irregular bones have different shapes to fulfill special functions in the human body (Figure 4-4). For example, the vertebrae provide a bony, protective tunnel for the spinal cord; offer several processes for muscle and ligament attachments; and support the weight of the superior body parts while enabling movement of the trunk in all three cardinal planes. The sacrum, coccyx, and maxilla are other examples of irregular bones.

Long bones form the framework of the appendicular skeleton (Figure 4-4). They consist of a long, roughly cylindrical shaft (also called the body, or diaphysis) of cortical bone, with bulbous ends known as condyles, tubercles, or tuberosities. A self-lubricating articular cartilage protects the
FIGURE 4-3  The human skeleton.

ends of long bones from wear at points of contact with other bones. Long bones also contain a central hollow area known as the medullary cavity or canal.

The long bones are adapted in size and weight for specific biomechanical functions. The tibia and femur are large and massive to support the weight of the body. The long bones of the upper extremity, including the humerus, radius, and ulna, are smaller and lighter to promote ease of movement. Other long bones include the clavicle, fibula, metatarsals, metacarpals, and phalanges.
FIGURE 4-4

A. The carpals are categorized as short bones. B. The scapula is categorized as a flat bone. C. The vertebrae are examples of irregular bones. D. The femur represents the long bones.

BONE GROWTH AND DEVELOPMENT

- Most epiphyses close around age 18, although some may be present until about age 25.

**Epiphysis**

growth center of a bone that produces new bone tissue as part of the normal growth process until it closes during adolescence or early adulthood.

Bone growth begins early in fetal development, and living bone is continually changing in composition and structure during the life span. Many of these changes represent normal growth and maturation of bone.

**Longitudinal Growth**

Longitudinal growth of a bone occurs at the epiphyses, or epiphyseal plates (Figure 4-5). The epiphyses are cartilaginous discs found near the ends of the long bones. The diaphysis (central) side of each epiphysis
continually produces new bone cells. During or shortly following adolescence, the plate disappears and the bone fuses, terminating longitudinal growth. Most epiphyses close around age 18, although some may be present until about age 25.

**Circumferential Growth**

Long bones grow in diameter throughout most of the life span, although the most rapid bone growth occurs before adulthood. The internal layer of the periosteum builds concentric layers of new bone tissue on top of existing ones. At the same time, bone is resorbed or eliminated around the circumference of the medullary cavity, so that the diameter of the cavity is continually enlarged. This occurs in such a way that both bending stresses and torsional stresses on the bones remain relatively constant (65).

These changes in bone size and shape are the work of specialized cells called osteoblasts and osteoclasts, which respectively form and resorb bone tissue. In healthy adult bone, the activity of osteoblasts and osteoclasts is largely balanced.

**Adult Bone Development**

There is a progressive loss of collagen and increase in bone brittleness with aging. Thus, the bones of children are more pliable than the bones of adults.

Bone mineral normally accumulates throughout childhood and adolescence, reaching a peak at about age 25–28 in women and age 30–35 in men (55). Following this peak, researchers disagree as to the length of time that bone density remains constant (62). However, an age-related, progressive decline in bone density and bone strength in both men and women may begin as soon as the early twenties (46). This involves a progressive diminishment in the mechanical properties and general toughness of bone, with increasing loss of bone substance and increasing porosity (15). Trabecular bone is particularly affected, with progressive disconnection and disintegration of trabeculae compromising the integrity of the bone’s structure and seriously diminishing bone strength (41).

These changes are much more pronounced in women than in men, however. In women, there is a notable decrease in both volume and density of cortical bone, and a decrease in the density of trabecular bone.
with aging (67). Approximately 0.5–1.0% of bone mass is lost each year, until women reach about age 50 or menopause (62). Following menopause, there appears to be an increased rate of bone loss, with values as high as 6.5% per year reported during the first five to eight years (36). Although similar changes occur in men, they do not become significant before a more advanced age. Women at all ages tend to have smaller bones and less cortical bone area than do men (65), although volumetric bone mineral density is similar for both genders (69).

**BONE RESPONSE TO STRESS**

Other changes that occur in living bone throughout the life span are unrelated to normal growth and development. Bone responds dynamically to the presence or absence of different forces with changes in size, shape, and density. This phenomenon was originally described by the German scientist Julius Wolff in 1892:

> The form of a bone being given, the bone elements place or displace themselves in the direction of functional forces and increase or decrease their mass to reflect the amount of the functional forces (79).

**Bone Modeling and Remodeling**

According to Wolff’s law, the densities and, to a much lesser extent, the shapes and sizes of the bones of a given human being are a function of the magnitude and direction of the mechanical stresses that act on the bones. Dynamic mechanical loading causes bones to deform or strain, with larger loads producing higher levels of strain. These strains are translated into changes in bone shape and strength through a process known as

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*Wolff's law indicates that bone strength increases and decreases as the functional forces on the bone increase and decrease.*

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*The structure of a long bone.*

remodeling. Remodeling involves resorption of fatigue-damaged older bone and subsequent formation of new bone (43). Bone modeling is the term given to formation of new bone that is not preceded by resorption, and is the process by which immature bones grow.

Adult bones gain or lose mass in accordance with Wolff's law. When strain on a bone exceeds a certain threshold, new bone is laid down at the strain sites, and overall bone mass and density are increased. When strain magnitudes stay below a lower threshold, bone remodeling occurs, with bone removed close to the marrow (18). Strain magnitudes in between these two thresholds occur in what is termed the "lazy zone" and do not trigger bone adaptation (73). Remodeling can occur in either "conservation mode," with no change in bone mass, or "disuse mode," with a net loss of bone mass characterized by an enlarged marrow cavity and thinned cortex (18). Bone is a very dynamic tissue, with the modeling and remodeling processes continuously acting to increase, decrease, or reshape bone.

The modeling and remodeling processes are directed by osteocytes, cells embedded in bone that are sensitive to changes in the flow of interstitial fluid through the pores resulting from strain on the bone (68). Dynamic loading resulting from high-level impact produces a high rate of deformation that best pushes fluid through the bone matrix (61). It is for this reason that activities involving high levels of impact are best at stimulating bone formation. In response to the motion of fluid within the bone matrix, osteocytes trigger the actions of osteoblasts and osteoclasts, the cells that respectively form and resorb bone (61). A predominance of osteoblast activity produces bone modeling, with a net gain in bone mass. Bone remodeling involves a balance of osteoblast and osteoclast action or a predominance of osteoclast activity, with associated maintenance or loss of bone mass. Approximately 25% of the body's trabecular bone is remodeled each year through this process (29). Strains resulting from an activity such as walking are sufficient to provoke bone turnover and new bone formation (80).

Thus, bone mineralization and bone strength in both children and adults are a function of stresses producing strains on the skeleton. Since body weight provides the most constant mechanical stress to bones, bone mineral density generally parallels body weight, with heavier individuals having more massive bones. Adults who gain or lose weight tend to also gain or lose bone mineral density (17). However, a given individual's physical activity profile, diet, lifestyle, and genetics can also dramatically influence bone density. Factors such as lean body mass, muscle strength, and regular participation in weight-bearing exercise have been shown to exert stronger influences on bone density than weight, height, and race (20, 38, 76). Dynamic loading during participation in gymnastics has been shown to affect bone size and strength more than muscle mass (16). Even in young, nonathletic children, bone appears to remodel in response to the presence or absence of physical activity (31).

The malleability of bone is dramatically exemplified by the case of an infant who was born in normal physical condition but missing one tibia, the major weight-bearing bone of the lower extremity. After the child was walking for a time, X-rays revealed that modeling of the fibula in the abnormal leg had occurred to the extent that it could not be distinguished from the tibia of the other leg (1).

Another interesting case is that of a construction worker who had lost all but the fifth finger of one hand in a war injury. After 32 years, the
metacarpal and phalanx of the remaining finger had been modeled to resemble the third finger of the other hand (58).

**Bone Hypertrophy**

Although cases of complete changes in bone shape and size are unusual, there are many examples of bone modeling, or bone hypertrophy, in response to regular physical activity. The bones of physically active individuals tend to be denser and therefore more mineralized than those of sedentary individuals of the same age and gender. Moreover, the results of several studies indicate that occupations and sports particularly stressing a certain limb or region of the body produce accentuated bone hypertrophy in the stressed area. For example, professional tennis players display not only muscular hypertrophy in the tennis arm but also hypertrophy of that arm’s radius (35). Similar bone hypertrophy has been observed in the dominant humerus of baseball players (49).

It also appears that the greater the forces or loads habitually encountered, the more dramatic the increased mineralization of the bone. In one study involving collegiate female athletes in basketball, volleyball, soccer, track, and swimming, the athletes participating in high-impact sports (basketball and volleyball) were found to have higher bone mineral densities and bone formation values than the swimmers (12). In another investigation, the bone mineral densities of trained runners and cyclists were compared to those of sedentary individuals of the same age (65). Compared to the nonexercisers, the runners were found to have increased bone density, although this was not true for the cyclists. Among older women, both yard work and weight training have been found to be strong predictors for bone density, with jogging, swimming, and calisthenics being weak predictors (70). On the whole, the research evidence suggests that physical activity involving impact forces is necessary for maintaining or increasing bone mass (51). Competitive swimmers, who spend a lot of time in the water where the buoyant force counteracts gravity, may have bone mineral densities lower than those of sedentary individuals (56).

**Bone Atrophy**

Whereas bone hypertrophies in response to increased mechanical stress, it displays the opposite response to reduced stress. When the normal stresses exerted on bone by muscle contractions, weight bearing, or impact forces are reduced, bone tissue atrophies through remodeling. When bone atrophy occurs, the amount of calcium contained in the bone diminishes, and both the weight and the strength of the bone decrease. Loss of bone mass due to reduced mechanical stress has been found in bedridden patients, sedentary senior citizens, and astronauts. Four to six weeks of bed rest can result in significant decrements in bone mineral density that are not fully reversed after six months of normal weight-bearing activity (5).

Bone demineralization is a potentially serious problem. From a biomechanical standpoint, as bone mass diminishes, strength and thus resistance to fracture also decrease, particularly in trabecular bone.

The results of calcium loss studies conducted during the Skylab flights indicate that urinary calcium loss is related to time spent out of the earth’s gravitational field. The pattern of bone loss observed is highly similar to that documented among patients during periods of bed rest, with greater bone loss in the weight-bearing bones of the
lumbar spine and lower extremity than in other parts of the skeleton (64). During one month in space, astronauts lose 1–3% of bone mass, or approximately as much bone mass as postmenopausal women lose in a year (8, 23).

It is not yet clear what specific mechanism or mechanisms are responsible for bone loss outside of the gravitational field. Research has consistently documented a negative calcium balance in astronauts and experimental animals during space flight, with reduced intestinal absorption of calcium and increased excretion of calcium (71). It is not known, however, whether this is caused by an increase in bone remodeling, a decrease in bone remodeling, or an imbalance between osteoblast and osteoclast activity (71). It appears that the normal balance between formation and resorption of bone becomes disturbed, with an initial increase in osteoclast activity followed by a prolonged decrease in osteoblast activity (40). One hypothesis is that these changes in bone remodeling are precipitated by changes in bone blood flow related to being outside of the gravitational field (10). More research on this topic is clearly needed.

It remains to be seen if measures other than the artificial creation of gravity can effectively prevent bone loss during space travel. Astronauts' current exercise programs during flights in space are designed to prevent bone loss by increasing the mechanical stress and strain placed
on bones using muscular force. However, the muscles of the body exert mainly tensile forces on bone, whereas gravity provides a compressive force. Therefore, it may be that no amount of physical exercise alone can completely compensate for the absence of gravitational force.

Recent research shows that resistive exercise combined with whole-body vibration may be an effective countermeasure for preventing muscle atrophy and bone loss during space flight (57). Researchers hypothesize that low-amplitude, high-frequency vibration stimulates muscle spindles and alpha-motoneurons (see Chapter 6), which initiate muscle contraction (34, 53). The effects of several months' intervention treatment with whole-body vibration appear to include improved bone mineral density resulting from increased bone deposition coupled with decreased bone resorption, with bone density particularly improved in the femur and tibia (30, 45).

Since joints support the body weight positioned above them, the magnitude of skeletal loading varies from joint to joint during both resistance exercise and vibration. Sample Problem 4.1 illustrates this point.

**OSTEOPOROSIS**

Bone atrophy is a problem not only for astronauts and bedridden patients but also for a growing number of senior citizens and female athletes. Osteoporosis is found in most elderly individuals, with earlier onset in women, and is becoming increasingly prevalent with the increasing mean age of the population (37). The condition begins as osteopenia, reduced bone mass without the presence of a fracture, but often progresses to osteoporosis, a condition in which bone mineral mass and strength are so severely compromised that daily activities can cause bone pain and fracturing (50).

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**SAMPLE PROBLEM 4.1**

The tibia is the major weight-bearing bone in the lower extremity. If 88% of body mass is proximal to the knee joint, how much compressive force acts on each tibia when a 600 N person stands in anatomical position? How much compressive force acts on each tibia if the person holds a 20 N sack of groceries?

**Solution**

Given: $wt = 600 \text{ N}$

(It may be deduced that weight = compressive force, $F_c$.)

Formula: $F_c$ on knees = $(600 \text{ N})(0.88)$

\[ F_c \text{ on one knee} = \frac{(600 \text{ N})(0.88)}{2} \]

\[ F_c \text{ on one knee} = 264 \text{ N} \]

\[ F_c \text{ with groceries} = \frac{(600 \text{ N})(0.88) + 20 \text{ N}}{2} \]

\[ F_c \text{ with groceries} = 274 \text{ N} \]
Postmenopausal and Age-Associated Osteoporosis

The majority of those affected by osteoporosis are postmenopausal and elderly women, although elderly men are also susceptible, with more than half of all women and about one-third of men developing fractures related to osteoporosis (32). Although it was once regarded as primarily a health concern for women, with the increasing age of the population, osteoporosis is now also emerging as a serious health-related concern for men (69). Risk factors for osteoporosis include being female, white or Asian ethnicity, older age, small stature or frame size, and family history of osteoporosis (32).

Type I osteoporosis, or postmenopausal osteoporosis, affects approximately 40% of women after age 50 (39). The first osteoporotic fractures usually begin to occur about 15 years postmenopause, with women suffering approximately three times as many femoral neck fractures, three times as many vertebral fractures, and six times as many wrist fractures as men of the same age (39).

This discrepancy occurs partially because men reach a higher peak of bone mass and strength than women in early adulthood, and partially because of a greater prevalence of disconnections in the trabecular network among postmenopausal women than among men (47).

Type II osteoporosis, or age-associated osteoporosis, affects most women and also affects men after age 70 (69). After age 60, about 90% of all fractures in both men and women are osteoporosis-related, and these fractures are one of the leading causes of death in the elderly population (50).

Although the radius and ulna, femoral neck, and spine are all common sites of osteoporotic fractures, the most common symptom of osteoporosis is back pain derived from fractures of the weakened trabecular bone of the vertebral bodies. Crush fractures of the lumbar vertebrae resulting from compressive loads created by weight bearing during activities of daily living frequently cause reduction of body height. Because most body weight is anterior to the spine, the resulting fractures often leave the vertebral bodies wedge-shaped, accentuating thoracic kyphosis (see Chapter 9). This disabling deformity is known as Dowager’s hump. Vertebral compression fractures are extremely painful and debilitating and affect physical, functional, and psychosocial aspects of the person’s life. As spinal height is lost, there is added discomfort from the rib cage pressing on the pelvis.

As the skeleton ages in men, there is an increase in vertebral diameter that serves to reduce compressive stress during weight bearing (47). Thus, although osteoporotic changes may be taking place, the structural strength of the vertebrae is not reduced. Why the same compensatory change does not occur in women is unknown.

Female Athlete Triad

The desire to excel at competitive sports causes some young female athletes to strive to achieve an undesirably low body weight. This dangerous practice commonly involves a combination of disordered eating, amenorrhea, and osteoporosis, a combination that has come to be known as the “female athlete triad.” This condition often goes unrecognized, but because the triad can result in negative consequences ranging from irreversible bone loss to death, friends, parents, coaches, and physicians need to be alert to the signs and symptoms.

As many as 62% of female athletes in certain sports display disordered eating behaviors, with those participating in endurance or artistic sports such as gymnastics and figure skating most likely to be involved (48). Prolonged disordered eating can lead to anorexia nervosa or bulimia nervosa, illnesses that affect 1–10% of all adolescent and college-age women (22).
Symptoms of anorexia nervosa in girls and women include body weight 15% or more below minimal normal weight for age and height, an intense fear of gaining weight, a disturbed body image, and amenorrhea. Symptoms of bulimia nervosa are a minimum of two eating binges a week for at least three months, a feeling of lack of control during binges, regular use of self-induced vomiting, laxatives, diuretics, strict dieting, or exercise to prevent weight gain, and excessive concern with body image and weight (22). Disordered eating behavior has been found to be strongly associated with both menstrual irregularity and low bone mineral density (9).

The relationship between disordered eating and amenorrhea appears to be related to a decrease in hypothalamic secretion of gonadotrophin-releasing hormone, which in turn decreases the secretion of luteinizing hormone and follicle-stimulating hormone, with subsequent shutting down of stimulation of the ovary (75). The prevalence of primary amenorrhea, with menarche delayed beyond 16 years of age, is less than 1% in the general population, but as high as 22% in sports such as cheerleading, diving, and gymnastics (48). Secondary amenorrhea, or the absence of three to six consecutive menstrual cycles, has been found to be present in 69% of dancers and 65% of long distance runners, as compared to 2–5% in the general population (48).

The link between cessation of menses and osteoporosis is estrogen deficiency, which increases bone resorption. Energy deficiency resulting from disordered eating is also likely to independently contribute to altered bone metabolism and reduced bone density (14). Although the incidence of osteoporosis among female athletes is unknown, the consequences of this disorder in young women are potentially tragic. Among one group of over 200 premenopausal female runners, those with amenorrhea had 10% less lumbar bone density than those with normal menses (25). This is of particular concern for adolescent athletes, because roughly 50% of bone mineralization and 15% of adult height are normally established during the teenage years (2). Not surprisingly, amenorrheic premenopausal female athletes have a high rate of stress fractures, with more fractures related to later onset of menarche (48). Moreover, the loss of bone that occurs may be irreversible, and osteoporotic wedge fractures can ruin posture for life.

**Preventing and Treating Osteoporosis**

Osteoporosis is neither a disease with acute onset nor an inevitable accompaniment of aging, but is the result of a lifetime of habits that are erosive to the skeletal system. Early detection of low bone mineral density is advantageous, because once osteoporotic fractures begin to occur, there has been irreversible loss of trabecular structure (60). Although proper diet, hormone levels, and exercise can work to increase bone mass at any stage in life, evidence suggests that it is easier to prevent osteoporosis than it is to treat it. The single most important factor for preventing or prolonging the onset of osteoporosis is the optimization of peak bone mass during childhood and adolescence (6, 9, 24, 32, 50, 74). Researchers hypothesize that weight-bearing exercise is particularly crucial during the prepubertal years, because the presence of high levels of growth hormone may act with exercise in a synergistic fashion to increase bone density (3, 6, 22, 33, 36). Activities involving osteogenic impact forces, such as jumping, have been shown to be effective in increasing bone mass in children (19).

Weight-bearing physical activity is necessary for maintaining skeletal integrity in both humans and animals. Importantly, studies show that a regular program of weight-bearing exercise, such as walking, can increase bone health and strength even among individuals with osteoporosis.
Because impact loading is particularly osteogenic, jumping in place, with 5–10 sets of 10 jumps done 3–5 times per week, is also recommended for maintenance of bone mass (77). Jumps should be performed with 10–15-second rest intervals between jumps, as this appears to enhance fluid flow within the bone matrix and the related stimulation of osteocytes, potentially doubling the effects of mechanical loading on bone building (21, 52). In practical terms, a very slow childhood game of hopscotch favors bone building over a fast one!

Increased dietary calcium intake exerts a positive influence on bone mass for women with a dietary deficiency, with the amount of calcium absorbed influenced positively by calcitriol (the active form of vitamin D) and negatively by dietary fiber (63). Although adequate dietary calcium is particularly important during the teenage years, unfortunately the median American girl falls below the recommended daily intake of 1200 mg per day by age 11 (13). A modified diet or calcium supplementation can be critical for the development of peak bone mass among adolescent females at a dietary deficiency. The role of vitamin D in enabling absorption of calcium by bone is also important, with over half the women receiving treatment for low bone density in North America having a vitamin D deficiency (26). Clinicians are now recognizing that a predisposition for osteoporosis can begin in childhood and adolescence when a poor diet interferes with bone mass development (7).

Other lifestyle factors also affect bone mineralization. Known risk factors for developing osteoporosis include physical inactivity; weight loss or excessive thinness; tobacco smoking; deficiencies in estrogen, calcium, and vitamin D; and excessive consumption of protein and caffeine (54, 62, 72, 78). A study of female twins, one of whom smoked more heavily than the other, showed that women who smoke one pack of cigarettes a day through adulthood will have a reduction in bone density of 5–10% by the time of menopause, which is sufficient to increase the risk of fracture (27). Although caffeine consumption may negatively affect bone mineral density among postmenopausal women who consume low amounts of dietary calcium, it has been shown not to affect bone mineral density among young women (11). Genetic factors also influence bone mass but do not appear to be as important as diet and exercise.

Recent, detailed studies of bone are increasingly showing that subtleties in bone microarchitecture may be more important in determining bone's resistance to fracture than bone mineral density (4, 59). In simple terms, bone quality may be more important in some ways than bone quantity. However, factors affecting bone structure within and around the trabeculae are currently unknown. Until much more is understood about osteoporosis, young women in particular are encouraged to maximize peak bone mass and to minimize its loss by engaging in regular physical activity and avoiding the lifestyle factors that negatively affect bone health.

**COMMON BONE INJURIES**

Because of the important mechanical functions performed by bone, bone health is an important part of general health. Bone health can be impaired by injuries and pathologies.

**Fractures**

A fracture is a disruption in the continuity of a bone. The nature of a fracture depends on the direction, magnitude, loading rate, and duration of the mechanical load sustained, as well as the health and maturity of the bone.

A greenstick fracture is incomplete, and the break occurs on the convex surface of the bend in the bone.

A fissured fracture involves an incomplete longitudinal break.

A comminuted fracture is complete and fragments the bone.

A transverse fracture is complete, and the break occurs at a right angle to the axis of the bone.

An oblique fracture occurs at an angle other than a right angle to the axis of the bone.

A spiral fracture is caused by twisting a bone excessively.

Under excessive bending loads, bone tends to fracture on the side loaded in tension.

the bone at the time of injury. Fractures are classified as simple when the bone ends remain within the surrounding soft tissues and compound when one or both bone ends protrude from the skin. When the loading rate is rapid, a fracture is more likely to be comminuted, containing multiple fragments (Figure 4-6).

Avulsions are fractures caused by tensile loading in which a tendon or ligament pulls a small chip of bone away from the rest of the bone. Explosive throwing and jumping movements may result in avulsion fractures of the medial epicondyle of the humerus and the calcaneus.

Excessive bending and torsional loads can produce spiral fractures of the long bones (Figure 4-6). The simultaneous application of forces from opposite directions at different points along a structure such as a long bone generates a torque known as a bending moment, which can cause bending and ultimately fracture of the bone. A bending moment is created on a football player’s leg when the foot is anchored to the ground and tacklers apply forces at different points on the leg in opposite directions. When bending is present, the structure is loaded in tension on one side and in compression on the opposite side, as discussed in Chapter 3. Because bone is stronger in resisting compression than in resisting tension, the side of the bone loaded in tension will fracture first.

Torque applied about the long axis of a structure such as a long bone causes torsion, or twisting of the structure. Torsion creates shear stress throughout the structure, as explained in Chapter 3. When a skier’s body rotates with respect to one boot and ski during a fall, torsional loads can cause a spiral fracture of the tibia. In such cases, a combined loading pattern of shear and tension produces failure at an oblique orientation to the longitudinal axis of the bone.

Since bone is stronger in resisting compression than in resisting tension and shear, acute compression fractures of bone (in the absence of osteoporosis) are rare. However, under combined loading, a fracture
resulting from a torsional load may also be impacted by the presence of a compressive load. An impacted fracture is one in which the opposite sides of the fracture are compressed together. Fractures that result in depression of bone fragments into the underlying tissues are termed depressed.

Since the bones of children contain relatively larger amounts of collagen than do adult bones, they are more flexible and more resistant to fracture under day-to-day loading than are adult bones. Consequently, greenstick fractures, or incomplete fractures, are more common in children than in adults (Figure 4-6). A greenstick fracture is an incomplete fracture caused by bending or torsional loads.

Stress fractures, also known as fatigue fractures, result from low-magnitude forces sustained on a repeated basis. Any increase in the magnitude or frequency of bone loading produces a stress reaction, which may involve microdamage. Bone responds to microdamage by remodeling: First, osteoclasts resorb the damaged tissue; then, osteoblasts deposit new bone at the site. When there is not time for the repair process to complete itself before additional microdamage occurs, the condition can progress to a stress fracture. Stress fractures begin as a small disruption in the continuity of the outer layers of cortical bone but can worsen over time, eventually resulting in complete cortical fracture.

In runners, a group particularly prone to stress fractures, about 50% of fractures occur in the tibia and approximately 20% of fractures are in the metatarsals, with fractures of the femoral neck and pubis also reported (28, 44). Increases in training duration or intensity that do not allow enough time for bone remodeling to occur are the primary culprits. Other factors that predispose runners to stress fractures include muscular fatigue and abrupt changes in either the running surface or the running direction (44).

**Epiphyseal Injuries**

About 10% of acute skeletal injuries in children and adolescents involve the epiphysis (42). Epiphyseal injuries include injuries to the cartilaginous
epiphyseal plate, the articular cartilage, and the apophysis. The apophyses are the sites of tendon attachments to bone, where bone shape is influenced by the tensile loads to which these sites are subjected. The epiphyses of long bones are termed pressure epiphyses and the apophyses are called traction epiphyses, after the types of physiological loading present. Both acute and repetitive loading can injure the growth plate, potentially resulting in premature closure of the epiphyseal junction and termination of bone growth.

Another form of epiphyseal injury, osteochondrosis, involves disruption of blood supply to an epiphysis, with associated tissue necrosis and potential deformation of the epiphysis. The cause of the condition is poorly understood. Osteochondrosis occurs most commonly between the ages of 3 and 10 and is more prevalent among boys than among girls (44).

Osteochondrosis of an apophysis, known as apophysitis, is often associated with traumatic avulsions. Common sites for apophysitis are the calcaneus and the tibial tubercle at the site of the patellar tendon attachment, where the disorder is referred to respectively as Sever’s disease and Osgood-Schlatter’s disease.

**SUMMARY**

Bone is an important and dynamic living tissue. Its mechanical functions are to support and protect other body tissues and to act as a system of rigid levers that can be manipulated by the attached muscles.

Bone’s strength and resistance to fracture depend on its material composition and organizational structure. Minerals contribute to a bone’s hardness and compressive strength, and collagen provides its flexibility and tensile strength. Cortical bone is stiffer and stronger than trabecular bone, whereas trabecular bone has greater shock-absorbing capabilities.

Bone is an extremely dynamic tissue that is continually being modeled and remodeled in accordance with Wolff’s law. Although bones grow in length only until the epiphyseal plates close at adolescence, bones continually change in density, and to some extent in size and shape, through the actions of osteoblasts and osteoclasts.

Osteoporosis, a disorder characterized by excessive loss of bone mineral mass and strength, is extremely prevalent among the elderly. It affects women at an earlier age and more severely than men. It is also present in an alarming frequency among young, eating-disordered, amenorrheic female athletes. Although the cause of osteoporosis remains unknown, the condition can often be improved through hormone therapy, avoidance of negative lifestyle factors, and a regular exercise program.

**INTRODUCTORY PROBLEMS**

1. Explain why the bones of the human body are stronger in resisting compression than in resisting tension and shear.

2. In the human femur, bone tissue is strongest in resisting compressive force, approximately half as strong in resisting tensile force, and only about one-fifth as strong in resisting shear force. If a tensile force of 8000 N is sufficient to produce a fracture, how much compressive force will produce a fracture? How much shear force will produce a fracture? (Answer: compressive force = 16,000 N; shear force = 3200 N)

3. Explain why bone density is related to an individual’s body weight.