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BIOMECHANICS OF BONE

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The purpose of the skeletal system is to protect internal organs, provide rigid kinematic links and muscle attachment sites, and facilitate muscle action and body movement. Bone has unique structural and mechanical properties that allow it to carry out these roles. Bone is among the body's hardest structures, only dentin and enamel in the teeth being harder. It is one of the most dynamic and metabolically active tissues in the body and remains active throughout life. A highly vascular tissue, it has an excellent capacity for self-repair and can alter its properties and configuration in response to changes in mechanical demand. For example, changes in bone density are commonly observed after periods of disuse and of greatly increased use; changes in bone shape are noted during fracture healing and after certain operations. Thus, bone adapts to the mechanical demands placed on it.

This chapter describes the composition and structure of bone tissue, the mechanical properties of bone, and the behavior of bone under different loading conditions. Various factors that affect the mechanical behavior of bone in vitro and in vivo are also discussed.

BONE COMPOSITION AND STRUCTURE

Bone tissue is a specialized connective tissue whose solid composition suits it for its supportive and protective roles. Like other connective tissues, it consists of cells and an organic extracellular matrix of fibers and ground substance produced by the cells. The distinguishing feature of bone is its high content of inorganic materials, in the form of mineral salts, that combine intimately with the organic matrix. The inorganic component of bone makes the tissue hard and rigid, while the organic component gives bone its flexibility and resilience.

The mineral portion of bone consists primarily of calcium and phosphate, mainly in the form of small crystals resembling synthetic hydroxyapatite crystals with the composition $Ca_{10}(PO_4)_6(OH)_2$. These minerals, which account for 65 to 70% of the bone's dry weight, give bone its solid consistency. Bone serves as a reservoir for essential minerals in the body, particularly calcium.

Bone mineral is embedded in variously oriented fibers of the protein collagen, the fibrous portion of the extracellular matrix. Collagen fibers are tough and pliable, yet they resist stretching and have little extensibility. Collagen composes approximately 95% of the extracellular matrix and accounts for about 25 to 30% of the dry weight of bone. A universal building block of the body, collagen is also the chief fibrous component of other skeletal structures. (A detailed description of the microstructure and mechanical behavior of collagen is provided in Chapters 2 and 3.)

The gelatinous ground substance surrounding the mineralized collagen fibers consists mainly of protein polysaccharides, or glycosaminoglycans (GAGs), primarily in the form of complex macromolecules called proteoglycans (PGs). The GAGs serve as a cementing substance between layers of mineralized collagen fibers. These GAGs, along with various noncollagenous glycoproteins, constitute about 5% of the extracellular matrix. (The structure of PGs, which are vital components of articular cartilage, is described in detail in Chapter 2.)

Water is fairly abundant in live bone, accounting for up to 25% of its total weight. About 85% of the water is found in the organic matrix, around the collagen fibers and ground substance, and in the hydration shells surrounding the bone crystals. The other 15% is located in canals and cavities that house bone cells and carry nutrients to the bone tissue.

At the microscopic level, the fundamental structural unit of bone is the osteon, or haversian system (Fig. 1–1). At the center of each osteon is a small channel, called a haversian canal, that contains blood vessels and nerve fibers. The osteon itself consists of a concentric series of layers (lamellae) of mineralized matrix surrounding the central canal, a configuration similar to growth rings in a tree trunk.

Along the boundaries of each layer, or lamella, are small cavities known as lacunae, each containing one bone cell, or osteocyte (see Fig. 1–1C). Numerous small channels, called canaliculi, radiate from each lacuna, connecting the lacunae of adjacent lamellae and ultimately reaching the haversian canal. Cell processes extend from the osteocytes into the canaliculi, allowing nutrients from the blood vessels in the haversian canal to reach the osteocytes.

At the periphery of each osteon is a cement line, a narrow area of cementlike ground substance composed primarily of glycosaminoglycans. The canaliculi of the osteon do not pass this cement line. Like the canaliculi, the collagen fibers in the bone matrix interconnect from one lamella to another within an osteon but do not cross the cement line. This intertwining of collagen fibers within the osteon undoubtedly increases the bone's resistance to mechanical stress and probably explains why the cement line is the weakest portion of the bone's microstructure (Dempster and Coleman, 1960; Evans and Bang, 1967).

A typical osteon is about 200 micrometers (μm) in diameter. Hence, every point in the osteon is no more than 100 μm from the centrally located blood supply. In the long bones, the osteons usually run longitudi

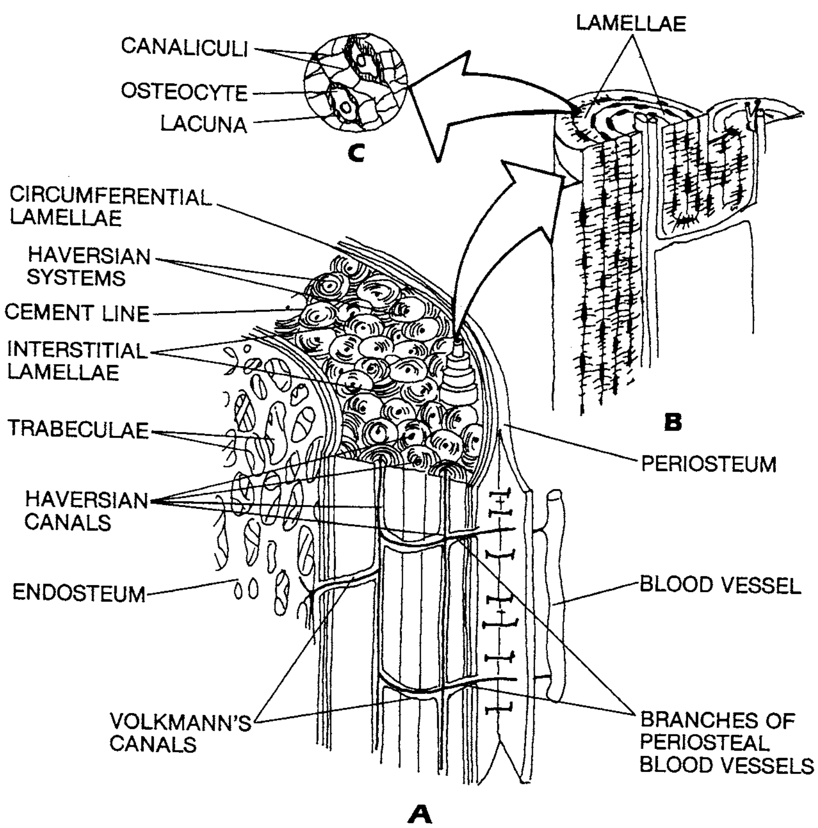


FIG. 1–1
A. The fine structure of bone is illustrated schematically in a section of the shaft of a long bone depicted without inner marrow. The osteons, or haversian systems, are apparent as the structural units of bone. In the center of the osteons are the haversian canals, which form the main branches of the circulatory network in bone. Each osteon is bounded by a cement line. One osteon is shown extending from the bone (20×). (Adapted from Bassett, 1965.) **B.** Each osteon consists of lamellae, concentric rings composed of mineral matrix surrounding the haversian canal. (Adapted from Tortora and Anagnostakos, 1984.) **C.** Along the boundaries of the lamellae are small cavities known as lacunae, each of which contains a single bone cell, or osteocyte. Radiating from the lacunae are tiny canals, or canaliculi, into which the cytoplasmic processes of the osteocytes extend. (Adapted from Tortora and Anagnostakos, 1984.)

nally, but they branch frequently and anastomose extensively with each other.

Interstitial lamellae span the regions between complete osteons (see Fig. 1–1A). They are continuous with the osteons and are just the same material in a different geometric configuration. As in the osteons, no point in the interstitial lamellae is farther than 100 μm from its blood supply. The interfaces between these lamellae contain an array of lacunae in which osteocytes lie and from which canaliculi extend.

At the macroscopic level, all bones are composed of two types of osseous tissue: cortical, or compact, bone and cancellous, or trabecular, bone (Fig. 1–2). Cortical bone forms the outer shell, or cortex, of the bone and has a dense structure similar to that of ivory. Cancellous bone within this shell is composed of thin plates, or trabeculae, in a loose mesh structure; the interstices between the trabeculae are filled with red marrow. Cancellous bone tissue is arranged in concentric lacunae-containing lamellae, but it does not contain haversian canals. The osteocytes receive nutrients through canaliculi from blood vessels passing through the red marrow. Cortical bone always surrounds cancellous bone, but the relative quantity of each type varies among bones and within individual bones according to functional requirements.

Since the lamellar pattern and material composition of cancellous and cortical bone appear identical, the basic distinction between the two is the degree of porosity. Biomechanically, the two bone types can be considered as one material whose porosity and density vary over a wide range (Carter and Hayes, 1977b). The difference in the porosity of cortical and cancellous bone can be seen in cross sections from human tibiae (Fig. 1–3). The porosity ranges from 5 to 30% in cortical bone and from 30 to over 90% in cancellous bone. The distinction between porous cortical bone and dense cancellous bone is somewhat arbitrary.

All bones are surrounded by a dense fibrous membrane called the periosteum (see Fig. 1–1A). Its outer layer is permeated by blood vessels and nerve fibers that pass into the cortex via Volkmann's canals, connecting with the haversian canals and extending to the cancellous bone. An inner, osteogenic layer contains bone cells responsible for generating new bone during growth and repair (osteoblasts). The periosteum covers the entire bone except for the joint surfaces, which are covered with articular cartilage. In the long bones, a thinner membrane, the endosteum, lines the central (medullary) cavity, which is filled with yellow fatty marrow. The endosteum

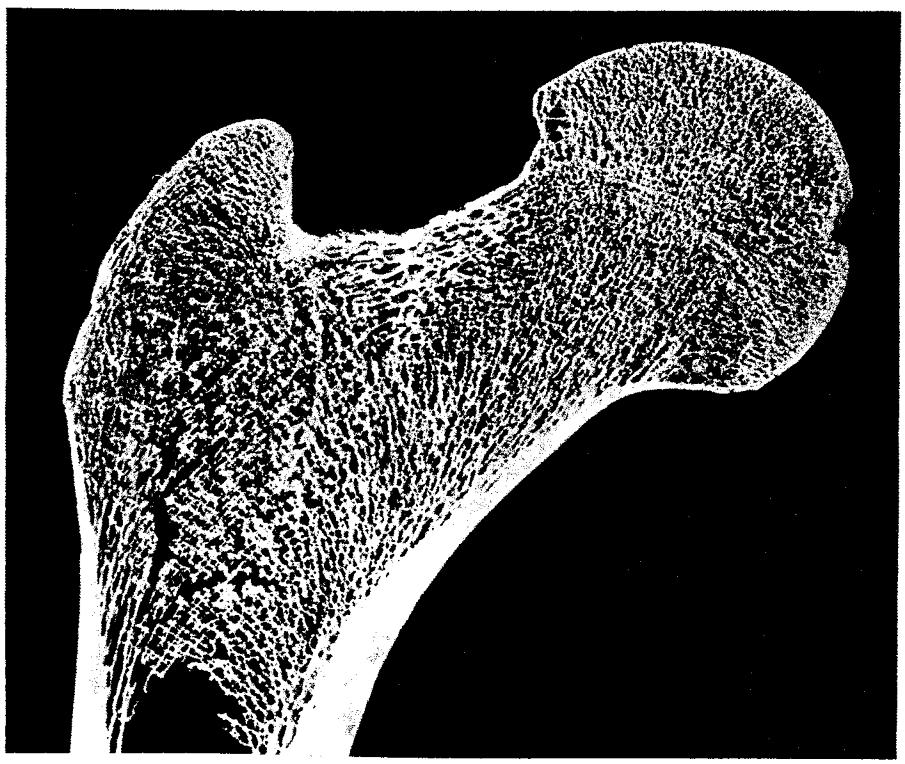
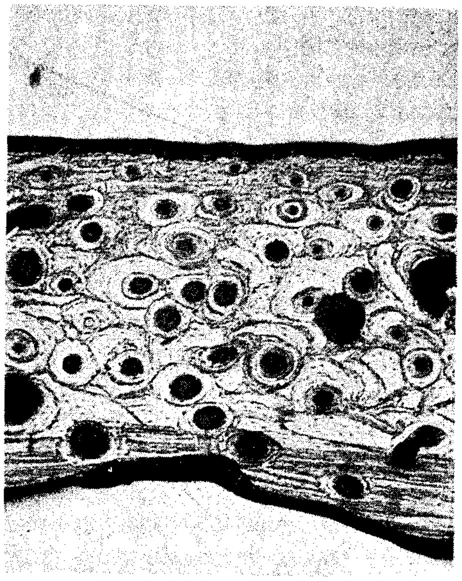
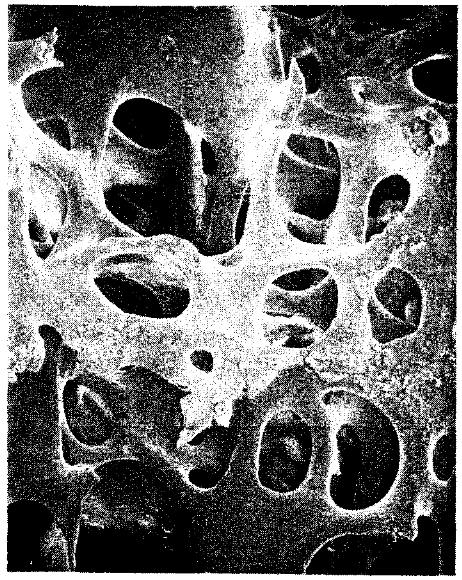


FIG. 1–2
Frontal longitudinal section through the head, neck, greater trochanter, and proximal shaft of an adult femur. Cancellous bone, with its trabeculae oriented in a lattice, lies within the shell of cortical bone. (Reprinted with permission from Gray, H.: Anatomy of the Human Body. 13th American Ed. Edited by C. D. Clemente. Philadelphia, Lea & Febiger, 1985.)



A



B

FIG. 1–3
A. Reflected-light photomicrograph of cortical bone from a human tibia (40×). (Courtesy of Dennis R. Carter, Ph.D.) **B.** Scanning electron photomicrograph of cancellous bone from a human tibia (30×). (Courtesy of Dennis R. Carter, Ph.D.)

contains osteoblasts and also giant multinucleated bone cells called osteoclasts, which play a role in the resorption of bone.

BIOMECHANICAL PROPERTIES OF BONE

Biomechanically, bone tissue may be regarded as a two-phase (biphasic) composite material, with the mineral as one phase and the collagen and ground substance as the other. In such materials (a nonbiologic example is fiberglass)—in which a strong, brittle material is embedded in a weaker, more flexible one—the combined substances are stronger for their weight than either substance alone (Bassett, 1965).

Functionally, the most important mechanical properties of bone are its strength and stiffness. These and other characteristics can best be understood for bone, or any other structure, by examining its behavior under loading, i.e., under the influence of externally applied forces. Loading causes a deformation, or a change in the dimensions, of the structure. When a load in a known direction is imposed on a structure, the deformation of that structure can be measured and plotted on a load-deformation curve. Much information about the strength, stiffness, and other mechanical properties of the structure can be gained by examining this curve.

A hypothetical load-deformation curve for a somewhat pliable fibrous structure, such as a long bone, is shown in Figure 1–4. The initial (straight line) portion of the curve, the elastic region, reveals the elasticity of the structure, i.e., its capacity for return-

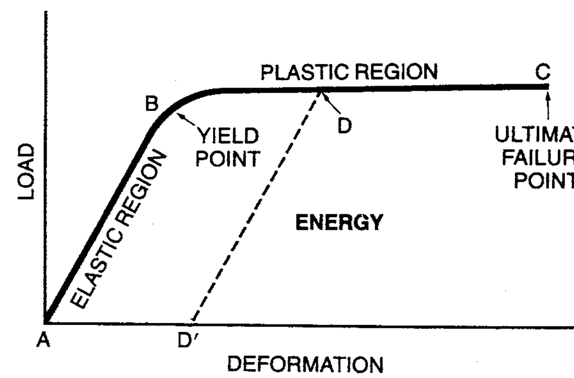


FIG. 1–4
Load-deformation curve for a structure composed of a somewhat pliable material. If a load is applied within the elastic range of the structure (A to B on the curve) and is then released, no permanent deformation occurs. If loading is continued past the yield point (B) and into the structure's plastic range (B to C on the curve) and the load is then released, permanent deformation results. The amount of permanent deformation that occurs if the structure is loaded to point D in the plastic region and then unloaded is represented by the distance between A and D′. If loading continues within the plastic range, an ultimate failure point (C) is reached.

ing to its original shape after the load is removed. As the load is applied, deformation occurs but is not permanent; the structure recovers its original shape when unloaded. As loading continues, the outermost fibers of the structure begin to yield at some point. This yield point signals the elastic limit of the structure. As the load exceeds this limit, the structure exhibits plastic behavior, reflected in the second (curved) portion of the curve, the plastic region. The structure will no longer return to its original dimensions when the load has been released; some residual deformation will be permanent. If loading is progressively increased, the structure will fail at some point (bone will fracture). This point is indicated by the ultimate failure point on the curve.

Three parameters for determining the strength of a structure are reflected on the load-deformation curve: (1) the load that the structure can sustain before failing, (2) the deformation that it can sustain before failing, and (3) the energy that it can store before failing. The strength in terms of load and deformation, or ultimate strength, is indicated on the curve by the ultimate failure point. The strength in terms of energy storage is indicated by the size of the area under the entire curve. The larger the area is, the greater the energy that builds up in the structure as the load is applied. The stiffness of the structure is indicated by the slope of the curve in the elastic region. The steeper the slope is, the stiffer the material.

The load-deformation curve is useful for determining the mechanical properties of whole structures such as a whole bone, an entire ligament or tendon, or a metal implant. This knowledge is helpful in the study of fracture behavior and repair, the response of a structure to physical stress, or the effect of various treatment programs; however, characterizing a bone or other structure in terms of the material that composes it, independent of its geometry, requires standardization of the testing conditions and the size and shape of the test specimens. Such standardized testing is useful for comparing the mechanical properties of two or more materials, such as the relative strength of bone and tendon tissue or the relative stiffness of various materials used in prosthetic implants. More precise units of measure can be used when standardized samples are tested, i.e., the load per unit of area of the sample (stress) and the amount of deformation in terms of the percentage of change in the sample's dimensions (strain). The curve generated is a stress-strain curve.

Stress is the load, or force, per unit area that develops on a plane surface within a structure in response to externally applied loads. The three units most commonly used for measuring stress in standardized samples of bone are newtons per centimeter squared (N/cm^2); newtons per meter squared, or pascals (N/m^2, Pa); and meganewtons per meter squared, or megapascals (MN/m^2, MPa).

Strain is the deformation (change in dimension) that develops within a structure in response to externally applied loads. The two basic types of strain are linear strain, which causes a change in the length of the specimen, and shear strain, which causes a change in the angular relationships within the structure. Linear strain is measured as the amount of linear deformation (lengthening or shortening) of the sample divided by the sample's original length. It is a nondimensional parameter expressed as a percentage (for example, centimeter per centimeter). Shear strain is measured as the amount of angular change (γ) in a right angle lying in the plane of interest in the sample. It is expressed in radians (one radian equals approximately 57.3 degrees) (International Society of Biomechanics, 1988).

Stress and strain values can be obtained for bone by placing a standardized specimen of bone tissue in a testing jig and loading it to failure (Fig. 1–5). These values can then be plotted on a stress-strain curve

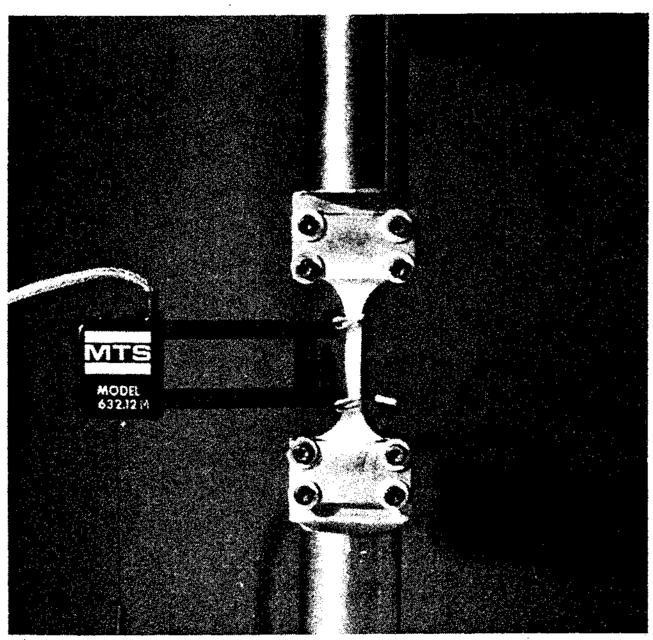


FIG. 1–5
Standardized bone specimen in a testing machine. The strain in the segment of bone between the two gauge arms is measured with a strain gauge. The stress is calculated from the total load measured. (Courtesy of Dennis R. Carter, Ph.D.)

(Fig. 1–6). The regions of this curve are similar to those of the load-deformation curve. Loads in the elastic region do not cause permanent deformation, but once the yield point is exceeded, some deformation is permanent. The strength of the material in terms of energy storage is represented by the area under the entire curve. The stiffness is represented by the slope of the curve in the elastic region. A value for stiffness is obtained by dividing the stress at any point in the elastic (straight line) portion of the curve by the strain at that point. This value is called the modulus of elasticity (Young's modulus). Stiffer materials have higher moduli.

Mechanical properties differ in the two bone types. Cortical bone is stiffer than cancellous bone, withstanding greater stress but less strain before failure. Cancellous bone in vitro does not fracture until the strain exceeds 75%, but cortical bone fractures when the strain exceeds 2%. Because of its porous structure, cancellous bone has a large capacity for energy storage (Carter and Hayes, 1976).

Stress-strain curves for cortical bone, metal, and glass illustrate the differences in mechanical behavior among these materials (Fig. 1–7). The variations in stiffness are reflected in the different slopes of the curves in the elastic region. Metal has the steepest slope and is thus the stiffest material.

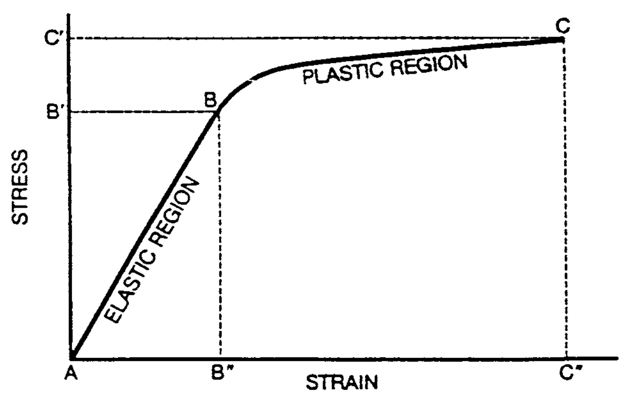


FIG. 1–6
Stress-strain curve for a cortical bone sample tested in tension (pulled). *Yield point (B):* point past which some permanent deformation of the bone sample occurred. *Yield stress (B'):* load per unit area sustained by the bone sample before plastic deformation took place. *Yield strain (B"):* amount of deformation withstood by the sample before plastic deformation occurred. The strain at any point in the elastic region of the curve is proportional to the stress at that point. *Ultimate failure point (C):* the point past which failure of the sample occurred. *Ultimate stress (C'):* load per unit area sustained by the sample before failure. *Ultimate strain (C"):* amount of deformation sustained by the sample before failure.

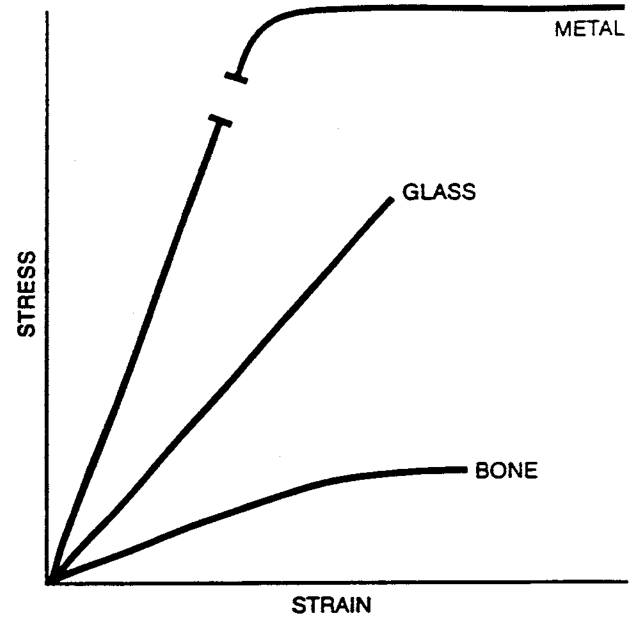


FIG. 1–7
Stress-strain curves for three materials. Metal has the steepest slope in the elastic region and is thus the stiffest material. The elastic portion of the curve for metal is a straight line, indicating linearly elastic behavior. The fact that metal has a long plastic region indicates that this typical ductile material deforms extensively before failure. Glass, a brittle material, exhibits linearly elastic behavior but fails abruptly with little deformation, as indicated by the lack of a plastic region on the stress-strain curve. Cortical bone, which possesses both ductile and brittle qualities, exhibits nonlinear elastic behavior. This behavior is demonstrated by a slight curve in the elastic region, which indicates some yielding during loading within this region. Cortical bone continues to deform before failure but to a lesser extent than does metal.

The elastic portion of the curve for glass and metal is a straight line, indicating linearly elastic behavior; virtually no yielding takes place before the yield point is reached. By comparison, precise testing of cortical bone has shown that the elastic portion of the curve is not straight but is slightly curved, indicating that bone is not linearly elastic in its behavior but yields somewhat during loading in the elastic region (Bonefield and Li, 1967).

After the yield point is reached, glass deforms very little before failing, as indicated by the absence of a plastic region on the stress-strain curve. By contrast, metal exhibits extensive deformation before failing, as indicated by a long plastic region on the curve. Bone also deforms before failing but to a much lesser extent than metal. The difference in the plastic behavior of metal and bone is due to differences in micromechanical events at yield. Yielding in metal (tested in tension, or pulled) is caused by plastic flow and formation of plastic slip lines; slip lines are formed when the molecules of the lattice structure of metal dislocate. Yielding in bone (tested in tension) is caused by debonding of the osteons at the cement lines and microfracture.

Materials are classified as brittle or ductile depending on the extent of deformation before failure. Glass is a typical brittle material, and soft metal is a typical ductile material. The difference in the amount of deformation is reflected in the fracture surfaces of the two materials (Fig. 1–8). When pieced together after fracture, the ductile material will not conform to its original shape whereas the brittle material will. Bone exhibits more brittle or more ductile behavior depending on its age (younger bone being more ductile) and the rate at which it is loaded (bone being more brittle at higher loading speeds).

Because the structure of bone is dissimilar in the transverse and the longitudinal directions, it exhibits different mechanical properties when loaded along different axes, a characteristic known as anisotropy. Figure 1–9 shows the variations in strength and stiffness for cortical bone samples from a human femoral shaft, tested in tension in four directions (Frankel and Burstein, 1970). The values for both parameters are highest for the samples loaded in the longitudinal direction. Although the relationship between loading patterns and the mechanical properties of bone throughout the skeleton is extremely complex, it can generally be said that bone strength and stiffness are greatest in the direction in which loads are most commonly imposed (Frankel and Burstein, 1970).

BIOMECHANICAL BEHAVIOR OF BONE

The mechanical behavior of bone—its behavior under the influence of forces and moments—is affected by its mechanical properties, its geometric characteristics, the loading mode applied, the rate of loading, and the frequency of loading.

BONE BEHAVIOR UNDER VARIOUS LOADING MODES

Forces and moments can be applied to a structure in various directions, producing tension, compression, bending, shear, torsion, and combined loading (Fig. 1–10). Bone in vivo is subjected to all of these loading modes. The following descriptions of these modes apply to structures in equilibrium (at rest or moving at a constant speed); loading produces an internal, deforming effect on the structure.

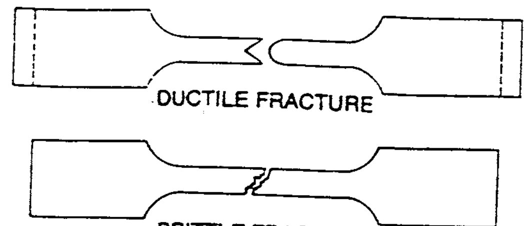


FIG. 1–8
Fracture surfaces of samples of a ductile and a brittle material. The broken lines on the ductile material indicate the original length of the sample, before it deformed. The brittle material deformed very little before fracture.

Tension

During tensile loading, equal and opposite loads are applied outward from the surface of the structure, and tensile stress and strain result inside the structure. Tensile stress can be thought of as many small forces directed away from the surface of the structure. Maximal tensile stress occurs on a plane perpendicular to the applied load (Fig. 1–11). Under tensile loading, the structure lengthens and narrows. At the microscopic level, the failure mechanism for bone tissue loaded in tension is mainly

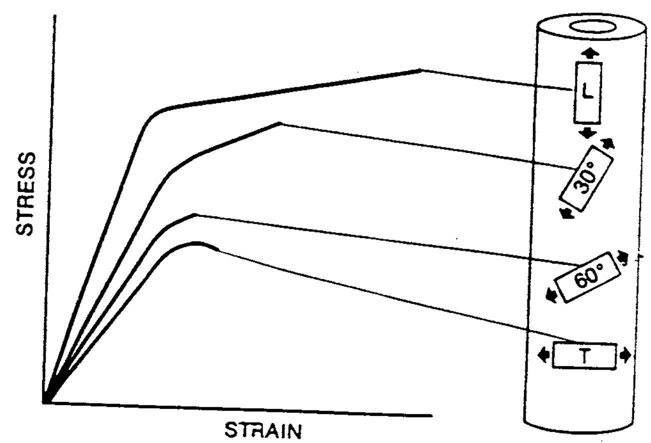


FIG. 1–9
Anisotropic behavior of cortical bone specimens from a human femoral shaft tested in tension (pulled) in four directions: longitudinal (L), tilted 30 degrees with respect to the neutral axis of the bone, tilted 60 degrees, and transverse (T). (Data from Frankel and Burstein, 1970.)

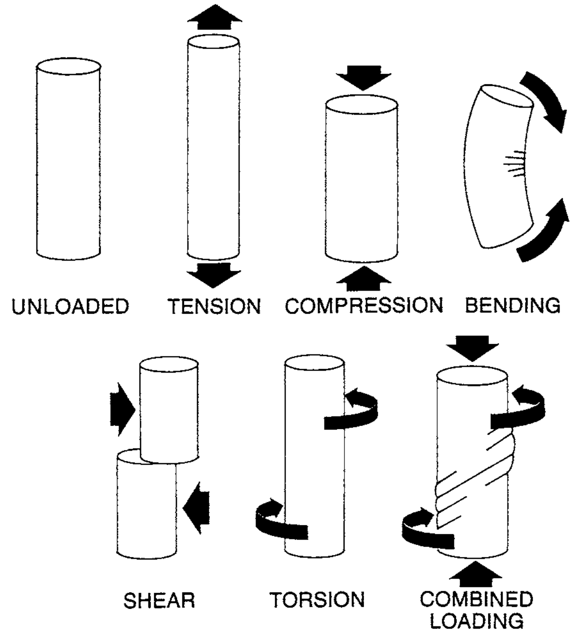


FIG. 1–10
Schematic representation of various loading modes.

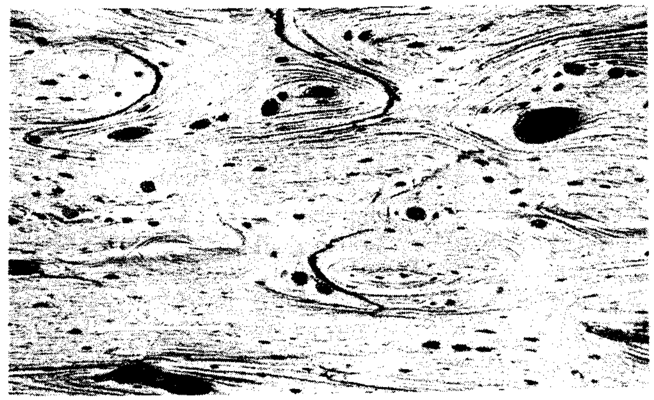


FIG. 1–12
Reflected light photomicrograph of a human cortical bone specimen tested in tension (30×). Arrows indicate debonding at the cement lines and pulling out of the osteons. (Courtesy of Dennis R. Carter, Ph.D.)

debonding at the cement lines and pulling out of the osteons (Fig. 1–12).

Clinically, fractures produced by tensile loading are usually seen in bones with a large proportion of cancellous bone. Examples are fractures of the base of the fifth metatarsal adjacent to the attachment of the peroneus brevis tendon and fractures of the calcaneus adjacent to the attachment of the Achilles tendon. Figure 1–13 shows a tensile fracture through the calcaneus; intense contraction of the triceps surae muscle produced abnormally high tensile loads on the bone.

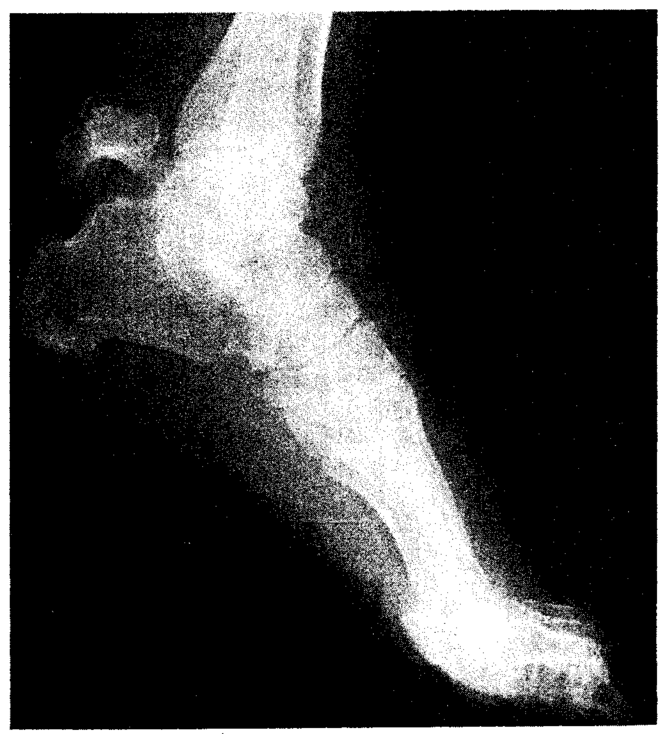


FIG. 1–13
Tensile fracture through the calcaneus produced by strong contraction of the triceps surae muscle during a tennis match. (Courtesy of Robert A. Winquist, M.D.)

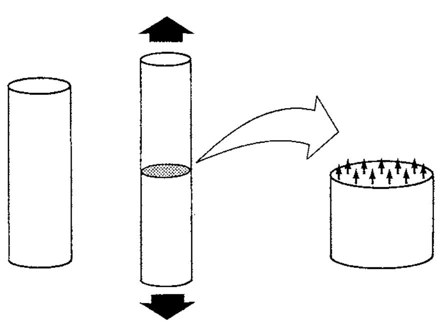


FIG. 1–11
Tensile loading.

Compression

During compressive loading, equal and opposite loads are applied toward the surface of the structure and compressive stress and strain result inside the structure. Compressive stress can be thought of as many small forces directed into the

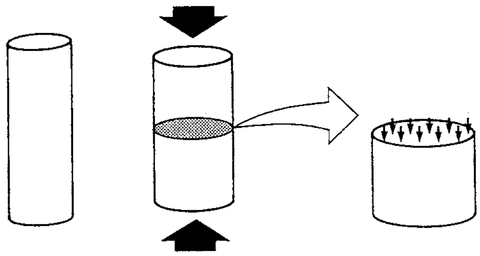


FIG. 1–14
Compressive loading.

surface of the structure. Maximal compressive stress occurs on a plane perpendicular to the applied load (Fig. 1–14). Under compressive loading the structure shortens and widens. At the microscopic level, the failure mechanism for bone tissue loaded in compression is mainly oblique cracking of the osteons (Fig. 1–15).

Clinically, compression fractures are commonly found in the vertebrae, which are subjected to high compressive loads. These fractures are most often seen in the elderly, whose bones weaken as a function of aging. Figure 1–16 shows the shortening and widening that took place in a human vertebra subjected to a high compressive load. In a joint, compressive loading to failure can be produced by abnormally strong contraction of the surrounding muscles. An example of this effect is presented in Figure 1–17; bilateral subcapital fractures of the femoral neck were sustained by a patient undergoing electroconvulsive therapy; strong contractions of the muscles around the hip joint compressed the femoral head against the acetabulum.

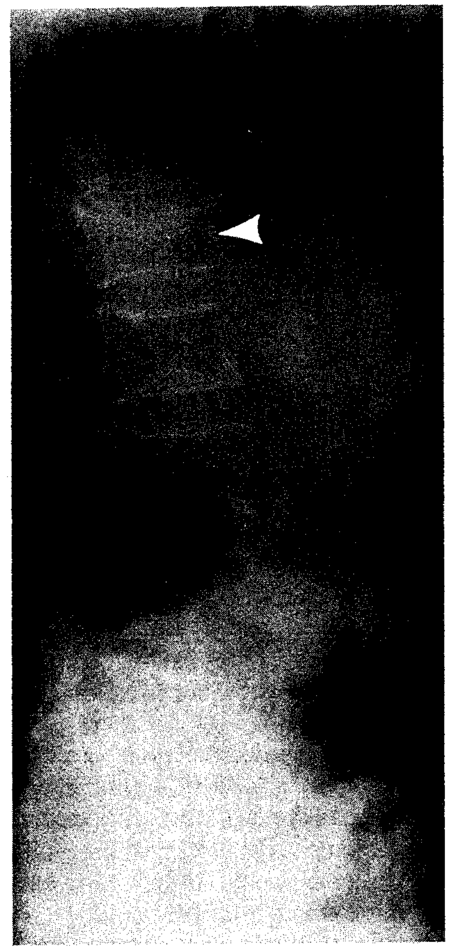


FIG. 1–16
Compression fracture of a human first lumbar vertebra. The vertebra has shortened and widened.

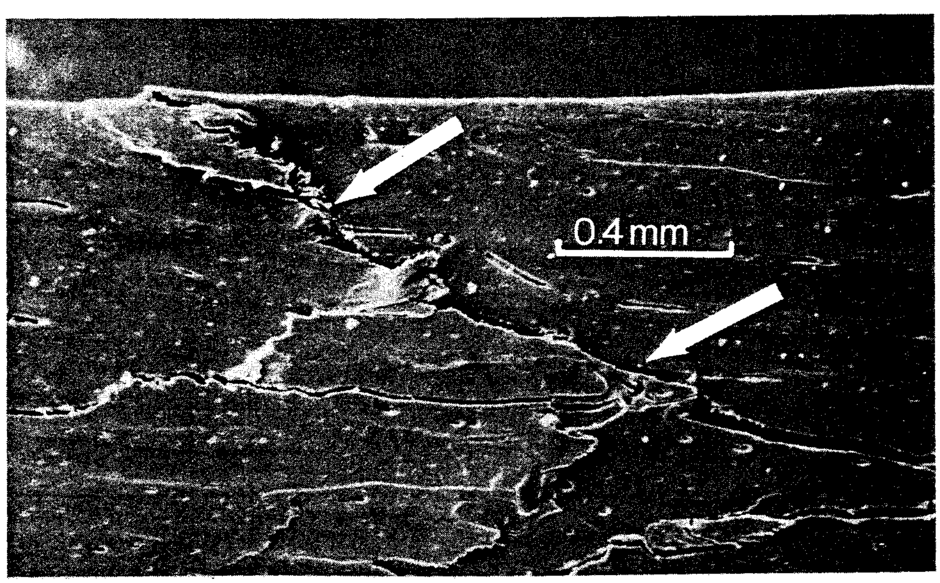


FIG. 1–15
Scanning electron photomicrograph of a human cortical bone specimen tested in compression (30×). Arrows indicate oblique cracking of the osteons. (Courtesy of Dennis R. Carter, Ph.D.)

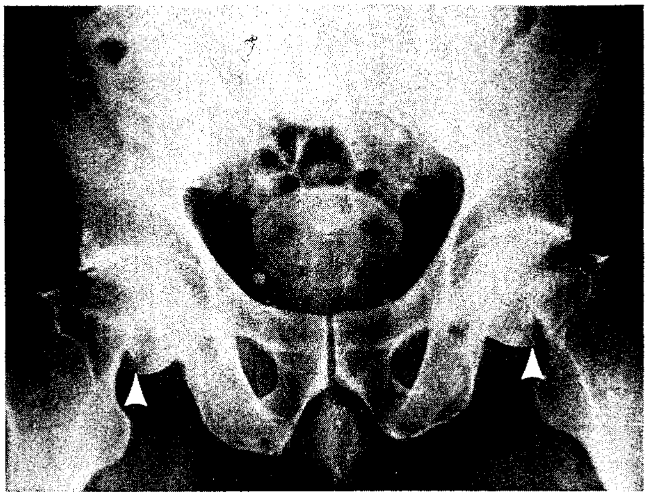


FIG. 1–17
Bilateral subcapital compression fractures of the femoral necks in a patient who underwent electroconvulsive therapy.

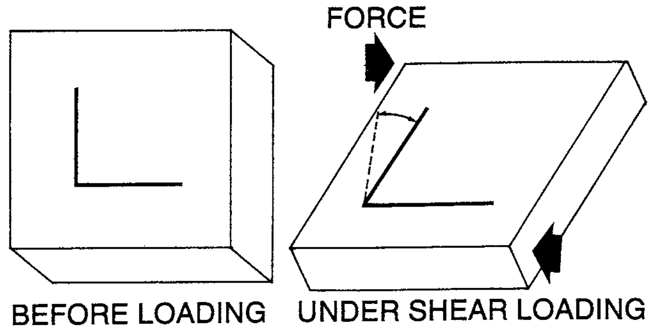


FIG. 1–19
When a structure is loaded in shear, lines originally at right angles on a plane surface within the structure change their orientation, and the angle becomes obtuse or acute. This angular deformation indicates shear strain. (Adapted from Frankel and Burstein, 1970.)

Shear

During shear loading, a load is applied parallel to the surface of the structure, and shear stress and strain result inside the structure. Shear stress can be thought of as many small forces acting on the surface of the structure on a plane parallel to the applied load (Fig. 1–18). A structure subjected to a shear load deforms internally in an angular manner; right angles on a plane surface within the structure become obtuse or acute (Fig. 1–19). Whenever a structure is subjected to tensile or compressive loading, shear stress is produced. Figure 1–20 illustrates angular deformation in structures subjected to these loading modes.

Clinically, shear fractures are most often seen in cancellous bone. Examples are fractures of the femoral condyles and the tibial plateau. A shear fracture of the tibial plateau is shown in Figure 1–21.

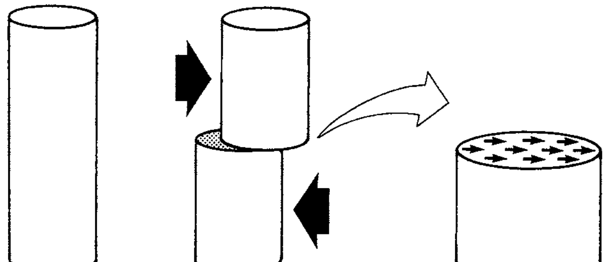


FIG. 1–18
Shear loading.

Human adult cortical bone exhibits different values for ultimate stress under compressive, tensile, and shear loading (Fig. 1–22). Cortical bone can withstand greater stress in compression than in tension and greater stress in tension than in shear (Reilly and Burstein, 1975). The value for the stiffness of a material under shear loading is known as the shear modulus rather than the modulus of elasticity.

Bending

In bending, loads are applied to a structure in a manner that causes it to bend about an axis. When a bone is loaded in bending, it is subjected to a combination of tension and compression. Tensile

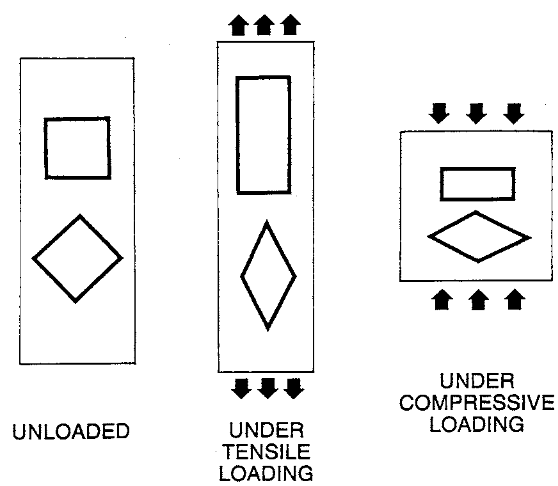


FIG. 1–20
The presence of shear strain in a structure loaded in tension and in compression is indicated by angular deformation. (Adapted from Frankel and Burstein, 1970.)

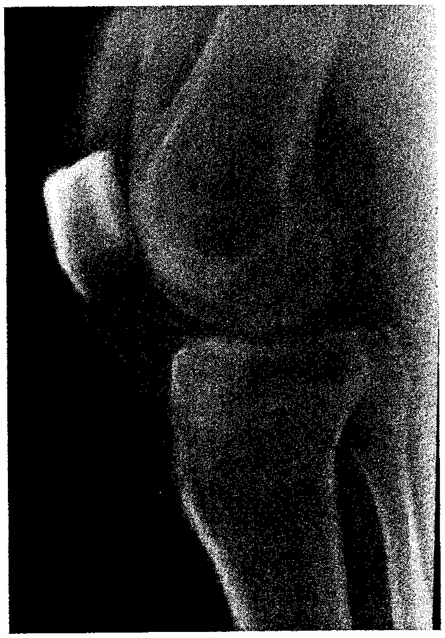


FIG. 1–21
Shear and compression fracture of the lateral tibial plateau. (Courtesy of Steven Lubin, M.D.)

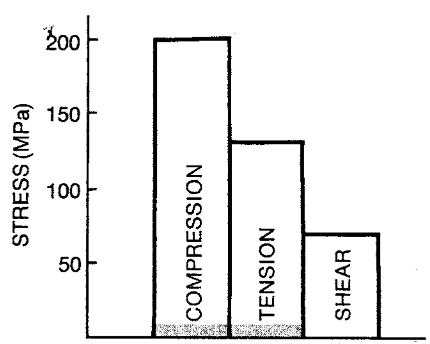


FIG. 1–22
Ultimate stress for human adult cortical bone specimens tested in compression, tension, and shear (average of data from Reilly and Burstein, 1975). Shaded area indicates ultimate stress for human adult cancellous bone with an apparent density of 35% tested in tension and compression (Carter, 1979).

stresses and strains act on one side of the neutral axis, and compressive stresses and strains act on the other side (Fig. 1–23); there are no stresses and strains along the neutral axis. The magnitude of the stresses is proportional to their distance from the neutral axis of the bone. The farther the stresses are from the neutral axis, the higher their magnitude. Because bone is asymmetrical, the tensile and compressive stresses may not be equal.

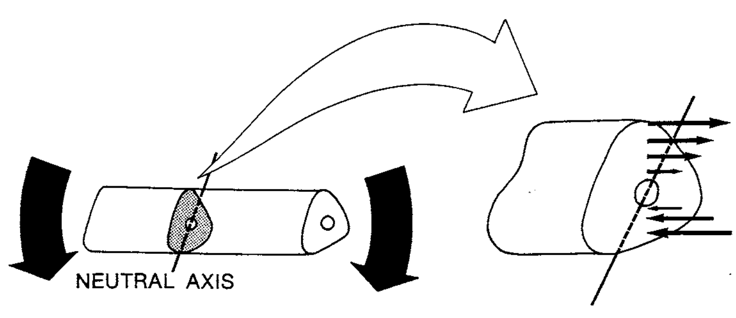
Bending may be produced by three forces (three-point bending) or four forces (four-point bending) (Fig. 1–24). Fractures produced by both types of bending are commonly observed clinically, particularly in long bones.

Three-point bending takes place when three forces acting on a structure produce two equal moments, each being the product of one of the two peripheral forces and its perpendicular distance from the axis of rotation (the point at which the middle force is applied) (see Fig. 1–24A). If loading continues to the yield point, the structure, if homogeneous and symmetrical, will break at the point of application of the middle force.

A typical three-point bending fracture is the "boot top" fracture sustained by skiers. In the "boot top" fracture shown in Figure 1–25, one bending moment acted on the proximal tibia as the skier fell forward over the top of the ski boot. An equal moment, produced by the fixed foot and ski, acted on the distal tibia. As the proximal tibia was bent forward, tensile stresses and strains acted on the posterior side of the bone and compressive stresses and strains acted on the anterior side. The tibia and fibula fractured at the top of the boot. Since adult bone is weaker in tension than in compression, failure begins on the side subjected to tension. Immature bone may fail first in compression, and a buckle fracture may result on the compressive side.

Four-point bending takes place when two force couples acting on a structure produce two equal moments. A force couple is formed when two parallel forces of equal magnitude but opposite direction are applied to a structure (see Fig. 1–24B). Because the

FIG. 1–23
Cross section of a bone subjected to bending, showing distribution of stresses around the neutral axis. Tensile stresses act on the superior side, and compressive stresses act on the inferior side. The stresses are highest at the periphery of the bone and lowest near the neutral axis. The tensile and compressive stresses are unequal because the bone is asymmetrical.



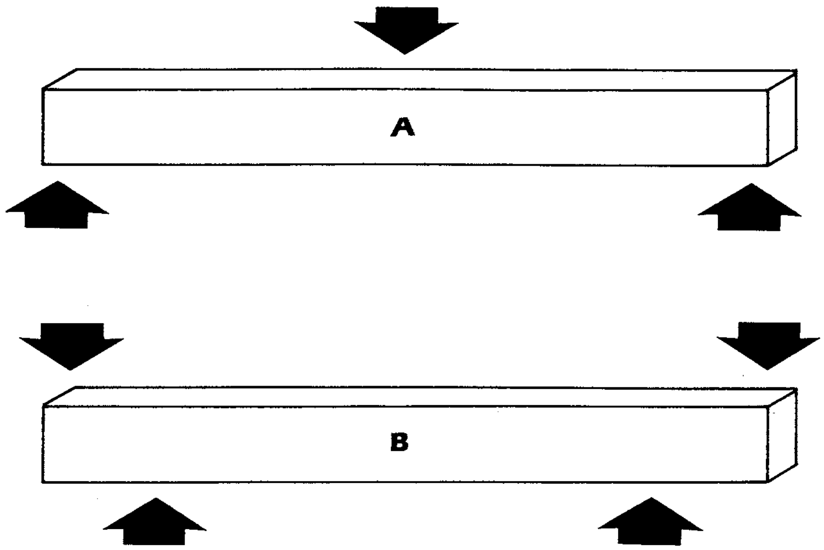


FIG. 1–24
Two types of bending. **A.** Three-point bending. **B.** Four-point bending.

magnitude of the bending moment is the same throughout the area between the two force couples, the structure breaks at its weakest point. An example of a four-point bending fracture is shown in Figure 1–26. A stiff knee joint was manipulated incorrectly during rehabilitation of a patient with a femoral fracture. During the manipulation, the posterior knee joint capsule and tibia formed one force couple and the femoral head and hip joint capsule formed the other. As a bending moment was applied to the femur, the bone failed at its weakest point, the original fracture site.

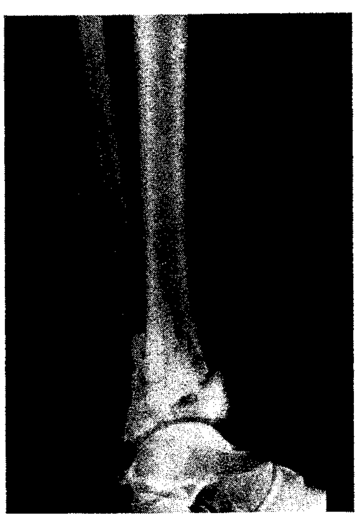


FIG. 1–25
Lateral roentgenogram of a "boot top" fracture produced by three-point bending. (Courtesy of Robert A. Winquist, M.D.)

Torsion

In torsion, a load is applied to a structure in a manner that causes it to twist about an axis, and a torque (or moment) is produced within the structure. When a structure is loaded in torsion, shear stresses are distributed over the entire structure. As in bending, the magnitude of these stresses is proportional to their distance from the neutral axis (Fig. 1–27). The farther the stresses are from the neutral axis, the higher their magnitude.

Under torsional loading, maximal shear stresses act on planes parallel and perpendicular to the neutral axis of the structure. In addition, maximal tensile and compressive stresses act on a plane diagonal to the neutral axis of the structure. Figure 1–28 illustrates these planes in a small segment of bone loaded in torsion.

The fracture pattern for bone loaded in torsion suggests that the bone fails first in shear, with the formation of an initial crack parallel to the neutral axis of the bone. A second crack usually forms along the plane of maximal tensile stress. Such a pattern can be seen in the experimentally produced torsional fracture of a canine femur shown in Figure 1–29.

Combined Loading

Although each loading mode has been considered separately, living bone is seldom loaded in one mode only. Loading of bone in vivo is complex for two principal reasons: bones are constantly subjected to multiple indeterminate loads, and their geometric structure is irregular. Measurement in vivo of the

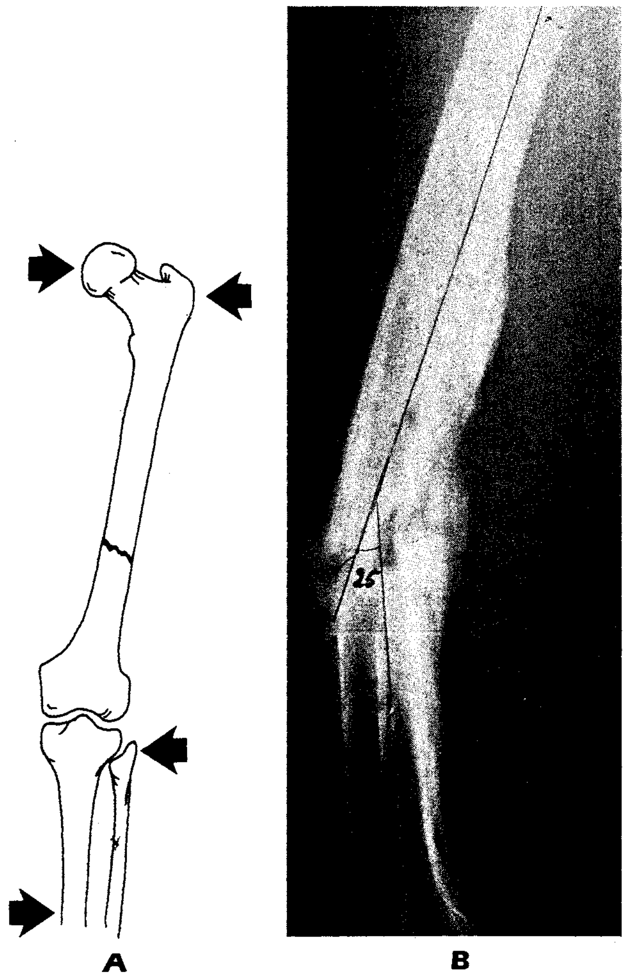


FIG. 1–26
A. During manipulation of a stiff knee during fracture rehabilitation, four-point bending caused the femur to fracture at its weakest point, the original fracture site. **B.** Lateral roentgenogram of the fractured femur. (Courtesy of Kaj Lundborg, M.D.)

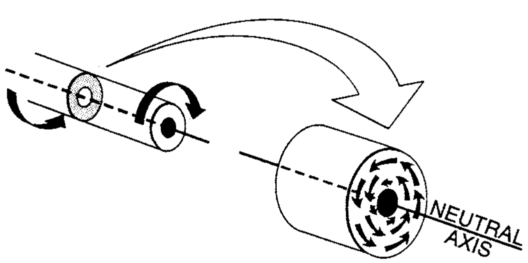


FIG. 1–27
Cross section of a cylinder loaded in torsion, showing the distribution of shear stresses around the neutral axis. The magnitude of the stresses is highest at the periphery of the cylinder and lowest near the neutral axis.

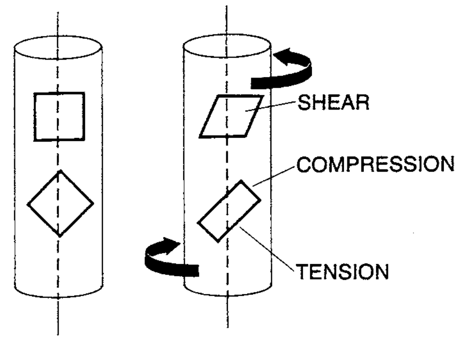


FIG. 1–28
Schematic representation of a small segment of bone loaded in torsion. Maximal shear stresses act on planes parallel and perpendicular to the neutral axis. Maximal tensile and compressive stresses act on planes diagonal to this axis.

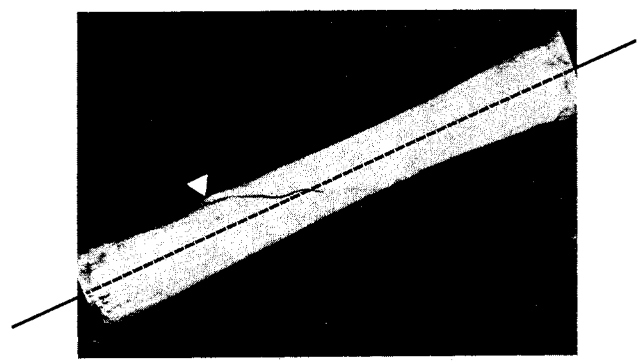


FIG. 1–29
Experimentally produced torsional fracture of a canine femur. The short crack (arrow) parallel to the neutral axis represents shear failure; the fracture line at a 30-degree angle to the neutral axis represents the plane of maximal tensile stress.

strains on the anteromedial surface of a human adult tibia during walking and jogging demonstrated the complexity of the loading patterns during these common physiologic activities (Lanyon et al., 1975). Stress values calculated from these strain measurements by Carter (1978) showed that during normal walking the stresses were compressive during heel strike, tensile during the stance phase, and again compressive during push-off (Fig. 1–30A). Values for shear stress were relatively high in the later portion of the gait cycle, denoting significant torsional loading. This torsional loading was associated with external rotation of the tibia during stance and push-off.

During jogging the stress pattern was quite different (Fig. 1–30B). The compressive stress predominating at toe strike was followed by high tensile stress during push-off. The shear stress was low through-

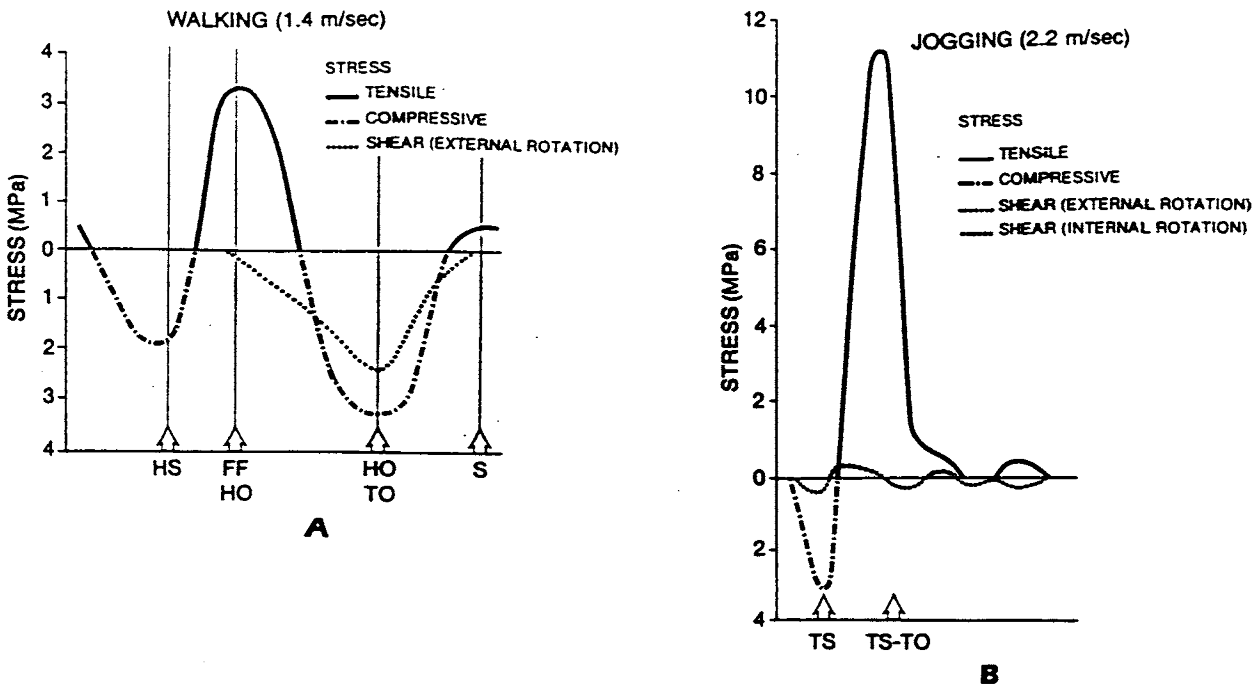


FIG. 1–30
A. Calculated stresses on the anteromedial cortex of a human adult tibia during walking. HS—heel strike; FF—foot flat; HO—heel-off; TO—toe-off; S—swing. (After Lanyon et al., 1975; courtesy of Dennis R. Carter, Ph.D.) **B.** Calculated stresses on the anteromedial cortex of an adult human tibia during jogging. TS—toe strike; TO—toe-off. (After Lanyon et al., 1975; courtesy of Dennis R. Carter, Ph.D.)

out the stride, denoting minimal torsional loading produced by slight external and internal rotation of the tibia in an alternating pattern. The increase in speed from slow walking to jogging increased both the stress and the strain on the tibia (Lanyon et al., 1975). This increase in strain with greater speed was confirmed in studies of locomotion in sheep, which demonstrated a fivefold increase in strain values from slow walking to fast trotting (Lanyon and Bourn, 1979).

Clinical examination of fracture patterns indicates that few fractures are produced by one loading mode or even by two modes. Indeed, most fractures are produced by a combination of several loading modes.

INFLUENCE OF MUSCLE ACTIVITY ON STRESS DISTRIBUTION IN BONE

When bone is loaded in vivo, contraction of the muscles attached to the bone alters the stress distribution in the bone. This muscle contraction decreases or eliminates tensile stress on the bone by producing compressive stress that neutralizes it either partially or totally.

The effect of muscle contraction can be illustrated in a tibia subjected to three-point bending. Figure 1–31A represents the leg of a skier who is falling forward, subjecting the tibia to a bending moment. High tensile stress is produced on the posterior aspect of the tibia, and high compressive stress acts on the anterior aspect. Contraction of the triceps surae muscle produces great compressive stress on the posterior aspect (Fig. 1–31B), neutralizing the great tensile stress and thereby protecting the tibia from failure in tension. This muscle contraction may result in higher compressive stress on the anterior surface of the tibia. Adult bone can usually withstand this stress, but immature bone, which is weaker, may fail in compression.

Muscle contraction produces a similar effect in the hip joint (Fig. 1–32). During locomotion, bending moments are applied to the femoral neck and tensile stress is produced on the superior cortex. Contraction

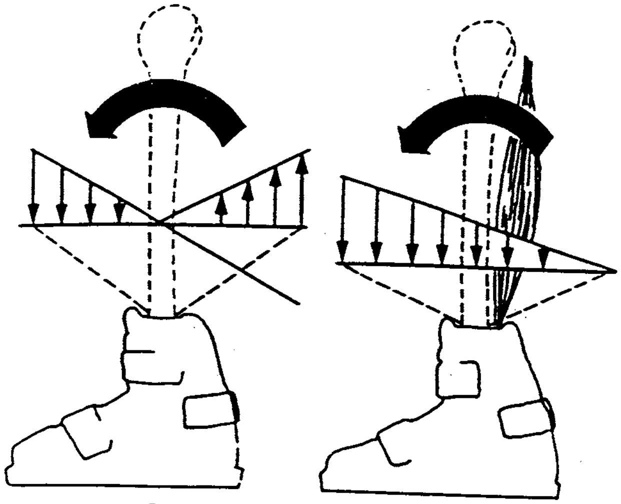


FIG. 1-31
A. Distribution of compressive and tensile stresses in a tibia subjected to three-point bending. **B.** Contraction of the triceps surae muscle produces high compressive stress on the posterior aspect, neutralizing the high tensile stress.

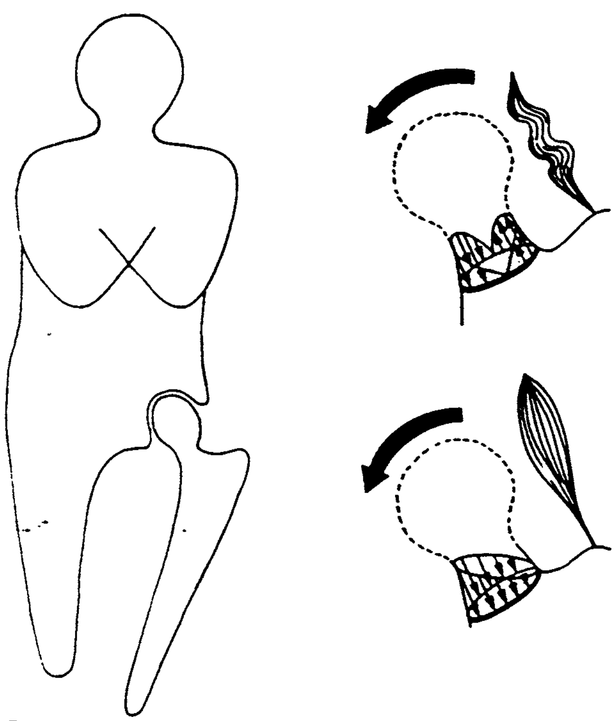


FIG. 1-32
Stress distribution in a femoral neck subjected to bending. When the gluteus medius muscle is relaxed (top), tensile stress acts on the superior cortex and compressive stress acts on the inferior cortex. Contraction of this muscle (bottom) neutralizes the tensile stress.

of the gluteus medius muscle produces compressive stress that neutralizes this tensile stress, with the net result that neither compressive nor tensile stress acts on the superior cortex. Thus, the muscle contraction allows the femoral neck to sustain higher loads than would otherwise be possible.

RATE DEPENDENCY IN BONE

Because bone is a viscoelastic material, its biomechanical behavior varies with the rate at which the bone is loaded (i.e., the rate at which the load is applied and removed). Bone is stiffer and sustains a higher load to failure when loads are applied at higher rates. Bone also stores more energy before failure at higher loading rates, provided that these rates are within the physiologic range.

The load-deformation curves in Figure 1-33 show the difference in the mechanical properties of paired canine tibiae tested in vitro at a high and a very low loading rate, 0.01 second and 200 seconds, respectively (Sammarco et al., 1971). The amount of energy stored before failure approximately doubled at the higher loading rate. The load to failure almost doubled, but the deformation to failure did not change significantly. The bone was about 50% stiffer at the higher speed.

The loading rate is clinically significant because it influences both the fracture pattern and the amount of soft tissue damage at fracture. When a bone fractures, the stored energy is released. At a low loading rate, the energy can dissipate through the formation of a single crack; the bone and soft tissues

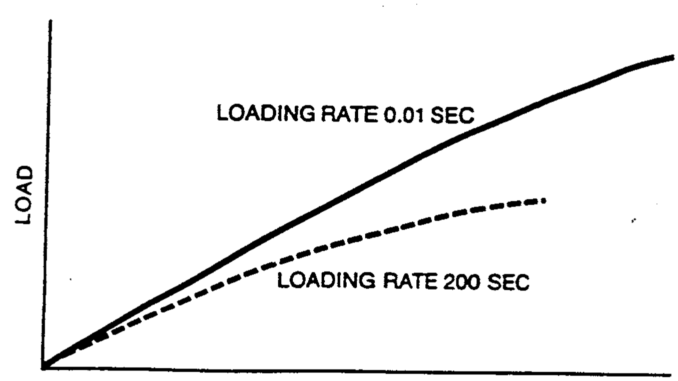


FIG. 1-33
Rate dependency of bone is demonstrated in paired canine tibiae tested at a high and a low loading rate. The load to failure and the energy stored to failure almost doubled at the high rate. (Adapted from Sammarco et al., 1971.)

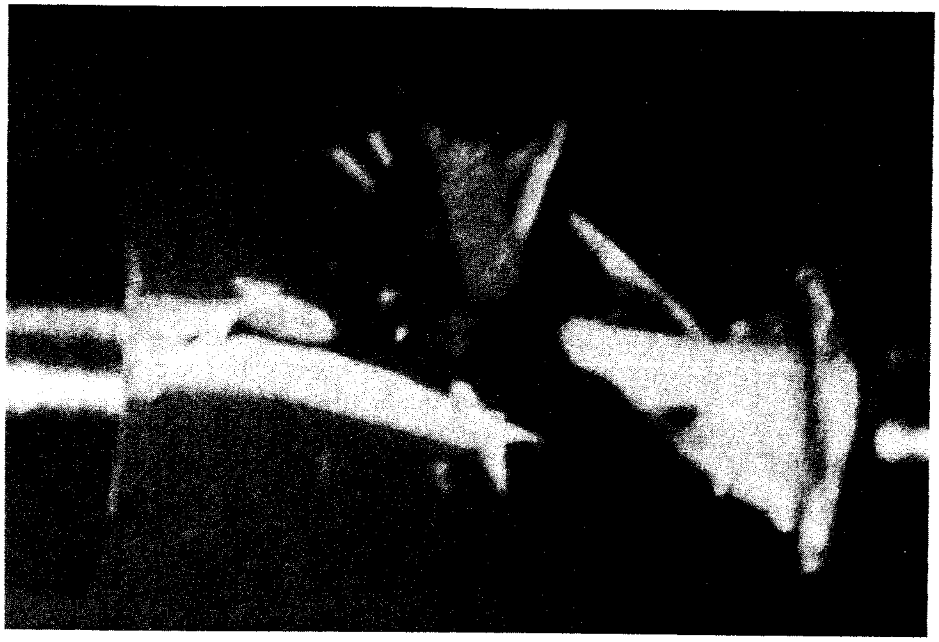


FIG. 1–34
Human tibia experimentally tested to failure in torsion at a high loading rate. Displacement of the numerous fragments was pronounced.

remain relatively intact, and there is little or no displacement of the bone fragments. At a high loading rate, however, the greater energy stored cannot dissipate rapidly enough through a single crack, and comminution of bone and extensive soft tissue damage result. Figure 1–34 shows a human tibia tested in vitro in torsion at a high loading rate; numerous bone fragments were produced, and displacement of the fragments was pronounced.

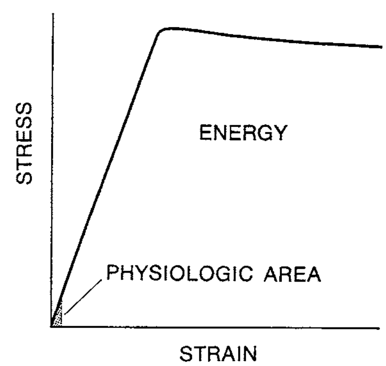


FIG. 1–35
Tensile strain values from a human adult tibia during jogging (Lanyon et al., 1975) have been plotted on a stress-strain curve for bone samples tested to failure in tension. A small proportion of the total energy storage capacity of the bone is utilized during this normal physiologic activity.

Clinically, bone fractures fall into three general categories based on the amount of energy released at fracture: low-energy, high-energy, and very high–energy. A low-energy fracture is exemplified by the simple torsional ski fracture; a high-energy fracture is often sustained during automobile accidents; and a very high–energy fracture is produced by very high–muzzle velocity gunshot.

Only a small proportion of the total energy storage capacity of bone is utilized during normal activity. Figure 1–35 illustrates just how little of this capacity is used during the normal physiologic activity of jogging.

FATIGUE OF BONE UNDER REPETITIVE LOADING

Bone fractures can be produced by a single load that exceeds the ultimate strength of the bone or by repeated applications of a load of lower magnitude. A fracture caused by repeated applications of a lower load is called a fatigue fracture and is typically produced either by few repetitions of a high load or by many repetitions of a relatively normal load.

The interplay of load and repetition for any material can be plotted on a fatigue curve (Fig. 1–36). For some materials (some metals, for example), the fatigue curve is asymptotic, indicating that if the load

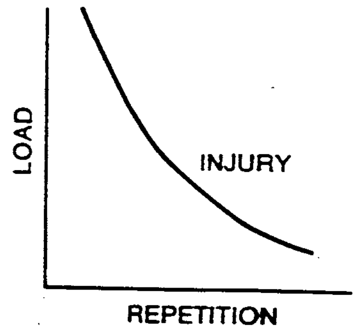


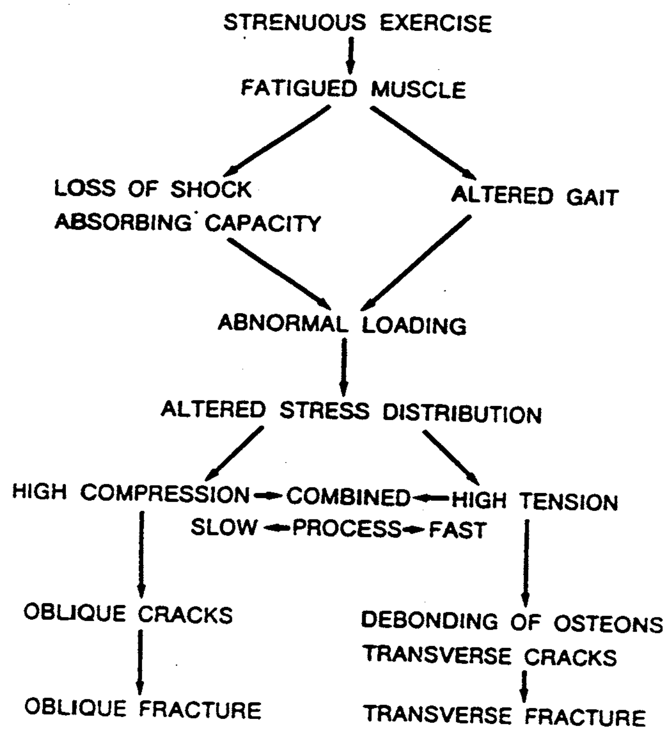
FIG. 1–36
The interplay of load and repetition is represented on a fatigue curve.

is kept below a certain level, theoretically, the material will remain intact, no matter how many repetitions. For bone tested in vitro, the curve is not asymptotic. When bone is subjected to repetitive low loads, it may sustain fatigue microfractures (Carter and Hayes, 1977a). Testing of bone in vitro also reveals that bone fatigues rapidly when load or deformation approaches the yield strength of the bone (Carter and Hayes, 1977a); that is, the number of repetitions needed to produce a fracture diminishes rapidly.

In repetitive loading of living bone, the fatigue process is affected not only by the amount of load and the number of repetitions but also by the number of applications of the load within a given time (frequency of loading). Since living bone is self-repairing, a fatigue fracture results only when the remodeling process is outpaced by the fatigue process, i.e., when loading is so frequent that it precludes the remodeling necessary to prevent failure.

Fatigue fractures are usually sustained during continuous strenuous physical activity, which causes the muscles to become fatigued and reduces their ability to contract. As a result they are less able to store energy and thus to neutralize the stresses imposed on the bone. The resulting alteration of the stress distribution in the bone causes abnormally high loads to be imposed, and a fatigue fracture may result. Bone may fail on the tensile side, the compressive side, or both sides. Failure on the tensile side results in a transverse crack, and the bone proceeds rapidly to complete fracture. Fatigue fractures on the compressive side appear to be produced more slowly; the remodeling is less easily outpaced by the fatigue process, and the bone may not proceed to complete fracture.

This theory of muscle fatigue as a cause of fatigue fracture in the lower extremities is outlined in the following schema:



INFLUENCE OF BONE GEOMETRY ON BIOMECHANICAL BEHAVIOR

The geometry of a bone greatly influences its mechanical behavior. In tension and compression, the load to failure and the stiffness are proportional to the cross-sectional area of the bone. The larger the area is, the stronger and stiffer the bone. In bending, both the cross-sectional area and the distribution of bone tissue around a neutral axis affect the bone's mechanical behavior. The quantity that takes into account these two factors in bending is called the area moment of inertia. A larger area moment of inertia results in a stronger and stiffer bone.

Figure 1–37 shows the influence of the area moment of inertia on the load to failure and the stiffness of three rectangular structures that have the same area but different shapes. In bending, beam III is the stiffest of the three and can withstand the highest load, because the greatest amount of material is distributed at a distance from the neutral axis. For rectangular cross sections, the formula for the area

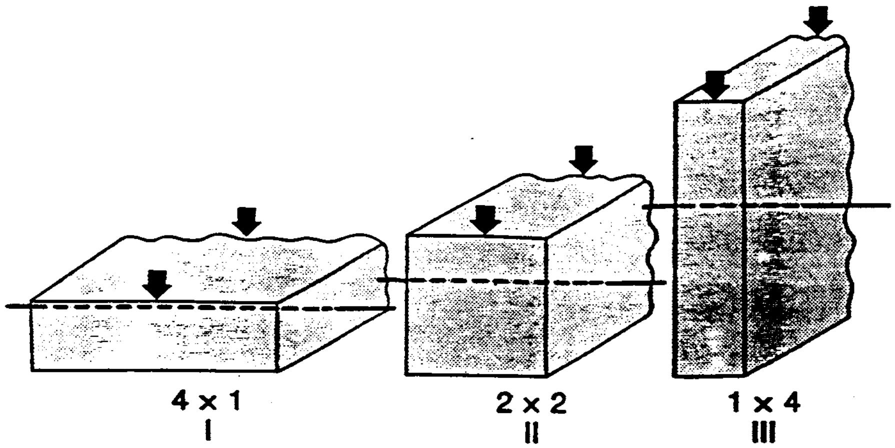


FIG. 1–37
Three beams of equal area but different shapes subjected to bending. For rectangular cross sections, the area moment of inertia is calculated by the formula $\frac{B \cdot H^3}{12}$, where B is the width and H, the height. The area moment of inertia for beam I is 4/12; for beam II, 16/12; and for beam III, 64/12. (Adapted from Frankel and Burstein, 1970.)

moment of inertia is the width (B) multiplied by the cube of the height (H^3) divided by 12:

$$\frac{B \cdot H^3}{12}$$

Because of its large area moment of inertia, beam III can withstand four times more load in bending than beam I.

A third factor, the length of the bone, influences the strength and stiffness in bending. The longer the bone is, the greater the magnitude of the bending moment caused by the application of a force. In a rectangular structure, the magnitude of the stresses produced at the point of application of the bending moment is proportional to the length of the structure. Figure 1–38 depicts the forces acting on two beams with the same width and height but different lengths: beam B is twice as long as beam A. The bending moment for the longer beam is twice that for the shorter beam; consequently, the stress magnitude throughout the beam is twice as high.

Because of their length, the long bones of the skeleton are subjected to high bending moments and, so, high tensile and compressive stresses. Their tubular

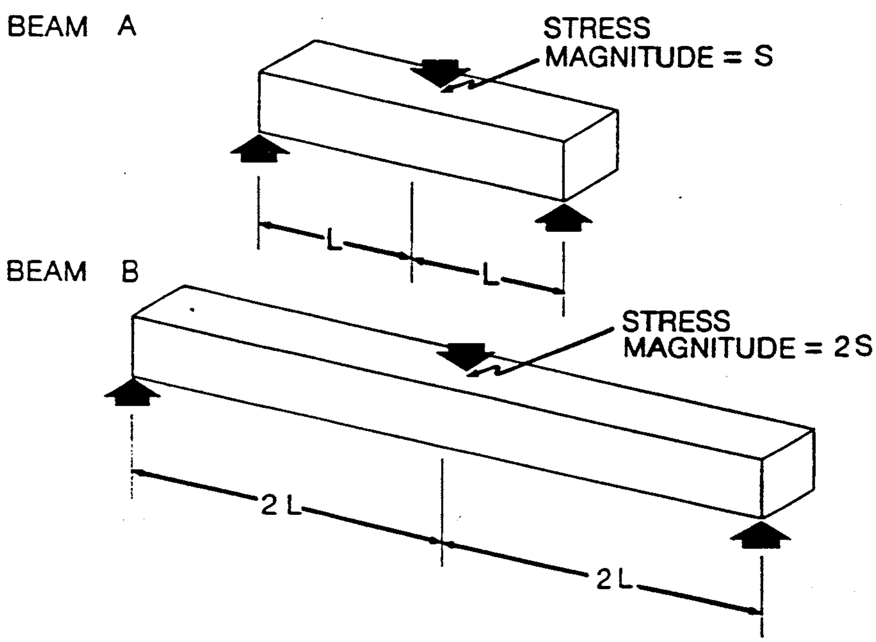


FIG. 1–38
Beam B is twice as long as beam A and sustains twice the bending moment. Hence, the stress magnitude throughout beam B is twice as high. (Adapted from Frankel and Burstein, 1970.)

shape gives them the ability to resist bending moments in all directions. These bones have a large area moment of inertia because much of the bone tissue is distributed at a distance from the neutral axis.

The factors that affect bone strength and stiffness in torsion are the same ones that operate in bending: the cross-sectional area and the distribution of bone tissue around a neutral axis. The quantity that takes into account these two factors in torsional loading is the polar moment of inertia. The larger the polar moment of inertia is, the stronger and stiffer the bone.

Figure 1–39 shows distal and proximal cross sections of a tibia subjected to torsional loading. Although the proximal section has a slightly smaller bony area than does the distal section, it has a much higher polar moment of inertia because much of the bone tissue is distributed at a distance from the neutral axis. The distal section, while it has a larger bony area, is subjected to much higher shear stress because much of the bone tissue is distributed close to the neutral axis. The magnitude of the shear stress in the distal section is approximately double that in the proximal section. Clinically, torsional fractures of the tibia commonly occur distally.

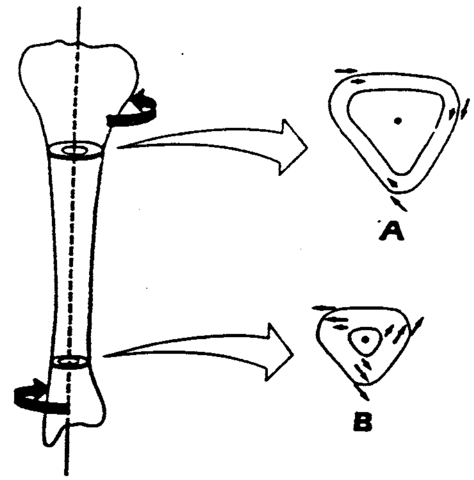


FIG. 1–39
Distribution of shear stress in two cross sections of a tibia subjected to torsional loading. The proximal section (A) has a higher moment of inertia than does the distal section (B), because more bony material is distributed away from the neutral axis. (Adapted from Frankel and Burstein, 1970.)

When bone begins to heal after fracture, blood vessels and connective tissue from the periosteum migrate into the region of the fracture, forming a cuff of dense fibrous tissue, or callus, around the fracture site, which stabilizes that area (Fig. 1–40A). The callus significantly increases the area and polar moments of inertia, thereby increasing the strength and stiffness of the bone in bending and torsion during the healing period. As the fracture heals and the bone gradually regains its normal strength, the callus cuff is progressively resorbed and the bone returns to as near its normal size and shape as possible (Fig. 1–40B).

Certain surgical procedures produce defects that greatly weaken the bone, particularly in torsion. These defects fall into two categories: those whose length is less than the diameter of the bone (stress raisers) and those whose length exceeds the bone diameter (open section defects).

A stress raiser is produced surgically when a small piece of bone is removed or a screw is inserted. Bone strength is reduced because the stresses imposed during loading are prevented from being distributed evenly throughout the bone and instead become concentrated around the defect. This defect is analogous to a rock in a stream, which diverts the water, producing high water turbulence around it (Fig. 1–41). The weakening effect of a stress raiser is particularly marked under torsional loading; the total decrease in bone strength in this loading mode can reach 60%.

Burstein and associates (1972) showed the effect of stress raisers produced by screws and by empty screw holes on the energy storage capacity of rabbit bones tested in torsion at a high loading rate. The immediate effect of drilling a hole and inserting a screw in a rabbit femur was a 74% decrease in energy storage capacity. After 8 weeks, the stress raiser effect produced by the screws and by the holes without screws had disappeared completely because the bone had remodeled: bone had been laid down around the screws to stabilize them, and the empty screw holes had been filled in with bone. In femora from which the screws had been removed immediately before testing, however, the energy storage capacity of the bone decreased by 50%, mainly because the bone tissue around the screw sustained microdamage during screw removal (Fig. 1–42).

An open section defect is a discontinuity in the bone caused by surgical removal of a piece of bone longer than the bone's diameter (for example, by the cutting of a slot during a bone biopsy). Because the outer surface of the bone cross section is no longer continuous, the bone's ability to resist loads is altered, particularly in torsion.

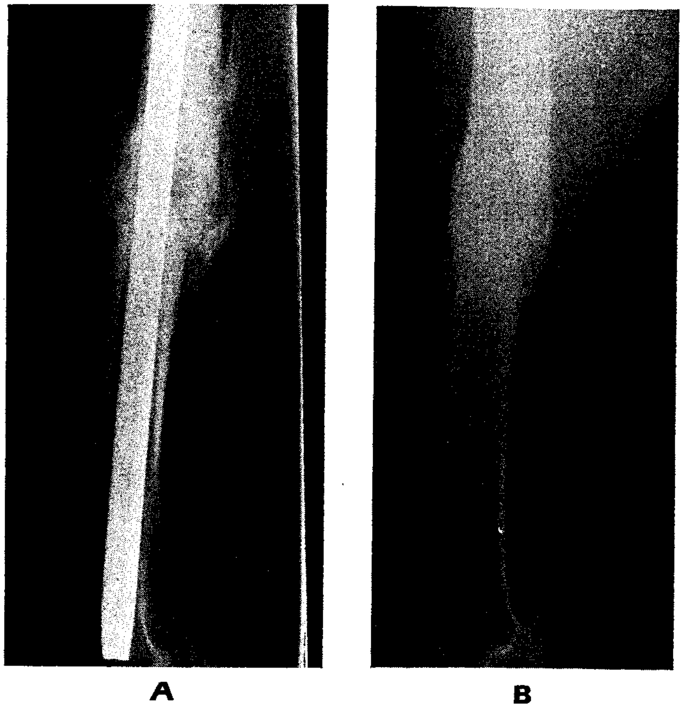


FIG. 1-40
A. Early callus formation in a femoral fracture fixed with an intramedullary nail. **B.** Nine months after injury the fracture has healed and most of the callus cuff has been resorbed. (Courtesy of Robert A. Winquist, M.D.)

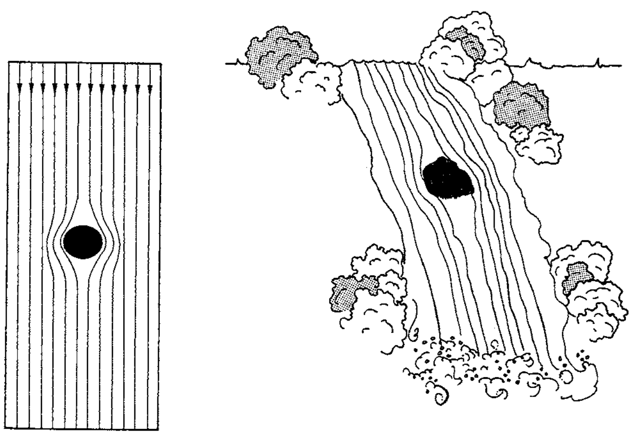


FIG. 1-41
Stress concentration around a defect; such a defect is analogous to a rock in a stream.

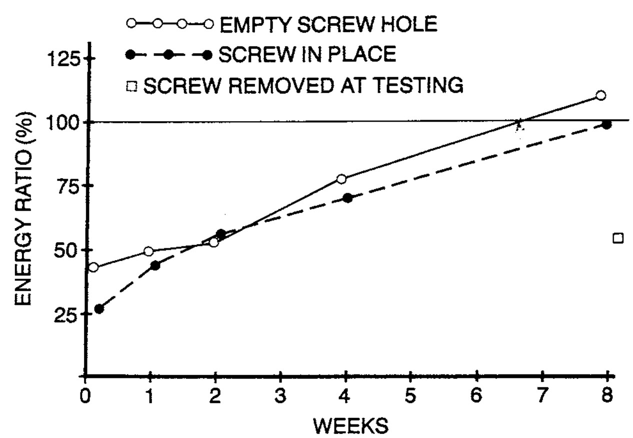


FIG. 1-42
Effect of screws and of empty screw holes on the energy storage capacity of rabbit femora. The energy storage for experimental animals is expressed as a percentage of the total energy storage capacity for control animals. When screws were removed immediately before testing, the energy storage capacity decreased by 50%. (Adapted from Burstein et al., 1972.)

In a normal bone subjected to torsion, the shear stress is distributed throughout the bone and acts to resist the torque. This stress pattern is illustrated in the cross section of a long bone shown in Figure 1-43A. (A cross section with a continuous outer surface is called a closed section.) In a bone with an open section defect, only the shear stress at the periphery of the bone resists the applied torque. As the shear stress encounters the discontinuity, it is forced to change direction (Fig. 1-43B). Through-

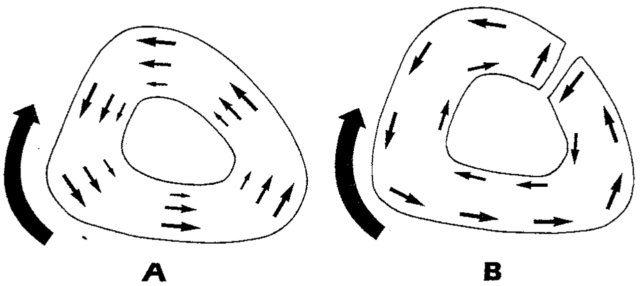


FIG. 1–43
Stress pattern in an open and closed section under torsional loading. **A.** In the closed section, all the shear stress resists the applied torque. **B.** In the open section, only the shear stress at the periphery of the bone resists the applied torque. (Adapted from Frankel and Burstein, 1970.)

out the interior of the bone, the stress runs parallel to to the applied torque, and the amount of bone tissue resisting the load is greatly decreased.

In torsion tests in vitro of human adult tibiae, an open section defect reduced the load to failure and energy storage to failure by as much as 90%. The deformation to failure was diminished by about 70% (Frankel and Burstein, 1970) (Fig. 1–44).

Clinically, surgical removal of a piece of bone can greatly weaken the bone, particularly in torsion. Figure 1–45 is a roentgenogram of a tibia from which a graft was removed for use in an arthrodesis of the hip. A few weeks after operation, the patient tripped while twisting in an attempt to rescue the Christmas ham from toppling onto the floor, and the bone fractured through the defect.

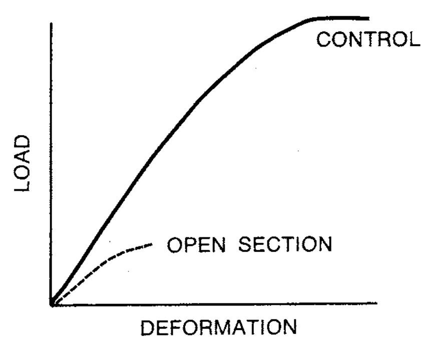


FIG. 1–44
Load-deformation curves for human adult tibiae tested in vitro under torsional loading. The control curve represents a tibia with no defect; the open section curve represents a tibia with an open section defect. (Adapted from Frankel and Burstein, 1970.)

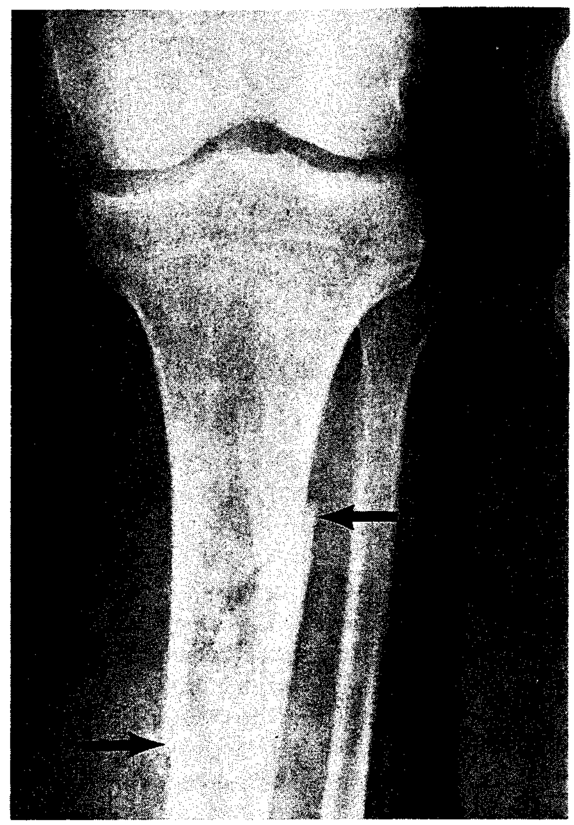


FIG. 1–45
A patient sustained a tibial fracture through a surgically produced open section defect when she tripped a few weeks after operation.

BONE REMODELING

Bone has the ability to remodel, by altering its size, shape, and structure, to meet the mechanical demands placed on it. This phenomenon, in which bone gains or loses cancellous and/or cortical bone in response to the level of stress sustained, is summarized as Wolff's law, which states that bone is laid down where needed and resorbed where not needed (Wolff, 1892).

If, because of partial or total immobilization, bone is not subjected to the usual mechanical stresses, periosteal and subperiosteal bone is resorbed (Jenkins and Cochran, 1969) and strength and stiffness decrease. This decrease in bone strength and stiffness was shown by Kazarian and Von Gierke (1969), who immobilized Rhesus monkeys in full-body casts for 60 days. Subsequent compressive testing in vitro of the vertebrae from the immobilized monkeys and from controls showed up to a threefold decrease in load to

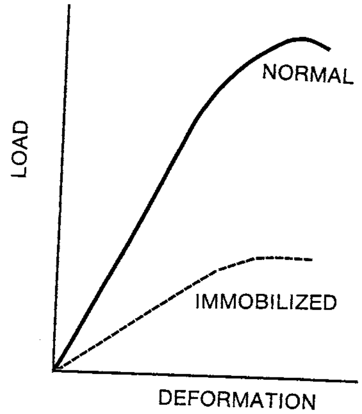


FIG. 1–46
Load-deformation curves for vertebral segments L5 to L7 from normal and immobilized Rhesus monkeys. (Adapted from Kazarian and Von Gierke, 1969.)

failure and energy storage capacity in the vertebrae that had been immobilized; stiffness was also significantly decreased (Fig. 1–46).

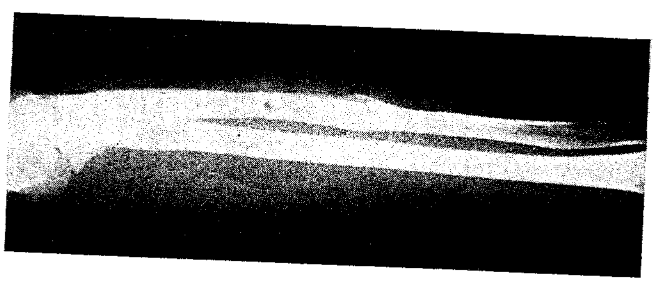
An implant that remains firmly attached to a bone after a fracture has healed may also diminish the strength and stiffness of the bone. In the case of a plate fixed to the bone with screws, the plate and the bone share the load in proportions determined by the geometry and material properties of each structure. A large plate, carrying high loads, unloads the bone to a great extent; the bone then atrophies in response to this diminished load. (The bone may hypertrophy at the bone-screw interface in an attempt to reduce micromotion of the screws.)

Bone resorption under a plate is illustrated in Figure 1–47. A compression plate made of a material approximately 10 times stiffer than the bone was applied to a fractured ulna and remained after the fracture had healed. The bone under the plate carried a lower load than normal; it was partially resorbed, and the diameter of the diaphysis became markedly smaller. A reduction in the size of the bone diameter greatly decreases bone strength, particularly in bending and torsion, as it reduces the area and polar moments of inertia. A 20% decrease in bone diameter may reduce the strength in torsion by 60%. Changes in bone size and shape illustrated in Figure 1–47 suggest that rigid plates should be removed shortly after a fracture has healed and before the bone has markedly diminished in size. Such a decrease in bone size is usually accompanied by secondary osteoporosis, which further weakens the bone (Slätis et al., 1980).

An implant may cause bone hypertrophy at its attachment sites. An example of bone hypertrophy around screws is illustrated in Figure 1–48. A nail plate was applied to a femoral neck fracture, and the bone hypertrophied around the screws in response to the increased load at these sites. Hypertrophy may also result if bone is repeatedly subjected to high mechanical stresses within the normal physiologic range. Hypertrophy of normal adult bone in response to strenuous exercise has been observed (Jones et al., 1977; Dalén and Olsson, 1974; Huddleston et al., 1980), as has an increase in bone density (Nilsson and Westlin, 1971).

A positive correlation exists between bone mass and body weight. A greater body weight has been associated with a larger bone mass (Exner et al.,1979). Conversely, a prolonged condition of weightlessness, such as that experienced during space travel, has

A



B

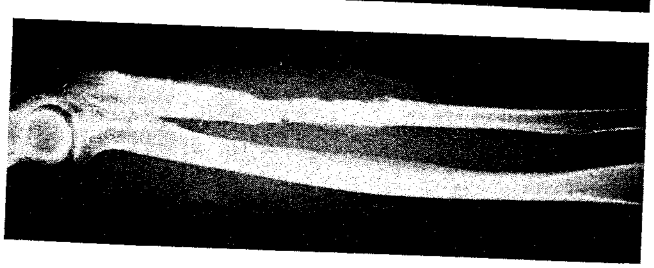


FIG. 1–47
Anteroposterior **(A)** and lateral **(B)** roentgenograms of an ulna after plate removal show a decreased bone diameter due to resorption of the bone under the plate. Cancellization of the cortex and the presence of screw holes also weaken the bone. (Courtesy of Marc Martens, M.D.)

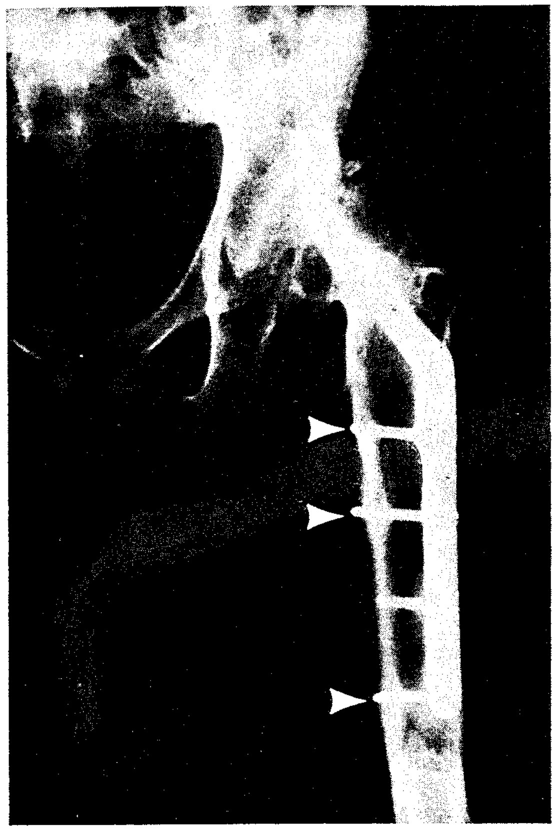


FIG. 1–48
Roentgenogram of a fractured femoral neck to which a nail plate was applied. Loads are transmitted from the plate to the bone via the screws. Bone has been laid down around the screws to bear these loads.

been found to result in a decreased bone mass in weight-bearing bones (Rambaut and Johnston, 1979; Gazenko et al., 1981).

DEGENERATIVE CHANGES IN BONE ASSOCIATED WITH AGING

A progressive loss of bone density has been observed as part of the normal aging process. The longitudinal trabeculae become thinner, and some of the transverse trabeculae are resorbed (Siffert and Levy, 1981) (Fig. 1–49). The result is marked reduction in the amount of cancellous bone and thinning of cortical bone. This decrease in the total amount of bone tissue and the slight decrease in the size of the bone reduce bone strength and stiffness.

Stress-strain curves for specimens from human adult tibiae of two widely differing ages tested in tension are shown in Figure 1–50. The ultimate stress was approximately the same for the young and the old bone. The old bone specimen could withstand only half the strain that the young bone could, indicating greater brittleness and a reduction in energy storage capacity.

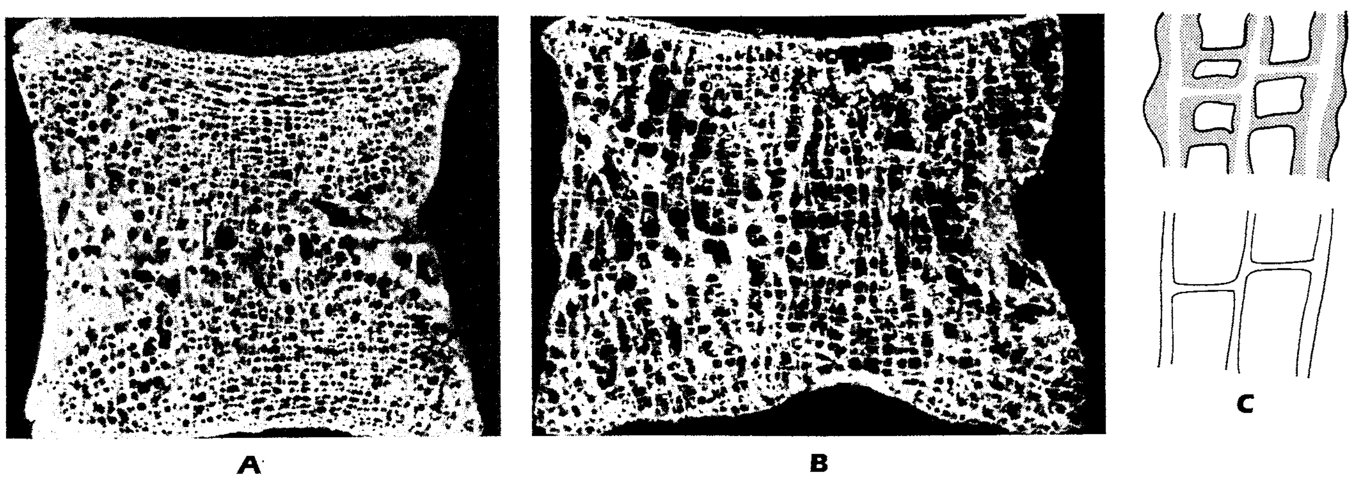


FIG. 1–49
Vertebral cross sections from autopsy specimens of young **(A)** and old **(B)** bone show a marked reduction in cancellous bone in the latter. (Reprinted with permission from Nordin, B.E.C.: Metabolic Bone and Stone Disease. Edinburgh, Churchill Livingstone, 1973.) **C.** Bone reduction with aging is schematically depicted. As normal bone (top) is subjected to absorption (shaded area) during the aging process, the longitudinal trabeculae become thinner and some transverse trabeculae disappear (bottom). (Adapted from Siffert and Levy, 1981.)

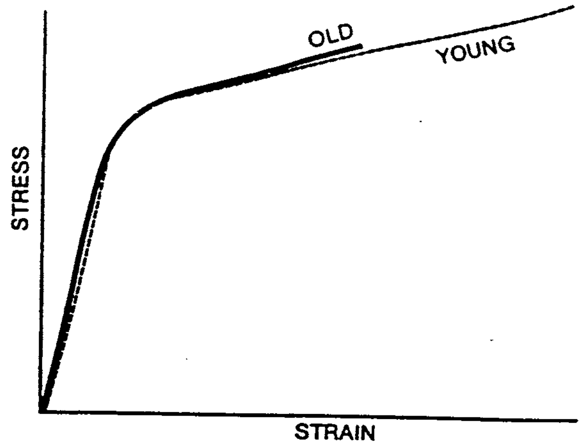


FIG. 1–50
Stress-strain curves for samples of adult human tibiae of two widely differing ages tested in tension. (Adapted from Burstein et al., 1976.)

SUMMARY

1. Bone is a two-phase composite material, inorganic mineral salts being one phase and an organic matrix of collagen and ground substance the other. The inorganic component makes bone hard and rigid, whereas the organic component gives bone its flexibility and resilience.
2. Microscopically, the fundamental structural unit of bone is the osteon, or haversian system, composed of concentric layers of mineralized matrix surrounding a central canal containing blood vessels and nerve fibers.
3. Macroscopically, the skeleton is composed of cortical and cancellous bone. Bone of both types can be considered as one material whose porosity and density vary over a wide range.
4. Bone is an anisotropic material, exhibiting different mechanical properties when loaded in different directions. Mature bone is strongest and stiffest in compression.
5. Bone is subjected to complex loading patterns during common physiologic activities such as walking and jogging. Most bone fractures are produced by a combination of several loading modes.
6. Muscle contraction affects stress patterns in bone by producing compressive stress that partially or totally neutralizes the tensile stress acting on the bone.
7. Bone is stiffer, sustains higher loads before failing, and stores more energy when loaded at higher rates.
8. Living bone fatigues when the frequency of loading precludes the remodeling necessary to prevent failure.
9. The mechanical behavior of a bone is influenced by its geometry (length, cross-sectional area, and distribution of bone tissue around the neutral axis).
10. Bone remodels in response to the mechanical demands placed on it; it is laid down where needed and resorbed where not needed.
11. With aging there is a marked reduction in the amount of cancellous bone and a decrease in the thickness of cortical bone. These changes diminish bone strength and stiffness.

REFERENCES

Bassett, C. A. L.: Electrical effects in bone. Sci. Am., *213*:18, 1965.

Bonefield, W., and Li, C. H.: Anisotropy of nonelastic flow in bone. J. Appl. Physics, *38*:2450, 1967.

Burstein, A. H., Reilly, D. T., and Martens, M.: Aging of bone tissue: Mechanical properties. J. Bone Joint Surg., *58A*: 82, 1976.

Burstein, A. H., et al.: Bone strength. The effect of screw holes. J. Bone Joint Surg., *54A*:1143, 1972.

Carter, D. R.: Anisotropic analysis of strain rosette information from cortical bone. J. Biomech., *11*:199, 1978.

Carter, D. R., and Hayes, W. C.: Bone compressive strength: The influence of density and strain rate. Science, *194*:1174, 1976.

Carter, D. R., and Hayes, W. C.: Compact bone fatigue damage. A microscopic examination. Clin. Orthop., *127*:265, 1977a.

Carter, D. R., and Hayes, W. C.: The compressive behavior of bone as a two-phase porous structure. J. Bone Joint Surg., *59A*:954, 1977b.

Carter, D. R., Schwab, G. H., and Spengler, D. M.: The effect of apparent density on the tensile and compressive properties of cancellous bone. Transactions of the 25th Annual Meeting, Orthopaedic Research Society, *4*:87, 1979.

Dalén, N., and Olsson, K. E.: Bone mineral content and physical activity. Acta Orthop. Scand., *45*:170, 1974.

Dempster, W. T., and Coleman, R. F.: Tensile strength of bone along and across the grain. J. Appl. Physiol., *16*:355, 1960.

Evans, F. G.: Bone and bones. J. Biomech. Eng., *104*:1, 1982.

Evans, F. G., and Bang, S.: Differences and relations between the physical properties and the microscopic structure of human femoral, tibial, and fibular cortical bone. Am. J. Anat., *120*:79, 1967.

Exner, G. U., et al.: Bone densitometry using computed tomography. Part I: Selective determination of trabecular bone density and other bone mineral parameters. Normal values in children and adults. Br. J. Radiol., *52*:14, 1979.

Frankel, V. H., and Burstein, A. H.: Orthopaedic Biomechanics. Philadelphia, Lea & Febiger, 1970.

Gazenko, O. G., Genin, A. M., and Yegorov, A. D.: Major medical results of the Salyut-6/Soyuz 185-day space flight. NASA NDB 2747. Proceedings of the XXXII Congress of the International Astronautical Federation, Rome, Italy, September 6–12, 1981.

Gray, H.: Anatomy of the Human Body. 13th American edition. Edited by C. D. Clemente. Philadelphia, Lea & Febiger, 1985.

Huddleston, A. L., Rockwell, D., Kulund, D. N., and Harrison, R. B.: Bone mass in lifetime tennis athletes. JAMA, *244*:1107, 1980.

International Society of Biomechanics: Quantities and Units of Measurement in Biomechanics, 1987 (unpublished).

Jenkins, D. P., and Cochran, T. H.: Osteoporosis: The dramatic effect of disuse of an extremity. Clin. Orthop., *64*:128, 1969.

Jones, H., Priest, J., Hayes, W., and Nagel, D.: Humeral hypertrophy in response to exercise. J. Bone Joint Surg., *59A*:204, 1977.

Kazarian, L. L., and Von Gierke, H. E.: Bone loss as a result of immobilization and chelation. Preliminary results in *Macaca mulatta*. Clin. Orthop., *65*:67, 1969.

Lanyon, L. E., and Bourn, S.: The influence of mechanical function on the development and remodeling of the tibia. An experimental study in sheep. J. Bone Joint Surg., *61A*:263, 1979.

Lanyon, L. E., Hampson, W. G. J., Goodship, A. E., and Shah, J. S.: Bone deformation recorded in vivo from strain gauges attached to the human tibial shaft. Acta Orthop. Scand., *46*:256, 1975.

Nilsson, B. E., and Westlin, N. E.: Bone density in athletes. Clin. Orthop., *77*:179, 1971.

Nordin, B. E. C.: Metabolic Bone and Stone Disease. Edinburgh, Churchill Livingstone, 1973.

Rambaut, P. C., and Johnston, R. S.: Prolonged weightlessness and calcium loss in man. Acta Astronautica, *6*:1113, 1979.

Reilly, D., and Burstein, A.: The elastic and ultimate properties of compact bone tissue. J. Biomech., *8*:393, 1975.

Sammarco, J., Burstein, A., Davis, W., and Frankel, V.: The biomechanics of torsional fractures: The effect of loading on ultimate properties. J. Biomech., *4*:113, 1971.

Siffert, R. S., and Levy, R. N.: Trabecular patterns and the internal architecture of bone. Mt. Sinai J. Med., *48*:221, 1981.

Slätis, P., Paavolainen, P., Karaharju, E., and Holmström, T.: Structural and biomechanical changes in bone after rigid plate fixation. Can. J. Surg., *23*:247, 1980.

Tortora, G. J., and Anagnostakos, N. P.: Principles of Anatomy and Physiology. 4th Ed. New York, Harper & Row, 1984.

Wolff, J.: Das Gesetz der Transformation der Knochen. Berlin, Hirschwald, 1892.

SUGGESTED READING

Whole Bones

Albright, J. A.: Bone: Physical properties. *In* The Scientific Basis of Orthopaedics. 2nd Ed. Edited by J. A. Albright and R. A. Brand. New York, Appleton-Century-Crofts, 1987, pp. 213–240.

Albright, J. A., and Skinner, H. C. W.: Bone: Structural organization and remodeling dynamics. *In* The Scientific Basis of Orthopaedics. 2nd Ed. Edited by J. A. Albright and R. A. Brand. New York, Appleton-Century-Crofts, 1987, pp. 161–198.

Asang, E.: Biomechanics of the human leg in alpine skiing. *In* Biomechanics IV. Edited by R. C. Nelson and C. A. Morehouse. Baltimore, University Park Press, 1974, pp. 236–242.

Asang, E.: Applied biomechanics of the human leg. A basis for individual protection from skiing injuries. Orthop. Clin. North Am., *7*:95–103, 1976.

Asang, E.: Experimental biomechanics of the human leg. A basis for interpreting typical skiing injury mechanisms. Orthop. Clin. North Am., *7*:63–73, 1976.

Bassett, C. A. L.: Electrical effects in bone. Sci. Am., *213*:18–25, 1965.

Burstein, A. H., and Frankel, V. H.: The viscoelastic properties of some biological materials. Ann. N.Y. Acad. Sci., *146*:158–165, 1968.

Burstein, A. H., et al.: Bone strength. The effect of screw holes. J. Bone Joint Surg., *54A*:1143–1156, 1972.

Carter, D. R.: Anisotropic analysis of strain rosette information from cortical bone. J. Biomech., *11*:199–202, 1978.

Currey, J.: The Mechanical Adaptations of Bones. Princeton, Princeton University Press, 1984, pp. 3–157.

Dalén, N., and Olsson, K.-E.: Bone mineral content and physical activity. Acta Orthop. Scand., *45*:170–174, 1974.

Dempster, W. T., and Coleman, R. F.: Tensile strength of bone along and across the grain. J. Appl. Physiol., *16*:355–360, 1960.

Evans, F. G.: Bone and bones. J. Biomech. Eng., *104*:1–5, 1982.

Evans, R. D. (ed.): Studies on the Anatomy and Function of Bone and Joints. New York, Springer-Verlag, 1966.

Exner, G. U., et al.: Bone densitometry using computed tomography. Part I: Selective determination of trabecular bone density and other bone mineral parameters. Normal values in children and adults. Br. J. Radiol., *52*:14–23, 1979.

Frankel, V. H.: The Femoral Neck: Function, Fracture Mechanisms, Internal Fixation. Springfield, Charles C Thomas, 1960.

Frankel, V. H., and Burstein, A. H.: Orthopaedic Biomechanics. Philadelphia, Lea & Febiger, 1970.

Frankel, V. H., and Burstein, A. H.: Biomechanics of the locomotor system. *In* Medical Engineering. Edited by C. D. Ray. Chicago, Year Book Medical Publisher, 1974, pp. 505–516.

Frost, H. M.: An Introduction to Biomechanics. Springfield, Charles C Thomas, 1976.

Hakim, N. S., and King, A. I.: Programmed replication of *in situ* (whole body) loading conditions during *in vitro* (substructure) testing of a vertebral column segment. J. Biomech., *9*:629–632, 1976.

Inman, V. T.: Functional aspects of the abductor muscles of the hip. J. Bone Joint Surg., *29A*:607–619, 1947.

Jenkins, D. P., and Cochran, T. H.: Osteoporosis: The dramatic effect of disuse of an extremity. Clin. Orthop., *64*:128–134, 1969.

Jensen, J. S., Hansen, F. W., and Johansen, J.: Tibial shaft fractures. A comparison of conservative treatment and internal fixation with conventional plates or AO compression plates. Acta Orthop. Scand., *48*:204–212, 1977.

Jones, H., Priest, J., Hayes, W., and Nagel, D.: Humeral hypertrophy in response to exercise. J. Bone Joint Surg., *59A*:204–208, 1977.

Kazarian, L., and Graves, G. A.: Compressive strength characteristics of the human vertebral centrum. Spine, 2:1–14, 1977.

Kazarian, L. E., and Von Gierke, H. E.: Bone loss as a result of immobilization and chelation. Preliminary results in *Macaca mulatta*. Clin. Orthop., *65*:67–75, 1969.

Lakes, R., and Saha, S.: Cement line motion in bone. Science, *204*:501–503, 1979.

Lanyon, L. E., Hampson, W. G. J., Goodship, A. E., and Shah, J. S.: Bone deformation recorded in vivo from strain gauges attached to the human tibial shaft. Acta Orthop. Scand., *46*:256–268, 1975.

Murphy, E. F., and Burstein, A. H.: Physical properties of materials including solid mechanics. *In* Atlas of Orthotics. Biomechanical Principles and Application. St. Louis, C. V. Mosby Co., 1975, pp. 3–30.

Netz, P., Eriksson, K., and Strömberg, L.: Ultimate failure of diaphyseal bone. An experimental study on dogs. Acta Orthop. Scand., *51*:583–588, 1980.

Piziali, R. L., and Nagel, D. A.: Modeling of the human leg in ski injuries. Orthop. Clin. North Am., *7*:127–139, 1976.

Robinson, R. A.: Bone tissue: Composition and function. Johns Hopkins Med. J., *145*:10–24, 1979.

Rosse, C., and Clawson, D. K.: Introduction to the Musculoskeletal System. New York, Harper & Row, 1970.

Rybicki, E. F., Simonen, F. A., and Weis, E. B.: On the mathematical analysis of stress in the human femur. J. Biomech., *5*:203–215, 1972.

Sammarco, G. J., Burstein, A. H., Davis, W. L., and Frankel, V. H.: The biomechanics of torsional fractures: The effect of loading on ultimate properties. J. Biomech., *4*:113–117, 1971.

Sandler, R. B., and Herbert, D. L.: Quantitative bone assessments: Applications and expectations. J. Am. Geriatr. Soc., *29*:97–103, 1981.

Skinner, H. C. W.: Bone mineralization. *In* The Scientific Basis of Orthopaedics. 2nd Ed. Edited by J. A. Albright and R. A. Brand. New York, Appleton-Century-Crofts, 1987, pp. 199–212.

Slätis, P., Paavolainen, P., Karaharju, E., and Holmström, T.: Structural and biomechanical changes in bone after rigid plate fixation. Can. J. Surg., *23*:247–250, 1980.

Strömberg, L., and Dalén, N.: Experimental measurement of maximum torque capacity of long bones. Acta Orthop. Scand., *47*:257–263, 1976.

Strömberg, L., and Dalén, N.: The influence of freezing on the maximum torque capacity of long bones. An experimental study on dogs. Acta Orthop. Scand., *47*:254–256, 1976.

Toridis, T. G.: Stress analysis of the femur. J. Biomech., *2*:163–174, 1969.

Cortical Bone

Bonefield, W., and Li, C. H.: Anisotropy of nonelastic flow in bone. J. Appl. Physics, *38*:2450–2455, 1967.

Burstein, A. H., Reilly, D. T., and Martens, M.: Aging of bone tissue: Mechanical properties. J. Bone Joint Surg., *58A*:82–86, 1976.

Burstein, A. H., Currey, J. D., Frankel, V. H., and Reilly, D. T.: The ultimate properties of bone tissue: The effects of yielding. J. Biomech., *5*:35–44, 1972.

Carter, D. R., and Hayes, W. C.: Compact bone fatigue damage. A microscopic examination. Clin. Orthop., *127*:265–274, 1977.

Carter, D. R., and Hayes, W. C.: The compressive behavior of bone as a two-phase porous structure. J. Bone Joint Surg., *59A*:954–962, 1977.

Currey, J. D.: The mechanical properties of bone. Clin. Orthop., *73*:210–231, 1970.

Dalén, N., Hellström, L.-G., and Jacobson, B.: Bone mineral content and mechanical strength of the femoral neck. Acta Orthop. Scand., *47*:503–508, 1976.

Enneking, W.: Principles of Musculoskeletal Pathology. Gainesville, FL, Storter Printing Company, 1970.

Evans, F. G.: Mechanical Properties of Bone. Springfield, Charles C Thomas, 1973.

Evans, F. G., and Bang, S.: Differences and relations between the physical properties and the microscopic structure of human femoral, tibial, and fibular cortical bone. Am. J. Anat., *120*:79–88, 1967.

Fredensborg, N., and Nilsson, B. E.: The bone mineral content and cortical thickness in young women with femoral neck fracture. Clin. Orthop., *124*:161–164, 1977.

Frost, H. M.: The Laws of Bone Structure. Springfield, Charles C Thomas, 1964.

Lakes, R., and Saha, S.: Long-term torsional creep in compact bone. J. Biomech. Eng., *102*:178–180, 1980.

Mueller, K. H., Trias, A., and Ray, R. D.: Bone density and composition. Age-related and pathological changes in water and mineral content. J. Bone Joint Surg., *48A*:140–148, 1966.

Nilsson, B. E., and Westlin, N. E.: Bone density in athletes. Clin. Orthop., *77*:179–182, 1971.

Pope, M. H., and Outwater, J. O.: The fracture characteristics of bone substance. J. Biomech., *5*:457–465, 1972.

Reilly, D. T., and Burstein, A. H.: The mechanical properties of cortical bone. J. Bone Joint Surg., *56A*:1001–1002, 1974.

Reilly, D. T., and Burstein, A. H.: The elastic and ultimate properties of compact bone tissue. J. Biomech., *8*:393–405, 1975.

Reilly, D. T., Burstein, A. H., and Frankel, V. H.: The elastic modulus for bone. J. Biomech., *7*:271–275, 1974.

Sedlin, E. D.: A rheological model for cortical bone. A study of the physical properties of human femoral samples. Acta Orthop. Scand., Suppl. *83*:1–87, 1965.

Viano, D., Helfenstein, U., Anliker, M., and Rüegsegger, P.: Elastic properties of cortical bone in female human femurs. J. Biomech., *9*:703–710, 1976.

Weaver, J. K.: The microscopic hardness of bone. J. Bone Joint Surg., *48A*:273–288, 1966.

Cancellous Bone

Behrens, J. C., Walker, P. S., and Shoji, H.: Variations in strength and structure of cancellous bone at the knee. J. Biomech., *7*:201–207, 1974.

Carter, D. R., and Hayes, W. C.: Bone compressive strength: The influence of density and strain rate. Science, *194*:1174–1176, 1976.

Carter, D. R., and Hayes, W. C.: The compressive behavior of bone as a two-phase porous structure. J. Bone Joint Surg., *59A*:954–962, 1977.

Carter, D. R., Schwab, G. H., and Spengler, D. M.: The effect of apparent density on the tensile and compressive properties of cancellous bone. Transactions of the 25th Annual Meeting, Orthopaedic Research Society, 4:87, 1979.

Chung, S. M. K., Batterman, S. C., and Brighton, C. T.: Shear strength of the human femoral capital epiphyseal plate. J. Bone Joint Surg., *58A*:94–103, 1976.

Enneking, W.: Principles of Musculoskeletal Pathology. Gainesville, FL, Storter Printing Company, 1970.

Evans, F. G.: Mechanical Properties of Bone. Springfield, Charles C Thomas, 1973.

Frankel, V. H., and Burstein, A. H.: Load capacity of tubular bone. *In* Biomechanics and Related Bio-engineering Topics. Edited by R. M. Kenedi. New York, Pergamon Press, 1965, pp. 381–396.

Frost, H. M.: The Laws of Bone Structure. Springfield, Charles C Thomas, 1964.

Galante, J., Rostoker, W., and Ray, R. D.: Physical properties of trabecular bone. Calcif. Tissue Res., *5*:236–246, 1970.

Hayes, W. C., and Carter, D. R.: Postyield behavior of subchondral trabecular bone. J. Biomed. Mater. Res., *7*:537–544, 1976.

Hayes, W. C., Boyle, D. J., and Velez, A.: Functional adaptation in the trabecular architecture of the human patella. Transactions of the 23rd Annual Meeting, Orthopaedic Research Society, 2:114, 1977.

Lindahl, O.: Mechanical properties of dried defatted spongy bone. Acta Orthop. Scand., *47*:11–19, 1976.

Pope, M. H., and Outwater, J. O.: The fracture characteristics of bone substance. J. Biomech., *5*:457–465, 1972.

Pope, M. H., and Outwater, J. O.: Mechanical properties of bone as a function of position and orientation. J. Biomech., *7*:61–66, 1974.

Siffert, R. S., and Levy, R. N.: Trabecular patterns and the internal architecture of bone. Mt. Sinai J. Med., *48*:221–229, 1981.

Weaver, J. K.: The microscopic hardness of bone. J. Bone Joint Surg., *48A*:273–288, 1966.

Weaver, J. K., and Chalmers, J.: Cancellous bone: Its strength and changes with aging and an evaluation of some methods for measuring its mineral content. I. Age changes in cancellous bone. J. Bone Joint Surg., *48A*:289–299, 1966.

Fatigue Fracture

Baker, J., Frankel, V. H., and Burstein, A. H.: Fatigue fractures: Biomechanical considerations. J. Bone Joint Surg., *54A*:1345–1346, 1972.

Burrows, H. J.: Fatigue infraction of the middle of the tibia in ballet dancers. J. Bone Joint Surg., *38B*:83–94, 1956.

Devas, M.: Stress Fractures. Edinburgh, London, and New York, Churchill Livingstone, 1975.

Frankel, V. H., and Hang, Y.-S.: Recent advances in the biomechanics of sport injuries. Acta Orthop. Scand., *46*:484–497, 1975.

Friedenberg, Z. B.: Fatigue fractures of the tibia. Clin. Orthop., *76*:111–115, 1971.

McBryde, A. M.: Stress fractures in athletes. J. Sports Med., *3*:212–217, 1976.

Walter, N. E., and Wolf, M. D.: Stress fractures in young athletes. Am. J. Sports Med., *5*:165–170, 1977.

Bone Remodeling

Abramson, A.: Bone disturbances in injuries to the spinal cord and cauda equina (paraplegia). Their prevention by ambulation. J. Bone Joint Surg., *30A*:982–987, 1948.

Bartley, M. H., Arnold, J. S., Haslam, R. K., and Jee, W. S. S.: The relationship of bone strength and bone quantity in health, disease, and aging. J. Gerontol., *21*:517–521, 1966.

Bassett, C. A. L.: Biologic significance of piezoelectricity. Calcif. Tissue Res., *1*:252–272, 1968.

Chamay, A., and Tschantz, P.: Mechanical influences in bone remodeling. Experimental research on Wolff's Law. J. Biomech., *5*:173–180, 1972.

Dalén, N., and Olsson, K.-E.: Bone mineral content and physical activity. Acta Orthop. Scand., *45*:170–174, 1974.

Franke, J., et al.: Physical properties of fluorosis bone. Acta Orthop. Scand., *47*:20–27, 1976.

Gjelsvik, A.: Bone remodeling and piezoelectricity—I. J. Biomech., *6*:69–77, 1973.

Horal, J., Nachemson, A., and Scheller, S.: Clinical and radiological long term follow-up of vertebral fractures in children. Acta Orthop. Scand., *43*:491–503, 1972.

Huddleston, A. L., Rockwell, D., Kulund, D. N., and Harrison, R. B.: Bone mass in lifetime tennis athletes. JAMA, *244*:1107–1109, 1980.

Jaworski, Z. F. G.: Physiology and pathology of bone remodeling. Cellular basis of bone structure in health and in osteoporosis. Orthop. Clin. North Am., *12*:485–512, 1981.

Jenkins, D. P., and Cochran, T. H.: Osteoporosis: The dramatic effect of disuse of an extremity. Clin. Orthop., *64*:128–134, 1969.

Jones, H. H., et al.: Humeral hypertrophy in response to exercise. J. Bone Joint Surg., *59A*:204–208, 1977.

Lanyon, L. E., and Bourn, S.: The influence of mechanical function on the development and remodeling of the tibia. An experimental study in sheep. J. Bone Joint Surg., *61A*:263–273, 1979.

Martin, R. B., Pickett, J. C., and Zinaich, S.: Studies of skeletal remodeling in aging men. Clin. Orthop., *149*:268–282, 1980.

Rambaut, P. C., and Johnston, R. S.: Prolonged weightlessness and calcium loss in man. Acta Astronautica, *6*:1113–1122, 1979.

Schock, C. C., Noyes, F. R., Mathews, C. H. E., and Crouch, M. M.: The effect of activity on surface remodelling in the Rhesus monkey rib and femur. Transactions of the 23rd Annual Meeting, Orthopaedic Research Society, 2:36, 1977.

Sharpe, W. D.: Age changes in human bone: An overview. Bull. N.Y. Acad. Med., *55*:757–773, 1979.