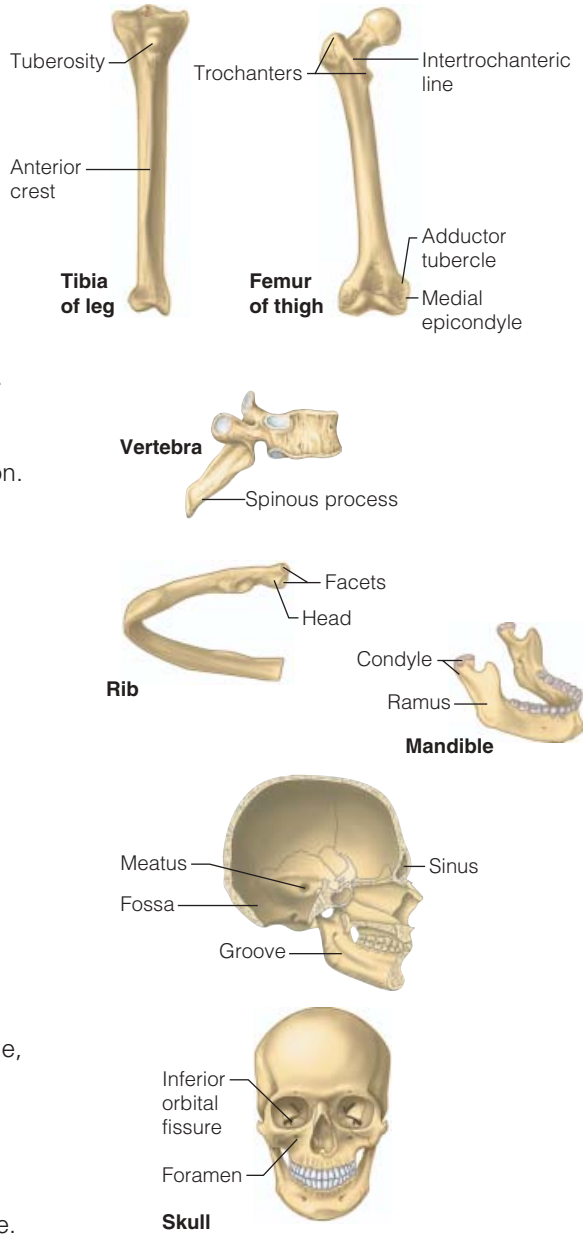
TABLE 5.1 Bone Markings

Name of bone marking	Description	Illustration
Projections that are sites of muscle and ligament attachment		
Tuberosity	Large, rounded projection; may be roughened.	
Crest	Narrow ridge of bone; usually prominent.	
Trochanter (tro-kan'ter)	Very large, blunt, irregularly shaped process. (The only examples are on the femur.)	
Line	Narrow ridge of bone; less prominent than a crest.	
Tubercle (too'ber-kl)	Small, rounded projection or process.	
Epicondyle	Raised area on or above a condyle.	
Spine	Sharp, slender, often pointed projection.	
Process	Any bony prominence.	
Projections that help to form joints		
Head	Bony expansion carried on a narrow neck.	
Facet	Smooth, nearly flat articular surface.	
Condyle (kon'dīl)	Rounded articular projection.	
Ramus (ra'mus)	Armlike bar of bone.	
Depressions and openings allowing blood vessels and nerves to pass		
Meatus (me-a'tus)	Canal-like passageway.	
Sinus	Cavity within a bone, filled with air and lined with mucous membrane.	
Fossa (fos'ah)	Shallow, basinlike depression in a bone, often serving as an articular surface.	
Groove	Furrow.	
Fissure	Narrow, slitlike opening.	
Foramen (fo-ra'men)	Round or oval opening through a bone.	



Bone Formation, Growth, and Remodeling

The skeleton is formed from two of the strongest and most supportive tissues in the body—cartilage and bone. In embryos, the skeleton is primarily made of hyaline cartilage, but in the young child most of the cartilage has been replaced by bone. Cartilage remains only in isolated areas such as the bridge of the nose, parts of the ribs, and the joints.

Except for flat bones, which form on fibrous membranes, most bones develop using hyaline

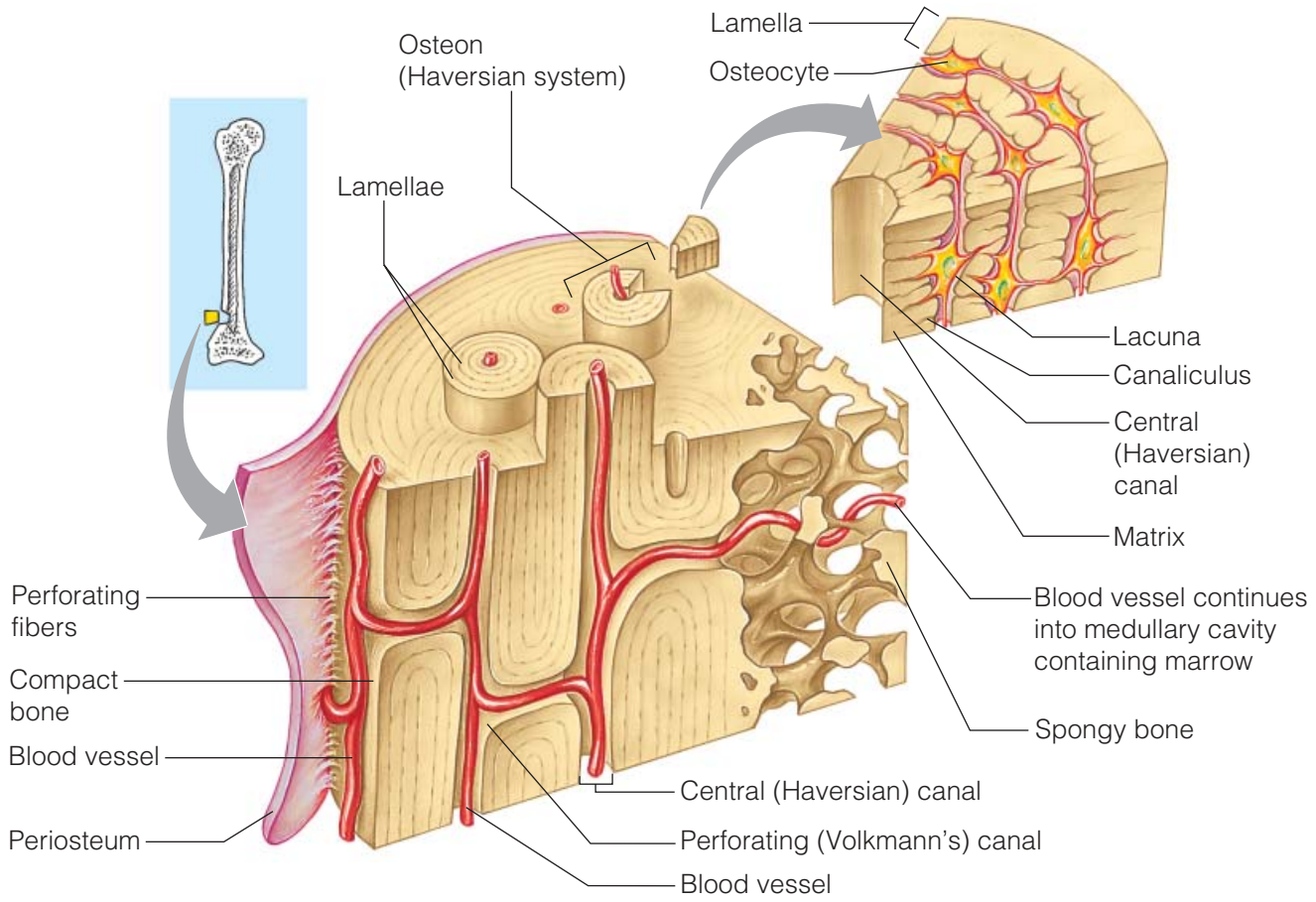
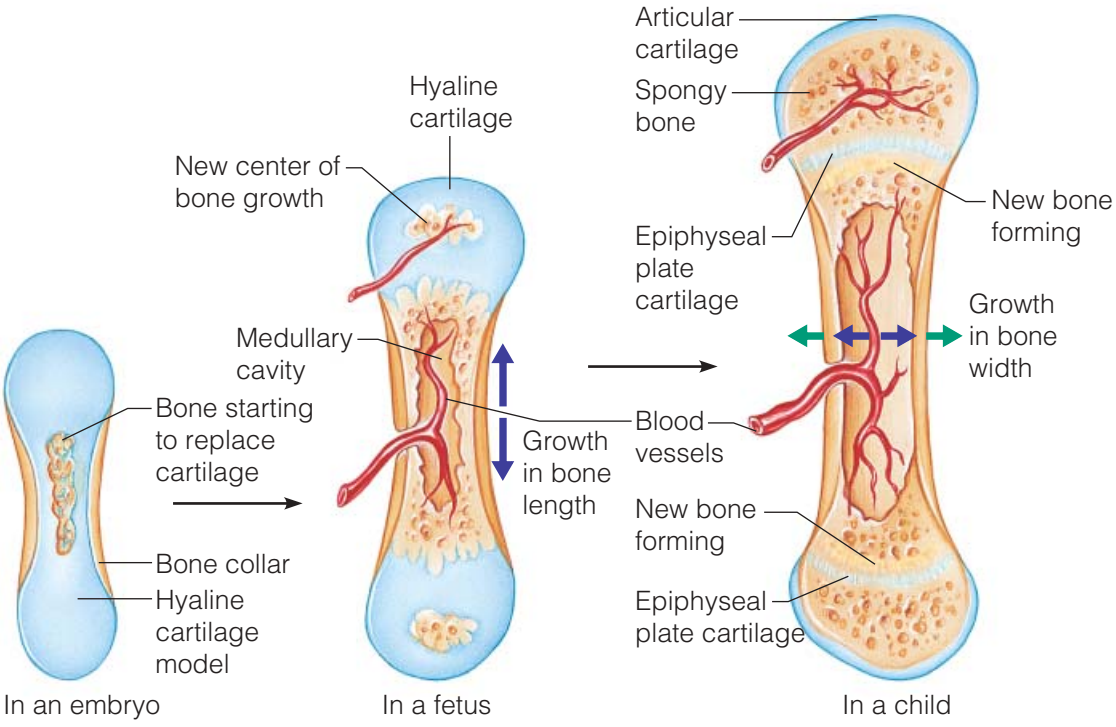


FIGURE 5.3 Microscopic structure of compact bone. Diagram of a pie-shaped segment of compact bone. (The inset shows a more highly magnified view.) Notice the position of osteocytes in lacunae (cavities in the matrix).

cartilage structures as their "models." Most simply, this process of bone formation, or **ossification** (os″ĭ-fĭ-ka′shun), involves two major phases (Figure 5.4a). First, the hyaline cartilage model is completely covered with bone matrix (a bone "collar") by bone-forming cells called **osteoblasts.** So, for a short period, the fetus has cartilage "bones" enclosed by "bony" bones. Then, the enclosed hyaline cartilage model is digested away, opening up a medullary cavity within the newly formed bone.

By birth or shortly after, most hyaline cartilage models have been converted to bone except for two regions—the **articular cartilages** (that cover the bone ends) and the **epiphyseal plates.** The articular cartilages persist for life, reducing friction at the joint surfaces. The epiphyseal plates provide for longitudinal growth of the long bones during childhood. New cartilage is formed continuously on the external face of the articular cartilage and on the epiphyseal plate surface that is farther away from the medullary cavity. At the same time, the old cartilage abutting the internal face of the articular cartilage and the medullary cavity is broken down and replaced by bony matrix (Figure 5.4b). Growing bones also must widen as they lengthen. How do they widen? Simply, osteoblasts in the periosteum add bone tissue to the external face of the diaphysis as osteoclasts in the endosteum remove bone from the inner face of the diaphysis wall (see Figure 5.4b). Since these two processes occur at about the same rate, the circumference of the long bone expands and the bone widens. This process by which bones increase in diameter is called *appositional growth*. This process of long-bone growth is controlled by hormones, most importantly *growth hormone* and, during puberty, the *sex hormones*. It ends during adolescence, when the epiphyseal plates are completely converted to bone.

Q *(a) What specific cell types form the bone collar? (b) What do you think a long bone would look like at the end of adolescence if bone remodeling did not occur?*



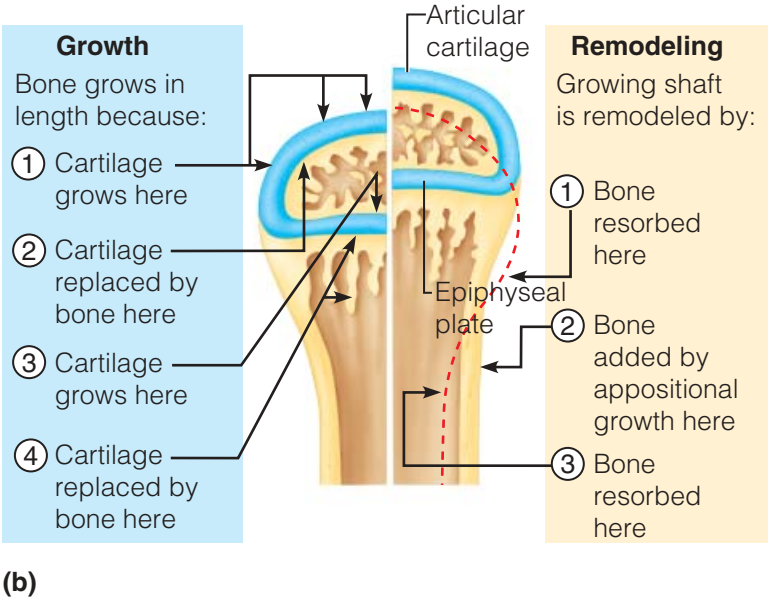
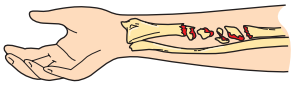
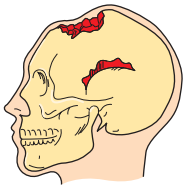
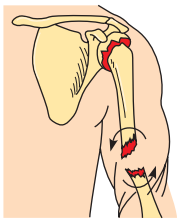
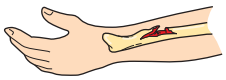


FIGURE 5.4 Long-bone formation and growth. (a) Stages of long-bone formation in an embryo, fetus, and young child. **(b)** The events indicated at the left depict the process of ossification that occurs at the articular cartilages and epiphyseal plates as the bone grows in length. The events indicated at the right reveal the process of appositional growth that occurs during long-bone growth to maintain proper bone proportions by enlarging long-bone diameter.

A *(a) Osteoblasts form the bone collar. (b) It would have a relatively short slender shaft with elongated clublike ends.*

TABLE 5.2 Common Types of Fractures

Fracture type	Illustration	Description	Comment
Comminuted		Bone breaks into many fragments.	Particularly common in the aged, whose bones are more brittle.
Compression		Bone is crushed.	Common in porous bones (i.e., osteoporotic bones).
Depressed		Broken bone portion is pressed inward.	Typical of skull fracture.
Impacted		Broken bone ends are forced into each other.	Commonly occurs when one attempts to break a fall with outstretched arms.
Spiral		Ragged break occurs when excessive twisting forces are applied to a bone.	Common sports fracture.
Greenstick		Bone breaks incompletely, much in the way a green twig breaks.	Common in children, whose bones are more flexible than those of adults.

Many people mistakenly think that bones are lifeless structures that never change once long-bone growth has ended. Nothing could be further from the truth; bone is a dynamic and active tissue. Bones are remodeled continually in response to changes in two factors: (1) calcium levels in the blood, and (2) the pull of gravity and muscles on the skeleton. How these factors influence bones is outlined next.

When blood calcium levels drop below homeostatic levels, the parathyroid glands (located in the throat) are stimulated to release parathyroid hormone (PTH) into the blood. PTH activates **osteoclasts,** giant bone-destroying cells in bones, to break down bone matrix and release calcium ions into the blood. On the other hand, when blood calcium levels are too high (*hypercalcemia* [hi″per-kal-se′me-ah]), calcium is deposited in bone matrix as hard calcium salts.

Bone remodeling is essential if bones are to retain normal proportions and strength during long-bone growth as the body increases in size and weight. It also accounts for the fact that bones become thicker and form large projections to increase their strength in areas where bulky muscles are attached. At such sites, osteoblasts lay down new matrix and become trapped within it. (Once they are trapped, they become osteocytes, or mature bone cells.) On the other hand, the bones of bedridden or physically inactive people tend to lose mass and to atrophy because they are no longer subjected to stress.

To explain the interaction between these two controlling mechanisms as simply as possible, PTH determines *when* (or *if*) bone is to be broken down or formed in response to the need for more or fewer calcium ions in the blood. On the other hand, the stresses of muscle pull and gravity acting on the skeleton determine *where* bone matrix is to be broken down or formed so that the skeleton can remain as strong and vital as possible.

Homeostatic Imbalance

Rickets is a disease of children in which the bones fail to calcify. As a result, the bones soften and a definite bowing of the weight-bearing bones of the legs occurs. Rickets is usually due to a lack of calcium