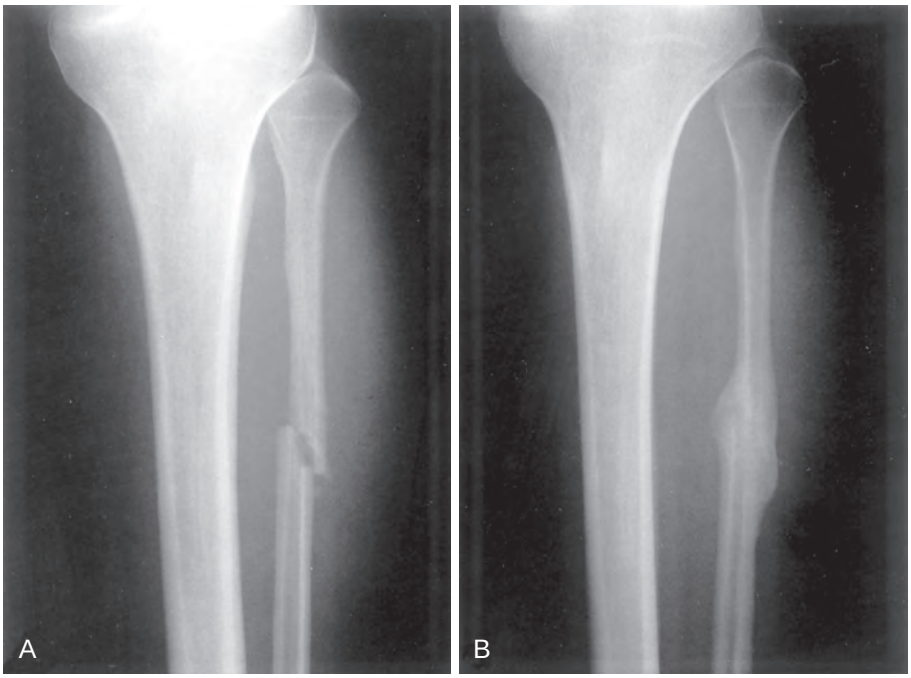

Figure 26-18 A, Recent fracture of the fibula. **B,** Marked callus formation 6 weeks later. (Courtesy Dr. Barbara Weissman, Brigham and Women's Hospital, Boston, Mass.)

forces, the portions that are not physically stressed are resorbed. In this manner the callus is reduced in size and the shape and outline of the fractured bone are reestablished as *lamellar bone*. The healing process is complete with restoration of the medullary cavity.

The sequence of events in the healing of a fracture can be easily impeded or even blocked. For example, displaced and comminuted fractures frequently result in some deformity. Inadequate immobilization permits movement of the callus and prevents its normal formation, resulting in *delayed union* or *nonunion*. If a nonunion persists, the malformed callus undergoes cystic degeneration, and the luminal surface can actually become lined by synovial-like cells, creating a false joint or *pseudoarthrosis*. A serious obstacle to healing is infection of the fracture site, which is especially common in open fractures. Malnutrition and skeletal dysplasia also hinder fracture healing. In children and young adults, near perfect union is the norm. In older adults, fractures often occur in the background of other bone disorders (e.g., osteoporosis and osteomalacia). In such settings, surgical immobilization is often needed for adequate repair.

Osteonecrosis (Avascular Necrosis)

Infarction of bone and marrow is a relatively common event that can occur in the medullary cavity or involve both the medulla and cortex. Most cases of bone necrosis stem from fractures or corticosteroid administration. A diverse set of other conditions also predispose to osteonecrosis (Table 26-5). All are believed to lead to vascular insufficiency through mechanical injury to blood vessels, thromboembolism, external pressure on vessels, or venous occlusion.

MORPHOLOGY

Regardless of etiology, medullary infarcts are geographic and involve the trabecular bone and marrow. The cortex is usually not affected because of its collateral blood flow. In subchondral infarcts, a triangular or wedge-shaped segment of tissue that has the subchondral bone plate as its base undergoes necrosis. The overlying articular cartilage remains viable, as it can access nutrients that are present in synovial fluid. Microscopically, dead bone is recognized by empty lacunae surrounded by necrotic adipocytes that frequently rupture. The released fatty acids bind calcium and form insoluble calcium soaps that may persist for life. In the healing response, osteoclasts resorb the necrotic trabeculae. Trabeculae that remain act as scaffolding for the deposition of new bone in a process known as creeping substitution. In subchondral infarcts the pace of this substitution is too slow to be effective, so there is collapse of the necrotic bone and distortion, fracture, and even sloughing of the articular cartilage (Fig. 26-19).

Table 26-5 Conditions Associated with Osteonecrosis

Alcohol abuse
Bisphosphonate therapy (especially jawbones)
Connective tissue disorders
Corticosteroid administration
Chronic pancreatitis
Dysbarism (the "bends")
Gaucher disease
Infection
Pregnancy
Radiation therapy
Sickle cell crisis (Chapter 14)
Trauma
Tumors