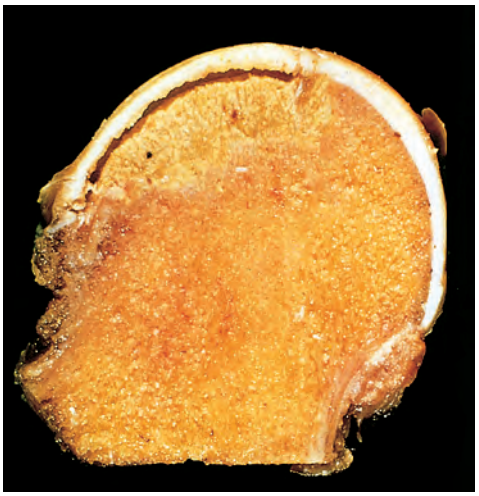

Figure 26-19 Femoral head with a subchondral, wedge-shaped pale yellow area of osteonecrosis. The space between the overlying articular cartilage and bone is caused by trabecular compression fractures without repair.

Clinical Course. **The symptoms depend on the location and extent of infarction.** Typically, subchondral infarcts cause pain that is initially associated only with activity but then becomes constant as secondary changes supervene. Subchondral infarcts often collapse and may lead to severe, secondary osteoarthritis. In contrast, medullary infarcts are usually small and clinically silent except when they occur in the setting of Gaucher disease, dysbarism (e.g., the "bends", Chapter 4), and sickle cell anemia. More than 10% of the 500,000 joint replacements performed annually in the United States are for treatment of complications of osteonecrosis.

Osteomyelitis

Osteomyelitis denotes inflammation of bone and marrow, virtually always secondary to infection. Osteomyelitis may be a complication of any systemic infection but frequently manifests as a primary solitary focus of disease. All types of organisms, including viruses, parasites, fungi, and bacteria, can produce osteomyelitis, but infections caused by certain pyogenic bacteria and mycobacteria are the most common. Currently in the United States, exotic infections in immigrants from developing countries and opportunistic infections in immunosuppressed individuals have made the diagnosis and treatment of osteomyelitis challenging.

Pyogenic Osteomyelitis

Pyogenic osteomyelitis is almost always caused by bacterial infections. Organisms may reach the bone by (1) hematogenous spread, (2) extension from a contiguous site, and (3) direct implantation. In otherwise healthy children, most osteomyelitis is hematogenous in origin and develops in the long bones. The initiating bacteremia may stem from seemingly trivial mucosal injuries, such as may occur during defecation or vigorous chewing of hard foods, or from minor infections of the skin. In adults, however, osteomyelitis more often occurs as a complication of open fractures, surgical procedures, and diabetic infections of the feet.

Staphylococcus aureus is responsible for 80% to 90% of the cases of culture-positive pyogenic osteomyelitis. These organisms express cell wall proteins that bind to bone matrix components such as collagen, which facilitates adherence of the bacteria to bone. *Escherichia coli*, *Pseudomonas*, and *Klebsiella* are more frequently isolated from individuals with genitourinary tract infections or who are intravenous drug abusers. Mixed bacterial infections are seen in the setting of direct spread or inoculation of organisms during surgery or into open fractures. In the neonatal period, *Haemophilus influenzae* and group B streptococci are frequent pathogens, and individuals with sickle cell disease are predisposed to *Salmonella* infection. In almost 50% of suspected cases, no organisms can be isolated.

The location of the bone infections is influenced by the osseous vascular circulation, which varies with age. In the neonate the metaphyseal vessels penetrate the growth plate, resulting in frequent infection of the metaphysis, epiphysis, or both. In children, localization of microorganisms in the metaphysis is typical. After growth plate closure, the metaphyseal vessels reunite with their epiphyseal counterparts and provide a route for the bacteria to seed the epiphyses and subchondral regions, which are common sites of infection in the adult.

MORPHOLOGY

Changes associated with osteomyelitis depend on the stage (acute, subacute, or chronic) and location of the infection. In the acute phase, bacteria proliferate and induce a neutrophilic inflammatory reaction. Necrosis of bone cells and marrow ensues within the first 48 hours. The bacteria and inflammation spread longitudinally and may percolate throughout the Haversian systems to reach the periosteum. In children the periosteum is loosely attached to the cortex. Thus, a sizable subperiosteal abscesses may form that can dissect for long distances along the bone surface. Lifting of the periosteum further impairs the blood supply to the affected region, contributing to the necrosis. The dead bone is known as a **sequestrum**. Rupture of the periosteum leads to a soft tissue abscess which can channel to the skin as a draining sinus. Sometimes the sequestrum crumbles, releasing fragments that pass through the sinus tract.

In infants, but uncommonly in adults, epiphyseal infection spreads through the articular surface or along capsular and tendoligamentous insertions into a joint, producing septic or suppurative arthritis, which can cause destruction of the articular cartilage and permanent disability. An analogous process involves the vertebrae, in which the infection destroys the hyaline cartilage end plate and intervertebral disc and spreads into adjacent vertebrae.

After the first week, chronic inflammatory cells release cytokines that stimulates osteoclastic bone resorption, ingrowth of fibrous tissue, and the deposition of reactive bone at the periphery. The newly deposited bone can form a shell of living tissue, known as an **involucrum,** around the segment of devitalized infected bone (Fig. 26-20). Several morphologic variants of osteomyelitis have eponyms. **Brodie abscess** is a small intraosseous abscess that frequently involves the cortex and is walled off by reactive bone. **Sclerosing osteomyelitis of Garré** typically develops in the jaw and is associated with extensive new bone formation that obscures much of the underlying osseous structure.