Bone infections and inflammations are among the most common and important indications for bone scintigraphy, the sensitivity of which has been firmly established (Duszynski et al. 1975; Gilday et al. 1975). Etiologically, the offending organisms are diverse, and the causes of nonspecific osteitides are obscure except for X- or γ-irradiation in radiation osteitis. For a scintigraphic description, bone infections may be categorized according to the site of initial affliction. Thus, pyogenic infection in bone marrow is termed osteomyelitis, and infections of the cortex, periosteum, and soft tissues are referred to, respectively, as infective osteitis or abscess, infective periostitis, and soft-tissue abscess. More often than not the three types of bone infection as well as soft-tissue infection become concurrent if the initial event is not brought under prompt control.

Clinically, bone infections may be classified into acute, subacute, and chronic, although attempts at sharp demarcation are for the most part unsuccessful and impracticable. Nonetheless, if well-isolated the acute bone infection is characterized by an abrupt, febrile, and painful onset with swelling, tenderness, and limited motion. In the chronic stage, symptoms and signs become poorly defined. Garré's sclerosing osteitis is a special form of chronic osteomyelitis. A group of inflammatory bone diseases of unknown cause are termed nonspecific osteitides. These include osteitis condensans ilii, osteitis pubis, condensing osteitis of the clavicle, Caffey's infantile cortical hyperostosis, and Paget's osteitis deformans. Bony proliferation and sclerosis are common features.

Before the advent of bone scintigraphy the imaging diagnosis of infective and inflammatory bone diseases was solely the domain of radiography. However, since Capitanio and Kirkpatrick in 1970 reported that acute osteomyelitis could be diagnosed by bone scintigraphy days earlier than by radiography, its usefulness has been systematically explored in infective bone diseases.

### 6.1 Acute Osteomyelitis

Osteomyelitis is primarily a disease of childhood and infancy, but a recent rise in its frequency is noted among the elderly (Waldvogel et al. 1970). The major offenders are *Staphylococcus aureus* in children, β-hemolytic streptococcus, *S. aureus* and *Escherichia coli* in neonates, and gram-negative bacilli in adults and drug abusers. One study of 348 adult patients with osteomyelitis by Waldvogel and Papageorgiou (1980) revealed that *S. aureus*, enteric species, and streptococcal organisms are causative in 60%, 29% and 8%, respectively. The organisms can reach bone by blood flow, by continuity from the infected soft-tissue focus, or by direct implantation through an open wound from needling, cutting, acupuncturing, or operation.

#### 6.1.1 Pathogenesis

In the hematogenous form, the infective process classically starts in the metaphysis of long bone by the lodgment of organisms in endarteries, in which a slow blood flow facilitates their entrapment. As bone and bone marrow become the loci of bacterial proliferation, tissues
react intensely with hyperemia, edema, cell infiltration, and suppuration, creating a milieu under pressure within the closed space of the bone marrow. Both the elevated intramedullary pressure and the proteolytic action of the offenders cause infective material to leak through the haversian and Volkmann’s canals of the cortex out to the subperiosteal layer, to produce an abscess. As a result, the affected bone becomes necrotized and the periosteum reacts with brisk new bone formation. The former is known as sequestrum and the latter involucrum. As shown in Fig. 6.1, the metaphyseal vascular supply, periosteal attachment, and histological characteristics of the physis in the long bone differ among infants under 1 year of age, children aged up to 16 years, and adults after bone fusion. During infancy, some of the metaphyseal arteries penetrate the growth cartilage to enter the epiphysis, carrying bacteria to that part. In children, however, such a tranochondral vasculature ceases to exist and, instead, more mature growth cartilage resists the bacterial spread from the metaphysis. This barrier delimits the infection to the metaphysis in the initial stage of an acute osteomyelitis (Fig. 6.2).

6.1.2 Radiographic Manifestations

The earliest but nonspecific radiographic change of acute osteomyelitis is soft-tissue swelling with the obliteration of the fat plane. It can be detected as early as 3 days after the sudden onset of disease. However, the direct sign of bone infection manifests more than a week later. The initial bone alterations are local osteopenia and osteolysis typically in the long bone metaphysis. With the rapid progress of the disease, infection spreads rampantly from cancellous bone to cortex and periosteum,