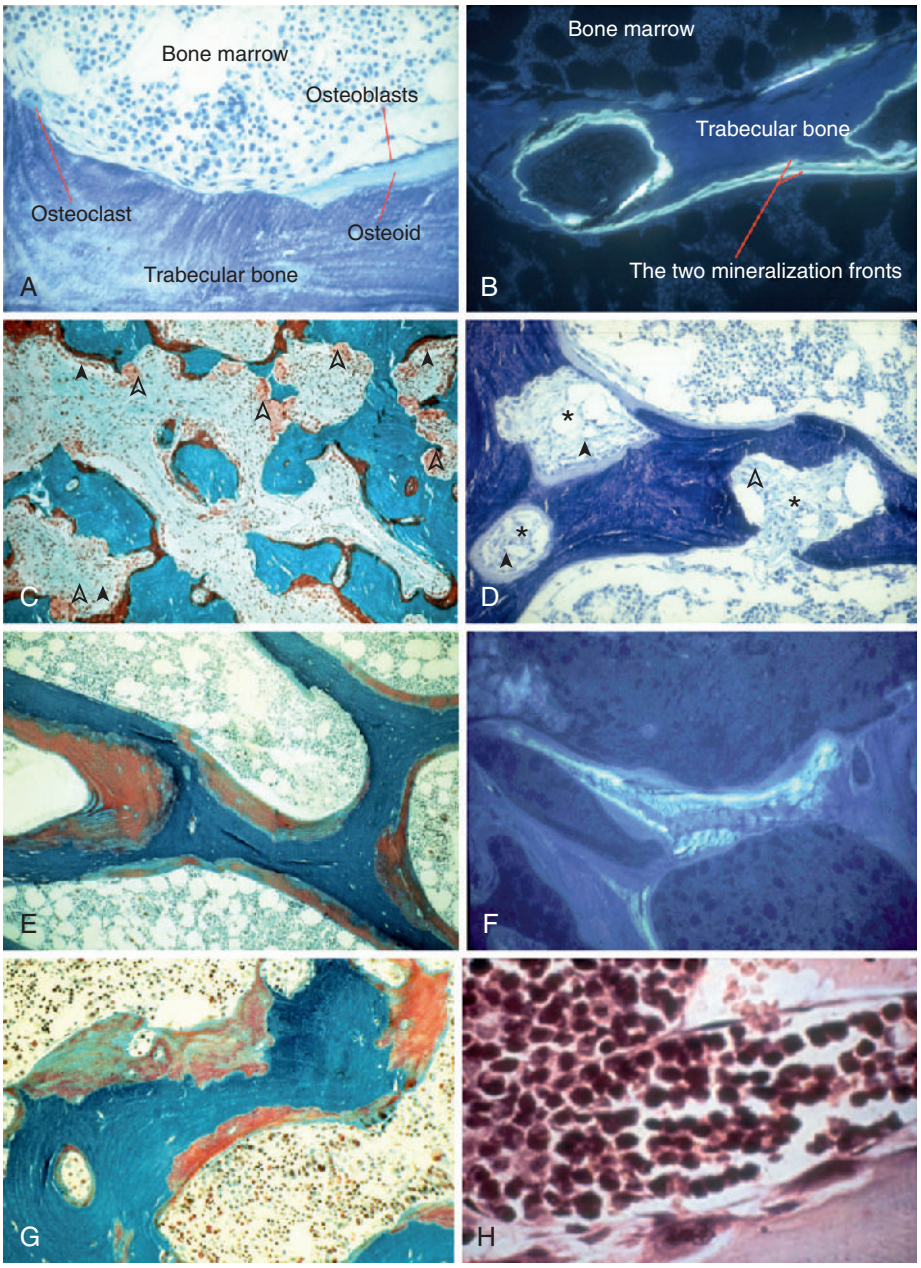

FIGURE 74-1 **A,** Normal bone histology, showing a normal bone-remodeling unit as seen in an undecalcified human anterior iliac crest biopsy. On the left, a multinucleated osteoclast has moved across the mineralized trabecular bone surface over the previous week or two, resorbing (removing) old bone. On the extreme right, the bone surface is covered by osteoid secreted by the overlying osteoblasts. In between the osteoclast- and osteoblast-covered surfaces of the trabecular bone are a large number of flat, fibroblastoid cells referred to as *lining cells.* No osteocytes are visible in this section. **B,** Tetracycline labeling of a bone biopsy from a patient with hyperparathyroid bone disease. Notice the bright yellow parallel lines on the trabecular bone surface. These lines represent the two sets of tetracycline labeling, which occurred 14 days apart. From these sets, the mineralization rate can be described in micrometers (microns) per day, the so-called *mineral apposition rate*, and it is increased dramatically in this example, as is typical of hyperparathyroid bone disease. Contrast with example **F**, which has no tetracycline labeling. **C,** Paget's disease. Enormous and abundant highly multinucleated osteoclasts *(open arrowheads)* are resorbing trabecular bone, and a comparably enormous number of osteoblasts *(closed arrowheads)* are making new but disorganized bone. The marrow space is replaced by fibrous cells. **D,** Primary hyperparathyroidism has the classic features of osteitis fibrosa cystica. Far more osteoid and osteoblasts *(closed arrowheads)* and osteoclasts *(open arrowhead)* exist than in the normal example (**A**). Three large microcysts *(asterisks)* have been created by aggressive osteoclastic bone resorption. These microcysts account for the *cystica* component of osteitis fibrosa cystica. The marrow space, particularly within the microcysts, is filled with fibroblasts, which make up the *fibrosa* component of osteitis fibrosa cystica. **E,** Osteomalacia or rickets. Notice the abundant quantities of partially and chaotically mineralized osteoid *(orange)*. These seams are the thick osteoid seams and represent osteoid that has been produced by osteoblasts but that cannot mineralize, which is the signature defect in osteomalacia and rickets. **F,** Tetracycline labeling reveals a complete absence of mineralization, diagnostic of osteomalacia or rickets. Compare with example **B**. **G,** Renal osteodystrophy. This photomicrograph of a biopsy from a patient on dialysis demonstrates many of the classic features of renal osteodystrophy, including evidence of aggressive osteoclastic bone resorption (i.e., numerous osteoclastic lacunae on the bone surface compared with the smooth surfaces in example **A**) and abundant, partially and chaotically mineralized areas of osteoid *(orange)*. **H,** Infiltrative bone disease as exemplified by multiple myeloma. The bone marrow is replaced by plasma cells, and two large osteoclasts in lacunae are actively resorbing the trabecular bone surface.