more difficult to do in the open medical care systems in the United States. In countries with single-payor systems, that approach does seem to be effective, as is also the case in closed health care systems in the United States.

The risk for future fracture after a first fracture is not linear. Highest risk occurs within the first 2 years after the first fracture. A recent large Medicare database study indicated that almost 20% of women will have a second fracture within 2 years after the first. Risk diminishes to less than half of that rate in the subsequent 3 years and declines to baseline thereafter for most fracture types, although risk after a vertebral or hip fracture may persist.

PATHOPHYSIOLOGY

■ BONE REMODELING

Osteoporosis results from bone loss due to age-related changes in bone remodeling as well as extrinsic and intrinsic factors that exaggerate this process. These changes may be superimposed on a low peak bone mass. Consequently, understanding the bone remodeling process is fundamental to understanding both the pathophysiology of osteoporosis **(Chap. 409)** and the effects of pharmacologic intervention. During growth, the skeleton increases in size by linear growth and by apposition of new bone tissue on the outer surfaces of the cortex **(Fig. 411-4)**. The latter process is called *modeling*, a process that also allows the long bones to adapt in shape to the stresses placed on them. Increased sex hormone production at puberty is required for skeletal maturation, which reaches maximum mass and density in early adulthood. Recent data suggest that delayed puberty may be associated with low bone mass that persists into adulthood. The sexual dimorphism in skeletal size becomes obvious after puberty, although true bone density remains similar between the sexes. Nutrition and lifestyle also play an important role in growth, although genetic factors primarily determine peak skeletal mass and density.