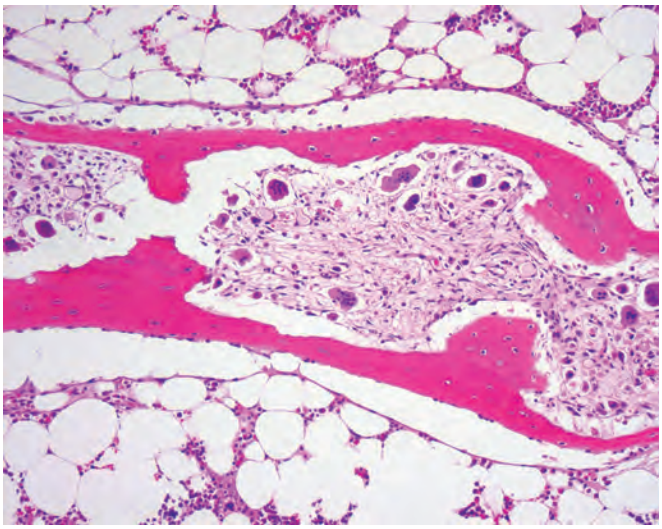

Figure 26-15 Hyperparathyroidism with osteoclasts boring into the center of the trabeculum (dissecting osteitis).

generalized, but is most severe in the phalanges, vertebrae and proximal femur. The increased osteoclast activity in hyperparathyroidism is most prominent in cortical bone (subperiosteal and endosteal surfaces) but medullary bone is not spared. Indeed, osteoclasts may tunnel into and dissect centrally along the length of the trabeculae, creating the appearance of railroad tracks and producing what is known as **dissecting osteitis** (Fig. 26-15). The marrow spaces around the affected surfaces are replaced by fibrovascular tissue. The correlative radiographic finding is a decrease in bone density or osteoporosis.

The bone loss predisposes to microfractures and secondary hemorrhages that elicit an influx of macrophages and an ingrowth of reparative fibrous tissue, creating a mass of reactive tissue, known as a **brown tumor** (Fig. 26-16). The brown color is the result of the vascularity, hemorrhage, and hemosiderin deposition, and it is not uncommon for the lesions to undergo cystic degeneration. The combination of increased bone cell activity, peritrabecular fibrosis, and cystic brown tumors is the hallmark of severe hyperparathyroidism and is known as **generalized osteitis fibrosa cystica (von Recklinghausen disease of bone)**.

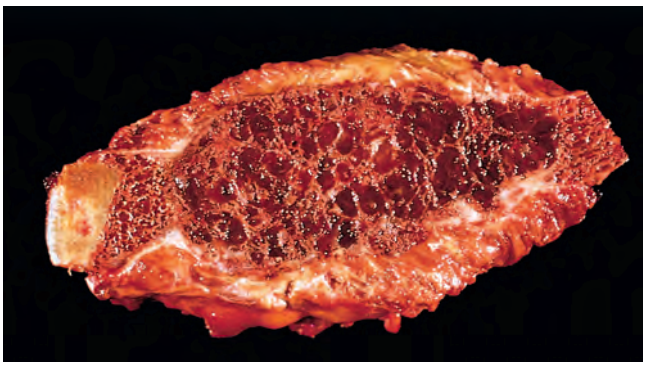

Figure 26-16 Resected rib, harboring an expansile brown tumor adjacent to the costal cartilage.

Osteitis fibrosa cystica is now rarely encountered because hyperparathyroidism is usually diagnosed on routine blood tests and treated at an early stage. Secondary hyperparathyroidism is usually not as severe or as prolonged as primary hyperparathyroidism, hence the skeletal abnormalities tend to be milder. Control of hyperparathyroidism allows the bony changes to regress or disappear completely.

Renal Osteodystrophy

The term *renal osteodystrophy* describes collectively the skeletal changes that occur in chronic renal disease, including those associated with dialysis. The manifestations are not unique, but include many of the entities described above including (1) osteopenia/osteoporosis (2) osteomalacia, (3) secondary hyperparathyroidism, and (4) growth retardation. As advances in medical technology have prolonged the lives of individuals with renal disease, its impact on skeletal homeostasis has assumed greater clinical importance. The various histologic bone changes in individuals with end-stage renal failure can be divided into three major types of disorders:

- *High-turnover osteodystrophy* is characterized by increased bone resorption and bone formation, with the former predominating.
- *Low-turnover or aplastic disease* is manifested by adynamic bone (little osteoclastic and osteoblastic activity) and, less commonly, osteomalacia.
- *Mixed pattern of disease* with areas of high turnover and low turnover.

Pathogenesis. Kidney disease causes skeletal abnormalities through three mechanisms (Fig. 26-17).

- **Tubular dysfunction.** The major tubular disease that affects the skeleton is renal tubular acidosis. The associated low pH dissolves hydroxyappatite, resulting in demineralization of the matrix and osteomalacia.
- **Generalized renal failure**, affecting glomerular and tubular function, leads to reduced phosphate excretion, chronic hyperphosphatemia, hypocalcemia and, ultimately *secondary hyperparathyroidism*. The resulting metabolic state is not completely analogous to primary

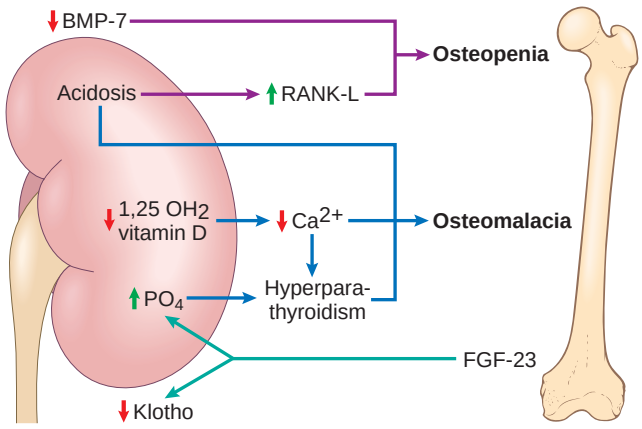

Figure 26-17 Mechanisms of renal osteodystrophy involves electrolyte levels and endocrine signaling between bone and kidney.