involving different oral phosphate binders have therefore been pursued to lower intestinal phosphate absorption early during the course of kidney disease and to thereby lower FGF23 levels. However, these approaches have been largely disappointing. Furthermore, there is concern as to whether supplementation with activated vitamin D analogues increases further the circulating FGF23 levels and their “off-target” effects in CKD patients.

TREATMENT

Chronic Kidney Disease

Therapy of CKD **(Chap. 311)** involves appropriate management of patients prior to dialysis and adjustment of regimens once dialysis is initiated. Attention should be paid to restriction of phosphate in the diet; avoidance of aluminum-containing phosphate-binding antacids; provision of an adequate calcium intake by mouth, usually 1–2 g/d; and supplementation with 0.25–1 μg/d calcitriol or other activated forms of vitamin D. The aim of therapy is to restore normal calcium balance to prevent osteomalacia and severe secondary hyperparathyroidism (it is usually recommended to maintain PTH levels between 100 and 300 pg/mL) and, in light of evidence of genetic changes and monoclonal outgrowths of parathyroid glands in CKD patients, to prevent secondary hyperparathyroidism from becoming autonomous hyperparathyroidism. Reduction of hyperphosphatemia and restoration of normal intestinal calcium absorption by calcitriol can improve blood calcium levels and reduce the manifestations of secondary hyperparathyroidism. Since adynamic bone disease can occur in association with low PTH levels, it is important to avoid excessive suppression of the parathyroid glands while recognizing the beneficial effects of controlling the secondary hyperparathyroidism. These patients should be closely monitored with PTH assays that detect only the full-length or bioactive PTH(1–84) to ensure that inactive, inhibitory PTH fragments are not measured. Use of oral phosphate-binding agents such as sevelamer lower blood phosphate levels in ESKD, but their use in earlier CKD stages does not seem to be beneficial in lowering blood phosphate levels and to prevent the rise in FGF23.