

Introduction

Acidosis has widespread effects on metabolism in patients with chronic renal failure. It increases protein degradation and amino acid oxidation (*Reaich et al., 1993*)

Acidosis is also associated with osteomalacia and osteopenia (*Mora Parma et al., 1983*). Acidosis may have a role in the development of hyperparathyroidism. This has been suggested by studies showing that acidosis enhances circulating parathyroid hormone (PTH), and optimal correction of acidosis in hemodialysis patients can delay the progression of hyperparathyroidism (*Lé Febvre et al., 1989*).

Reduced serum concentration of the active metabolite of vitamin D = 1,25(OH)₂D have been implicated in mediating the acidosis-induced metabolic bone disease (*Langman, 1989*). The low 1,25(OH)₂D serum concentration have been attributed to diminished renal 1 α -hydroxylase activity (*Reddy et al., 1982*), although not all studies have been consistent with an impaired conversion of 25(OH)D to 1,25(OH)₂D (*Kraut et al., 1983*).
