The bone disease associated with chronic renal failure in the past has often been allowed to progress to the point of producing grotesque skeletal malformations. It is doubtful whether this attitude was ever entirely justifiable but with recent developments both in the treatment of chronic renal failure and in the production of Vitamin D metabolites, it is certainly indefensible now. It is important therefore, to identify renal osteodystrophy at an early stage in its development so that appropriate treatment can be given which will prevent the pain and disability of the advanced disease.

Therefore, some years ago we decided to improve the early identification and treatment of this condition and much of the data contained in this report have been obtained by Dr. R.J. Postlethwaite and Dr. L. Hill, working jointly from the Department of Child Health and the Department of Medicine in Manchester.

Identification of early bone disease

Thirty-one children have been studied who exhibited all degrees of chronic renal failure but were not being dialysed and had not been transplanted; nor did they have renal tubular diseases as the primary cause of their chronic renal failure.

Of these 27 children, 17 showed histological evidence of renal osteodystrophy on bone biopsy. Increased osteoclastic activity was an almost universal finding but many also had thickened osteoid seams and evidence of a calcification defect. Twelve of these 17 histologically proven cases showed radiological evidence of bone disease. Twelve patients also showed serum parathormone concentrations > 1 ng/ml (11 exceeded 2 ng/ml), and all of these had evidence of radiological
abnormalities. A parathormone level of < 1 ng/ml was regularly associated with normal radiological appearances even if there was biopsy evidence of bone disease.

Serum parathormone concentrations correlated rather poorly with serum inorganic phosphate levels. Correlation between serum alkaline phosphatase concentrations and severe bone disease was somewhat better but there were several examples of hyperparathyroidism accompanied by levels of alkaline phosphatase which fell within the wide range of normality for children. Thus neither phosphate nor alkaline phosphatase concentration in the serum is a good predictor of bone disease when one looks at cross-sectional data of this type. However, if serum alkaline phosphatase concentrations are measured sequentially on individual patients, increasing levels (even within the normal range) may be better indicators of developing bone disease.

There was a good correlation between serum parathormone levels and serum creatinine concentrations, \((R = 0.82)\) and serum creatinine concentrations of > 1 mg/dl were not associated with high serum parathormone levels (Postlethwaite et al 1976); Chan & DeLuca (1979) obtained rather similar data.

These studies demonstrated that short of doing a bone biopsy, serum parathormone concentrations were one of the earliest indicators of bone disease. However, careful examination of x-rays of the hands and wrists, especially a search for sub-periosteal erosions was almost as sensitive. Moreover, such early radiological changes commonly preceded any clinical evidence such as bone pain or deformity and for clinical as opposed to research purposes, our present procedure is to rely heavily on radiological diagnosis of early but distinct disease and to use this as our indication for starting therapy. Of course this attitude assumes that histological bone disease and hyperparathyroidism without radiological changes are innocuous as well as presymptomatic situations. There have been reports (Chesney et al, 1980) that appropriate treatment of renal osteodystrophy improves growth substantially and if it could be shown that this growth improvement were present even in the pre-symptomatic stage of the disease then much earlier detection and treatment would be needed. It is also conceivable that an increased