100 cal/kg per day. Calcium cascinate was expensive and difficult to get and despite the reduced sodium intake, cardiac failure during recovery was a frequent problem. Furthermore, in the light of more recent knowledge of energy requirements for catch-up growth, the energy content of the previous diet was suboptimal.

Consequently, a number of diets were designed with varying amounts of sodium and energy. These were used to treat a series of malnourished children who were then closely followed clinically and biochemically during recovery. In particular, changes in estimated blood volume and in urinary excretion of sodium were measured. It was found that the children who subsequently developed cardiac failure had a significantly low sodium urinary excretion during the initial 5 day collection. This low sodium excretion was associated with large increases in estimated blood volume during the time when oedema was being lost.

An inexpensive diet was designed containing dried skim milk reinforced with energy derived mainly from fat. This was not associated with an increased incidence of cardiac failure when given at a concentration of 100 kcal/kg. After the loss of oedema the diet was given at a rate of 150 kcals/kg with good results.

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The diagnosis of Leigh’s encephalomyelopathy was suspected during life in a small-for-dates infant with severe neurological abnormalities, persistent lactic acidosis, and hyperalaninaemia. L-Glutamine therapy produced biochemical improvement but the infant died after smallpox vaccination. Necropsy confirmed the diagnosis and detailed enzymatic analysis of the liver revealed a deficiency of pyruvate carboxylase. The significance of this finding in relation to diagnosis and possible therapy was discussed.

Rat model for study of growth in renal failure. C. Chantler, R. D. Adelman, R. C. MacDonell, and M. A. Holliday. Department of Paediatrics, University of California, San Francisco, and Department of Paediatrics, Guy’s Hospital, London.

A rat model has been developed to study growth in uraemia. The model allows growth to be observed over short periods and will enable the study of manipulations in diet and treatment on growth to be examined within a controlled experimental design. The growth and food intake of male and female rats rendered uraemic by 80% nephrectomy was observed between 40 and 70 days of age. The uraemic rats gained significantly less weight and consumed significantly fewer calories than control sham operated rats; tail growth was also less in the uraemic rats. The effect of lowering blood urea by reducing protein intake was studied; no improvement in growth or calorie intake was observed even though blood urea levels were comparable with those found in control rats. Uraemic rats were gavaged with corn oil and improvement in growth was noted. This model should prove useful for studying other factors which have been implicated in the growth failure of uraemia.

Growth and dietary intake of children with chronic renal insufficiency. P. R. Betts and G. Magrath introduced by R. H. R. White. The Children’s Hospital, Birmingham.

Growth retardation in children with chronic renal failure is of increasing importance now that replacement treatment is possible by haemodialysis or transplantation. The relations between growth, dietary intake, and degree of renal insufficiency have been studied in 30 children as part of a larger project. The children have been divided into groups A and B.

Group A. 20 children in whom the onset of renal insufficiency occurred in early infancy as a result of either congenital defects or other causes. The majority of these children were on or below the 3rd centile for height irrespective of creatinine clearance. However, they grew steadily along their centile unless they developed renal osteodystrophy. Marked pubertal delay occurred in 2 children.

Group B. 10 children with normal renal function in early infancy but who developed a progressive nephropathy during childhood. Growth retardation in these children was not so marked. They grew normally along their centile except for those with a rapidly progressive nephropathy whose height velocity fell abnormally. Puberty progressed satisfactorily in 3 children.

There was significant reduction in the calorie, protein, and vitamin D intake of these children from that recommended for their age, and of calorie intake compared with children of their own height. It appears that the onset of renal insufficiency in early infancy has a more deleterious effect on growth than that in later years. The restriction in calorie intake in these children may, in part, be responsible for their growth retardation.


A continuous sampling technique has been used to investigate growth hormone (GH) secretion during nocturnal sleep and after insulin-stimulated hypoglycaemia (ISH) in 8 small, normal children (group A) and 6 children with clinical evidence of GH deficiency (group B). In group A there was good correlation between GH response to sleep and ISH. Sleep GH secretion, but not ISH-GH response increased with age. All group B children had low nocturnal GH secretion; ISH-GH secretion was similarly low in 4, but normal in 2 children. A relation has been shown between impaired nocturnal GH secretion, irrespective of ISH-GH response and clinical features of GH deficiency. Sleep-related GH secretion may be an important component of normal growth.