

late rickets or osteomalacia adhered to a chapati-free diet for 7 weeks, substituting unleavened bread of lower extraction. All 10 subjects showed prompt evidence of biochemical healing with subsequent biochemical relapse on resuming a normal diet. On the other hand, while 37 out of a sample of 66 Pakistani and Indian children resident in Glasgow, between 9 to 16 years of age, showed biochemical, radiological, or clinical evidence of late rickets, only one of a sample of 23 children taking vitamin D supplements showed a minimal depression of serum calcium. Similarly, a survey of the small Pakistani community in Stornoway, where atmospheric pollution is minimal, showed no biochemical evidence of rickets or osteomalacia in 42 subjects who were examined. In order to further elucidate the role of vitamin D deficiency, a competitive protein binding assay for 25-hydroxycholecalciferol (25HCC) was developed. It was found that normal Asians had 25HCC levels less than half those of Caucasians, while Asians with florid rickets had undetectable levels (limit of detectability 0.8 ng/ml).

It seems probable, therefore, that the high dietary phytate content combined with vitamin D deficiency is responsible for the marked prevalence of rickets and osteomalacia among Asian immigrants. A possible unifying hypothesis was presented for discussion.

D. BARLTROP. London. 'Neonatal calcium metabolism.' The mechanisms responsible for neonatal hypocalcaemia remain imperfectly understood. Dietary factors have been implicated including the fat content of milk and the mineral composition, and, additionally, the responsiveness of the newborn parathyroid gland has been questioned. Hitherto research has been limited by the lack of suitable methods of investigation so that the major pathways of calcium metabolism in the newborn have not been well defined. The application of a stable (nonradioactive) isotope of calcium to this problem was described. The findings in infants were presented which described the true absorption of calcium from the gut together with the urinary loss and deposition in bone. In addition, the magnitude of the exchangeable calcium pool in the newborn has been estimated. The findings have revealed an unsuspected excretion of calcium into the gut which may be an important factor. Analysis of meconium suggested that this faecal endogenous excretion also occurs *in utero*. Conventional balance studies and plasma calcium determinations are inadequate for the study of the responses to new infant milk formulae.

J. DOBBING (and Jean Sands). Manchester. 'Growth retardation and the human fetal brain.' It is inevitable that knowledge of the effects of undernutrition on the fetal human brain should be largely derived from experimental animals. It is to this extent speculative. Such speculation demands a careful examination of the validity of inter-species extrapolation.

The main difficulty is the different timing of birth in the various species rather than differences in brain development processes themselves. We now feel in a better position to make the calculations, based on our

own surveys of quantitative brain growth in man, as well as in rats, pigs, and guinea pigs.

Several fallacies in previous reasoning are now thought to exist and were discussed. Principal among these are, firstly, the widely held view that the human brain growth spurt is a mainly prenatal process. We showed that this is not so. Secondly, the false assumption that fetal brain growth in the rat is equivalent to that in humans. Restricting fetal brain growth in rats imposes constraint at a stage of brain development comparable with that in human fetal life before 18 weeks of gestation, and unless this difference in timing is taken into account serious mistakes will arise.

These matters were discussed in the light of their relevance to the cerebral consequences of human fetal growth retardation.

W. HAMILTON. Glasgow. 'Aminoglutethimide in the treatment of congenital adrenal hyperplasia.'

C. C. BAILEY introduced by G. M. Komrower. Manchester. 'Linear and skeletal growth in congenital adrenal hyperplasia.' Of 35 children diagnosed as congenital adrenal hyperplasia, 18 of the salt-losing variety have been followed from birth; the present ages ranging between 2½ and 17 years. A full description of the treatment (long acting preparations of glucocorticoid and mineralocorticoid during the first year of life followed by oral prednisolone and fludrocortisone) and the clinical progress were given with details of the criteria for control of therapy. This includes measurements of oxosteroids and pregnanetriol excretion.

Height achievement and skeletal maturity has been assessed and all the heights found to be on or below the 25th centile line, though bone age has varied appreciably.

An explanation of these findings was offered with suggestions concerning the early treatment of these children.

A. ROBINSON (and Bridgett O'Connell) introduced by B. M. Laurance. London. 'Parameters for monitoring growth in children with congenital adrenal hyperplasia.' Publications have stressed that adequate control of congenital adrenal hyperplasia with steroids results in normal growth. Of 20 children with 21-hydroxylase deficiency, 18 salt losers and 2 non-salt losers, referred to the Endocrine Clinic at Queen Elizabeth Hospital for Children, London, only 2 were above the median for height and 13 were on or below -2 SD. By plotting height velocity, the information already available on a linear growth chart is amplified. In 8 of the children evidence was presented that height velocity can be a valuable addition to the usual parameters for controlling treatment. The increased velocity of growth in 4 children whose linear height was on or below the third centile increased so much when the steroid dose was reduced that salt losing crises might have been anticipated. Velocity charts were shown to be useful sometimes in prospectively adjusting the dose of cortisone treatment of congenital adrenal hyperplasia in order to achieve optimal growth.

H. VALASSI-ADAM. Hellenic Paediatric Society. 'Immunoglobulin levels in children with thalassaemia.'