visual field defects in young children'. Homonymous hemianopia is a common accompaniment of infantile hemiplegia, affecting approximately 25% of cases, and occurs more frequently when the condition is acquired after birth.

Young children often strongly resist occlusion of one eye, so that testing for uniocular visual field defects is very difficult. Homonymous field defects can, however, be assessed with both eyes open simultaneously and are thus much more readily noted in young children provided that the examiner remembers to employ the necessary simple tests. Visual stimuli such as brightly coloured toys or balls are introduced into the periphery of the field of vision, watching for refixation movements of the eyes on to the target. Such stimuli should be introduced from behind the child's head, either singly into first one and then the other lateral half of the binocular visual field looking for asymmetry of response or simultaneously and repeatedly into both temporal fields seeking for evidence of a constant preference towards one side for refixation. If a target is held across the upper part of both halves of the field of vision, and a second target is introduced from the defective side into the lower half, refixation will occur on to this second target only when it crosses the midline into the seeing half of the visual field.

Homonymous hemianopia is associated with reading difficulties in some cases, especially when the right halves of the fields of vision are affected. It is also known that such difficulties are more commonly found with right hemiplegia than with left hemiplegia even in the absence of visual field defects, so that reading problems may be a direct result of the dominant hemisphere lesion rather than indirect on account of difficulties in scanning along lines of print. Homonymous field defects are a particular hazard to children when learning to cross roads, and may be a bar to issue of a driving licence in adult life. Many affected children nevertheless seem to compensate very adequately for their handicap, either by frequent rapid movements of both eyes towards the blind side, or by adopting an abnormal head posture, turning the face towards the affected side.

P. J. MILLA introduced by R. M. Hardisty. London. 'Control of infection in children with leukaemia and lymphomas'. Patients with leukaemia and lymphomas are particularly susceptible to infection when the disease is in relapse; this frequently compromises the administration of antileukaemic therapy and is now the commonest cause of failure to achieve remission. 51 new patients, referred to The Hospital for Sick Children during 1972 for initial treatment, formed the subject of this study. The most important factor in the pathogenesis of infection was neutropenia: 45 patients were neutropenic during induction therapy and 28 of these developed an infection, 12 having proven septicaemia. 2 patients died from an overwhelming infection before remission could be induced. The time spent with infection was inversely proportional to the level of circulating neutrophils, serious infections being commoner at lower neutrophil levels. Gram-negative organisms cause the majority of severe infections, which may be rapidly fatal unless adequate treatment is instituted promptly.

In an attempt to reduce the incidence of infection, a simple regimen of topical antiseptics and antibiotics combined with oral nonabsorbable antibiotics (group I; 14 patients) was compared with a control group on normal ward nursing (group II; 16 patients). Septicaemia occurred in 14% of patients in group I and 38% in group II. 20% of septicaemias in group II, but none in group I, were due to pseudomonas. Of the total number of days during which patients were neutropenic, 15% were spent with infection in group I, and 32% in Group II. These preventive measures form a useful part of the supportive care of such children and are conducive to a high remission rate.

M. K. STRELLING. Plymouth. 'Should low birthweight infants be given supplements of folic acid?' A recommendation that supplementary folic acid be given to infants below 2·0 kg birthweight is based on evidence of the frequency of deficiency, definition of the infant most at risk, and consideration of the maternal folate state.

Further analysis of a population of special care babies showed that 24% of those under 2·0 kg developed folate deficiency severe enough to cause megaloblastic change on buffy-coat blood films.

A low Hb was an unreliable guide to folate lack; Hb was over 9 g/100 ml in 7 of the 19 affected infants. Red cell folate levels were usually, but not invariably, low at diagnosis.

The risk of megaloblastic change was greatest in infants of lowest birthweight, irrespective of maturity, and was especially high in those who were small-for-dates, including the smaller of twins. Deficiency developed rapidly after birth and was most marked in the second and third months. Evidence for folate depletion other than megaloblastic change was found in two infants with haemolytic anaemia.

The average daily folate intake of megaloblastic infants was probably less than 20 μg but a full haematological response and rise in red cell folate required 120 to 480 μg of intramuscular folic acid a day.

Maternal folate deficiency was associated with low infant red cell folate levels from birth until 5 to 10 weeks, but maternal folic acid treatment was associated with normal infant levels. Tissue folate stores of the smallest infants, even if augmented by supplementary folic acid during pregnancy, would be unlikely to meet their requirements beyond 1 to 2 months.

Infants below 2·0 kg at birth probably need additional folic acid from 2 to 4 months and a daily oral dose of 50 to 100 μg may be appropriate.

J. A. FORD (W. B. McIntosh and M. G. Dunnigan) introduced by I. D. Riley. Glasgow. 'Aetiology of Asian rickets and osteomalacia in the United Kingdom.' Recent evidence suggests that late rickets and osteomalacia in Indian and Pakistani immigrants is due to a high intake of dietary phytate combined with a suboptimal intake of vitamin D. 10 Pakistani subjects with

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.'