under treatment, requiring 350–1150 mg phenylalanine daily to keep their blood levels between 2–8 mg/100 ml.

The treatment of these children was monitored by the use of Guthrie tests collected by the health visitor in the home at the request of the paediatrician, the frequency of testing depending on the efficiency of control of blood levels. In the HPA infants (excluding PKU) when blood phenylalanine levels registered as <2 mg/100 ml on two or more consecutive occasions, a trial period of 2–3 days on a normal diet was given and the Guthrie test repeated.

In distinguishing classical PKU from persistent HPA, phenylalanine load tests may be helpful in doubtful cases. In the normal child, blood and urinary phenylalanine levels revert to normal basal levels 4 hours after a dose of 100 mg/kg phenylalanine. In PKU, infants' peak levels are found between 1 and 4 hours after giving the load, and the basal level is not reached until 24–48 hours. It has been reported that in HPA peak levels are reached as in PKU between 1 and 4 hours, but basal levels are regained within 24 hours.

It is of the utmost importance that those undertaking the management of infants with HPA are aware of the variants that occur within this group. Apart from classical PKU, insufficient data are available to classify these different types. Liver biopsy for the estimation of phenylalanine hydroxylase is the only means of studying them at present and this is not a practical procedure. Nor do we know what is the critical level of blood phenylalanine that results in brain damage. It is our policy at present to treat all infants whose blood phenylalanine is persistently 12 mg/100 ml or higher. Until more is known about these variants, there is a case for centralization, for both Guthrie testing and management of these infants. It is apparent that apart from classical PKU, these patients with HPA variants may at any time become capable of metabolizing phenylalanine in a normal manner and if this is not recognized, there is a

danger of developmental retardation and failure to thrive due to prolonged hypophenylalaninaemia.

Herpes Zoster Serum in Chicken-pox Contacts with Depressed Immunological Responses. A. E. Caunt, E. G. Hall, and D. Mainwaring (*Department of Pathology, Alder Hey Children's Hospital, Liverpool L12 2AP*). Chicken-pox occurring in patients during the course of treatment of malignant disease, including leukaemia, with cytotoxic agents and steroids may be a severe and sometimes fatal disease. The ineffectiveness of human normal immunoglobulin and convalescent chicken-pox immunoglobulin in the treatment of such cases was illustrated. The high and persisting levels of neutralizing antibody to varicella-zoster virus after clinical herpes zoster indicated that post-zoster serum might be more effective. Promising results were reported following the treatment of each of 4 patients with approximately 400 ml of post-zoster serum. 5 further patients who had been exposed to chicken-pox were treated during the incubation period with 200 ml of serum. None of these developed the disease.

The Virulence of Group B Streptococci in the Newborn, and Possible Difficulties in their Identification. K. B. Rogers (*Department of Microbiology, The Children's Hospital, Ladywood Middleway, Birmingham B16 8ET*).

Medical Research Council Trials of Treatment in Neuroblastoma and Nephroblastoma. A. E. Claireaux (*Department of Pathology, The Hospital for Sick Children, Great Ormond Street, London W.C.1*). The current position regarding the arrangements for these trials was reported, and matters which would involve pathologists were discussed.

Familial Gaucher's Disease. D. G. Fagan (*Department of Pathology, The University, Dundee*).