Muscle Haemorrhage in Babies Born by Breech Presentation. Z. Ralis (Children’s Hospital, Western Bank, Sheffield). The study is based on the necropsy dissections of 86 babies and the clinical examination of 50 surviving babies born by breech presentation. A control group comprised 38 newborns who died after vertex presentation.

In most of the breech-born babies haemorrhages were found into the muscles of the lower back and lower extremities, and in some cases there was damage to nerves and joints. The amount of bleeding was often great, particularly in premature infants. In the damaged muscles there were necrotic changes and in older babies fibrosis. The frequency, size, and site of haemorrhage in each muscle was estimated and often formed typical patterns.

It was suggested that: (1) in the most severe cases the amount of damaged muscle tissue could contribute to death due to the crush syndrome, and the amount of haemorrhage to hypovolaemic shock; and (2) the local final consequences of the muscle damage could be responsible for some 'congenital' deformities of lower limbs.

Effects of Damage to the Arterial Wall in Infancy. C. L. Berry (The Hospital for Sick Children, Great Ormond Street, London W.C.1). (To be publish-ed.)

Obesity—or Cushing’s Syndrome. R. W. Logan (Yorkhill Children’s Hospital, Glasgow). A case record of a 9-year-old girl was presented where a diagnosis of Cushing’s syndrome had been made. This was based largely on measurement of plasma ‘cortisol’ concentrations, both as Porter-Silber chromogens and by a fluorimetric technique. The close agreement between the increased levels as measured by both procedures indicated that 11- and 21-hydroxylations were intact. The failure of response to dexamethasone, ACTH, and tetracosactrin together with the macroscopical and histological appearance of the tumour tissue, led to the conclusion that an adrenal cortical carcinoma was responsible for the condition. Measurement of the cortisol secretion rate confirmed this that was increased at 75 mg/day. After removal of the tumour and radiotherapy, the patient is at present in good health.

Neonatal Enteritis Due to a Providencia Organism. H. Kohler and P. Kite (The Maternity Hospital, Hyde Terrace, Leeds 2). Two premature neonates severely ill with diarrhoea were observed on the same ward within a period of 9 days. Providencia A was isolated from the stools of both babies. Antibiotic and supporting treatment were followed by recovery. Providencia—a subgroup allied to the genus Proteus—is rarely identified as a pathogen. Its affiliation to Proteus is established by the formation of phenylpyruvic acid (PPA) from phenylalanine, an essential reaction for the diagnosis of this subgroup. Omission of this test may, in the past, have led to the organism being disregarded and perhaps labelled with the new meaningless name ‘paracolon bacillus’.

Ultramicroscopical Appearance of Pneumocystis carinii. A. E. Claireaux (The Hospital for Sick Children, Great Ormond Street, London W.C.1). Studies were made on ultrathin sections derived from the lungs of two patients with pneumocystic pneumonia. One lung specimen had been obtained by biopsy and then deep frozen for a number of years and the other specimen was obtained at necropsy. Both provided suitable material for electron microscopy. In each, numerous cystic organisms measuring 2-5μ in diameter were found. These had thick walls and contained glycogen particles, mitochondria, and nuclear material. Some of the cysts were very crescentic in outline. No free trophozoite forms were seen.

Observations on the Cytokinetics of Malignant Tumours in Children. W. A. Ahern (General Hospital, Newcastle upon Tyne). With the advent of chemotherapeutic drugs which act on the metabolic processes of DNA synthesis and of mitosis, it is becoming important to gain some knowledge of malignant cell population kinetics. The preliminary results of a continuing study of cell kinetics were presented. Metaphase arrest was accomplished by intravenous colcemid given four hours before biopsy or excision of the tumour (in the latter case a small piece of tumour was removed before excision of the main mass to obviate ischaemic effects). The tissue was processed in the usual way and a count of metaphases was made. On the simplifying assumption that the tumour was growing exponentially, the cell cycle time was calculated from

\[ t_c = \frac{1}{n_2} (t_{ma}/I_{ma}) \]

where \( t_{ma} \) represents the duration of exposure to colcemid and \( I_{ma} \) is a metaphase index.

Cell cycle times were thus shown to average 3 days in a group of mainly embryonic tumours. During the same period serial measurements were made on radiologically visible secondary deposits in lung. These showed a wide scatter of volume-doubling times; the mean value was 20 days. The wide discrepancy between cell cycle times and over-all volume doubling times which has been observed by others in adult human neoplasms and in experimental tumours suggests that only a proportion of the cell population is proliferating at any one time and that there may be a heavy mortality among tumour cells in general.

Immunological Studies in Acute Leukaemia. R. N. P. Sutton (King’s College Hospital, Denmark Hill, London S.E.5). Immunoglobulins (IgG, IgM, and IgA) were estimated in sera from mothers and sibs of children with acute lymphoblastic leukaemia and in sera from appropriate controls. The distribution of IgA titres was significantly lower in the sibs of leukaemic children than in control children, and that of IgM was significantly higher in mothers of leukaemic children than in control mothers. These abnormalities might result from a genetically determined familial immunological abnormality, or might be non-specific changes resulting from virus infection. Their relation to leukaemia was discussed, and the EB virus, already associated with