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 published.)

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 that this 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 2–5 μ 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. Aherne (*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

$$t_c = 1n2(t_{ma})/I_{ma}$$

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

Burkitt's lymphoma and with infectious mononucleosis, was mentioned as a possible candidate virus.

Action of Vincristine Sulphate on Gliomas. G. Pearse and L. P. Lassman (*General Hospital, Newcastle upon Tyne*). Film.

Cell Culture Studies in Cystic Fibrosis. G. B. Reed, A. D. Bain, and W. M. McCrae (*Royal Hospital for Sick Children, Edinburgh*). Fibroblasts cultured from skin biopsies of children with cystic fibrosis and their parents were studied, using a variety of methods including a simple histochemical stain for metachromasia, toluidine blue O. With Eagle's medium supplemented with pooled human serum, the degree of cellular metachromasia (per 1000 cells surveyed) found in parents and controls was similar, but in affected children there was a greater degree of metachromasia. The degree of metachromasia in the cultured cell was compared with the chemical content of acid mucopolysaccharide and with the *in vitro* incorporation of ^{35}S .

Up to the present it has not been possible to relate the degree of metachromasia to cell chemical content nor to the isotope uptake.

Preliminary studies of cultured histiocytes derived from the buffy coat of peripheral blood specimens of controls, parents, and affected patients have been carried out. Despite *in vitro* conditions similar to those employed for fibroblast cultures, it has not been possible to distinguish at present, by metachromatic staining, any significant difference between controls, parents, or affected patients.

Further studies are necessary in order to understand the mechanisms leading to cytoplasmic metachromasia in cultured cells and in order to investigate the relation of this metachromasia to a particular disease, in this case cystic fibrosis of the pancreas.

Wolman's Disease. A. J. Barson (*Department of Pathology, University of Manchester*). A case of Wolman's disease was described in a female infant who died at 7 weeks of age in the Hospital for Sick Children, Toronto. This is a rare familial disorder of lipid metabolism in which there is an accumulation of esterified cholesterol in many abdominal organs, especially the small intestine. Characteristically both adrenal glands are calcified.

This infant presented at 1 week of age with persistent vomiting and abdominal distension. A laparotomy performed at 2 weeks of age failed to show a mechanical obstruction, but the diagnosis was made from tissue biopsied at this time and confirmed later at necropsy.

For the first time in this disease large quantities of ceroid were demonstrated in the liver, spleen, adrenals, lymph nodes, and particularly within the lamina propria of the small intestine. It is postulated that the ceroid resulted from the oxidation of the accumulated cholesterol esters. Because ceroid is so inert, it acts as an impermeable seal progressively impairing intestinal absorption.

Accessory Lungs with Foregut Connexion.

A. A. M. Gibson (*Yorkhill Children's Hospital, Glasgow*). A case was reported of a male baby with bilateral accessory lungs who died at the age of 7 weeks from infection. The accessory lungs were situated symmetrically in the posterior thorax on either side of the midline just above the diaphragm. Each lung was single-lobed and lay in a separate pleural cavity. A fistula resembling a normal main bronchus connected the lungs with the posterior wall of the stomach. The blood supply to the lungs was from an accessory artery arising from the abdominal aorta, and the venous return was to the portal vein. There was also a congenital diaphragmatic hernia on the left side.

Osmiophilic Inclusions in the Human Lung in the Perinatal Period. G. Gandy and W. Jacobson (*Addenbrooke's Hospital, Cambridge*). Published in *Archives of Disease in Childhood*, under the title 'Hyaline Membrane Disease' (1970, 45, 289).

Oxygen-induced Bronchiolar Changes in Infancy.* R. C. Rosan, T. C. Durbridge, M. M. Bieber, and M. C. Cogan (*Department of Pathology, Stanford Medical School, California, U.S.A.*). 80–100% oxygen at atmospheric pressure causes similar sequential changes in the bronchioles of infant humans, guinea-pigs, mice, and young adult rats. The earliest cellular changes are reactive. Within a few days, there is necrosis. Metaplasia appears in 1–2 weeks, and later there is hyperplasia. This chemical bronchiolitis results in irregular aeration, lobular distension, and finally lobular emphysema in survivors of more than 2 weeks. In such survivors, histological and clinical evidence of pulmonary hypertension is the rule. It is feasible to follow the progress of the disease in humans by exfoliative cytology, in which case increasing abnormality of cells is the rule. Increasing difficulty with secretions is also common, due both to loss of an effective sheet of respiratory mucosa and to increased secretion of viscous mucus. The latter can be documented by electrophoresis.

The development of a metaplastic bronchiolar mucosa is a prominent part of late adaptation to chronic oxygen intoxication. These cells are characteristically rich in ribosomes. Even as early as 4 days it can be shown histologically, autoradiographically, and by electron microscopy that there are many ribosomes in surviving mucosal cells. Our data show conclusively that early oxygen toxicity does not decrease net ribosome synthesis in homogenates of whole lung examined on sucrose gradients. On the contrary, it appears that a period of ribosomal synthesis precedes the development of metaplasia by several days. When these 'early' ribosomes are examined by molecular techniques, we find that their protein structure is anomalous and that their response to stimulation by polyuridine and polyadenine is erratically defective. The data are the same for infant humans and guinea-pigs. These features suggest that ribosomes may play a hitherto unsuspected role in the regulation of

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