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 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 laparatomy 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, Standford 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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morphological expression. We have, therefore, an interesting model of injury, the same for man and animals, in which an early lesion in an organelle (ribosome) is associated with a later lesion in tissue (metaplasia) and finally with disruption of organ function (emphysema). The model also suggests a reconsideration of the principles which guide the administration of high tensions of oxygen to newborn infants.

Morphology of Late Stages of Hyaline Membrane Disease. S. Ranström (*Sahlgrenska Hospital, Gothenberg, Sweden*). Beginning about the 5th day there is a proliferation of fibroblasts in the alveolar septa, but with rather scanty formation of collagen fibres. At the same time alveoli begin to disappear, with persistence of at least some of the capillaries. The hyaline material is gradually resorbed, partly in the form of fragmentation, partly as carnification, with formation of new capillaries in the lung tissue. The end result is a severe structural change with disappearance of alveoli, leaving only the alveolar ducts open and separated by broad septa of connective tissue rich in capillaries and poor in collagen. As the bulk of the lung capillaries are no longer in direct contact with the epithelium, a great deal of blood passing through the lung vessels is shunted past the much diminished gas-exchanging surface. These changes in the late stages of the respiratory distress syndrome seem too severe to make normal restoration of lung likely.

Perinatal Pathology of Costochondral Junction in Relation to Pulmonary Hyaline Membranes. B. I. Ivemark and B. Robertson (*Department of Paediatric Pathology, Karolinska Institute, Stockholm, Sweden*). Histological examination of the costochondral junction in a consecutive series (Robertson and Ivemark, 1969) of 100 neonatal necropsy cases showed a statistically significant correlation ($p < 0{\cdot}001$) between a certain type of growth disturbance, characterized by rarefaction of the trabecular pattern in the metaphyses, and the presence of pulmonary hyaline membranes. In the same series, cases with trabecular rarefaction also showed a high incidence of abnormal dental development, with proliferation of the outer enamel epithelium and cord-like persistence of the dental ledge. The cause of this correlation is not clear. Possibly, the trabecular rarefaction and the dental lesions represent a non-specific pattern of growth arrest, secondary to the severe metabolic derangement occurring in fatal cases of the idiopathic respiratory distress syndrome. An alternative explanation of the association between these particular metaphysial, dental, and pulmonary lesions would be a hitherto unrecognized common aetiological mechanism, and this is being studied.

REFERENCE

Robertson, B., and Ivemark, B. (1969). Abnormalities of the costochondral junction in cases of perinatal death, with special reference to hyaline membrane disease. *Acta Pathologica Microbiologica Scandinavica*, **77**, 172.

Dilatation of the Renal Tract in Children with Neurospinal Dysraphism. M. Forbes and A. R. Wilcock (*The Children's Hospital, Sheffield*). Wilcock and Emery (1970) in a recent necropsy study showed the predominance of dilatation lesions of the renal tract in children with meningomyelocele. The incidence of these deformities increased steeply with the age at death, starting at 6% in stillbirths and rising to 35% in children dying at 5 years.

The aetiology of dilated ureters in these children is probably multifactorial, but some workers have suggested that faulty pelvic autonomic innervation may be responsible.

The intrinsic innervation of normal and dilated ureters was studied by the acetylcholine esterase method and formalin-induced catecholamine fluorescent technique. The histological findings of this study were described (Forbes, Underwood, and Emery, 1969, 1970). No qualitative difference was found between normal and dilated ureters in either their adrenergic or cholinergic innervation.

It was concluded that abnormalities of intrinsic innervation did not appear to contribute significantly to dilatation of the ureters in children with meningomyelocele.

REFERENCES

Forbes, M., Underwood, J., and Emery, J. L. (1969). The intrinsic innervation of the dilated ureter in children with meningomyelocele. *Developmental Medicine and Child Neurology*, Suppl., **20**, 49.

——, ——, and —— (1970). The intrinsic nerve plexes of the human ureter. A histochemical study of dilated and normal ureters, with particular reference to children with meningomyeloceles. *British Journal of Urology*, **42**, 158.

Wilcock, A. R., and Emery, J. L. (1970). Deformities of the renal tract in children with meningomyelocele and hydrocephalus, compared with those of children showing no such central nervous system deformities. *British Journal of Urology*, **42**, 152.