REFERENCES

- Bernstein, J., and Meyer, R. (1967). Parenchymal maldevelopment of the kidney. In *Practice of Pediatrics*, vol. III, chapter 26. Ed. by V. C. Kelley. Harper and Row, Hagerstown, Maryland.
- 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.

Fat-laden Macrophages in Cerebrospinal Fluid as an Indication of Brain Damage in Children.

D. C. Chester, J. L. Emery, and S. R. Penny (*Department of Pathology, The Children's Hospital, Western Bank, Sheffield 10*). The occurrence of fat-laden cells in areas of degenerating brain is well known and such cells can escape into the cerebrospinal fluid. The appearance of these cells in stained smears from cerebrospinal fluid was described.

Over a period of one year, all cerebrospinal fluids cultured in the laboratories of the Sheffield Children's Hospital were examined for fat-laden cells. Differential counts were done on positive specimens.

Of 867 fluids examined, fat-laden cells were seen in 336, the majority showing only small numbers of these cells. Correlation of clinical information and laboratory findings suggested the following.

(a) When the cerebrospinal fluid contained less than 10% fat-laden cells, most of the children recovered with no obvious brain damage.

(b) When more than 30% of the cells in the cerebrospinal fluid contained fat droplets, most of the children died and survivors showed evidence of severe brain damage. When intermediate levels of fat-laden macrophages were found, the clinical picture was variable but most of the surviving children showed cerebral symptoms at a later stage.

Examination of cerebrospinal fluid for fat-laden cells is a simple, inexpensive procedure, and may have prognostic significance.

Pseudomonas aeruginosa Bronchopneumonia.

A. J. Barson (*University Department of Pathology, Williamson Building, Brunswick Street, Manchester 13*). Published in *Archives of Disease in Childhood*, under the title 'Fatal *Pseudomonas aeruginosa* Bronchopneumonia in a Children's Hospital' (1971, **46**, 55).

Is Respirator Lung a Distinct Syndrome? D. G. Fagan (*Department of Pathology, The University, Dundee*).

Insulin Secretion and Islet Cell Morphology of Human Fetal Pancreas. L. E. Olding (*University of Uppsala, Dag Hammarskjolds vag. 17 Uppsala, Sweden*).

Paediatric Pathology in the Children's Hospital, Saigon, 1969-70. D. A. Stanley (*Royal Liverpool Children's Hospital, Myrtle Street, Liverpool 7*).

Familial Dyschondroplasia with Visceral Involvement. A. H. Cameron (*Department of Pathology, The Children's Hospital, Ladywood Middleway, Birmingham 16*).

Granulomatous Disease with Acid-fast Bacilli.

H. B. Marsden (*Royal Manchester Children's Hospital, Pendlebury, Manchester M27 1HAO*). The paper described two children of Indian stock born in the United Kingdom, a boy aged 3 years 10 months and his sister aged 2 years 6 months. The boy had a large left tonsillar swelling which did not respond to treatment with PAS and INAH. Generalized lymphadenopathy and necrosis of tibiae, femora, and the right clavicle developed together with pyrexia, high neutrophil leucocytosis, and a rash. Investigations for immunological abnormality and leucocyte function were negative. Gland biopsy from the neck showed fibrosis and plasma cell reaction with small polymorph foci. Culture of the gland yielded a branching acid-fast, as yet unidentified, bacillus sensitive to tetracycline and gentamicin.

The sister showed a similar picture of lymphadenopathy and rash without bone disease. Treatment with tetracycline produced a dramatic improvement in both children although glandular enlargement responding to gentamicin recurred in the boy after three months. Antibody was detected in high concentration to the acid-fast bacillus in both children by FA and agglutination of a formalinized suspension. 15 controls including the parents were negative.

Squamous Epithelium in the Respiratory Tract of Children with Tracheo-oesophageal Fistula, and 'Retention Lung'.

J. L. Emery and A. J. Haddadin (*Department of Pathology, The Children's Hospital Western Bank, Sheffield 10*). Serial blocks from 35 children with tracheo-oesophageal fistula showed that 25 had extensive areas of squamous epithelium in the trachea.

The squamous change occurred principally in the muscular segment of the trachea but in a considerable number of children extended throughout the whole length of the trachea and into the bronchi and around the whole perimeter of the trachea.

A detailed survey of the cause of death in 50 children with tracheo-oesophageal fistula showed that many of the deaths previously ascribed to pneumonia were apparently due to the lack of ciliated epithelium in the bronchial air passages and the retention within the lung of cellular debris and inhaled mucus. This appeared to be the major cause of death in children with isolated tracheo-oesophageal fistula.

The histological appearance of retention lung was discussed and it was pointed out that this change is nonspecific and possibly forms one of the facets of respirator lung. The condition is important to recognize clinically as the most rational treatment would appear to be pulmonary lavage.

Disseminated Ectopic Calcification in a New-born Infant.

F. A. Langley (*Department of Pathology, St. Mary's Hospital for Women and Children, Whitworth Park, Manchester 13*). This infant was born to a mother who was aged 20 and had been suffering from systemic lupus erythematosus for 3 years. Treatment by aspirin and chloroquine was stopped when she became