Paediatric Pathology Society

Proceedings of the Sixteenth Annual Meeting

The Sixteenth Annual Meeting was held in Belfast on 16 and 17 October 1970.

Scientific Communications

Membranoproliferative Glomerulonephritis. E. F. Glasgow (*Department of Pathology, Children's Hospital, Ladywood Middleway, Birmingham B16 8ET*). Membranoproliferative glomerulonephritis forms a small distinctive group among patients with renal disease. This report considers 50 such patients, 26 children and 24 adults. Follow-up indicated a chronic progressive course. All showed characteristic appearances on needle-biopsy. These consisted of enlargement of tufts, variable lobularity, and especially a combination of mesangial expansion coupled with diffuse capillary wall thickening. The mesangial enlargement was due to mesangial cell proliferation and increase in matrix. The latter was fibrillary on light microscopy and had basement membrane-like consistency on electron microscopy. The mesangial material appeared to extend around the lumen of the capillaries between basement membrane and endothelial lining. Thus on electron microscopy the capillary walls were thickened due to subendothelial layers of basement membrane-like material, islands of cytoplasm, and irregular thickening of lamina densa. In early biopsies the capillary wall thickening was not present in all loops. The degrees of proliferation and mesangial increase are variable and where the former is slight the glomeruli may, on light microscopy, give an appearance of epimembranous nephropathy, but the location of the subendothelial aggregations is characteristic in membranoproliferative glomerulonephritis. The degree of lobularity, the tubular and interstitial changes, and the occurrence of 'fibrin cap' lesions were probably dependent on the severity and stage of the disease process, and all the features were well demonstrated on light microscopy of ultra-thin silver-stained sections of epon-embedded biopsy material.

Light Microscopy of Epon-embedded Kidney Biopsies. J. Huber (*Afd. Pathologische Anatomie der Medische Faculteit Rotterdam, P.O. Box 1738, Rotterdam*).

Renal Venous Thrombosis. A. M. MacDonald (*Department of Pathology, Royal Hospital for Sick Children, Yorkhill, Glasgow C.3*). The lesions of renal venous thrombosis were demonstrated and an attempt was made to show histologically the progress of the lesion and the pathological effects. It was emphasized

that renal venous thrombosis is probably always bilateral but the degree of thrombosis may vary in each kidney and the speed and extent of thrombus formation is also variable.

The Nephrotic Syndrome in Infants. F. Alexander (*Royal Belfast Hospital for Sick Children, Falls Road, Belfast BT12 6BE*). (To be published.)

Renal Dysplasia in Neurospinal Dysraphism. M. Forbes and J. L. Emery (*Department of Pathology, The Children's Hospital, Western Bank, Sheffield 10*). Wilcock and Emery (1970), in their necropsy survey of the relationship of renal tract deformity to abnormalities of the central nervous system, found an incidence of 6.9% of cysts and dysplasia in children with meningo-myelocele as compared with only 1.3% in those with no abnormality of the central nervous system. The present study was undertaken to determine if histological examination of the kidneys, using fairly rigid criteria, from a further series of cases of neurospinal dysraphism corroborated the above figures.

A total of 197 necropsy kidneys were examined. 97 were obtained from 50 children with neurospinal dysraphism. 5 of these had primary renal tract deformities such as horseshoe kidney, unilateral agenesis, and ectopia; and 17 had dilatation deformities, such as hydronephrosis and hydroureter. 100 control kidneys were obtained from 50 children with no abnormality of the central nervous system or renal tract on macroscopical examination.

The kidneys were weighed, bisected longitudinally, the reniculi counted, and the whole of the cut surface blocked, sectioned, and examined for evidence of dysplasia. The criteria used were those described by Bernstein and Meyer (1967), only primitive ducts and ductules and/or metaplastic cartilage being taken as evidence of embryonic maldevelopment. 6 of the 50 cases of neurospinal dysraphism fulfilled those criteria, i.e. 12%. There were no cases of dysplasia among the control series.

This histological study confirmed that the incidence of renal dysplasia was higher than previously reported in cases of neurospinal dysraphism. Since only 1/50th of the kidney was sampled, it seems reasonable to suggest that the true incidence is even higher in these children. As dysplastic kidneys have been reported as being more susceptible to pyelonephritis and hypertensive change, early investigation of the renal tract becomes even more important in children with dysraphia.

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