Fused adrenal glands. A. A. M. Gibson. Department of Pathology, Royal Hospital for Sick Children, Glasgow G3 8SJ.

Fusion of two adrenal glands is a rare congenital anomaly. In the past 3 years, however, 10 cases of fused adrenals were observed at necropsy; only 2 other cases were found in the previous 17 years. In this series there was a constant association with spina bifida and meningomyelocele, the number of these cases being only 50% greater in each of the later 3 years compared with the earlier period (a total of 199 cases in 20 years). There was also a striking association with primary renal anomalies, such as unilateral renal agenesis (4 cases) and fused kidneys (3 cases), which occurred in 58% of cases with fused adrenals compared with 5% in control cases of spina bifida and meningomyelocele with normal adrenals and with 1% in all other necropsies. There was no significant difference in the level or the extent of the spinal defect between those with fused and those with normal adrenals, but there was a significant increase in severe kyphoscoliosis in cases with fused adrenals. Fused adrenals in the context of spina bifida are regarded as an indication of the severity of the teratogenic insult. The high incidence of such severe cases in recent years remains unexplained.

The fused glands were joined in the midline, usually behind the aorta, giving a butterfly outline and the histological structure was normal.

Quantitative assessment of histological changes in renal biopsies from children. R. A. Risdon. Department of Morbid Anatomy, The Hospital for Sick Children, Great Ormond Street, London W.C.1.

Evaluation of the histological changes found in kidney biopsies obtained during the investigation of children with renal disease is complicated by the fact that in many instances the abnormalities seen by light microscopy are slight or absent, and may be prone to subjective errors of interpretation.

In this study a number of percutaneous renal biopsies taken from 59 children who presented with a nephrotic syndrome, 24 children with idiopathic recurrent haematuria, and 22 children with renal involvement after Henoch-Schönlein purpura have been submitted to quantitative assessment, including differential counts of the various types of cell composing the renal glomeruli. These counts were found to be particularly valuable in the interpretation of slight degrees of 'proliferative' change in the renal glomeruli, the presence or absence of which may be difficult to establish subjectively.

In the biopsy specimens from children with Henoch-Schönlein nephritis and with recurrent haematuria, proliferative changes, where present, were either 'diffuse', involving all the glomeruli examined, or 'focal', involving only some glomeruli and sparing others. Despite the usual descriptions of a focal glomerulonephritis in these conditions, differential glomerular cell counts indicate that a diffuse proliferative glomerulonephritis is far commoner.

In children with recurrent haematuria renal biopsies showed morphologically normal glomeruli in the

majority of cases. Proliferative changes were found only when significant proteinuria accompanied the haematuria.

Among the children with Henoch-Schönlein nephritis, a very good correlation was shown between the degree of structural damage revealed on renal biopsy and the subsequent clinical course.

Basilar artery thrombosis and giant cell arteritis. H. B. Marsden. Department of Pathology, Royal Manchester Children's Hospital, Pendlebury, Manchester M27 1HA.

A case of basilar artery thrombosis in a girl aged 3 years was reported. She was an only child, the father having died from Hodgkin's disease. There was a short history of slight headache and dizziness for about 2 weeks. On the day of admission to hospital the headache became very severe and there was gradual loss of consciousness over the next few hours. Right-sided hemiplegia was present at first and later the patient became decerebrate. Angiograms were of little help and CSF showed 20 leucocytes/mm³ with protein 20 mg/100 ml. The child died after 2 days, and at necropsy was found to have thickening of the basilar artery over a distance of about 7.5 mm. Microscopically there was acute arteritis of the basilar artery with recent and organized thrombus. Multinucleate giant cells were associated with fragmentation and swelling of the elastic lamina. No evidence of arteritis or other disease was noted elsewhere in the body. Necrotizing arteritis is well recognized in childhood but giant cell arteritis must also be considered in this age group.

Hypothalamic syndrome caused by 'pinealoma' occupying the third ventricle. J. N. Cox. Institut Universitaire de Pathologie, 40 Boulevard de la Cluse, Geneva.

An 8-year-old Tunisian female was transferred to the Paediatric Department, Geneva, in hypoglycaemic coma and died within 24 hours. She had presented symptoms of diabetes insipidus for over a year, but it was only within the month before her transfer that the clinical diagnosis of a cerebral tumour in the region of the third ventricle was made. This was confirmed at necropsy. The tumour filled the third ventricle with seedlings in the dilated lateral ventricles, occupied the hypothalamus, and infiltrated the optic chiasma and lamina terminalis. It extended into the fourth ventricle by way of the cerebral aqueduct, infiltrating the superior and inferior colliculi. The histological picture was that of a 'pinealoma', also known as anisomorphic pinealocytoma (Zülch, 1956) or atypical teratoma (Russell and Rubinstein, 1971; Dayan *et al.*, 1966). Serial sections of the pineal gland showed continuity of the tumour with its anterior portion, the remainder of the gland being normal (Wildi and Frauchiger, 1965). The long clinical evolution and the importance of the tumour mass in the hypothalamus and third ventricle (explaining the hypothalamic syndrome) with little or no involvement of the pineal gland favour the hypothesis of a slow growing tumour originating

outside this body, and therefore this tumour should not be considered a true pinealoma.

REFERENCES

- Dayan, A. D., Marshall, A. H. E., Miller, A. A., Pick, F. J., and Rankin, N. E. (1966). Atypical teratomas of the pineal and hypothalamus. *Journal of Pathology and Bacteriology*, **92**, 1.
- Russell, D. S., and Rubinstein, L. J. (1971). *Pathology of Tumours of the Nervous System*, 3rd ed. Arnold, London.
- Wildi, E., and Frauchiger, E. (1965). Modifications histologiques de l'épiphyse humaine pendant l'enfance, l'âge adulte et le vieillissement. *Progress in Brain Research*, **10**, 218.
- Zülch, K. J. (1956). Biologie und Pathologie der Hirngeschwülste. In *Handbuch der Neurochirurgie*, Vol. III. Ed. by H. Olivecrona and W. Tonnies. Springer, Berlin.

Necrotizing enterocolitis. S. F. Cahalane and B. Moore. The Children's Hospital, Temple Street, Dublin 1.

Necrotizing enterocolitis is a condition predominantly affecting premature newborn infants but occurring occasionally in older children, and is probably identical with ischaemic colitis, a condition of old age. The aetiology is unknown but splanchnic shock is known to cause a similar condition in experimental animals. It has been suggested that infection and immune reaction may play a part.

Clinical manifestations are vomiting followed by abdominal distension and bloody diarrhoea. Mortality is high whether conservative or surgical treatment is adopted. 10 cases from the necropsy files of this hospital were analysed according to clinical and pathological features. 7 were males, and ages varied from a few hours to 3½ years. Only 3 were premature. One or more adverse perinatal factors had occurred in each of the 5 newborn cases. Exchange transfusion, which is known to be implicated in some cases, had been carried out in only one of the series.

The small bowel was involved in all 10 cases, the stomach in 2, the appendix in 1, and various parts of the colon in 5 cases. The bowel was dilated, hyperaemic, and showed a greyish mucosal pseudomembrane on gross inspection. Perforation had occurred in 3 cases.

Microscopically there was mucosal necrosis in all cases and submucosal haemorrhage in 8. Intravascular thrombi in the bowel wall were found in 3 cases and there was an inflammatory exudate in 3. The most interesting finding in other organs was the presence of intra-alveolar haemorrhage.

Only one case had been diagnosed before death. The x-ray picture does not appear to be specific.

Deaths during the first year of life in children born in Malmö, 1966-70. G. Östberg and Ingrid Bjerre. Department of Pathology, Malmö General Hospital, Malmö, Sweden.

The study was undertaken as a preliminary to a clinical follow-up of low birthweight children. 146 children below 2500 g birthweight died (13.2% of all live births in the same weight group) and 84 over 2500 g. The total neonatal mortality in Malmö (260,000 inhabitants, annual birth rate 14/1000) was 0.95%.

The death rate was nearly 100% for infants below 1000 g weight and 28 weeks' gestation, successively

falling in higher 500 g groups. Nearly all the low birthweight children died within 6 days. 45 of 84 with a weight over 2500 g died within 6 days and 27 lived more than one month.

Necropsies were performed on all but one. They were placed in 5 diagnostic groups: malformations 64 cases; 'various' (erythroblastosis, infections) 32 cases; haemorrhage (brain, lungs, adrenals) 31 cases; hyaline membrane disease 61 cases; and immaturity 42 cases. In the last 3 groups the children were mostly grossly immature. Birthweight and gestational age were lowest in the immaturity group (mean values 909 g, 188 days), were intermediate in the group with hyaline membrane disease (1827 g, 222 days), and were highest in the malformation group (2968 g, 266 days). Immature infants died within the first week, those with hyaline membrane disease during days 1, 2, and 3, and malformed children more evenly distributed throughout the year. Maternal complications with uterine bleeding from placenta praevia or ablatio were more common in the groups with haemorrhage, hyaline membrane disease, and immaturity.

Fatal complications of THAM (tris-buffer) administration in the newborn. Helga Rehder and E. Heiming. Division of Paediatric Pathology, University Institute of Pathology, 53 Bonn-Venusberg, West Germany.

In the course of treatment of two newborn girls with respiratory acidosis, THAM (tris-buffer) was inadvertently infused into one of the umbilical arteries. The first child died on the 9th day. Necropsy revealed extensive skin necrosis of the back and left lower extremity. The internal organs showed haemorrhagic necrosis secondary to thrombosis and vascular damage. The other child survived, but cutaneous scars, sciatic nerve palsy, and necrosis of the femoral head on one side persisted. Several months later the infant was operated on for vesico-vaginal fistula and pyonephrosis: one kidney was removed and found to be fibrosed and scarred, with its parenchyma much diminished. Similar scarring was found in other internal organs and this was interpreted as a sequel to haemorrhagic necroses similar to those seen in the first case.

Lymphoreticular aggregates in infant's lungs. J. L. Emery and F. Dinsdale. Department of Pathology, Children's Hospital, Western Bank, Sheffield 10.

Hypoglycaemia in the newborn. K. M. Laurence and A. D. Griffiths. Department of Child Health, Welsh National School of Medicine, Heath Park, Cardiff, and Department of Paediatrics, Nevill Hall Hospital, Abergavenny, Wales.

Standard celloidin-embedded brain sections (cerebrum, midbrain, pons, medulla, cerebellum, and spinal cord) were studied from 17 infants who died after a period of hypoglycaemia (true blood glucose <20 mg/100 ml) during the first 24 hours of life. Nearly all cases were treated with intravenous glucose immediately