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

REFERENCES

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, 1960-70. 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' (erythrobastosis, 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, sciatric 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 cellobioid-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
the low blood glucose was found, and in only one case was untreated hypoglycaemia present for more than 24 hours. 17 cases who had similar birthweights and comparable clinical hypoxia, but normal blood glucose levels, were used as controls. Attention was confined to the neuronal changes.

Histologically, both groups were characterized by the presence of classical ‘ischaemic’ nerve cell change, the cells involved showing either homogenizing cell change or pyknotic nuclei and a shrunken hyperchromatic cytoplasm. The involvement was most marked in the thalamus, Ammon’s horn, dentate nucleus, brainstem nuclei, with more variable changes in the Purkinje cells and the spinal cord and less involvement of the cerebral cortex. The frequency and severity of these changes at the various sites varied slightly but not significantly between the two groups.

It is concluded that exposure to a limited degree of hypoglycaemia in the newborn period, especially if treated within 24 hours, produces no excess CNS damage over and above that produced by coincident hypoxia which was obviously present in all the 34 cases examined. The distribution of neuronal changes conforms almost precisely with the pattern of selectively vulnerable areas which is the hallmark of all hypoxias. The exception is the severity of change found in the brainstem, which is probably a reflection of the higher basal metabolic rate of the brainstem in the newborn.