Paediatric Pathology Society

Proceedings of the Nineteenth Annual Meeting

The Nineteenth Annual Meeting was held in Glasgow on 19 and 20 October 1973, at the Royal Hospital for Sick Children.

Scientific communications

Hydatidiform mole as example of failed pre-natal selection. D. I. Rushton. Department of Pathology, Birmingham Maternity Hospital, Edgbaston, Birmingham B15 2TG.

Placenta membranacea. J. Pryse-Davies. Bernhard Baron Research Laboratories, Queen Charlotte's Maternity Hospital, Goldhawk Road, London W6 0XG.

A case of placenta membranacea was described in a 16-year-old girl who bled in early pregnancy and required up to 10 l. blood by transfusion and aborted at 20 weeks' gestation. In retrospect, the condition could have been diagnosed by ultrasound examination at 11 weeks' menstrual age. (To be published elsewhere.)

Prognosis of germ cell tumours of the ovary in children. F. A. Langley and J. K. Steward. Department of Pathology, St. Mary's Hospital, Whitworth Park, Manchester M13 0JH.

Adrenal changes in stillborn infants. M. J. Becker. Laboratory of Pathological Anatomy, University of Amsterdam, Wilhelmina Gasthuis, Amsterdam, Holland.

From an extensive study of the causes of intrauterine death it evolved that the distribution of fat in the fetal zone of the adrenal cortex could give an indication of the mode of death of the fetus. Three different types of fat distribution were recognized.

Type I, characterized by absence of fat in the fetal zone, correlated with normal placentas and the clinical history of an acute complication. In contrast, type III, characterized by fatty transformation of the complete fetal zone, correlated with placentas with severe circulatory disturbances and a clinical picture of toxæmia, i.e. chronic fetal distress. Type II was an intermediate type, characterized by a fetal zone showing an irregular fatty transformation, with the main concentration around the central vein leaving a clear zone under the definitive cortex. Type II correlated with a subacute mode of death, like infections and blood group incompatibility.

The study showed that the pattern of fat distribution in the fetal zone of the adrenal cortex is indicative of the

mode of death—acute, subacute, or chronic—and therefore may have clinical significance.

Myocardial infarction in the newborn. N. J. Brown. Department of Pathology, Southmead Hospital and Royal Hospital for Sick Children, Bristol B510 5NB.

Myocardial infarction in infancy can be due to anomalous origin of the coronary artery. Much more rare is its occurrence when the origin of the coronary arteries is normal; 2 such cases were presented.

Twelve hours after normal birth the first infant became cyanosed and shocked with cardiac enlargement, baffling ECG, and evidence of nonfunctioning left ventricle. Myocardial infarction was suspected and he died next day. Necropsy revealed a massive recent anterior myocardial infarct. The coronary arteries were macroscopically normal, but histologically there appeared to be an embolus in the anterior descending branch. This could have been paradoxical embolism from the ductus venosus as suggested by Berry (1970).

The second infant developed heart failure when aged 5 hours with a highly abnormal ECG. Myocarditis was suspected and he died next day. Necropsy revealed a large recent anterior myocardial infarct. Histologically there was muscle necrosis but no myocarditis. The coronary arteries were normal apart from mild perivascular fibrosis of doubtful significance. Birth asphyxia, trauma, disseminated intravascular coagulation, arterial calcification, and supravalvular aortic stenosis were all excluded as aetiological factors and no satisfactory explanation of the infarct can be offered.

REFERENCE

Berry, C. L. (1970). Myocardial infarction in a neonate. *British Heart Journal*, **32**, 412.

Polyclonal gammopathy and lymphoproliferation after transfer factor in severe combined immunodeficiency disease. J. Huber, E. W. Gelfand, R. Barnal, M. C. Crookston, and K. H. Shumak. Hospital for Sick Children, 555 University Avenue, Toronto 101, Ontario, Canada.

Transfer factor was administered to a 10-month-old infant with severe combined immunodeficiency disease in an attempt to stimulate cell-mediated immunity. No change in thymus-dependent lymphocyte function was observed. However, 3 weeks after transfer factor there was a marked increase in the leucocyte count with the appearance of mature plasma cells in the peripheral blood and bone marrow. Serum IgM rose to 2000 mg/100 ml; several blood group antibodies, including