Scottish Paediatric Society

At the Annual General Meeting held at the Royal Hospital for Sick Children, Glasgow, on 22 November 1974, Dr. D. M. Douglas was elected President in succession to Professor J. O. Forfar. Dr. E. N. Coleman was re-elected Secretary and Treasurer.

The titles of the clinical demonstrations were as follows.

Family with structural abnormalities of chromosome 1. J. R. Graham Pole and M. A. Ferguson-Smith. Royal Hospital for Sick Children, Glasgow.

Familial vitamin-D resistant rickets. R. P. C. Barclay. Paediatric Division, Stobhill General Hospital, Glasgow.

Aortic stenosis and acute renal failure. M. Blair (introduced). Royal Hospital for Sick Children, Glasgow.

Case of chronic hepatitis. I. D. Hodsman (introduced). Royal Hospital for Sick Children, Glasgow.

Dystrophia myotonica in the newborn. J. B. P. Stephenson and I. C. Ferguson. Royal Hospital for Sick Children, Glasgow.

Scientific communications

Testicular activity in infants with sex chromosome abnormalities. S. G. Ratcliffe and C. S. Corker (introduced). MRC Clinical and Cytogenetics Unit, Western General Hospital, Edinburgh.

Experimental work in the rat and the hamster has shown that sexual differentiation of the male includes a stage when the central nervous system is androgenized and that this is critical for normal male sexual behaviour. Until recently this was thought to occur during the intra-uterine development of the human fetus, but in 1973, Forest, Cathiard, and Bertrand showed a burst of testicular activity in the first 3 months of postnatal life in the human male infant. The present communication confirmed this observation in healthy male infants and its absence in female infants. The investigation had been extended to include 4 infants with sex chromosome abnormalities in view of the known problems of gender identity and impaired sexual functioning in this group of individuals.

REFERENCE

Forest, M. G., Cathiard, A. M., and Bertrand, J. A. (1973). Evidence of testicular activity in early infancy. *Journal of Clinical Endocrinology and Metabolism*, 37, 148.

Tocopherol and anaemia of prematurity. W. R. McWhirter. Paediatric Department, Ninewells Hospital, Dundee.

A double-blind controlled trial of tocopherol administration over a period of one week from the age of 4 weeks was conducted to determine whether it might be

effective in preventing anaemia of prematurity. 37 low birthweight infants were included in the trial, of whom 18 were in the treated group; 19 acted as controls and received a dummy suspension. All infants in the trial received iron and folic acid supplements from the age of 3 weeks. The dosage of tocopherol was 100 mg/day. Measurement of the serum level at the ages of 4 and 5 weeks showed satisfactory absorption of tocopherol. There was also an increase in the Hb levels of the treated group over those of the control group, up to the age of 12 weeks ($P < 0.05$). There was, however, no difference in the reticulocyte counts of the two groups. Hb fell to 7.5 g/dl or less in 9 of the control group but in only one of the treated group ($P < 0.025$). 4 of the control group but none of the treated group required a blood transfusion.

Changes in blood volume associated with exchange transfusion. J. C. Maclaurin, J. B. S. Coulter (introduced), and A. M. Hoby (introduced). Paediatric Department, Glasgow Royal Maternity Hospital, Rottenrow, Glasgow.

Using ^{125}I -labelled albumin with a blood volume computer, estimations of blood volume, plasma volume, and red cell mass had been made on 10 infants before and after exchange transfusion for rhesus isoimmunization. Serum electrolytes and proteins were estimated at the same time and compared with the levels found in donor blood. The results indicated that during exchange transfusion a rise in blood volume occurs, the mean increase being 19 ml/kg body weight ($t=3.06$, $P < 0.01$). This rise is shown to be largely due to an increase of red cell mass ($t=4.17$, $P < 0.005$), the plasma volume remaining unchanged ($t=0.23$, $P > 0.80$). Donor protein levels were higher than those of the infants. During exchange transfusion a slight but significant increase in albumin levels occurred. The sodium levels of ACD blood were markedly above plasma levels which increased during transfusions. It seemed likely that increases in oncotic pressure were largely responsible for the demonstrated rise in blood volume. The deficit of 10–20 ml purposely created during exchange transfusion may well be inadequate.

Cerebrospinal fluid lactate and lactate pyruvate ratios after convulsions and acute hypoxic episodes. H. Simpson, E. L. George (introduced), and A. Habel. Royal Hospital for Sick Children, Edinburgh.

Cerebrospinal fluid lactate, pyruvate, and acid base variables were measured in 52 children after convulsions (febrile convulsions 31; miscellaneous convulsions 21), and in 15 after a severe hypoxic episode (cardiac or

respiratory arrest, or severe circulatory failure). The results were compared with those obtained from 15 controls. A mean lactate concentration of 1.63 ± 0.06 mmol/l, pyruvate 0.12 ± 0.005 mmol/l, and lactate-pyruvate ratio of 13.7 ± 0.5 were found in control patients. Results in the febrile convulsion and miscellaneous convulsion groups did not differ significantly from control values. Within the convulsion groups 15 patients had had repeated convulsions before study and in this group the mean lactate and lactate-pyruvate ratios were significantly higher than values obtained in

controls, namely, mean lactate 2.13 ± 0.20 mmol/l ($0.05 > P > 0.02$), lactate-pyruvate ratio 17.6 ± 2.0 ($0.05 > P > 0.02$). The most striking abnormalities were seen after severe episodes of hypoxia, namely mean lactate 5.75 ± 0.95 mmol/l ($P < 0.01$) and lactate-pyruvate ratio 22.8 ± 2.6 ($P < 0.01$). These results indicated that prolonged and repeated convulsions result in cerebral hypoxia. Severe and often lethal cerebral hypoxia after cardiac or respiratory arrest is usually reflected by an increase in the lactate concentration and lactate-pyruvate ratio in cerebrospinal fluid.