Scottish Paediatric Society

At the Summer Meeting held in the Medico-Chirurgical Hall, Foresterhill, Aberdeen, on 1 June 1973, the President, Professor J. O. Forfar, was in the Chair.

The titles of the clinical demonstrations were as follows:

Sanfilippo syndrome. D. H. Mellor. University Department of Child Health, Foresterhill, Aberdeen.

Kinky hair disease. P. Howard (introduced). Royal Aberdeen Children's Hospital, Foresterhill, Aberdeen.

School phobia. I. Lowit (introduced). Royal Aberdeen Children's Hospital, Foresterhill, Aberdeen.

Seckel's bird-headed dwarfism. P. Duffy (introduced). University Department of Child Health, Foresterhill, Aberdeen.

Calabar swellings. Rowan Adams (introduced). Dermatology Department, Royal Aberdeen Children's Hospital, Foresterhill, Aberdeen.

A paper was given on the Scottish Social Work Act in practice entitled 'Authority for urgent therapy against parental opposition' by Valerie J. Marrian, Perth Royal Infirmary, Perth.

Scientific communications

Effect of correction of primary late metabolic acidosis on weight gain in premature infants. H. E. Churmuckly (introduced) and W. M. McCrae. Elsie Inglis Hospital, Edinburgh.

A proportion of premature infants develop metabolic acidosis at 1 to 3 weeks of age ('primary late metabolic acidosis of prematurity'). This is coincident with the period during which premature infants show retarded initiation of weight gain or, in some cases, slow weight gain. The object of this study had been to observe the effect of correction of late primary metabolic acidosis by the administration of oral sodium bicarbonate on the rate of weight gain. 25 consecutively born premature infants (appropriate for dates) at a maternity hospital from 1 November 1972–28 February 1973 were admitted to the trial. Preliminary evidence suggested that correcting this type of metabolic acidosis reduced the period of retarded initiation of weight gain. The treated infants were at the same centile in weight at their E.D.D. as at birth and, in certain cases, at a higher centile than their birth centile.

Study of enzyme induction in epileptic children. D. C. Davidson, J. A. Ford, and W. B. McIntosh (all introduced). Division of Medical Paediatrics, Stobhill General Hospital, Glasgow.

Many pharmacological agents, including anti-convulsant drugs, can stimulate the activity of hepatic microsomal enzymes. This phenomenon is known as

enzyme induction. The authors had investigated outpatient (25) and institutionalized (18) epileptics on anticonvulsant therapy for evidence of enzyme induction by estimating the urinary excretion of D-glucaric acid and plasma γ -glutamyl-transpeptidase activity. 23 normal children on no medication were also studied. Urinary excretion of D-glucaric acid was significantly increased in both epileptic groups, being greater in the inpatients than in the outpatients. Plasma γ -glutamyl-transpeptidase activity was also increased in both epileptic groups, but no significant correlation was found between this and D-glucaric acid excretion. As D-glucaric acid excretion is an established index of enzyme induction, this investigation suggested that plasma γ -glutamyl-transpeptidase activity is a less reliable measure of enzyme induction in man. 36% of the outpatients and 44% of the inpatients had biochemical rickets, presumably the result of accelerated breakdown of vitamin D by liver enzyme induction. This high incidence of rickets emphasized the need for biochemical screening for evidence of rickets in such patients.

Testicular function tests: an integrated approach. W. Hamilton. Royal Hospital for Sick Children, Glasgow.

The estimation of urinary 17-oxosteroids is a sufficiently imprecise procedure by which to estimate testicular function as to render it almost valueless. An improvement would be to estimate urinary and plasma testosterone levels but even then, were the levels to be low, it would not be possible to say whether there was an inborn error of testosterone synthesis or simply testicular atrophy. Human chorionic gonadotrophin (HCG) has a luteinizing hormone action on the testes. By administering HCG and thereafter fractionating the urinary 17-oxosteroids, it is possible to determine whether testosterone synthesis is normal quantitatively and qualitatively and, if not, where the biosynthetic block lies. The results can be later confirmed by estimating the testicular enzymes from biopsy material. Illustrative examples were given. In a case of bilateral undescended testes the HCG test had indicated testicular atrophy (low basal levels of all precursors of testosterone and no rise following HCG administration); at operation, testes could not be located in the abdomen. In a case of an XY-female, the HCG test had shown an increase in precursors of testosterone without a corresponding rise in testosterone. From biopsy material the defect was shown to be deficiency of 17,20 desmolase. Hence, because of inadequate testosterone, a male fetus had been feminized. In a case of testicular feminization the