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Scottish Paediatric Society

At the Annual General Meeting held at Western General Hospital, Edinburgh, on Friday, 18 November 1977, the President, Dr R. A. Shanks, was in the Chair. Dr E. N. Coleman was re-elected Secretary and Treasurer.

Clinical demonstrations

Ewing's tumour of rib. J. Y. Q. Mok (introduced). Royal Hospital for Sick Children, Edinburgh.

Neonatal obstructive jaundice: two families. L. McNaught (introduced). Western General Hospital, Edinburgh.

Phaeochromocytoma: two cases. P. J. Latham (introduced). Royal Hospital for Sick Children, Edinburgh.

Big kidney and big liver. I. I. Smith (introduced). Western General Hospital, Edinburgh.

Paraneoplastic encephalopathy. J. A. Sills (introduced). Royal Hospital for Sick Children, Edinburgh.

Scientific communications

Endocrine status of children in prolonged remission from malignant disease. D. A. C Barter, W. Hamilton, and M. L. N. Willoughby. Departments of Haematology and Child Health, Royal Hospital for Sick Children, Glasgow.

With the longer survival of children treated for acute lymphoblastic leukaemia (ALL) and the improved results obtained in those with solid tumours, long-term sequelae require consideration. A preliminary study was made of 24 children in long-term remission from ALL (18) or miscellaneous advanced solid tumours (6). In the ALL group were 9 children treated with prophylactic craniospinal irradiation, 6 with cranial irradiation, and 3 with no radiotherapy; all had received chemotherapy. The 6 children in the other group had had at least 2 years' chemotherapy but neither cranial nor craniospinal irradiation.

GH assays were performed after insulin-induced hypoglycaemia at a mean of 24·5 months (median 18 months) after the end of treatment. The overall incidence of partial (17%) and complete (67%) GH deficiency was 83%; only 2 children showed significant growth failure (<3rd centile). GH deficiency correlated with age at diagnosis, all patients in the ALL group aged under 7 years showing deficiency. In the solid tumour group the number of patients was insufficient to show clearly whether here also GH deficiency was a characteristic of the younger patients. All T3, T4, and TSH levels were in the euthyroid range. Gonadotrophin levels (FSH and LH) were normal except for 2 girls who had received abdominal irradiation and in whom grossly raised values suggested ovarian failure. That GH deficiency was discovered in 19 of the 24 patients was disturbing. The mechanism of its production and questions of persistence and clinical significance were receiving further study. A move towards less intensive chemotherapy and less CNS irradiation for some patients with ALL might prove beneficial.

Patterns of drug prescribing for children in hospital. G. W. Rylance, T. A. Moreland (introduced), L. J. Christopher (introduced), and I. H. Stevenson (introduced). Departments of Child Health and Pharmacology and Therapeutics, Ninewells Hospital, Dundee.

The drugs prescribed for children in Tayside teaching hospitals in 1974 and 1975 were surveyed. The data from two paediatric subgroups aged 4 weeks to under 3 years and 3 to 13 years were compared with respect to pharmacological drug classes and individual drug use. The results were also compared with similar data on adult patients. Similar proportions of paediatric (57%) and adult (61%) age groups received drugs, but less than three drugs were prescribed for children (mean 2·5) compared with twice that number for adults. Seven classes of drugs accounted for almost four-fifths of drugs prescribed for children, but only for two-fifths of the total drug use in adults. There was greater use of antihistamine/sedatives, anticonvulsants, decongestants, and mucolytic drugs in children, while the reverse was true for diuretics, potassium chloride, cardioactive agents, hypnotics, and tranquillisers. Antimicrobials accounted for approximately one-third of total drugs used in children and these were used more in the 4 week–3 year age group. One-half of all children receiving drugs had at least one anticonvulsant; the penicillins alone accounted for 65% of all antimicrobials used in children.

Depigmented hair: the earliest sign of tuberous sclerosis. R. C. McWilliam (introduced) and J. B. P. Stephenson. Royal Hospital for Sick Children, Glasgow.

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Early diagnosis of tuberous sclerosis is required to allow appropriate management including precise genetic counselling. Until now the earliest useful external sign has been the ash-leaf shaped depigmented macule or 'white spot'; these pigmented macules may not be visible in the early months of life, particularly in fair-skinned individuals or in the absence of sun tan. Patches of white hair or grey hair may be found in adults and older children with tuberous sclerosis, and it has been speculated that such depigmented hair tufts might have been congenital rather than acquired. We studied 4 children with tuberous sclerosis with one or more patches of depigmented scalp hair and in each case these were noticed by the parents at birth. In one case the finding of a tuft of white hair predated the appearance of white macules by many months. A tuft of white scalp hair is a useful new sign of tuberous sclerosis in the newborn and young child; the hair should be examined as carefully as the skin when early organic seizures are unexplained.

Prostaglandin test for GH deficiency. W. Hamilton. Royal Hospital for Sick Children, Glasgow.

Prostaglandin activates membrane adenyl cyclase to increase cAMP from ATP. GH–RH which causes the release of preformed growth hormone from the pituitary, acts in a similar fashion. It was not unexpected therefore that prostaglandin was shown to increase the circulating levels of growth hormone in sheep. Prostaglandin (PGE_2) IV (30 μg/kg) was given to 33 children with short stature and the circulating levels of GH, TSH, LH, FSH, cortisol, and glucose, measured. Where there was no increase in GH levels after PGE_2, neither was there a positive response to the insulin hypoglycaemia test (IHT). However, 6 patients unresponsive to IHT showed a positive response to PGE_2. It was suggested, therefore, that GH deficiency may be due either to a hypothalamic lesion (hypothalamic unresponsiveness to glucoprivation) or to a lack of pituitary synthesis of GH (both IHT and PGE_2 test negative). PGE_2 does not act through hypoglycaemia, for in all patients the plasma glucose increased 3- to 4-fold over basal levels. Also, PGE_2 caused an increase in circulating TSH levels but had no effect on plasma cortisol, LH, or FSH levels. In selected cases of short stature due to GH deficiency as measured by the IHT, PGE_2 might prove useful in therapy.

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