E. M. Ross. Royal Hospital for Sick Children, Bristol. ‘Convulsive problems in the National Child Development Study’. Some 15,000 children currently resident in England, Scotland, and Wales were born in the week 3–9 March 1958. They have been followed prospectively since birth to the present by their school teachers, health visitors, and school doctors. This information is the basis of the National Child Development Study (Davie, Butler, and Goldstein, 1972).

A sub-study of those who have had convulsive episodes revealed that 21/1000 have had one or more febrile convulsions and a further 4·1/1000 have had undoubted recurrent febrile seizures (Ross, 1973). These are lower figures than were recorded in earlier British studies but accord well with recent Scandinavian experience. The hospital paediatricians who have cared for these children were asked to supply confirmatory data including details of diagnosis, electroencephalographic findings, patterns of drug treatment, aetiological factors, nature of attacks, restrictions on activity, and prognosis.

These children have been shown to be a diverse group of individuals with little in common. The suggestion is made that the generic word ‘epilepsy’ tends to obscure their paediatric needs.

REFERENCES


P. D. Waters and C. G. Newman. Queen Mary’s Hospital, Roehampton. ‘Writing performance at different ages in normal and in limb-handicapped children of normal intelligence’. Written communication is required in basic learning, conceptualization, and in the development of language and other skills. The fullest realization of educational potential is of particular importance to young people with physical handicap.

Writing ability was studied in physically handicapped pupils, for the most part suffering from limb reduction handicap. In the absence of normal standards to serve as controls, these had first to be established. We reported the writing speeds, under set conditions, of some 1250 normal children going on to GCE, O or A levels, aged from 8–18 years. There is a steady increase in average speed from 5 words/min at 8 years to 22/min at 18 years.

531 returns from schools for the physically handicapped received so far show a wide range but a wide agreement between half and one quarter of the ‘normal’ speeds. The deficit increases with age, since the handicapped children tend to lack the increased increase of 1½–2 words/min per year after age 12 years of the ‘normal’ group.

A pilot study of 6 severely handicapped children with good intelligence showed that in their case it was possible to increase their writing performance to normal by the use of special equipment.

While the ‘normal’ group attended different schools, there were no grounds to suppose that the quality of education received by the handicapped pupils was responsible for the poor writing performance. Over 95% of the teachers of the handicapped pupils affirmed that in their view their pupils’ education was being retarded by impaired writing performance. This finding represents an important challenge.

P. S. Harper. University of Wales, Cardiff. ‘Congenital myotonic dystrophy in Britain’. A study had been undertaken of patients in Britain with myotonic dystrophy (dystrophia myotonica) in whom symptoms were present from the neonatal period. 53 cases from 39 sibships were studied. In all cases the mother was the affected parent and no instance of a new mutation or of paternal transmission was found. The grandparental sex ratio was equal. 19 neonatal deaths occurred in the sibships, many with features suggesting they were affected. There were 31 apparently unaffected sibs. Reduced fetal movements and hydramnios were present in more than a third of affected pregnancies, with hypotonia and respiratory problems as the main neonatal presenting features. The results of the study support the hypothesis that a maternal environmental factor is responsible for the congenital form of myotonic dystrophy and that affected individuals are also genetically affected. Neonatal death may be commoner than is at present recognized. Viral studies of serum and cultured placental material have not shown a viral cause for the postulated maternal factor; a transplacental metabolic factor remains the likeliest explanation, but is as yet unproved.

Hilary Scott. Hammersmith Hospital, London. ‘Outcome of severe birth asphyxia’. There have been few reports on the later prognosis for infants who survive very severe birth asphyxia. A follow-up study of such children who were born at Hammersmith Hospital during a 6-year period, 1966–1971, is presented. They were selected either because they were apparently ‘stillborn’ (Apgar 0 at 1 minute) or because spontaneous respirations were not established within 20 minutes of birth, despite intensive resuscitation by intratracheal intubation with intermittent positive pressure ventilation, and in most cases external cardiac massage and the administration of intravenous alkali. 23 of the 48 children so selected survived the neonatal period and have been followed from 2–7 years. Particular attention has been paid to developmental progress, neurological sequelae, and intelligence. 16 of the 23 children have no detectable abnormality, and have a normal intelligence or development quotient. One child is of border-line normal intelligence, and 6 children have cerebral palsy. 6 of the 7 children surviving apparent ‘stillbirth’ are normal. An attempt has been made to analyse the antepartum and intrapartum factors associated with the birth asphyxias, and to correlate them with eventual outcome.

British Paediatric Endocrine Group

J. G. Ratcliffe. Royal Infirmary, Glasgow. ‘Application of radioimmunoassay of serum TSH, T3, and T4 to paediatric thyroid problems’. Specific and
sensitive radioimmunoassays for serum TSH, total T3, and T4 have been developed in recent years. Compared with previous in vitro methods, these offer considerable advantages for the diagnosis of thyroid status in paediatric practice. They require only small sample volumes (100 μl for TSH, 25 μl for T3 and T4), no preliminary extraction, and the thyroid hormone assays can be completed within a working day. In the T4 and T3 assays, bound hormones are displaced from serum binding proteins by 8-anilino-1-naphthalene sulphonic acid. Raised TSH (<20 μU/100 ml) with reduced T4/T3 ratios, are characteristic of childhood hypothyroidism. TSH and T4 assays are valuable in the diagnosis of hypothyroidism in infancy when the accurate assessment of bone age may be difficult. In hyperthyroidism, T4 and T3 levels are raised, but fail to normal within 3 months when the condition is secondary to maternal thyrotoxicosis. In the normal neonate, the TSH level rises rapidly within minutes of birth and is associated with marked increments in T4 and smaller increases in T3 levels during the first week of life. It is not yet clear whether the spurt of TSH release is related to the low T3 levels in cord blood.

B. M. LAURANCE. Queen Elizabeth Hospital for Children, London E.2. ‘TSH stimulation test as an aid to determining thyroid status during thyroxine administration’. 131I uptakes after thyroid stimulating hormone (TSH) injections in 3 children who were receiving thyroxine are reported. The test at age 3 years in a girl who had had thyroxine since the age of 10 days suggested hypothyroidism and in a girl of 13 years 4 months who had had thyroxine since the age of 8 years suggested euthyroidism. The latter remained clinically and biochemically euthyroid 8 months after she had stopped her thyroxine.

This test obviates the need to stop the drug in order to prove the diagnosis and seems to be of value in distinguishing hypothyroidism from euthyroidism. A boy of 7 years 3 months receiving growth hormone which he had had since the age of 4 years and thyroxine which he had had since the age of 7 years showed a low normal uptake of 131I after TSH. Other thyroid function tests done before the boy had started thyroxine suggested a low thyroid reserve and he has benefitted from thyroxine treatment.

The test seems capable of distinguishing primary and secondary hypothyroidism from euthyroidism in a child who is receiving thyroxine, but is less valuable in distinguishing between the primary and secondary forms of this condition.

J. M. PARKIN. Royal Victoria Infirmary, Newcastle. ‘Spontaneous remission in a patient with pseudo-hypoparathyroidism’. The case history of a girl of normal stature and intelligence who presented at the age of 12 years with spontaneous tetany was presented. Biochemical abnormalities included serum calcium of less than 6 mg/100 ml and serum phosphate of over 6 mg/100 ml, and raised alkaline phosphatase. Bone biopsy and parathormone infusion tests supported the diagnosis of target organ failure of response to parathormone. Her symptoms and biochemical abnormalities were controlled with large doses of vitamin D, which after 5 years were gradually withdrawn. The patient has remained symptom free and biochemically normal for 2 years without treatment.

C. G. D. BROOK. Institute of Child Health, London. ‘Growth in children with 45XO Turner’s syndrome.’ Mixed longitudinal growth data from 64 patients with Turner’s syndrome and chromosome constitution 45XO were presented. Mean birth length (47.6 cm, SD 2.8) and mean birthweight (2.8 kg, SD 0.5) were both significantly below expected. Height was increasingly behind that of normal children and there was no evidence of an adolescent growth spurt, even in those children in whom pubic hair appeared (68%). This raises the question of what induces the height spurt in normal girls at puberty, which has been assumed to be due, at least in part, to secretion of adrenal androgens.

The effects of treatment with oestrogens were analysed in 18 patients. Though a small spurt in growth was induced in some patients, especially the younger ones, treatment scarcely affected ultimate stature, since without treatment slow growth continued until well after age 20. There was no evidence that the age at which oestrogens were administered made any difference to the effects on growth (χ² = 13.52, 15 df, NS).

The final heights of patients were compared to those of their parents and a linear regression remarkably similar to that of normal subjects was found. Thus, it was possible to predict the height of a patient from the heights of her parents with an accuracy of ±4.3% in 95% of cases.

D. G. D. BARR. Western General Hospital, Edinburgh. ‘Bone deficiency in Turner’s syndrome measured by metacarpal dimensions’. 184 hand x-rays from 67 individuals with Turner’s syndrome, age range 4 months to 25 years, were measured for metacarpal cortical thickness, metacarpal diameter, medullary width, and bone age.

Results showed that under 11 years of age the children are significantly underheight and retarded in bone age and there is already significant reduction in cortical thickness and medullary diameter. Diameter is significantly more reduced than cortical thickness, suggesting that the major defect at this age is a failure of outer (periosteal) apposition.

Comparison of results under 11 years of age with cases aged 11–25 years shows that with age there is further stunting of linear growth, further retardation of bone age, and significantly greater reduction in cortical thickness with a significant increase in medullary width. Medullary widths approach normal values for age which means that for bones of this size there is relative medullary dilatation. This is consistent with a lack of the steroid-mediated phase of endosteal apposition normally occurring at puberty. In the 11–25 year age group cases on oestrogen therapy show a slight but significant improvement in cortical thickness as compared with untreated cases.