sensitive radioimmunoassays for serum TSH, total T₄, and T₃ 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 44 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 45 XO Turner’s syndrome.’ Mixed longitudinal growth data from 64 patients with Turner’s syndrome and chromosome constitution 45 XO 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 (66%). 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 ($\chi^2 = 13.52$, 15d f, 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 $\pm 4\cdot 3\%$ 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 metacarpal 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.