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 T₂), 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 sulphonate acid. Raised TSH (<20 μU/100 ml) with reduced T₂/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 spur 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 (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 (χ² = 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 ±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 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.
Patients with Turner’s syndrome enter adult life with an overall deficiency of compact bone. Further studies are needed to assess the long-term significance of this and the possible influence on it of oestrogen therapy.

P. H. CHAPMAN. Royal Hospital for Sick Children, Glasgow. ‘Prognostic significance of androgen excration as measured by testicular function test’. In this investigation Leydig cell function was assessed by measuring plasma testosterone, urinary testosterone, and urinary androgens before and during stimulation with human chorionic gonadotrophin (6000 IU/day intramuscularly) for 3 days. From experience it was found that several of the measured androgens correlate well with the phenotype of the patient, and may have prognostic value. Representative cases will be shown to demonstrate these points, which may be stated biochemically thus: 5a-Androstan-3a, 17β-diol (5a-A-diol) is a hepatic metabolite of both testosterone and 5α-dihydrotestosterone and at puberty the urinary excretion of 5α-A-diol increases rapidly, the rise being related to an increased utilization of testosteron by testosterone-dependent tissues. Thus, a good androgenic status means adequate testosterone production in association with a high urinary excretion of 5α-A-diol.

5β-Androstan-3a, 17β-diol (5β A-diol) is associated with the development of the external genitalia. Good genital status is indicated by a high urinary excretion of 5β A-diol. When 5β A-diol is high, even in association with small external genitalia, the indication is that the external genitalia will develop provided there is an adequate supply of testosterone. 5β A-diol then may have prognostic value.

5α Androstan 3βol, 17-one (epiandrosterone) is a 17-oxygenoid metabolite of dehydroepiandrosterone (DHA) having retained the 3β-hydroxyl group. If the general tissue metabolism cannot utilize testosterone, as an alternative DHA becomes the principal anaabolic hormone and the urinary excretion of epiandrosterone then increases. When testosterone is utilized as the anabolic hormone urinary epiandrosterone is low. Thus, a low urinary excretion of epiandrosterone indicates good somatic status.

D. B. GRANT. The Hospital for Sick Children, Great Ormond Street, London. ‘Two cases of microgenitism with rudimentary testes’. Two patients, aged 3 months and 1 month, with the syndrome of rudimentary testes and microgenita (Bergada et al., 1962) were presented. In both patients an extreme degree of microgenita was associated with an empty, hypoplastic scrotum. Both showed a normal male karyotype (XY).

HCG stimulation (5000 units × 3 days) was carried out in one patient. There was no significant change in either plasma testosterone or urinary steroids after HCG. At laparotomy tests could not be identified in either patient. In view of the extreme microgenita it was decided to rear both patients as girls and vulvoplasty with division of the scrotum was carried out by Mr. D. I. Williams.

G. M. KOMROWER. Royal Manchester Children’s Hospital. ‘Precocious puberty in association with pineal seminoma’. Case history of sexual precocity in a male child of 9 years 5 months. Duration of symptoms 4 months. Features: great increase of height and muscle bulk—deepened voice—pubic and facial hair—considerable penile enlargement without corresponding testicular growth. No behavioural or neurological symptoms or signs.

Initial investigations revealed a significant increase of urinary 17-ketosteroids, testosterone, and gonado steroids; a marked rise of plasma testosterone and alkaline phosphatase. Initial bone age was 9½ years but within 2½ months this advanced to 13 years with a growth spurt of 5-6 cm. X-ray of skull showed calcification in the pineal region and detailed studies showed a clearly defined and isolated pineal tumour. Suprarenal and thyroid function was normal but high levels of human growth hormone were determined. Cyproterone acetate therapy was started but after one month the boy complained of headache, and on iophendylate ventriculography there was evidence of encroachment on the aqueduct of Sylvius and the tumour was removed—apparently intact. It was a pinealoma of the malignant seminoma type. After the operation the boy developed a homonymous hemianopia which subsequently has improved.

Measurements of luteinizing hormone and testosterone have been made before and during cyproterone therapy and also after the removal of the tumour. Further studies were initiated to determine whether the pinealoma was acting as an autonomous tumour or whether its effect was produced by hypothalamic disturbance. The boy has had a course of deep x-ray treatment.

D. C. L. SAVAGE. Department of Child Health, Dundee. ‘Excretion of individual adrenocortical steroids in obese children’. (To be published.)

P. H. W. RAYNER and J. H. COURT. Institute of Child Health, Birmingham. ‘Effect of dietary restriction and anorectic drugs on linear growth in childhood obesity’. Simple obesity in childhood is associated with advancement of linear growth. The effect of a reduced calorie intake and anorectic drugs on the growth of obese children has received less attention.

The growth of 26 obese children (17 girls aged 3 years 9 months to 10 years 3 months, and 9 boys aged 3 years 10 months to 12 years 1 month) has been studied over periods of at least one complete year. Growth velocity, expressed as a percentage of the 50th centile velocity for age, has been analysed in terms of sex, weight change, skinfold thickness change, and therapeutic regimen. Three therapeutic regimens were assessed: diet (1000 cal) alone, or diet plus amphetamine derivatives (chlorphentermine, diethyl propion), or diet plus fenfluramine.

REFERENCE