J. F. N. Taylor introduced by Dr. R. E. Bonham Carter (London). 'Prognosis Following Balloon Atrial Septostomy and Subsequent Management of Transposition of the Great Arteries.' In transposition of the great arteries the pulmonary and systemic circulations are independent; but there must be an adequate communication between the two circulations, preferably at atrial level. An adequate atrial septal defect may be created satisfactorily during the first 3 months of life by the technique of balloon atrial septostomy described by Rashkind and Miller (1966). This procedure carries a very much lower mortality in this age group than the Blalock-Hanlon operation.

During the ensuing months following satisfactory palliation there is a continuing declining survival, indicating the need for further treatment. Analysis of the first 150 operations for rearrangement of atrial flow by the Mustard technique performed since February 1965 shows that in simple transposition of the great arteries (i.e. with no additional lesions than an atrial communication) there is a 90% operative survival rate; age range 3 months to 20 years. The survival rate is similar if the infant group (children less than one year old) is separated out.

It is thus suggested that in simple transposition of the great arteries successful palliation by balloon septostomy should be followed by rearrangement of atrial flow at about one year of age (when most of the infants weigh 5–7 kg), as the survival rate from operation is at least as high as the natural survival rate during the ensuing year.

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J. M. H. Buckler (Leeds). 'The Growth Hormone Response to Exercise.' To be published.

G. J. A. I. Snodgrass introduced by Dr. L. Stimmmer (London). 'Intravenous Glucagon as a Test of Growth Hormone (GH) Secretory Capacity.' To be published elsewhere.

P. H. W. Rayner (Birmingham). 'The Use of Clomiphene Citrate to Assess Pituitary Gonadal Function in Males with Delayed Puberty.'

Clomiphene citrate, a non-steroidal oestrogen analogue, is capable of inducing the release of luteinizing hormone (LH) from the pituitary in normal adult males. A preliminary assessment of the value of this response as a test of the pituitary Leydig cells axis has been performed in boys with delayed puberty or in whom abnormal pubertal development was anticipated.

The urinary and plasma testosterone response to the oral administration of clomiphene citrate (100 mg once daily for 6 days) has been measured by a competitive protein binding assay in 12 male subjects (age range 9–22 years). Measurements were continued for 2 days after the drug was stopped. The following diagnostic categories were studied: (1) hypogonadotrophic hypogonadism 2 subjects, (2) isolated pituitary GH deficiency 2 subjects, (3) constitutional short stature with delayed puberty 3 subjects, (4) Prader Willi syndrome 2 subjects, (5) cryptorchidism 2 subjects, and (6) penoscrotal hypospadias 1 subject.

Basal urinary testosterone levels were below the normal pubertal range (0·4–6·0 µg/24 hr) in all except 2 patients. Both hypogonadotrophic patients and both patients with the Prader-Willi syndrome showed no response. The two youngest patients, aged 9 and 11 years, also showed no significant response. The remaining 6 patients showed maximal urinary testosterone levels after clomiphene stimulation ranging from 4·7 to 36·4 µg/24 hr indicating a normal pituitary-Leydig cell axis; in 3 patients the maximum response occurred 2 days after clomiphene was discontinued. There was an increase in the maximum urinary testosterone levels recorded after clomiphene with increasing chronological age and increasing sexual maturity.

These results demonstrate that clomiphene citrate administration may provide a basis for a clinically useful test of pituitary-Leydig cell axis in males with pubertal abnormality. Further studies are required during normal puberty, and on the dose and duration of clomiphene required to obtain an optimum response.

D. C. L. Savage, Constance C. Forsyth, Eileen McCafferty, and Jenny Cameron (Dundee). 'Excretion of Individual 17-oxosteroids and Corticosteroids in the Urine during Childhood and Adolescence.'

There are few reports of fractionation of adrenal metabolites in the urine of children. We have studied the individual 17-oxosteroids and the α-keto metabolites of cortisol and corticosterone during a 24-hour period in 83 normal children and adolescents and 10 adults by paper chromatography using Bush systems. The normal results, which have their own physiological interest, provide a basis on which the effect of various disease states on adrenal metabolism may be compared.

There is an increase as the child grows older in the excretion of the total 17-oxosteroids, the 11-deoxy-17-oxosteroids (dehydroepiandrosterone, androstenedione, androstosterone) and the 11-oxo-17-oxosteroids (11β-hydroxy-androsterone, 11β-hydroxy-aetiocholanolone, 11-oxo-aetiocholanolone, 11-oxo-androstenedione). Dehydroepiandrosterone is detectable by this method at 6 years of age. During childhood the increase in excretion of the total 17-oxosteroids appears to be related to body weight but since below the age of 10 years 60% of the total assay is non-steroid chromagen interpretation of this data with respect to adrenal metabolite excretion must be cautious; furthermore, the 11-deoxy-17-oxosteroids show no such relationship. There is a preferential degradation of the 17-oxosteroids to 5α derivatives initially before puberty is clinically detectable but which continues through puberty and occurs earlier in the girls than the boys.