Annotations

Excessive growth

Tall is beautiful? Tallness is generally considered healthy, handsome, and desirable and it is indeed statistically correlated with intelligence, success, and social class. In the United States bishops are 4.5 cm taller than rural ministers, and presidents of major universities 2.5 cm taller than those of small colleges, in Scotland there is a 5.3 cm difference between upper and lowest social classes.¹ The secular trend to greater height in the population has raised our sights and few parents now worry that their tall normal children will reach a socially unacceptable height. On the other hand, excessive growth at any age requires full assessment.

Clinical assessment²

The further height deviates from the population mean the more likely it is to reflect underlying disease. Clinical assessment of growth must also take account of genetic height potential, growth rate or velocity, and stage of maturation. Both parents determine equally height potential so the midpoint between the 2 parental centiles, the 'mid-parental centile', indicates the mean height of the population of children they might theoretically beget. Deviation from the mid-parental rather than the 50th centile, therefore, indicates deviation from expected height for an individual. The most discriminating indicator of growth is the height velocity, for which 2 accurate measurements separated by a known and reasonably long time period are required. An abnormally increased, or decreased, rate of growth reliably indicates current mischief. Maturation is best estimated from the bone age and pubertal development, and in children with early or delayed puberty the growth rate may more appropriately be compared with the norm for the stage of maturation than for the chronological age. If such assessment suggests that a child is indeed too tall or growing too fast the cause must be sought.

Prenatal growth acceleration^{3 4 5}

For most macrosomic babies no cause is apparent but excessive fetal growth is characteristic of several conditions. Infants born to mothers with poorly controlled diabetes are not only obese but also overgrown and advanced in maturation, probably as a result of excessive prenatal insulin secretion in

response to maternal hyperglycaemia. Insulin may also be a factor in the prenatal overgrowth of the Beckwith-Wiedemann or exomphalos-macroglossia-gigantism syndrome in which hyperinsulinaemic hypoglycaemia is a frequent early problem. In Sotos syndrome of cerebral gigantism birthweight and length are increased (mean values 3.9 kg and 55.2 cm), a rapid growth velocity is maintained for the first 2–3 years of life, and final height is often increased in spite of relatively early puberty. Other syndromes of prenatal overgrowth have been described by Weaver and Marshall. Prenatal hyperthyroidism may also cause accelerated growth and skeletal maturation.

Disorders of the sex chromosomes^{5 6}

Boys with XXY Klinefelter's syndrome are tall: their increase in height is due to excessively long legs, is apparent well before puberty, and their final height is on average 10 cm increased. The characteristic habitus, often in association with small, firm testes, may be the only clue to the diagnosis of this common condition before puberty. A similar phenotype may be seen in boys with mosaic XXY or even normal genotypes. Boys with an XYY genetic construction may have a very similar build. Early diagnosis of these conditions permits anticipation of the many physical, psychological, and emotional problems to which these boys are prone. Girls with an XXX karyotype also tend to be tall.

Obesity

Childhood obesity is associated with increased height and advanced bone age.⁷ Since obese children enter puberty early their final height is not increased and they may simply be expressing optimal growth, but acceleration in height velocity does occur during periods of rapid weight gain.⁸ A useful clinical adage states that the tall obese child is probably normal but the short obese child may well have an underlying disorder.

Skeletal disorders^{5 6}

In Marfan's syndrome the limbs and the extremities are long and thin and throughout childhood there

is a decreased ratio of upper to lower body segments. Increased length may be present at birth. A similar build may be seen in homocystinuria; in the mucosal neuroma syndrome (multiple endocrine neoplasia, type III) in which neural crest abnormalities may include neuromata, medullary carcinoma of the thyroid, and pheochromocytoma; and in congenital contractural arachnodactyly.

Endocrine disorders⁹

The long limbed eunuchoid build of the adult agonadal patient is caused by continued growth of the limbs in the absence of the gonadal hormones that normally fuse the epiphyses.

Severe overgrowth occurs in those endocrine disorders in which there is inappropriate secretion before puberty of hormones acting on the skeleton. Gigantism resulting from increased growth hormone secretion from a pituitary adenoma is extremely rare but proportionate overgrowth without advance of the bone age may be the only early sign. In prepubertal children adrenal androgen excess causes rapid growth and skeletal maturation in association with signs of virilisation: this may result from virilising congenital adrenal hyperplasia, late onset adrenal hyperplasia, or an androgen secreting adrenal tumour. In idiopathic premature adrenarche there is often slight acceleration of growth and advance in the bone age.

Premature testicular androgen production is usually caused by activation of the gonadal axis in true precocious puberty but may rarely result from an autonomous testicular tumour. The source of androgen may be apparent from clinical examination of the testes as they are bilaterally enlarged in true

precocious puberty, asymmetrical in the presence of a testicular tumour, and small when the androgen is of adrenal origin.

Centrally mediated precocious puberty in girls is also associated with acceleration of growth and skeletal maturation. Thyroid hormones in excess may cause accelerated growth and maturation but this is seldom a major feature of thyrotoxicosis in childhood.

Thus growth that is too early, too rapid, or disproportionate must alert paediatric diagnostic antennae.

References

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