to be available, both casualty clinicians and radiologists. It is unlikely that there will ever be enough radiologists to staff accident and emergency departments, particularly at the times when head injuries are most common. The onus must continue to fall on casualty clinicians, who need to be more clearly instructed both in the clinical assessment of head injury and in the indications for and the interpretation of skull radiographs. As might be expected they tend to be over-cautious; in one audit false-positive readings of radiographs were twice as common as false-negative readings.²⁰ It should be remembered, however, that junior medical staff at present rarely receive any formal instruction in the interpretation of skull x-ray films. Head injury is so common and potentially so dangerous that it might be wise to require medical staff in accident departments to participate in some form of self-instruction in how to read skull x-ray films; this could be provided by a tape/slide unit. These doctors must not only learn to recognise skull fractures; they also need to know when films are too poor to be a safe basis for diagnosis and be empowered to request repeat films after consultation with the radiographer. It is essential for the safety of patients that clinicians, radiologists, and radiographers should collaborate in solving this difficult problem.

Unless it becomes possible to provide adequate radiological facilities inevitably many patients will be unnecessarily admitted to hospital. Given reasonable radiography it would be appropriate to admit, from those patients fully conscious and without neurological signs, only those who have been shown to have a fracture or in whom a fracture is suspected and cannot, for one reason or another, be excluded.

As resources become more restricted it becomes all the more important to ensure that they are allocated wisely, to benefit as many patients as possible. It is doubtful whether a significant proportion of resources should be deployed to avoid a very occasional event, because this might raise medicolegal questions, particularly if this entails denying treatment of undoubted

benefit to many other patients.²¹ None the less, it must be concluded that in most of the cases reviewed here, because they had come under scrutiny for medicolegal reasons, there were clear clinical indications that they should have been more carefully managed. Before legal considerations are blamed for forcing doctors to practice defensive medicine we should remember that lawyers are supposed to judge our actions by what is normal careful practice.

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Problems of Childhood

Puberty: precocious and delayed

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Endocrine control of puberty

Gonadotrophins and sex hormones are secreted during infancy and childhood, and a feedback system operates between the hypothalamus, the pituitary, and the gonads. It is, however, set at a low level, so that the small amounts of sex hormones secreted by the gonads are enough to inhibit the secretion of hypothalamic releasing hormone and pituitary gonadotrophins. The onset of puberty is triggered by a decreasing sensitivity of the hypothalamus to sex hormones, so that releasing factor and gonadotrophin secretion rises and leads in turn to higher levels

of sex hormones, which bring about the physical changes of puberty. The precise explanation for the decrease in hypothalamic sensitivity is unknown, but it is influenced by genetic factors and nutritional state and may be related to the attainment of a critical body weight. There is a secular trend in the onset of puberty: the age at the menarche, for instance, has decreased progressively since records were first kept, and small differences exist between racial groups and social classes. These differences, however, are not significant in clinical practice.

Age at onset and events of normal puberty

GIRLS

The onset of puberty occurs about 18 months earlier in girls than in boys. The first event is usually breast development, which occurs at the age of 11, followed by the growth of pubic hair at 11½ and the menarche at 13 to 13½. The pubertal growth

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spurt is usually over before menstruation begins. The time taken to pass through puberty is on average three years. The normal range for the age when puberty starts is wide, so that early or precocious puberty in girls may be defined as the onset of pubertal development before the age of $8\frac{1}{2}$, and puberty may be regarded as delayed if no development has occurred by $13\frac{1}{2}$ or if the menarche has not occurred by $16\frac{1}{2}$. The time taken to pass through puberty is not related to the age at which it begins.

BOYS

Enlargement of the genitalia in boys begins at an average age of $11\frac{1}{2}$, followed shortly afterwards by pubic hair growth. A wide range in the age of starting puberty is also apparent in boys, so that puberty may be defined as precocious when it occurs before the age of 10 and delayed when later than 14. The timing of the growth spurt in relation to other pubertal events is later in boys than girls, and most boys do not complete their growth spurt until development of the genitalia is almost complete.

Stages of pubertal development

It is often valuable to stage the development of puberty so that progress between examinations may be assessed objectively. The following scheme¹ may be used.

BOYS: GENITAL DEVELOPMENT

Stage 1—In preadolescence the testes, scrotum, and penis are of about the same size and proportion as in early childhood.

Stage 2—The scrotum and testes grow, and the skin of the scrotum reddens and changes in texture. There is little or no enlargement of the penis.

Stage 3—The penis begins to grow, but first mainly in length. There is further growth of the testes and scrotum.

Stage 4—The penis continues to grow and begins to broaden, and development of the glans is evident. The testes and scrotum are larger, and the scrotum skin is darkened.

Stage 5—The genitalia are adult in size and shape.

GIRLS: BREAST DEVELOPMENT

Stage 1—In preadolescence only the papillae are raised.

Stage 2—In the breast-bud stage the breast and papilla rise to a small mound. There is enlargement of the areola diameter.

Stage 3—There is further enlargement and elevation of breast and areola, with no separation of their contours.

Stage 4—The areola and papilla project to form a secondary mound above the level of the breast.

Stage 5—In the mature stage only the papilla projects, the areola having receded to the general contour of the breast.

BOTH SEXES: PUBIC HAIR

Stage 1—In preadolescence the vellus over the pubes is not further developed than that over the abdominal wall—that is, there is no pubic hair.

Stage 2—There is a sparse growth of long, slightly pigmented downy hair, straight or slightly curled, chiefly at the base of the penis or along the labia.

Stage 3—The hair is much darker, coarser, and more curled and spreads sparsely over the junction of the pubes.

Stage 4—The hair is adult in type, but the area is considerably smaller than in the adult. There is no spread to the medial surface of the thighs.

Stage 5—The hair is adult in quality and type and distributed in the horizontal (or classically "feminine") pattern. It has spread to the medial surface of the thighs but not up the linea alba or elsewhere above the base of the inverse triangle. (Spread up the linea alba occurs late and is rated as stage 6.¹)

Precocious puberty

The term precocious puberty refers to the physical features of puberty occurring at an inappropriately early age. It may arise in two ways. In true precocious puberty the changes result from a premature increase of pituitary gonadotrophic secretion and a consequent increase in gonadal sex hormones—that is, in the same manner as in normal puberty. In false or pseudo-puberty the changes result from gonadal or adrenal sex hormone secretion independent of pituitary gonadotrophins or from ingesting sex hormones such as oral contraceptives.

GIRLS

True precocious puberty is more common in girls than boys. The changes are identical with those of normal puberty. The onset may be associated with cerebral conditions such as encephalitis or hydrocephalus, but in most cases no definable cause is found and the term idiopathic or constitutional is used. Careful neurological examination should, however, be performed. Growth is accelerated and tallness may be a presenting feature. Intravaginal examination is not usually required, rectal examination being adequate to assess the size of the uterus and exclude an ovarian tumour. X-ray examination of the skull and electroencephalography should be performed. Skeletal maturation (bone age) will be advanced, and serum gonadotrophins (luteinising and follicle-stimulating hormones) and serum oestrogens will be in the pubertal or adult range. Pseudopuberty resulting from oestrogen-secreting ovarian tumours, such as granulosa-cell or adrenal tumours, is rare but should be suspected if a pelvic or abdominal swelling is found or calcification is seen on abdominal radiography. Such tumours may also secrete androgens and lead to virilisation. Pubertal development resulting from oestrogen ingestion is usually transient.

Incomplete forms of pubertal development may occur. Breast enlargement without other features of puberty—precocious thelarche—usually occurs in young girls. Growth and bone age are not advanced and the breast development usually regresses over one or two years. Breast enlargement in both precocious and normal puberty is commonly unilateral at first, and a local lesion need not be suspected.

Precocious adrenarche (pubarche) refers to the precocious growth of pubic and axillary hair without other signs of puberty. It results from the early secretion of adrenal androgens, and normal puberty follows, usually at the normal age.

Vaginal bleeding may sometimes be the first recognised sign of precocious or normal puberty, but in all cases where blood loss has occurred without definite features of puberty a vaginal foreign body such as cotton-wool or toilet tissue or a local cause such as vaginitis or polyps should be excluded by an experienced gynaecologist.

BOYS

The features of true precocious puberty in boys are those of normal puberty with gradual bilateral enlargement of the testes. Rapid growth, acne, or voice change may be the reason for parents seeking medical advice. Bone age is advanced and serum gonadotrophins and androgens are in the pubertal or early adult range. An underlying cerebral abnormality is likely, and neurological investigations such as electroencephalography and pneumoencephalography are indicated, even in the absence of neurological signs.

Pseudopuberty may sometimes arise from androgen-producing testicular tumours—for instance, a Leydig-cell tumour. One testis is usually enlarged, while the other remains infantile. Androgens of adrenal origin from adrenal tumours, or more commonly congenital adrenal hyperplasia, lead to precocious pubertal development. In the latter condition an inborn error of metabolism—the inability to synthesise cortisol—leads to

increased pituitary corticotrophin production and excess adrenal androgen secretion. Both testes remain infantile, although all the other features of normal puberty may be present.

EFFECTS AND MANAGEMENT

Precocious puberty leads to rapid growth, but the advanced rate of skeletal maturation results in early epiphyseal fusion and growth arrest. Eventual adult height may be below normal. Although the onset of menstruation may be early in girls, reproductive life is usually normal and the menopause occurs at the normal age. In both sexes psychological problems may arise because of advanced height and pubertal development. Embarrassment is common, and early menstruation requires careful explanation and a sympathetic, understanding approach by parents and teachers. Motor skills and co-ordination do not advance as rapidly as growth, and precocious children may appear clumsy and awkward. Their mature appearance may lead to an unrealistic estimate of their academic ability at school.

Sexual awareness and sexuality are not usually advanced, although in girls early pregnancy is a risk and may arouse considerable parental anxiety. If the condition is due to a primary gonadal or adrenal tumour or other organic disorder treatment should obviously be directed to the primary condition. Treatment of true or cerebral precocity may be indicated if psychological problems arise or if menstruation proves difficult to manage. Drugs that suppress the secretion of pituitary gonadotrophins or block sex hormone action may be used, such as medroxyprogesterone acetate, cyproterone acetate, tamoxifen citrate, or danazol. They are usually effective in producing some regression of secondary sexual development and in inhibiting menstruation, but their effect on the advance of growth and bone age is disappointing.

Delayed puberty

During adolescence children become increasingly aware of their physical appearance, and failure to conform with changes occurring in their contemporaries may rapidly cause psychological distress. A common case in which medical advice may be sought is that of the teenager who is simply a "late maturer" with constitutional delay in pubertal development. Relatively short stature is often present owing to delay in the pubertal growth spurt. A familial pattern of similar development is usually present. Careful examination often shows early signs of puberty, and reassurance may be given.

Delayed or total pubertal failure may be due to conditions affecting the hypothalamus and pituitary or the gonads. Puberty may also be delayed in chronic conditions, such as chronic renal disease, cystic fibrosis, or diabetes, or by emotional disturbance. Failure of pituitary gonadotrophic secretion may occur as an isolated trophic hormone deficiency, such as hypogonadotrophic hypogonadism. It is more common in boys and is associated with tallness, eunuchoid body proportions, and infantile testes. Inherited forms may occur. When gonadotrophic deficiency occurs as part of panhypopituitarism pubertal failure is associated with short stature due to growth hormone deficiency. The commonest pituitary abnormality in childhood is an isolated growth hormone deficiency, in which, because of delay in maturation, puberty is often delayed despite ultimately normal gonadotrophic secretion. Pituitary damage may result from various causes including tumours such as craniopharyngioma, tuberculous meningitis, and trauma.

Some rare conditions associated with delayed or total pubertal failure are thought to have a hypothalamic basis. They include the Prader-Willi syndrome (obesity, short stature, hypotonia, and mental retardation) and the Laurence-Moon-Biedl syndrome (obesity, short stature, polydactyly, diabetes insipidus, and retinitis pigmentosa).

GONADAL ABNORMALITIES

Girls—Ovarian failure due to ovarian dysgenesis, which occurs in the chromosomal abnormality Turner's syndrome (45 XO), is the commonest cause of pubertal failure in girls. The associated features, such as neck webbing, cubitus valgus, epicanthic folds, coarctation of the aorta, or short stature, may alert attention during childhood. Mosaic forms (XOXX) occur and may lack these characteristic features but amenorrhoea is invariable. Some growth of pubic hair may occur from adrenal androgens but breast development is absent. Nuclear sexing and chromosomal analysis should be performed to confirm the diagnosis. Primary ovarian failure from other causes is rare.

Boys—Bilateral cryptorchidism is not usually associated with delayed puberty, although fertility may be impaired. True bilateral anorchism is rare. One normal testis is enough for normal pubertal development. In Klinefelter's syndrome (47 XXY) secondary sexual development may be deficient. Body habitus is eunuchoid with long legs and the testes are small and soft. Gynaecomastia is common.

OBESE CHILDREN

Obese children, especially boys, are often unjustifiably thought to be prone to delayed puberty and hypogonadism. In simple exogenous obesity growth is accelerated and puberty usually occurs slightly early. Genitalia may appear small due to the deposition of suprapubic fat. When obesity is associated with short stature, however, a pathological condition and the likelihood of delayed puberty should be considered.

INVESTIGATION OF DELAYED PUBERTY

A careful physical and neurological examination should be performed. Skull x-ray examination and more-specialised neuroradiology may be indicated unless the cause is clearly an abnormality of the gonads. Since the pituitary gonadal feedback is operative during childhood raised levels of serum luteinising and follicle-stimulating hormones may be found then in patients with gonadal failure. The ability of the pituitary to secrete these gonadotrophins may be assessed by administering hypothalamic gonadotrophin releasing hormone, which is now available. Gonadal production of sex hormones may be assessed by measuring serum sex hormone levels after stimulation with human chorionic gonadotrophins. Nuclear sexing and chromosomal analysis will exclude sex chromosome abnormalities.

TREATMENT

Simple delay in pubertal development may need no treatment other than reassurance. If eventual pubertal failure can be confirmed during childhood sex hormone replacement therapy may be given at the appropriate age and psychological disturbance minimised. In boys, replacement therapy with testosterone is best given as a depot preparation such as testosterone oenanthate by intramuscular injections every two or three weeks; alternatively methyltestosterone may be given as a daily sublingual tablet. In girls, ethinyloestradiol is given by mouth daily for the first three weeks of each month.

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