Problems of Puberty and Adolescence *

Richard W. B. Ellis, M.D., F.R.C.P. **

The period of transition from childhood to adulthood is one of gre t importance socially and to the individual. It is of particular concern to the paediatrician, the school medical officer, and to those responsible for the health of adolescents employed in industry. Whilst most children reach and pass puberty without any major disturbance, others have emotional or physical difficulties which may lead to conflict with authority, as represented by parents, school or society in general. For this conflict, when it occurs, society is at least partly to blame. Parents may find difficulty in striking a balance between allowing the greater freedom demanded by the adolescent, and keeping a restraining hand on activities which may prove antisocial. At the same time, Western society has tended to make the age of adult responsibility and marriage later and later, whilst the physical changes of puberty, with their associated sexual drive, are actually occurring earlier. In a recent study of sexual maturation of Edinburgh schoolchildren, it was found that the average age of first menstruation was approximately 18 months earlier than at the beginning of the century –13.4 years (Provis and Ellis, 1956) compared with 15 years in an earlier Edinburgh study. The youngest age at which marriage is permissible in Great Britain is 16 years, and the age at which marriage is considered socially and economically desirable is considerably later.

It is understandable that the frustrations and tensions of adolescence have been prolonged and increased. These have resulted in an increase in juvenile delinquency, and particularly in house-breaking, assault, and sexual offences. There has also been a significant increase in the incidence of suicide in adolescence. The present century has witnessed a change from an authoritarian, repressive upbringing of children to one which is much more largely permissive. Whilst society has adjusted more or less successfully to the greater freedom allowed to children, it has been less successful in providing sociallyacceptable outlets for the adolescents who are the product of this earlier freedom.

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** Professor of Child Life and Health, University of Edinburgh.

The period of puberty is marked by three major changes. The first of these consists of a complete readjustment of endocrine balance, during which the gonads, which have been virtually dormant since infancy, produce powerful hormonal stimuli resulting in the appearance of secondary sexual characters.

The second major change is the pubertal spurt of growth. Growth at this time is more rapid than at any time since early infancy. The increased velocity of growth commonly starts with the earliest signs of pubescence and lasts approximately eighteen months. With the occurrence of menstruation in girls and spertmatogenesis in boys, growth in height decelerates and finally ceases with the fusion of the epiphyses. Increase in weight and musculature commonly continues into adult life.

Thirdly, the profound emotional changes linked with the arousal of the sexual drive are ones which are at first canalised and controlled with difficulty.

Disturbance may occur in one or all of these aspects of development. Even where there is no deviation from the normal, the period from the first onset of sexual development to full maturity is likely to last at least three or more years and requires changing disciplines and sympathetic understanding. It should be emphasised, also, that the age at which the successive stages of puberty occur varies very widely in normal individuals. Any large group of 14 year old boys is likely to show a proportion who are still completely undeveloped, a middle group who are showing the early signs of sexual development and may be described as pubescent, and a number who are in a stage of advanced adolescence with the stature and development of young men. Since the educational system is based on age and intelligence rather than physical development, these teen-age groups of widely different maturity present a real problem for the school, and the individuals may prove an embarrassment to their contemporaries.

In considering the readjustment of endocrine balance, a problem which is not yet completely solved is the mechanism by which the changes of puberty are normally initiated. We know that at a particular point in time for each individual the anterior pituitary begins to pour out gonadotropic hormones in increasing amount, and the gonads, which have been inactive and have shown minimal growth since infancy, respond by rapid enlargement and themselves produce active hormones for the first time. It has been suggested that the gonads only respond when they are 'ripe' and ready to do so, but the weight of evidence is in favour of the stimulus coming from the pituitary and in fact it is possible to produce gonadal development artificially by

the injection of gonadotrophic hormones during early childhood. Almost certainly the pituitary itself is stimulated to produce gonadotrophins by some part of the central nervous system, possibly the hypothalamus or tuber cinereum.

In this case we are thrown back on the assumption that the central nervous system itself undergoes a process of ripening. When the necessary stage of maturation is reached, the stimulus to sexual development is relayed from the hypothalamus to the anterior pituitary, and thence to the gonads and adrenal cortex.

In girls, androgens (or male hormones) are responsible for growth of the clitoris, which is the female counterpart of the penis, the labia majora, and the appearance of body hair. Imbalance, or excess of of androgens, may cause acne in girls or excessive hair growth.

The presence of these sex-inappropriate hormones may explain some of the sex-inappropriate behaviour during early puberty observed in both sexes. The pubescent boy may show feminine characteristics, or the girl male ones. These are commonly transient, but may be sufficiently embarrassing to the child or worrying to the parents for medical advice to be sought. In the great majority of cases, hormone treatment is contraindicated, since the disturbances are likely to be corrected when the hormonal drive becomes predominantly sex-appropriate. The administration of testosterone to boys or oestrogens to girls at puberty carries a risk of premature fusion of the epiphyses before the normal growth potential has been fulfilled.

The pubertal readjustment of endocrine balance presents certain features which may be compared to minor manifestations of endocrine disorder. Thus the rapid and ungainly growth of the hands, feet, nose and maxilla which is commonly observed in boys simulates the changes of acromegaly. Pubescent girls frequently show a temporary enlargement of the thyroid gland associated with tachycardia, nervousness and vasovagal instability, comparable to the early manifestations of thyrotoxicosis. Hirsuties and obesity may suggest some degree of hyperadrenalism. In girls, the onset of menstruation may be preceded by discomfort and the menses may be irregular before the adult cycle is properly established. Other patients may complain of symptoms indicating recurrent hypoglycaemia. The voracious appetite which is commonly encountered at puberty is explained by the demands of extremely rapid growth.

Whilst all these manifestations are to be regarded as physiological, they are often sufficient to cause disturbance of physical and emotional wellbeing, if not actual distress. The adolescent cannot be expected to feel or behave either like a well-adjusted child or adult. Those

responsible for the care of adolescents should realise that the various symptoms of which they may complain are very real to the patient though there may be no reason for invalidism or alarm. Rapid growth necessitates not only adequate nutrition but also sufficient rest and sleep.

At the same time it must be remembered that the readjustment of endocrine balance at puberty may be associated with the activation of latent tuberculosis or the appearance of diabetes. Indeed many cases of childhood diabetes will become unstabilised with the onset of puberty, and require much more careful control throughout this phase of development. Cases of rheumatic carditis are also liable to become decompensated during the period of pubertal growth.

Before considering the cause of retarded puberty (or infantilism) and the much rarer cases of precocious puberty, it must be emphasised that there is a very wide variation in the age at which puberty occurs in normal children. Thus in one study of age-onset of menstruation in healthy girls, it was found that the extremes were $9\frac{1}{2}$ and $17\frac{1}{2}$ years, with an average of $13\frac{1}{2}$ years. For convenience, three stages of development may be recognised: non-pubescent in which no signs of puberty are present, pubescent, and adolescent. The pubescent phase is defined in girls as the presence of early breast development before menstruation has occurred. In boys, the pubescent phase is represented by early genital development and/or presence of pubic hair. Adolescence in girls is reached with the onset of mentsruation and in boys with advanced genital development, presence of pubic and axillary hair, and broken voice. The earliest signs of puberty are found to occur on average two years earlier in girls than boys, and the pubertal spurt of growth also occurs correspondingly earlier. Using these maturity gradings, the percentage of boys and girls non-pubescent, pubescent and adolescent in each year of age can be determined. It was found in a study of Edinburgh children that 50 % of girls were non-pubescent and 50% more mature at 11.3 years, and that the median age of adolescence (as indicated by onset of menstruation) was 13.4 years. In the case of boys, the corresponding ages were 13.4 and 15.4 years.

Most of the recent North American studies indicate that puberty occurs appreciably earlier there than in Great Britain. In the tropics, there is considerable evidence that puberty occurs on average later than in temperate climates, although this is contrary to popular belief. It may well be that inadequate nutrition in the tropics is primarily responsible for the late occurrence of puberty, but there is exper-

imental evidence that sexual development of rats can be delayed by keeping animals in a hot moist atmosphere.

Whilst the onset of puberty, like adult stature, is under general genetical control, it can certainly be modified by environmental factors. In addition to the possible influence of climate, inadequate nutrition will tend to delay the onset of puberty and optimum nutrition accelerate it. The earlier onset of puberty at the present day compared with that at the beginning of the century in many Western countries can confidently be attributed to the raised standard of living. Chronic disease of an essential organ, such as chronic renal disease, coeliac disease, congenital heart disease will all tend to delay sexual development, growth and ossification. More general diseases such as congenital syphilis, chronic malaria and Leishmaniasis will have a similar effect.

For sexual development to occur at all it is essential that there should be functional integrity of the endocrine organs concerned. These include not only the gonads but the anterior pituitary and the thyroid. In cretinism, adequate and early replacement therapy with thyroid may be expected to bring about normal sexual development and growth. In primary hypopituitarism the prognosis both as regards growth and maturation is poor, and the majority of such cases show a primary hypogonadism in addition to the pituitary defect which does not respond adequately to the administration of gonadotrophin. Secondary hypopituitarism due to intracranial tumour will be indicated by the presence of papilloedema and temporal hemianopia, and will require surgical treatment. When both gonads are absent, hormone therapy cannot do more than induce the appearance of secondary sexual characters and cause fusion of the epiphyses. It is doubtful if hormone therapy is of any value whatever in the treatment of undescended testicle.

Precocious puberty is very much rarer than infantilism, and may be genetically determined and familial or occur without other evidence of gross endocrine pathology. Tumours of the gonads, floor of the fourth ventricle, pineal body, or adrenal cortex are possible causes which should be excluded. The condition is also occasionally seen in congenital or acquired disease of the central nervous system involving the tuber cinereum or hypothalamus. Adrenal hyperplasia will produce virilism rather than true precocious puberty, and should be treated with cortisone in the first instance.

In conclusion I wish to emphasise that puberty should be regarded as a phase of development having problems, strains and symptoms peculiar to itself; that during puberty latent disease may be exacerbated; and that the age of onset of puberty varies very widely be-

tween normal individuals. Primary endocrine disease is perhaps the least common cause of late onset of puberty, and before embarking on any form of endocrine treatment it is always essential to determine whether the condition is really abnormal, and if so whether it is not accounted for primarly by malnutrition or chronic disease of organs other than the endocrine glands.