Charles G D Brook

Growth Assessment in Childhood and Adolescence

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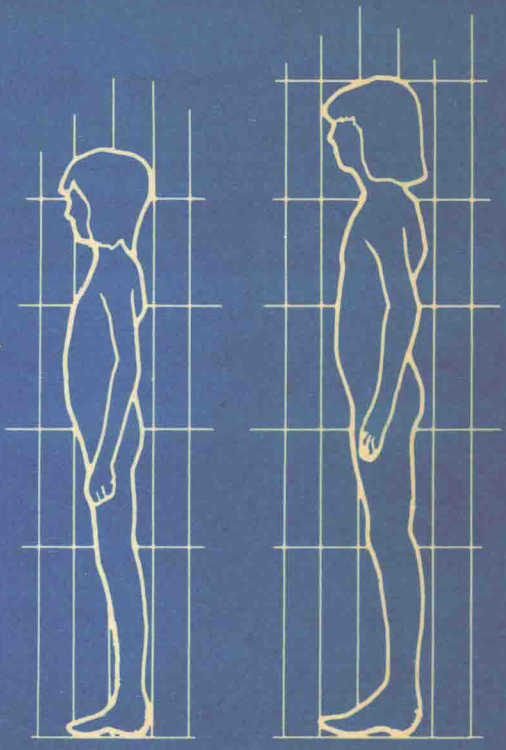

Growth Assessment in Childhood and Adolescence

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Growth Assessment in Childhood and Adolescence

Preface

This book arises from my training with Professor J. M. Tanner, whose contribution to the study of human growth is unique. It is written in the belief that growth assessment makes such a major contribution to clinical paediatric practice that its principles should be widely available and employed by all workers in medicine and allied professions who have the care of the health of children. This book aims to put growth assessment — the basic skill of paediatrics — into the market place.

The philosophy of my paediatric practice owes much to the work and thoughts of Professor Tanner, Professor O.H. Wolff, Dr R. J. K. Brown and Dr Stephen Herman and I thank them for their unwitting help. Miss Lynette Napper started the secretarial grind which underlies the writing of a book: I am grateful for her help in getting things started but the major work has been that of Mrs Sue Shorvon whose unfailing cheerfulness in the face of my demands has made it all fun. I give her my very special thanks.

CHARLES G. D. BROOK
THE MIDDLESEX HOSPITAL 1982

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CHAPTER 1

Prenatal Growth

Growth begins at conception and is at its fastest between conception and birth; because of this, the prenatal part of the growth process is the part most vulnerable to adverse environmental circumstances and the time when such circumstances have the most long-lasting and severe effects. Paradoxically, it is also the period about which we have least knowledge and on which we are least able to exert control. Nevertheless, advances in perinatal morbidity and mortality will only be achieved by a better understanding of what is going on at this time.

A principal reason for our lack of knowledge stems from lack of access to the normal fetus between the time when social abortions are performed and the time when normal babies are born. Advances in ultrasound have made a considerable difference to this situation and ultrasound is the most reliable method by which to bring an infant to delivery appropriately grown and with a normal growth potential following a gestation period of 40 weeks from the date of the last menstrual period. Unfortunately, a single late ultrasonic measurement is probably insufficient to detect poor growth and repeated measurements are needed (Ellis & Bennett 1981).

Measurements of crown-rump length by ultrasound indicate that fetal growth is exponential up to twelve weeks and thereafter linear (Campbell 1976, Birkbeck 1976). Crown-rump and crown-heel lengths increase proportionately in the early weeks although, as fetal age advances, the legs get disproportionately longer. Biparietal and occipitofrontal measurements of skull diameter correlate closely with measurements of length (Birkbeck 1976). Growth in length during the first trimester proceeds at a

sufficiently reliable rate to allow predictions of gestational age from a single determination of length at this time (Robinson 1973). Later, because length measurements become more difficult, skull diameter must be used, but this too provides reliable information on which to base an estimate of gestational age (Campbell & Newman 1971), always assuming that the pregnancy is a normal one.

Comparisons of measurements made by ultrasound (Robinson 1973) with actual measurements made on aborted fetuses (Birkbeck 1976) and with birth length standards (Lubchenko *et al* 1966) show remarkable agreement. Fig. 1.1 shows the growth curve of the human fetus and Fig. 1.2 the rate of growth in length during gestation drawn by me from these data. These are technically known as distance and velocity curves respectively.

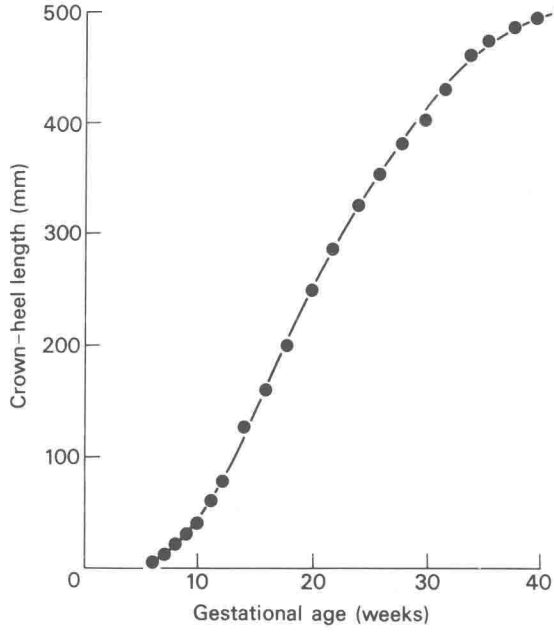

Fig. 1.1 Growth curve of human fetus.

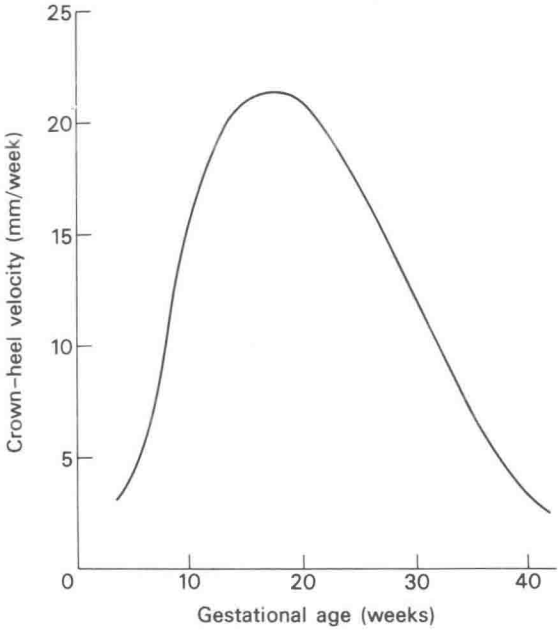

Fig. 1.2 Velocity curve of fetal growth in length.

Fetal weight gain naturally follows growth in length, but the peak velocity of weight gain occurs later than that of height because of the contribution made to weight by the various internal body organs. Of these, the brain is by far the biggest contributor, although its weight does have a significant relationship to other bodily dimensions (Jordaan 1976). Fetal body composition is relevant to the nursing of newborn infants: the increased water content of preterm babies together with their greater surface area: weight ratio and the fact that their skin readily loses water are all highly relevant. In this context, it is important to realise that the baby born at 32 weeks is not at all in the same situation two weeks later at a postmenstrual age of 34 weeks, as the baby just being born at 34 weeks. For this reason measurements of physiological functions made after birth have to take account of both gestational age and extrauterine age.

In spite of the fact that all babies are weighed at birth, it has to be admitted by any practising physician that this is done more or less by convention and because it is easier than measuring length, rather than because it produces useful information. The increased birthweight of the premature infant of the diabetic mother makes all the points necessary to indicate what a useless measurement weight is as an estimate of maturity or of fetal well being. In the newborn nursery day-to-day weight changes are useful because they are sufficient in magnitude to allow longitudinal estimates of growth to be made. I doubt, however, whether many babies would come to serious harm if routine weighing were abandoned in all but sick babies. Measurement of neonatal length is not difficult and an excellent instrument is available (Fig. 1.3, Davies & Holding 1972). Tape measurements of length are not worth making.

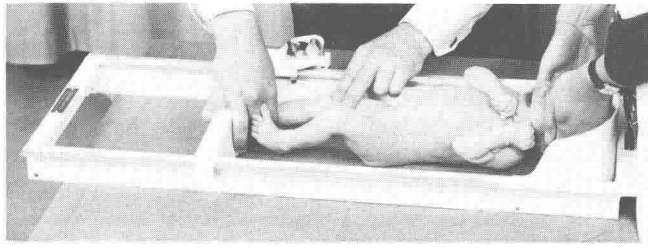

Fig. 1.3 Measuring length of a newborn with a Harpenden neonatometer. (Available from Holtain Ltd., Crosswell, Crymych, Dyfed SA41 3UF, Wales.)

Brain growth is, of course, by far the most important event of prenatal life. The relationship between the increase in head circumference and brain development in the normally growing infant during the first year of postnatal life is well established and probably holds for the intrauterine period as well (Brandt 1976). The major spurt in human brain growth begins in mid-pregnancy and brain cells continue to increase in number into the second postnatal year, myelination continuing the process still further into the third and fourth years (Dobbing 1976).

Even then, functional maturity is far from complete, as the increase in subsequent performance of children with late-treated hypothyroidism shows (Money *et al* 1978). The problem of studying brain growth is that structure and function may be quite separate. While the concept of the vulnerability of the developing brain is well accepted (Dobbing 1981), access to affected human material is severely restricted and extrapolations from animal material may be inappropriate.

Birth is incidental to the growth process as long as environmental circumstances within or without the uterus remain optimal. Because they often do not remain so and because this has effects on the growth of the baby, both birthweight (which is an indirect measure of birth length) and length of gestation are associated with perinatal mortality. To have the highest chance of surviving, an infant should be born between 38 and 41 weeks of gestation, weighing between 3400 and 4200 grams (Goldstein & Peckham 1976). Birth outside these ranges is associated with increased mortality and morbidity and antenatal care is directed at trying to achieve delivery of babies within these ranges as often as possible. The care of premature and small-for-dates infants seeks to optimise the environment and thus to maximise the chances of survival with normal function.

INFLUENCES ON FETAL GROWTH

Apart from sexual differentiation, *the fetal testis* causes a substantial difference between males and females in terms of size for a given postmenstrual age and males are always bigger than females. This applies both to primiparous and multiparous mothers and for infants of different races. From a practical point of view it is therefore important to use sex-specific standards when considering the growth of a newborn infant.

The contribution of *genetics* to fetal growth is probably not all that great. The correlation in birthweight between two successive siblings is in the order of 0.5 but much of

this rather surprisingly low correlation must be due to maternal size. Height and mid-pregnancy weight affect birthweight and should be allowed for when comparing different infants (Tanner & Thomson 1970).

Nutrition before and during pregnancy may influence fetal growth. Clear-cut relationships are difficult to show in societies where obvious malnutrition is uncommon and diet variability considerable, but there are suggestions that minor degrees of subclinical malnutrition, especially of vitamins and trace metals, may contribute to the incidence of congenital malformation. The problem is that such relationships are at the limit of detection and much work needs to be done to establish whether supplementing the diets of pregnant women will improve the outcome of their pregnancies. When nutritional disasters occur, on the other hand, the effect on fetal growth is obvious (Stein & Susser 1975).

Obese women tend to have heavy infants and this is probably due to increased amounts of subcutaneous fat (Whitelaw 1976). Extreme obesity, because it represents a very abnormal situation in the mother, may lead to obstetric hazard and very high increments of maternal weight may lead to a decrease in the weight of the newborn (Curr 1962). Attempts are being made to develop equations for maternal measurements obtained at mid-pregnancy which will identify mothers carrying fetuses at risk from malnutrition. Such predictors of fetal growth should make a substantial improvement on perinatal morbidity (Metcalfe 1978).

Smoking is now well recognised to be an adverse fetal growth factor and the effects of smoking on subsequent learning ability and growth are alarming (Butler & Goldstein 1973). Whether such effects are caused by direct toxicity or by secondary effects on maternal behaviour is not clear, but there is no doubt that cessation of smoking in women of childbearing age would have a very definite beneficial effect on their offspring. Unfortunately for mothers-to-be, the balance of evidence is that drugs and alcohol and excessive ingestion of tea and coffee may also be found to be important when considering the production

of perfect children.

Altitude above sea level may be important to fetal growth, although the influence of other factors, such as the size of the mother, has not yet been adequately explored. Interaction of socioeconomic factors with malnutrition, smoking, obesity and so on is exceedingly complex but what *is* certain is that the effects of poor environmental circumstances are probably more important to fetal growth than most of the influences which come within the remit of doctors to alter. This is unfortunately true of much of the promotion of child health: housing, nutrition and education are much more important than traditional medical care.

Hormonal factors are the principal modulators of the growth process and endogenous hormone production, placental, and maternal hormones are all important. The interchange of the feto-placental unit with the maternal endocrine system is of considerable relevance here and is poorly understood. At present we are at the stage of describing changing concentrations of hormones in infants, but as changes in receptors and in the catabolism of hormones are certainly equally important to mechanisms of hormone action, we are only at a qualitative and not very informed stage of understanding fetal endocrinology.

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CHAPTER 2

Growth in Childhood

The growth curve of the human child has never been better exemplified than by the growth of the child of Count Philippe de Montbeillard between the years 1759 and 1777 (Tanner 1962). It is reproduced in Fig. 2.1. In the upper part of the figure, the heights of the child are plotted at six-monthly intervals against his chronological age, giving a distance curve; in the lower part increments over each six months are converted into annual rates and are plotted against chronological age to make a velocity curve. Growth in infancy is rapid but rapidly decelerating so that it describes an arc with a relatively short radius. The deceleration seen immediately after birth is a continuation of that seen in the fetal period and illustrated in Fig. 1.2 (p.3).

Between two and three years of age, the deceleration changes in magnitude and the years of middle childhood are years of steady and gradually decelerating growth, although there is a small increase in growth rate (possibly associated with the production of adrenal androgens (adrenarche)), and a discontinuation of deceleration, which has been called the mid-childhood growth spurt. This short-lived increase in growth rate, which can be clearly seen in Fig. 2.1, occurs mainly in boys and is the subject of study at present (Tanner & Cameron 1980). Overall, however, during the prepubertal years the growth curve assumes the shape of an arc with a relatively long radius. The slow deceleration is brought to an end by the adolescent growth spurt which rises to a peak and then disappears when epiphyseal fusion has been completed. The adolescent growth spurt adds approximately 20 cm to that of a boy, so the height at which puberty begins is important to the determination of final height.

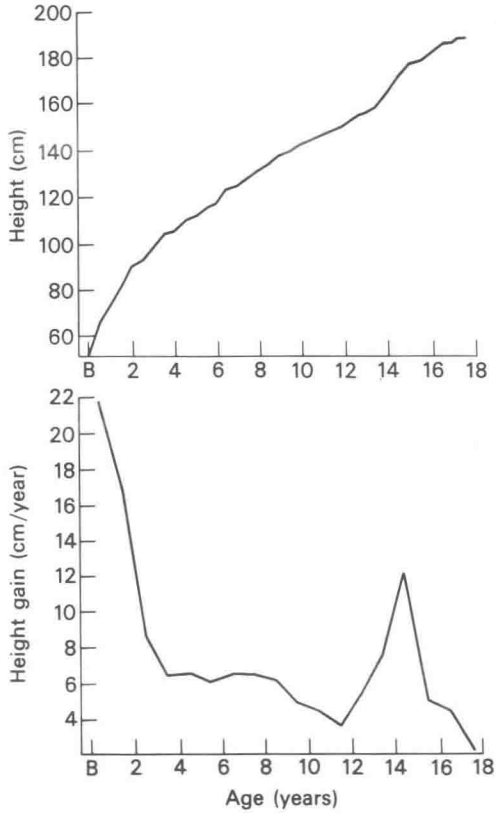

Fig. 2.1 Growth of the son of Count Philippe de Montbeillard, 1759–77. In the upper panel the height is plotted at six monthly intervals. In the lower panel the height increments over each successive six months are converted to annual rates and plotted against age. Reproduced with permission.

The growth of all children follows a curve similar to the one illustrated and, apart from deviations which are due to pathological processes, the spread in the height of growing children results from differences in genetic endowment of height and to differences in timing. Obviously the spread of normality increases as children get bigger, that is, they fan out, and those destined to be taller grow more quickly

and vice versa. There is some rearrangement of ranking of birth lengths, but at every age the standard deviation of height distribution represents a relatively fixed percentage of the mean. The shapes of individual curves are remarkably uniform and the regularity of growth can be demonstrated by the ability to derive an equation containing only five parameters which describes growth in stature from age two until maturity (Preece & Baines 1978).

Boys grow a little faster than girls before puberty and gain about 1.5 cm in prepubertal growth but the major difference between the growth of boys and girls results from the difference in timing of the adolescent growth spurt. Fig. 2.2 is taken from the work of Largo and colleagues (1978), who analysed the adolescent growth

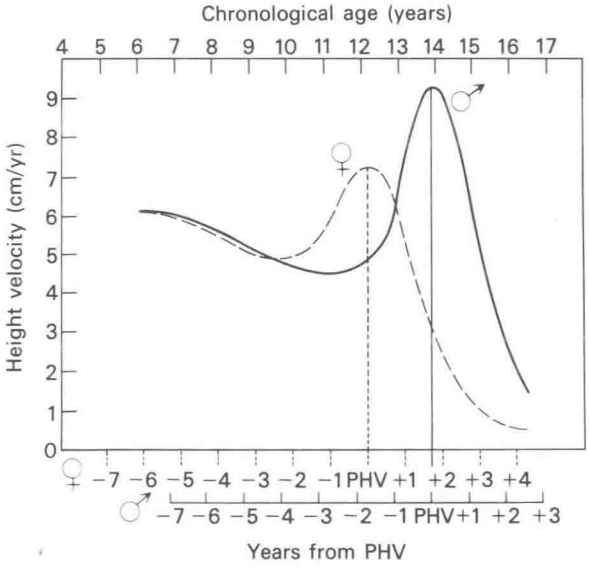

Fig. 2.2 The adolescent growth spurt in girls and boys. Peak height velocities (PHV) occurred at age 12 in girls and 14 in boys and the scale at the base of the figure indicates velocity measurements for years before and after PHV. (Data from Largo *et al* 1978). Reproduced with permission.