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The Physical Status: The Use and Interpretation of Anthropometry in Adolescence

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Catch-Up Growth during Adolescence

Key Words
Catch-up growth
Adolescence

Abstract
Various chronic diseases and malnutrition cause growth failure in childhood and adolescence; following recovery, catch-up growth may occur. The extent to which growth failure can be compensated for depends on the timing, severity and duration of the growth failure, as well as on the aetiology and pathogenesis of the disease restricting growth and development. There are three types of catch-up growth. In type 1, when growth restriction ceases, growth occurs to such an extent that the height deficit is rapidly eliminated. Once the original growth curve is attained, growth proceeds normally. In type 2, when growth restriction ceases, there is a delay in growth and somatic development. However, growth continues for longer than usual, compensating for the growth arrest. Type 3 is a mixture of types 1 and 2, and all three types may be complete or incomplete. Two factors make it difficult to record catch-up growth during adolescence: the large variability in timing, expression and duration of pubertal growth and somatic development, and the relationship between the measurement error and the increase in growth observed within a defined time period. To avoid data collection and analysis problems, prospective and long-term study design should be considered. Ideally, data collection should be started in the prepubertal period and continue until final adult height is reached. High technical standards and well-trained personnel should be used. A variety of parameters should be assessed to obtain different dimensions of the growth process and pubertal development. Data analysis should be related not only to chronological age, but also to biologically oriented time-scales, such as bone age, pubertal staging and age at peak height velocity.

Introduction

In the first clinical report on catch-up growth, Bauer [1] described growth delay and subsequent catch-up growth in 19 children with nephrotic syndrome. The mean of their heights for age and sex was exactly average for the population when the disease began; when it ended the mean height had sunk to the 12th centile. During recovery, growth returned not only to normal, but was accelerated. After 12 months, the mean height reached the 25th centile and after 18 months the 30th centile. In 1963, the term catch-up growth was introduced into the medical literature by Prader et al. [2]. Prader's group described 5 children whose growth had been slowed by illness or starvation. When growth restriction stopped, there was a rapid phase of growth until the children reached their pre-illness growth curve.

Since 1963, much research on catch-up growth has been published [3–6]. While catch-up growth in infancy and childhood is a well defined phenomenon, during ado-

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lescence it is less well-defined. During adolescence, compensatory growth processes are difficult to record because of the variability in timing, duration and expression of growth and somatic development and the relationship between measurement errors and the increments observed within a defined time period.

Definition

A prerequisite for catch-up growth is canalization [7]. Normal growth is a fairly regular process which canalizes the growth of an organism within certain limits. Regularity in growth is the result of a dynamic and complex system of control which makes growing organisms return to their growth paths after deviation.

In clinical terms, canalization means that the individual growth curve follows the centile curves of growth charts. The degree of canalization varies among the various growth parameters. Anthropometric measurements of the head follow the centile channels most closely. Height and bone age do so moderately, while weight, circumferences of the extremities and skinfold thickness often cross the centile curves. The degree of canalization of growth parameters also depends on chronological age. While height in the prepubertal period displays a high degree of canalization, growth staying within centile channels during adolescence is exceptional. In most children, growth does not remain in the centile channels because of different biological timetables and expression of pubertal growth. Some children have their adolescent growth spurts early and some late and these phase differences lead to the centile curves being crossed. The lack of canalization during adolescence makes the recording of catch-up growth more difficult than in the prepubertal period.

Taking into account this limitation of growth canalization during adolescence, the phenomenon of catch-up growth may still be recognizable. Growth has a tendency to return to its original channel if it has been pushed off its intrinsically determined course. Three types of catch-up growth can be distinguished [4].

Type 1

When growth restriction ceases, growth velocity increases to such an extent that the height deficit is quickly eliminated. Velocity may increase up to 4 times the mean velocity of the corresponding chronological age. When the original curve is attained, growth proceeds normally (fig. 1). The various growth parameters are not equally affected by growth restriction (table 1).

Table 1. Growth failure and catch-up growth: sensitivity of growth parameters

	Growth restriction	Catch-up growth
Skinfold thickness	++++	++
Arm/calf circumference	+++	++
Weight	+++	++++
Height	++	++
Bone age	++	+
Head circumference	+	+++

+ Indicates the degree to which growth parameters are affected by growth restriction and catch-up growth.

Type 1 catch-up growth is common in infancy and childhood but data prior to the onset of growth restriction are needed for its identification. The phenomenon is usually reported as a case study and seldom in group statistics. In adolescence, type 1 catch-up growth can only be recognized in individuals followed carefully from childhood to adulthood, due to the variable course of growth and somatic development.

Type 2

When growth restriction ceases, there is a delay in growth and somatic development. Growth continues for longer than usual (fig. 1), so that ultimately the growth arrest is compensated. Type 2 catch-up growth has only a small or no increase of velocity compared with the mean velocity for chronological age. It occurs in children with a constitutional growth delay. Late maturers show a retardation in height growth, bone age and pubertal development which is compensated for by a longer than normal growth period.

Type 3

Type 3 catch-up growth is a mixture of types 1 and 2. When growth restriction ceases, there is an increase in growth velocity as well as a delay and prolongation of growth.

Comparison of Types

Each type of catch-up growth may be complete or incomplete, completely or partially restoring the stature to normal (fig. 2). In adolescence, complete catch-up is defined by reaching final adult height within the range of target height. During adolescence, catch-up growth types 2 and 3 are the most common, though in many studies it is

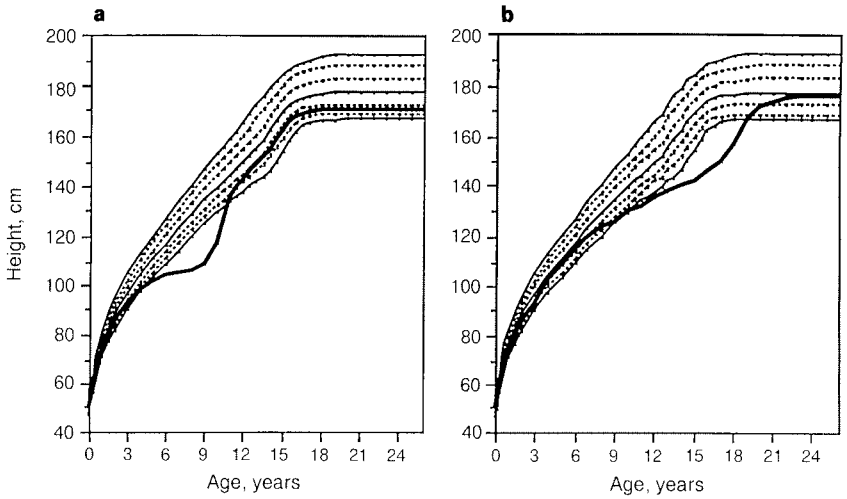

Fig. 1. Catch-up growth: type 1 (**a**) and type 2 (**b**).

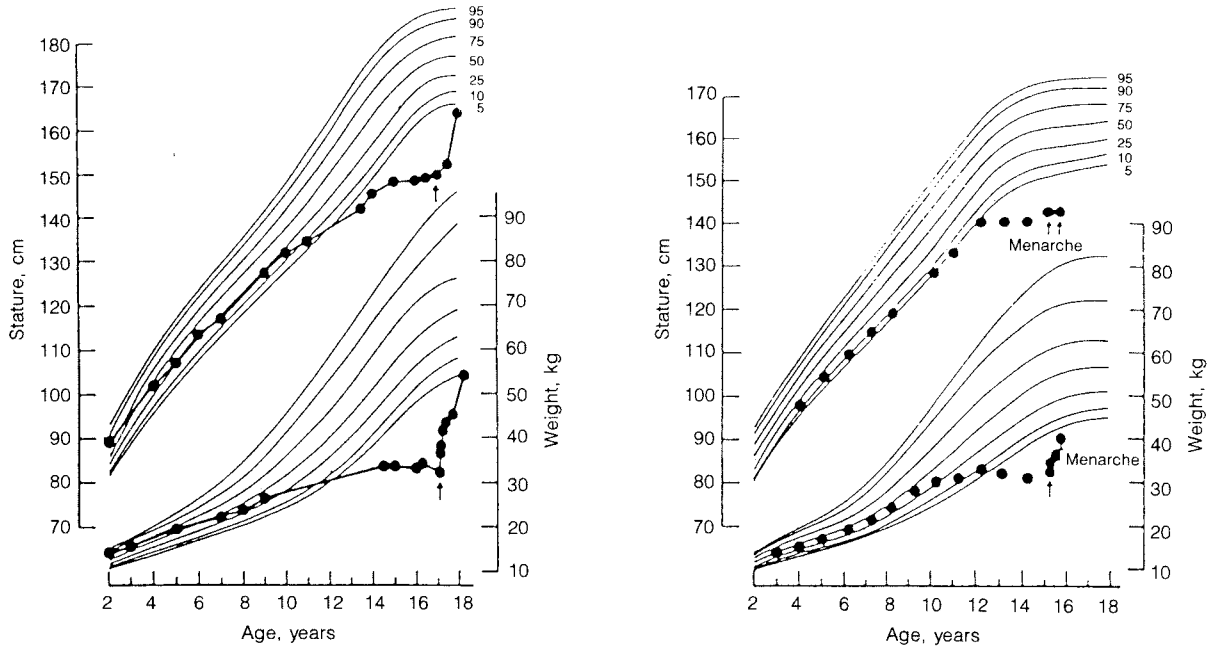

Fig. 2. Growth failure in 2 patients as a result of self-imposed restriction of caloric intake arising from a fear of becoming obese. Incomplete catch-up growth is observed after adequate nutritional intake was implemented. Note that inadequate weight gain preceded diminished linear growth and adequate weight gain preceded an acceleration of linear growth. The arrows indicate initiation of nutritional therapy [with permission, 8].

not known whether catch-up growth is complete or incomplete because of variations in pubertal growth and incomplete data collection.

Mechanism

Various theories have been put forward to explain the mechanism of catch-up growth in humans [9]. Animal experiments first shed light on the regulation of growth [10]. An excellent review on recent research was published by Mosier [11].

Peripheral tissues have some capacity for growth regulation. Growth hormone and insulin-like growth factor-1 (IGF-1) receptors are present in cartilage. IGF-1 is found in various tissues, suggesting that it is synthesized locally. Binding proteins for growth hormone and for IGF-1 may modulate the effects of these factors.

Central control of catch-up growth has been extensively studied in rats. The secretion of growth hormone increases with an increase in integrated growth hormone secretion when fasted rats are refed [12]. After neonatal irradiation of a narrow mid-line zone of the head, the sensor for body size appears to be reset. Among the brain structures investigated, the dorsomedial hypothalamic nucleus is the likeliest candidate for the site of a true body size set point mechanism. When irradiated rats were fasted and refed they caught up, but only to the preset stunted curve [13]. In contrast to non-irradiated rats there is no increase in growth hormone during the catch-up phase in the irradiated rats, implying that catch-up growth is not dependent on growth hormone secretion alone. The experiments indicate that head irradiation is reset to a smaller body size without major alterations of metabolism or nutrition. This is illustrated by the observation that rats with lesions of the dorsomedial hypothalamic nucleus develop a normal food and water intake when referred to body weight and normal plasma glucose, insulin, free fatty acids, glycerol, corticosterone, somatomedin activity and protein parameters.

Pharmacological agents can also influence the set point; body size is reset after perinatal administration of thyroxine or androgen in rats, and permanent resetting to a lower body weight may occur after treatment with the environmental toxin 2,3,4,7-tetrachlorodibenzo-*p*-dioxin in rats and guinea pigs. Transient changes in the set point may result from treatment with certain neuropharmacological agents. Fenfluramine, an anorectic, may reduce body weight in rats, independently of its effect of reducing food intake.

Functional factors seem to influence the set point as well. The body weight of ground squirrels varies over an annual cycle; efforts to change the cyclic body weight set points have evoked metabolic weight defence. Dormice, with shorter body weight cycles, undergo cyclic changes in feeding efficiency corresponding to the weight cycle. Body weight set point changes in female rats have been shown to correlate with the oestrus cycle and body size is influenced by the in utero location of the fetus.

Causes of Growth Failure and Subsequent Catch-Up Growth

Catch-Up Growth as a Normal Phenomenon

Short-term changes in growth leading to minor deviations from the centile channels are part of normal development. Temporary delays and subsequent catch-up growth due to seasonal changes and episodes of ordinary acute illness occur at all ages.

Disease-Related Causes

Catch-up growth has been described following many diseases, particularly chronic conditions [14]. Some of the most recent articles reporting on disease-related catch-up growth are listed in table 2. References to previous studies can be found in these articles. The intensity and duration of growth failure and of the subsequent catch-up growth varies between diseases, and even within the same clinical condition, catch-up growth may differ between patients.

Malnutrition-Related Catch-Up Growth

The impact of the various forms of malnutrition on growth in infancy and childhood has been studied in detail [27–29]. Much less is known about the effect of malnutrition in infancy and childhood on adolescent growth. There is strong evidence that malnutrition causes a delay in the onset of pubertal growth and development [30–35]. However, the extent to which timing, duration, and severity of food deprivation during infancy, childhood and adolescence may lead to compromised adult stature is largely unknown. Sparse evidence suggests that severe caloric deprivation early in life, probably within the first 5 years of age, is needed for permanent stunting.

Adolescence, as a period of accelerated growth, may be particularly influenced by nutritional factors. Other studies [32, 36] indicate that puberty may also serve as a catch-up period when childhood growth losses are re-

Table 2. Disease-related growth failure and subsequent catch-up growth (recent references)

Disease	Authors	Year	Ref.
Gastrointestinal disease			
Crohn's disease	Booth	1991	15
Cystic fibrosis	Booth	1991	15
Coeliac disease	Booth	1991	15
Renal disease			
Chronic renal failure	Fennell et al.	1990	16
Renal transplantation	Schaefer et al.	1990	17
Respiratory disease			
Asthma	Balfour-Lynn	1987	18
Liver disease			
Transplantation	Chin et al.	1991	19
Heart disease	Poskitt	1987	20
Endocrinological disorders			
Growth hormone deficiency	Vanderschueren-Lodeweyckx et al.	1987	21
Constitutional delay of growth and adolescence	Crowne et al.	1991	22
Connective tissue			
Juvenile chronic arthritis	Allen et al.	1991	23
Metabolism			
Bartter syndrome	Proesmans et al.	1988	24
Genetics			
Turner's syndrome	Bergmann et al.	1990	25
Childhood cancer	Sklar	1991	26

Table 3. Growth parameters used to assess growth failure and catch-up growth

Height
Predicted adult height
Target height
Predicted adult height/target height
Weight
Weight for height
Skinfold thickness
Arm/calf circumference
Bone age
Bone age/chronological age
Cortical thickness
Secondary sexual characteristics
Menarche
Testicular size

Parameters may be related to chronological age, bone age, pubertal staging and age of peak height velocity.

Table 4. Standard deviation of the error of measurement [38]

	Girls	Boys
Weight, g	440	430
Supine length, mm	4.0	5.2
Standing height, mm	5.0	4.7
Head circumference, mm	2.4	2.3
Upper arm circumference, mm	3.7	4.5
Calf circumference, mm	3.9	4.0
Triceps skinfold thickness, mm	1.4	1.1
Subscapular skinfold thickness, mm	0.6	0.6

gained. Estimates of body fat as well as direct anthropometry indicate that the onset of puberty is not size-related during chronic childhood malnutrition.

Measuring Growth

A variety of reasons make the assessment of growth difficult during adolescence. The period of pubertal growth is associated with major changes in all components of body structure and composition. Pubertal growth accounts for approximately 16% of adult height, and body weight almost doubles. In addition to these changes, the measurement error and the variability of growth and somatic development during puberty make the recording of a temporary phenomenon, such as catch-up growth, a difficult undertaking.

Various parameters of growth and pubertal development have been used to record growth failure and catch-up growth (table 3). Of these parameters, height is possibly the best indicator of the final outcome following growth failure and catch-up growth.

Anthropometric Measurements

High technical standards and well-trained investigators are of paramount importance in studies on pubertal growth and development [8, 37]. The study of catch-up growth requires the calculation of velocities or increments of growth parameters. Velocities and increments are based on the difference between two distance measurements taken within a certain time interval. In the calculation of velocities, the measurement error is of major significance (table 4). The standard deviations of the error of measurement not only reflect the various sources of error caused by measuring procedures, but also the individual variability, such as diurnal and seasonal oscillations and spontaneous short-term changes of velocity [39].

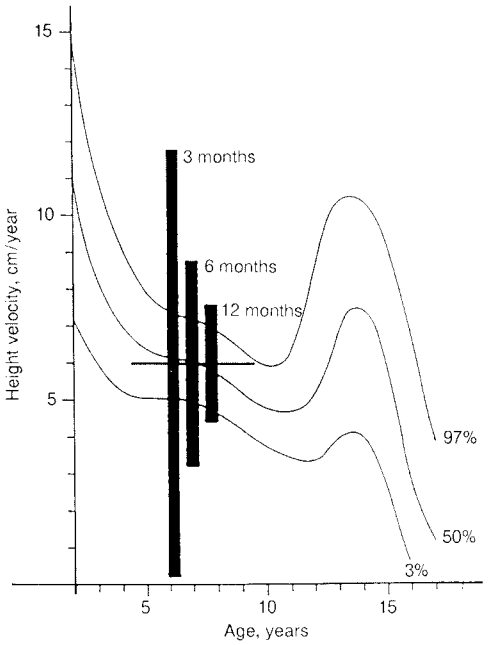

Fig. 3. 95% confidence intervals for true height velocity in a boy with an observed yearly increment of 6 cm, calculated from height increments in observation periods of 3, 6 and 12 months. The error of measurement is 4.7 mm.

Assuming that the errors of two measurements are statistically independent, the standard deviation of the error of increment can be obtained by multiplying the standard deviation of the error of measurement by the square root of 2 (=1.4) and dividing the result by the time interval (years) between the two measurements. The significance of the measurement error and of the time interval for the error of increment is demonstrated in fig. 3.

In the First Zurich Longitudinal Study the standard deviation of the error of standing height measurement was 4.7 mm for boys. With an observed yearly increment of 6 cm the error of increment is as follows: for an observation period of 3 months the 95% confidence interval is 12 cm, approximately twice as large as the normal range of height velocity at this age. For time intervals of 6 and 12 months the corresponding confidence intervals are 6 and 3 cm, respectively. It should be emphasized that the anthropometrist in the First Zurich Longitudinal Study was an extremely reliable person with 20 years' experience in anthropometry. Measurement errors taken by a less experienced person may easily exceed 5 mm.

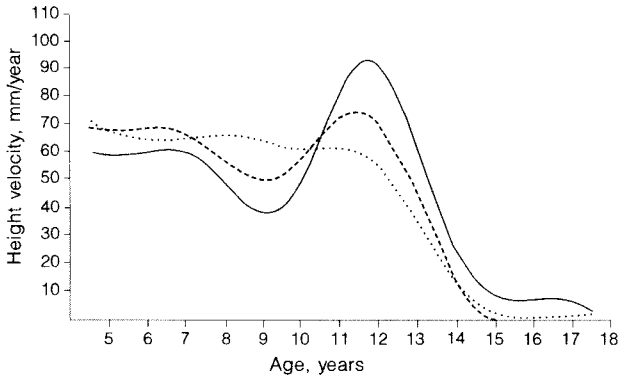

Fig. 4. Individual height velocity curves of 3 girls with variable expression of the adolescent growth spurt [40].

The timing, expression and duration of growth parameters are variable during adolescence [40]. The pubertal growth spurt in boys may occur as early as 12 years or as late as 15.8 years while in girls the spurt is even more variable. The duration of the adolescent growth spurt ranges from 1.3 to 8.2 years in girls and from 2.6 to 6.5 years in boys. About one third of girls display a peak increase in height of less than 2 cm, some of them have virtually no growth spurt (fig. 4).

Weight, weight for height and skinfold thickness are also variable during adolescence and depend on the timing of pubertal development. The increase of subcutaneous fat tissue and muscle tissue is closely related to the pubertal growth spurt [41].

Weight and height do not show a steady relationship because of the dramatic changes in body composition. Using standards which disregard pubertal development, an individual may be judged too light or too heavy for their height simply by virtue of pubertal status. At the age of 12 or 13 years, girls in an advanced stage of pubertal development are about 10 kg heavier than girls of the same height in the first stages of pubertal development.

Predicted Adult Height and Target Height

Predicted adult height, target height and the difference between the two are often used in studies of catch-up growth during the pubertal period. Predicted adult height is calculated either by the method of Bayley and Pinneau [42] or that of Tanner et al. [43]. The former method is based on actual height and bone age; the latter also takes midparent height and menarcheal age into account. The genetic growth potential of a child remains unknown, though the range of heights within which 95% of the off-

springs of particular parents will fall can be estimated. The following formula for target height recommended by Tanner et al. [44] is used in most studies:

target height + (father's height + mother's height)/2
+ 6.5 cm for boys, −6.5 cm for girls.

The 95% confidence interval for the expected adult stature is target height ± 8.5 cm, independent of sex. In most cases the wide range in target height of 17 cm does not allow a decisive judgement on the completeness of catch-up growth to be made. In addition, this formula does not take into account the secular trend and environmental influences which may change from generation to generation and, therefore, affect midparent height [45].

Bone Age

All children finally reach skeletal maturity regardless of the magnitude of the growth failure and of the incompleteness of catch-up growth. In most studies, bone age is determined either according to the TW2 method [43] or according to the Greulich and Pyle method [46]. In most studies intra- and interobserver reliability are not reported, though the expertise of the investigator may greatly influence the results.

Increments in bone age and the ratio of bone age to chronological age play a role in estimating whether catch-up growth will be complete or incomplete. It is usually assumed that bone age advances by 12 months every year, though the variability of yearly bone age increments limits the usefulness of this assumption. At the age of 10–11 years, for example, bone age may increase by 5 or 28 months and between 12 and 13 years bone age may advance by 2 or 36 months.

Pubertal Development

Delay in growth is often associated with a delay in pubertal development. Thus, assessing the pubertal status is an important part of studies on catch-up growth in adolescence [47, 48]. In most studies, pubertal development is evaluated according to Tanner [41].

Pubertal development is extremely variable. In girls and boys, pubertal stages display a variability of 4.5–6.5 years. For example, genital stage G2 may be present at 9–10 years or not before 14–15 years of age. The interval between pubertal stages also varies considerably. In girls and boys the standard deviations of the intervals vary between 0.8 and 1.4 years. One girl may proceed from breast stage B2 to menarche within 10 months, while in another girl menarche may occur more than 4 years after the initiation of breast development.

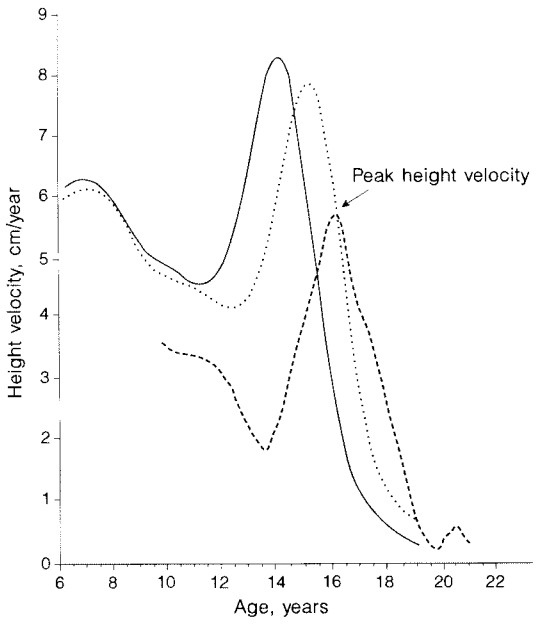

Fig. 5. Synchronized mean height velocity curves of 15 pubertal boys with chronic renal failure (----) and healthy children maturing at average (——) and late age (·····) [with permission, 17].

Biologically Based Time Scales

Since growth and somatic development are highly variable with respect to chronological age, efforts have been made to find more biologically based time-scales. Height growth was based on a bone age timetable. Growth parameters such as height, weight or skinfold thickness were related to pubertal staging and menarche [32]. An approach which took the characteristics of pubertal growth into account best was time-scaling, based on the age of peak height velocity [17] (fig. 5).

Conclusion

If all the methodological aspects are taken into account, a prospective and long-term study design is essential when investigating catch-up growth during adolescence. Data collection should be started in the prepubertal period and continued until final adult height is reached.

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