

tients who have Bloom syndrome suffer growth impairment and immunodeficiency and are at increased risk of developing many different malignancies, including leukemias, lymphomas, and solid tumors. Children born with Werner syndrome present with premature aging, atherosclerosis, diabetes, cataracts, and increased risk of soft-tissue sarcomas. Rothmund-Thomson syndrome involves a pathognomonic rash called poikiloderma and predisposes to osteosarcoma. In ataxia-telangiectasia, caused by mutations in the *ATM* gene, patients develop truncal

ataxia in early childhood and oculocutaneous telangiectasias by age 5. They are immunodeficient and are at increased risk for leukemias, lymphomas, and solid tumors as well as central nervous system tumors. Carriers are at increased risk for breast cancer.

Comment: Another cancer worth mentioning in the context of family risk is Wilms tumor (WT). Although most cases are sporadic, some are associated with genetic syndromes (Beckwith-Wiedemann, WAGR [Wilms tumor, aniridia, genitourinary anomalies, devel-

opmental delay], Denys-Drash, congenital aniridia), and approximately 5% are familial. Sporadic and syndromic WT is associated with inactivating mutations of the *WT1* gene, a tumor suppressor, on chromosome 11. Familial WT, which follows a pattern of autosomal dominant inheritance with incomplete penetrance, has been associated with mutations on chromosomes 17 (*FWT1*) and 19 (*FWT2*).

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In Brief

Growth

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Author Disclosure

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Growth during childhood is tightly regulated and depends on the proper functioning of multiple systems. The process is affected by perinatal factors, including maternal nutrition and uterine size; genetic growth potential inherited from parents; and nutrition throughout childhood. Growth also is affected by the interplay of multiple hormones, including growth hormone (GH), thyroid hormone, insulin, and sex hormones, all of which have varying influence at different stages of growth. Despite all these factors, final adult height generally is restricted throughout the human population to a rela-

tively narrow range: 95% of Americans have a final adult height that falls within only a 6% to 8% variation from the mean. Because final adult height and growth are so well regulated, a deviation from normal expected patterns of growth often can be the first indication of an underlying disorder. Carefully documented growth charts, therefore, can serve as powerful tools for monitoring the overall health and well-being of patients. Key to diagnosing abnormal growth is an understanding of normal growth, which can be classified into four primary areas: fetal, postnatal/infant, childhood, and pubertal.

Fetal growth, influenced by maternal nutrition, uterine size, or restrictions, as well as by insulin and insulin growth factors, actually may have long-lasting effects throughout life. For example, small-for-gestational age and preterm infants have reduced insulin sensitivity later in life that, in turn, has been linked to earlier onset of puberty.

Following birth, growth continues at a rapid rate. Although healthy term

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infants may lose up to 10% of birth-weight within the first days after birth, they quickly regain this weight by 2 weeks of age. This initial weight loss is seen particularly in exclusively breastfed infants when a mother's milk supply is not fully "in" until several days after birth. Subsequently, infants gain as much as 20 to 30 g/day for the first 3 postnatal months. As a result, most term infants triple their birthweight by 1 year of age. GH and thyroid hormone play large roles during this rapid phase of postnatal growth. Other major influences include insulin and overall nutrition.

Growth subsequently slows during childhood. Although birthweight triples by 1 year of age, 3 to 4 years are required to double birth length. Along with nutrition, GH and thyroid hormone continue to be the primary influences on growth.

Growth during puberty, when sex hormones become a significant factor, accounts for approximately 17% of total adult height. A slight deceleration in linear growth accompanies the onset of puberty, followed immediately by a rapid acceleration of growth and corresponding weight gain. Girls, on average, enter puberty at age 9 years and reach peak growth during Sexual Maturity Rating 2 to 3 or about 2.5 years into puberty. Boys tend to enter puberty later, on average at age 11 years, and their growth spurt also occurs at a later point in puberty, usually Sexual Maturity Rating 3 to 4. The later onset of puberty and the later male growth spurt allow for additional growth, with males ultimately being an average 5 in taller than females.

Accurate measurements are key to tracking the growth of a child and require appropriate, properly functioning, and well-maintained equipment. Scales should be calibrated regularly. Children should be weighed while undressed to their underwear or diaper. A child who will not be still can be weighed in a parent's arms, with the parent's weight then subtracted from

the total to determine the child's weight. Length, or supine height, should be measured in infants and toddlers younger than age 2 years. Beyond that age, standing heights should be used. For optimal supine measurements, the child should be lying with legs fully extended, the head resting against an inflexible board, and a moveable footboard used to determine the length. Standing heights should be taken with a wall-mounted stadiometer because measurements obtained with the flexible arms on balance scales often are inaccurate. For patients who are unable to stand, several techniques can be used to measure height. Arm span is a good substitute for height or height can be estimated by adding measurements from the base of the heel to the knee, from the knee to the hip, and from the hip to the top of the head.

Measurements should be plotted on a growth curve. Standardized growth curves can be obtained from either the Centers for Disease Control and Prevention or the World Health Organization website. Specific growth charts available for special populations, such as low-birthweight and very low-birthweight preterm infants as well as for patients who have trisomy 21, Turner syndrome, Klinefelter syndrome, and achondroplasia, among others, should be used for affected children. Although a preterm infant can be plotted during the first few years after birth on a standard growth chart at the corrected gestational age, rather than chronologic age, the use of charts specifically for preterm infants is preferred.

Several principles apply when interpreting a growth curve. First, an individual child should be considered in terms of his or her expected growth potential. For example, a child tracking along the 5th percentile for height whose parents are both short and healthy does not raise concern, but if the parents are both close to 6 ft tall, investigation may be warranted. An estimate of genetic growth potential

can be obtained using a weighted average of the parental heights called the mid-parental height (MPH):

For boys: $[\text{father's height (cm)} + \text{mother's height (cm)} + 13]/2$ or $[\text{father's height (in)} + \text{mother's height (in)} + 5]/2$

For girls: $[\text{father's height (cm)} - 13 + \text{mother's height (cm)}]/2$ or $[\text{father's height (in)} - 5 + \text{mother's height (in)} + 5]/2$

Predicted range: ± 8.5 cm or 3 in for 2 SD from MPH

The next principle applicable to assessing growth is that children tend to grow at predictable rates and track along a growth percentile curve. Shifts across two or more percentile lines may indicate an abnormality in growth and, therefore, point toward a wide variety of disease processes. At times, however, shifting along the growth curve can be normal and even expected. As noted earlier, birth size tends to be a reflection of maternal factors and in utero conditions rather than genetic growth potential. As a result, shifts on the growth curve toward a child's genetic potential between 6 and 18 months of age are common. Often, small infants born to tall parents begin catch-up growth around 6 months of age until they reach a linear growth curve that better matches their expected growth. Large infants born to small parents may have a deceleration in growth, usually at 12 to 18 months of age, slowly shifting downward on the linear growth curve until they reach their new growth trajectory. Although such shifts early in life are expected and can even be anticipated, shifts across two or more percentile lines on the growth curve after age 3 to 4 years are uncommon and most likely represent an abnormality of growth.

Well-documented growth charts can help distinguish among different types of abnormal growth. In malnourished children, be it from chronic disease, malabsorption, or neglect, a drop is seen first

on the weight curve, followed by decreases in height percentile and finally head circumference. Children who present with primary linear growth problems often have some congenital, genetic, or endocrine abnormality. Frequently, children who have primary endocrine abnormalities, such as hypothyroidism or GH deficiency, maintain normal or even elevated weight-for-height measurements while height trends downward on the growth curve.

Growth charts also can help with the diagnosis of familial short stature (FSS) and constitutional growth delay (CGD). In FSS, height and weight generally are within the normal range in the first 2 to 3 years after birth. Height then drifts downward across growth percentile lines until reaching a growth curve that fits the child's genetic potential. Children who have FSS tend to follow a growth curve that parallels normal curves at a lower percentile line. After their initial drop off the growth curve, they have normal growth velocities of at least 5 cm/y and normal bone ages, enter puberty within normal age ranges, and have normal pubertal growth spurts. The result is a

short final adult height consistent with MPH expectations.

A different growth pattern is seen in children who have CGD and who experience a slowing of their growth velocity during the first 3 years after birth, with both height and weight crossing several percentile lines. Such children subsequently demonstrate normal or near-normal growth velocity during the prepubertal years. The bone age is delayed in children who have CGD; if height is plotted at the bone age rather than chronologic age, the child usually is at a percentile consistent with the predicted MPH range. Puberty is delayed by several years, so affected children appear to fall further off the height curve during early adolescence. Ultimately, after completing puberty in the late teens to early 20s, children who have CGD achieve adult height in the normal range, although sometimes they are slightly shorter than expected for MPH.

No current discussion of growth in childhood can avoid the issue of obesity. Body mass index (BMI) is the standard measure of overweight and obesity, despite the limitation that it does not differentiate lean muscle from fat. BMI is

calculated as weight in kilograms divided by height in meters squared. A BMI for age between the 85th and 95th percentiles defines overweight, and a BMI greater than the 95th percentile defines obesity. At the opposite end of the spectrum, a BMI below the 5th percentile is considered a sign of underweight and warrants an investigation of its own. However, weight-for-height under the 5th percentile is a better indicator of malnutrition. Another useful parameter is the ideal body weight, which measures weight as a percent of the median weight-for-height ratio at the patient's age.

Comment: The most fundamental tasks of childhood are growth and development. Much of the pleasure we take in our work as pediatricians comes from watching over these dynamic processes, and our success most often can be measured by how well the children we look after grow and develop, emphasizing the importance of measuring and assessing carefully and accurately.

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