

# In Brief

## Short Stature

**Essentials of Growth Diagnosis.** Rosenfeld RL. *Endocrinol Metab Clin North Am.* 1996;25:743–758

**Disorders of Growth Hormone/Insulin-Like Growth Factor Secretion and Action.** Cohen P, Rosenfeld RG. In: Sperling M, ed. *Pediatric Endocrinology.* 2nd ed. Philadelphia, Pa: WB Saunders; 2002:211–288.

**Childhood and Adolescent Obesity: Prevalence and Significance.** Styne DM. *Pediatr Clin North Am.* 2001;48:823–854

**The Basics for the Diagnosis and Management of Short Stature: A Pediatric Endocrinologist's Approach.** MacGillivray MH. *Pediatr Ann.* 2000;29:570–576

Pediatricians monitor growth as a reflection of a child's overall health. A disturbance of linear growth may manifest as short stature that is immediately obvious when a child walks through the door or more subtly, when growth is evaluated in the context of other family members or the child's history of growth. Although short stature may represent a normal variant, it also may signify a serious illness. Clinicians must recognize patterns of aberrant linear growth and sort through the diagnostic features of the many possible causes.

A useful classification scheme separates causes of short stature into those directly involving the growth plate and those in which the growth plate is affected secondarily. Osteochondrodysplasias, such as achondroplasia and spondyloepiphyseal dysplasia, act directly on the growth plate. The mechanism of growth retardation in chromosomal abnormalities, such as Russell-Silver or Down syndrome, has not yet been elucidated clearly but is believed

to be intrinsic to the growth plate. Conditions in which growth retardation is a secondary phenomenon include malnutrition, metabolic and endocrine disorders, and severe systemic illnesses.

In cases of genetic short stature and constitutional delay of growth, the underlying process may involve a combination of direct and indirect effects on the growth plate. Regardless, both conditions generally are recognized as normal variants. The first step in diagnosing either condition is to compare the child's growth to the parents' and siblings' patterns of growth. With genetic short stature, other family members are short, and the child grows along a curve consistent with others in the family. Constitutional delay of growth is characterized by short stature in the preadolescent period, delayed puberty with a delayed growth spurt, and eventual catch-up growth with attainment of normal adult height.

Absolute height, longitudinal growth data, and a child's height relative to family serve as screening tools in the initial identification of children who have potential growth disturbances. Although a single height measurement removed from the context of longitudinal growth is of limited value in sorting through the various causes of growth disorders, a height more than 2 standard deviations below the mean for age always merits further consideration. Similarly, a child of any height whose growth has fallen across more than 2 percentiles on standard growth curves over a period of at least 6 months may have an underlying growth disorder. At least 6 months of observation are necessary to verify the significance of a downward shift because such a falloff over a shorter period may be within the

range of measurement error or the result of intercurrent illness or seasonal variation in the rate of growth. Finally, further evaluation is warranted for any child whose height clearly is inconsistent with that of siblings or whose growth velocity is unlikely to lead to an adult height within the range predicted by parental heights. Target height may be estimated by taking the average of both parents' heights and adding or subtracting 6.5 cm for a boy or girl, respectively.

Once a child who has an apparent growth disturbance is identified by using these criteria, a focused history and physical examination may be sufficient for diagnosis. With skeletal dysplasias primarily involving the long bones, such as achondroplasia, the limbs are disproportionately short compared with the trunk. The contrary is true with spondylo-dysplasias, which feature greater shortening of the trunk than of the limbs. Specific genetic syndromes have characteristic dysmorphic features and associated anomalies. Among the endocrine abnormalities causing growth failure, hypothyroidism and hypercortisolism usually present with other characteristic features, but short stature may be the only manifestation of a defect in the growth hormone axis.

A weight-for-height measurement helps to distinguish between short stature from caloric deprivation or systemic illness and growth retardation from an endocrine abnormality. When both weight and height are low but weight is disproportionately diminished, malnutrition and chronic illness are the prime concerns. Children who have endocrine disorders, genetic short stature, or constitutional delay of growth have either normal or even generous weights for

height. Obesity in a short child should heighten suspicion for an endocrine or genetic disorder because exogenous obesity is associated with tall stature.

Height velocity, which is the rate of linear growth, is critical in distinguishing pathologic from benign causes of short stature. In particular, height velocity is normal in cases of genetic short stature and constitutional delay of growth, but it is slowed in children who have growth hormone deficiency. A downward shift across more than 2 percentile channels on standard growth charts, representing a deceleration in growth velocity, warrants further evaluation at any age. However, a downward shift that remains within 2 percentiles may be normal in a child younger than the age of 2 years or older than 12 years. Whereas intrauterine factors predominantly determine birth length and weight, genetics and the environment prevail after birth. In the first 2 years after birth, the equilibration of these influences may manifest

as an accelerating or decelerating growth velocity. Crossing growth percentiles can be acceptable during adolescence because the timing of the growth spurt varies with pubertal development, rather than directly with age. Children in whom the onset of puberty is relatively late initially may appear to fall off their growth curves only to accelerate back once puberty begins.

Although the cause of short stature may be obvious with dysplastic and genetic syndromes, differentiating among genetic short stature, constitutional delay of growth, and growth hormone deficiency demands careful attention to the growth chart and, in particular, to height velocity. A child's height velocity can be calculated from longitudinal growth data and compared with available graphs standardized for age.

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**Comment:** Part of what makes pediatrics special is that our patients are on an upward curve. Health for them means accomplishing the fundamental tasks of childhood: growth and development. Dr Chianese describes one useful classification scheme for short stature that hinges on whether the growth plates are affected primarily or secondarily. I offer another. Short children who are developing normally are failing to grow; children who are neither growing nor developing along the normal upward curves are failing to thrive. The term "failure to thrive" rightfully has its critics, but it has long been a paradigm for the interplay of biologic and psychosocial forces on the health of children. It reminds us that growth hormone is necessary but not sufficient for a successful childhood.

*Henry M. Adam, MD  
Editor, In Brief*

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Jennifer Chianese  
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