

## Long-Term Growth in Juvenile-Acquired Hypothyroidism: The Failure to Achieve Normal Adult Stature

Hypothyroidism was diagnosed and treated in 18 girls and six boys with a mean age of approximately 10.5 years and a mean bone age of 6.1 years. At diagnosis, heights were  $4.05 \pm 0.5$  standard deviations (SD) and  $3.15 \pm 0.5$  SD below the 50th percentile in girls and boys, respectively. Prior to deceleration of growth the mean height of all patients was less than  $\pm 0.3$  SD from the 50th percentile. The bone age at diagnosis closely matched the age at which deceleration of growth began, which suggests that the bone age at diagnosis corresponds well with the onset of severe hypothyroidism. L-thyroxine was given at  $3.4 \pm 0.3$   $\mu\text{g}/\text{kg}/\text{day}$  for treatment. Serial bone age determinations were available in most cases.

Mature heights were  $2.1 \pm 0.2$  SD below the 50th percentile. Differences between the predicted mature heights and the actual mature heights were  $7.7 \pm 6.0$  cm and  $6.7 \pm 5.5$  cm for females and males, respectively. The loss occurred primarily in the first 18 months of treatment and correlated significantly with the duration of hypothyroidism and the height SD at diagnosis. There was no correlation between the loss in mature height and the

chronologic, height, or bone ages at diagnosis.

The authors demonstrated that catch-up growth is incomplete after treatment of long-standing juvenile hypothyroidism. In brief, these patients rarely achieve their full genetic growth potential. The authors conclude that the possible etiologies for this deficit include: (1) overtreatment; (2) prolonged hypothyroidism, which diminishes the potential for catch-up growth; and (3) puberty coinciding with initiation of therapy, which results in completion of skeletal maturation prior to the completion of catch-up growth.

The thyroid function tests did not indicate overtreatment. Loss in predicted height during the first 18 months of treatment occurred in children who did not exhibit pubertal changes. The authors suggest that multiple factors may be involved but a delay in therapy is a critical factor in limiting catch-up growth that underscores the need for early recognition of hypothyroidism.

Rivkees SA, Bode HA, Crawford JD. *N Engl J Med* 1988;318:599-602.

**Editor's comment**—Failure to

*reach expected adult heights in patients with prolonged juvenile hypothyroidism has been apparent to most pediatric endocrinologists but data documenting its occurrence and extent have been lacking. Rivkees et al have provided us with those data, thereby permitting postulations that can be tested to be made. They are to be congratulated for their contribution.*

*The decision to be made now is what to do for the next patient with prolonged hypothyroidism so that he or she can achieve the height inherent in his or her genetic potential. I would use a lower dose of thyroxine than that used by the authors, as 3  $\mu\text{g}/\text{kg}/\text{day}$  in older children may be more than is necessary to attain a euthyroid state. Alternative approaches might include the use of an analogue of leutinizing-hormone-releasing hormone(a) to block puberty and/or the addition of growth hormone. These latter approaches, if chosen, should be used within rigid protocol guidelines and, therefore, should not be considered by most of us unless we are willing and able to establish and follow such a protocol.*

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