

Management of Idiopathic GH-Deficient Patients During Puberty

At the Fifth International Symposium Regarding Growth and Growth Disorders, Berlin, April 1988, Price et al presented data from their clinic and from the literature regarding the growth of patients with growth hormone deficiency (GHD) during spontaneous or induced puberty. Boys with idiopathic GHD had a significantly later onset of puberty (15.0 to 15.9 years) than normal boys (11.5 to 12.0 years). The peak height velocity (PHV) occurred at 16.0 to 16.4 years, compared with 14 years in normal boys. The bone ages were comparable (13.5 to 14.0 years) at the time of PHV in the two groups. The PHV was less

in the GHD group and the total gain after G2 sex development was 17.0 to 22.8 cm (means of four groups of GHD patients studied at different centers) vs 27.4 cm in normals and 18.0 cm in boys with constitutional delayed growth (CDG). The loss in final height SD score (-2 to -2.5) may reflect pretreatment loss rather than a failure of adequate treatment during puberty, because the total gain after G2 sex development was comparable to that of boys with CDG. The GH treatment was unsophisticated by modern methods, with fixed doses independent of body size given two to three times per week. Data regarding girls were very limited and, therefore, are not reported here.

With respect to treatment, the authors did not encourage an increased dose of GH during puberty

because significantly increased growth velocity occurs spontaneously in boys with isolated GHD and because of the greater cost of larger doses. They urged, however, that daily rather than intermittent doses be used. They also stated the need for further information before dose schedules for pubertal patients can be firmly recommended.

Manipulation of puberty was recommended in patients who have GHD and either gonadotropin deficiency or sexual precocity. In the former, the authors recommended strongly that physicians consider the induction of puberty at 14 to 15 years of age in boys and 13 to 14 years of age in girls. They also suggested that this approach be considered in patients with isolated GHD if puberty has not developed spontaneously. Their ar-

guments are based on the psychological need of adolescents to develop at these chronologic ages and the disproportionate stature that develops if treatment is prolonged. Six boys with luteinizing hormone deficiency who were not treated with testosterone until late had an SD score for mean leg length/sitting height of 1.4, compared with 0.6 in isolated GHD. Low doses of testosterone (25 mg twice monthly) are advised in boys to counteract the shorter pubertal duration of 2.7 vs 4.2 years observed when 100 mg was given monthly. Estrogen, 2 to 5 $\mu\text{g}/\text{d}$, was recommended for girls. The authors suggested that delay of puberty with gonadotropin-releasing

hormone analogs should be considered in patients with sexual precocity, but they readily admit that data are not available to evaluate the effectiveness of delaying epiphyseal fusion in order to increase height.

Price DA, Shaleta SM, Clayton PE. *Acta Paediatr Scand* 1988;347 (suppl):44-51.

Editor's comment—*The fact that testosterone, endogenous or exogenous, stimulates growth in GH-deficient patients, and that Laron dwarfs have an adolescent growth spurt, support the concept that the growth spurt at adolescence is derived, at least in part, from a direct action of testosterone on the*

growth plate. The second, and possibly more influential, action is via the increased GH secretion that occurs under the influence of testosterone. Whether additional GH should be given to GH-deficient patients while they are passing through adolescence is still debatable. Each case should be individualized, and the decision to treat should be made on the basis of current height, bone age, mid-parental height, and cost. Some very short GH-deficient patients certainly should be given the opportunity to grow maximally while passing through adolescence and should be considered for additional GH treatment.

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