

Predictive Factors in the Determination of Final Height in Boys With Constitutional Delay of Growth and Puberty

Albanese and Stanhope hypothesized that boys with constitutional delay of growth and puberty (CDGP) form a heterogeneous diagnostic category composed of children with varying degrees of impairment of final height. Consequently, they analyzed the patterns of growth in height and the changes in body proportions in 78 prepubertal or early pubertal boys with CDGP. The characteristics of those in this group were that the chronological age was ≥ 13 years and bone age delay was >1.5 years. These boys were treated for 4 months with either 50 mg of sustained-action testosterone every 2 weeks or 1.25 mg daily of oxandrolone, or received neither drug. The mean height standard deviation score (SDS) was -2.7 ± 0.7 (140.6 ± 8.6 cm) at the initial evaluation and -2.0 ± 0.9 (160.5 ± 6.7 cm) at final height. The latter was significantly below either the mean predicted adult height or the corrected midparental height (MPH), although much overlap occurred. The final height of 45 (58%) of the 78 patients did not achieve the target height range. Of the 33 (42%) of patients whose final heights fell within the target height range, the heights of only 3 (0.7%) exceeded the corrected MPH.

At final height, several (26%) of the boys had eunuchoid habitus, with short spines relative to lower limb lengths at diagnosis and at final height. Using multiple regression analyses, the authors determined that standing height, growth velocity, and the difference between the sitting height and the subischial leg length present at the initial evaluation could be used as predictors of impaired final height. Neither the chronological age, the delay in bone age at the initial examination, nor treatment for 4 months with androgens influenced this analysis.

The authors conclude that decreased spinal growth is present in many boys with CDGP, and that the presence of a short spine relative to leg length suggests that final adult stature will be impaired.

Editor's comment: As the authors point out, the 78 boys studied represent only a fraction of those with CDGP, and possibly only those with the most severe impairment of growth were followed in the authors' clinic. Therefore, their conclusions may be applicable only to a subset of patients with CDGP. Nevertheless, the observation of impaired prepubertal spinal growth leading to impaired final height prompts the question whether some patients with CDGP have a subtle spinal chondrodystrophy. The report indicates the need to routinely measure sitting heights or upper to lower ratios in such patients.

Allen W. Root, MD

2nd Editor's comment: The authors were unable to explain the failure to achieve target height in 58% of their patients. Speculation is appropriate that this subgroup may have a variant of CDGP, one with growth hormone insufficiency that is not revealed by pharmacologic tests of growth hormone secretion, or they possibly may have an unclassified skeletal dysplasia. The authors also conclude that treatment with androgens, at the doses used, does not improve final height but only accelerates the growth spurt. They do suggest that the use of low doses of oxandrolone may prevent reduced spinal growth and, consequently, improve final height. In this editor's opinion, the problem with some of these speculations is that only 4 months of androgen therapy were used, and this short period of therapy may not affect either predicted height or ultimate height. Earlier androgen therapy over a prolonged period but at a dose absolutely not higher than that recommended by the authors may be beneficial in increasing ultimate height. Studies need to be done concerning this.

Robert M. Blizzard, MD

Albanese A, Stanhope R. *J Pediatr* 1995;126:545-550.

Zinc Deficiency in a Breast Fed Premature Infant

Zinc is an essential element for a variety of biochemical functions of the human body, including normal function of skin, the gastrointestinal system, and the immune and central nervous system (CNS) systems. Individuals with severe zinc deficiency present with erosive skin changes, particularly of the face and anogenital area, and with alopecia of scalp hair. Failure to thrive, irritability, and immunodepression are also common.

Several causes of zinc deficiency syndrome are known. These include: (1) deficient exogenous zinc supply, either from breast milk when the mother is deficient in zinc or in her diet; (2) increased intestinal or urinary zinc loss; (3) inadequate absorption in preterm infants; and (4) poor storage. Since breast milk usually is a good source of zinc, severe zinc deficiency in full-term infants is very rare. However, a number of investigators have reported zinc deficiency in breast-fed, preterm infants (Aggett et al; Bilinski et al; Buehning and Goltz).

A recent paper by Heinen et al reports a typical case of a preterm infant who was exclusively breast-fed and suffered from severe zinc deficiency syndrome. The neonatal period was complicated by bronchopulmonary dysplasia, cerebral hemorrhage with subsequent hydrocephaly, and ventriculitis. A zinc-containing formula was given only for the first 4 days of life. After that he was breast-fed and received parenteral nutrition without zinc supplements. At 20 weeks, erosive skin changes, developmental retardation, and muscular hypotension were noted. Blood zinc levels measured in both mother and infant were significantly low. Oral zinc therapy was instituted. Marked improvement in the skin lesions occurred in 2 days. Heinen et al concluded that a diet based exclusively on breast milk may in some cases, depending on the mother's nutritional status, lack sufficient zinc and lead to severe zinc deficiency in the infant.