

diagnosis of hypophysitis was made by pathologic studies in all patients. Light microscopy revealed lymphoplasmacytic infiltrate accompanied by varied numbers of neutrophils, eosinophils, and macrophages. Preserved cells were grouped in small islands surrounded by inflammatory infiltrate or fibrous tissue. Immunocytochemistry performed in 14 cases revealed the presence of GH and prolactin in all but 1 patient. There was absence of corticotropin immunoreactivity in 5 patients; of these, 3 had adrenal insufficiency and 1 was receiving treatment with steroids. In addition to confirming the findings of light microscopy, electron microscopy (8 patients) identified lactotroph cell hyperplasia or hyperactivity in 3 patients (1 male and 2 females, pregnancy related). In the 3 postmortem examinations, gross pituitary atrophy along with adrenal atrophy (presumably secondary to pituitary-target organ dysfunction) was found. The authors concluded that lymphocytic hypophysitis should be considered in the differential diagnosis of females presenting with pituitary enlargement in the peripartum period, in patients presenting with GH deficiency or excess associated with autoimmune disorders, and in patients presenting with rapidly enlarging pituitary masses with or without pituitary hormone dysfunction.

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Thodou E, et al. *J Clin Endocrinol Metab* 1995;80:2302-2311.

**Editor's comment:** This is an important compilation of patients with lymphocytic hypophysitis that showed the great diversity and heterogeneity of the clinical picture of this disorder. The diagnosis of lymphocytic hypophysitis was confirmed by biopsy in all instances. The authors advocate conservative treatment on the basis of clinical suspicion to avoid aggressive surgical intervention. However, it was the experience of the authors, as well as of others, that there is no way to elucidate the final diagnosis before surgery. Antipituitary antibody testing in 2 patients produced negative results. Thus, the validity of these measurements to diagnose lymphocytic hypophysitis cannot be relied upon for diagnostic purposes. This group of patients with hypophysitis did not include children with hypopituitarism; therefore, it is hard to ascertain whether autoimmune hypophysitis occurs in children diagnosed with GH deficiency who do not exhibit pituitary masses on imaging studies and in whom the diagnosis of idiopathic GH deficiency is made. Most of the literature of lymphocytic hypophysitis relates to middle-aged patients, with a higher prevalence of females than males.

## Final Height and Predicted Height in Boys With Untreated Constitutional Growth Delay

The authors reexamined 49 males at a mean chronologic age of 22.9 years (range, 20.4 to 31.2 years) who presented to their clinic at a mean age of 13.3 years (range, 7.3 to 16.4 years) and were diagnosed with constitutional delay of growth (CDG). The reexamination included measurements of standing height, using a Harpenden stadiometer, and testicular volume. At initial presentation, the diagnosis of CDG was made by documenting a standing height <5th percentile for chronologic age and a bone age retarded by 1 year or more in a boy who was born at term and had a birth weight of 2,500 g. Seventy-five percent of the boys had a history of late maturing parents. Heights of both parents were recorded. No patient with dysmorphic features, systemic disease, nutritional disorders, or suspected hormone deficiency was included in the sample. None of these men had received any chronic medical treatment, including anabolic steroids, during the intervening years. At the initial visit, the bone ages were determined by the methods of Greulich and Pyle and of Tanner-Whitehouse Mark II (TW2). Height predictions were calculated by the Bayley-Pinneau, TW2, and Roche-Wainer-Thissen (RWT) methods. Target height (TH) was defined as midparental height with 6.5 cm added and with 1 standard deviation (SD) defined as 4.25 cm. Paired *t*-tests and linear analyses were used for comparisons.

At the reexamination, the measured final height of these men was within the lower range of normal for the population, but significantly below their THs (by an average of 1.7 cm). There was a good correlation between the final height SD score (SDS), the initial bone age deficit, and the initial height SDS for bone age. No endocrine disorders became evident in these men and the mean testicular volume on reexamination was 19.0 mL (range, 10.3 to 25 mL). Predicted height by the Bayley-Pinneau method did not differ from the mean final

height, and predictions by all 3 methods and by TH were significantly positively correlated with final height. Height predictions were not more accurate in boys with advanced versus younger chronologic age at initial presentation.

Sperlich M, et al. *Eur J Pediatr* 1995;154:627-632.

**Editor's comment:** This study provides important information for the pediatric endocrinologist counseling boys with CDG and their families. The authors have demonstrated that there is a good correlation between predicted heights, THs, and final heights. However, in untreated men with CDG the final heights are significantly lower than the THs. Interestingly, both the final heights and the THs were within the lower range of the population norm, suggesting a component of familial short stature present in these men. Their data suggest that final height in untreated CDG patients may be compromised. A table in the article summarizes similar findings from 10 other studies of final height in untreated men with CDG. All but one demonstrate a final height significantly lower than the TH.

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**2nd Editor's comment:** In GGH, Vol. 11, No. 4, page 2, an article entitled "Predictive Factors in the Determination of Final Height in Boys With CDGP" was abstracted. The ultimate heights recorded in this article were less than expected for midparental height. You may wish to review the data abstracted from the article and the editorial comments by 2 of our editors in GGH 11:4.

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