

Growth Hormone Secretory Dynamics in Turner's Syndrome

Ross, Long, Loriaux, and Cutler have examined growth hormone (GH) output, somatomedin-C determinations, and bone ages in 30 patients with Turner's syndrome (TS), ages 2 to 20 years. The findings have been compared with those of 17 normal subjects, ages 4 to 17 years. The mean GH concentrations during day and night (specimens collected every 20 minutes), the peak amplitudes, and the peak frequencies were similar in girls with TS who were less than 8 years of age and in age-matched controls. The mean 24-hour GH levels in this group were actually higher in patients with TS than in controls (4.6 ± 0.7 ng/ml v 2.9 ± 0.2 ng/ml), although these values were not statistically significant.

TS patients who were more than 9 years old had lower mean GH concentrations during both day and night, compared with age-matched controls ($p < 0.005$). Patients also had a significant decrease in the peak amplitude of GH release as compared with normals. Interestingly, when the TS patients between the ages of 9 and 20 were compared with each other, there was no significant difference between the day and night mean GH levels, peak amplitudes, or peak frequencies.

Normal females in this age range have greater nocturnal elevation and amplitude, but not peak frequency.

All 21 patients with TS who were stimulated with arginine and insulin had peak GH concentrations > 10 ng/ml. Serum IGF-1 concentrations were stated to be significantly decreased in those with TS between 6 and 12 years of age when compared with normals. However, none of the IGF-1 determinations was in the GH-deficient range.

The authors also present data indicating that bone ages are delayed in TS children of all ages. The delay in 14 girls, 6 to 10 years of age, was 1.4 yrs ± 0.3 SEM. The difference in bone ages between the normal population and the patients with TS increased during the adolescent years. The mean values for patients with TS, 11 to 17 years of age, were decreased by approximately three years, as compared with controls.

A significant increase in GH secretion during normal puberty has been observed in some, but not all, normal subjects. The authors propose that in these sexually infantile girls the role of estrogen would be consistent with the observation that integrated concentrations of GH did not increase at pubertal age. They

also state that since short stature in children with TS is observed at all ages, the cause of short stature is most likely multifactorial. The authors conclude that a relative deficiency of GH in pubertal patients with TS may contribute to their adult short stature.

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Editor's comment—*The data presented are not surprising, but documentation that there is a difference in GH secretion between normals and patients with TS during the adolescent years is a significant contribution. Although many earlier studies in normal children do not indicate an increase of mean GH concentrations in normals at the onset of adolescence, recent studies utilizing testosterone in boys with constitutional growth delay strongly suggested that more GH is released in the presence of testosterone, and other studies suggest estrogen increases GH concentrations.*

In this study the authors found that the mean 24-hour GH determinations in their normal controls, 8 years of age and younger, was 2.9 ± 0.2 ng/ml, v 5.7 ± 0.8 ng/ml in the

9- to 17-year-old controls. The mean GH levels in TS decreased between childhood and adolescence from 4.6 ± 0.7 ng/ml to approximately 2.5 ng/ml. The difference in the secretion of GH by the girls with TS in the two age ranges is not statistically significant. The observed difference in the mean GH secretory rates, therefore, is primarily related to an increase in GH secretion in normal female adolescents and is not surprising.

The discrepancy of the somatomedin-C determinations during adolescence is also probably related to the absence of sex hormones in the TS patients. The authors found a mean level of approximately 0.85 U/ml for the 11 TS patients who were 11 years of age and older. If estrogen were administered to girls in this age group, the somatomedin-C levels would very likely increase and approach those seen in normal female adolescents.

That bone age is delayed in TS patients is also not surprising, since sex hormones contribute to skeletal maturation after the age of about 9 years. Patients with TS do not have sex steroids present, and, therefore, the clinical observation of delayed skeletal maturation discussed by the authors is expected.