

Growth Hormone Secretion in Turner's Syndrome

Twenty-four growth hormone (GH) profiles in 26 girls with Turner syndrome were compared with those of 26 normally growing short children (18 males, 8 females) and 24 slowly growing short children (17 males, 7 females). All patients studied were prepubertal and less than 12 years of age (study 1). A randomly selected subgroup of 13 Turner girls was restudied during treatment with ethinyl estradiol 0.05 µg/kg/d. Separate samples were obtained, and GH was measured by immunoradiometric assay (IRMA).

A second trial (study 2) was done with 45 girls with Turner syndrome, aged 6.7 to 18.9 years, submitted to continuous blood sampling. A different IRMA kit was used for GH measurements. These patients were divided into 4 subgroups:

1. age less than 12 years, no treatment;
2. age more than 12 years, no treatment and no spontaneous breast development;
3. age more than 12 years, spontaneous breast development; and
4. age more than 12 years, treated with ethinyl estradiol 0.1 µg/kg/d.

Time series analysis of the results was done by Fourier transformation. In addition, the mean GH level of each profile was used for estimation of the differences between groups and for correlation with clinical situations.

In study 1, the mean 24-hour serum concentrations of the Turner girls and of the normally growing short children were both significantly higher than those of the slowly growing short children ($P < 0.0001$). In the Fourier analysis, the dominant periodicity of GH secretion was similar in the 3 groups of children, but the oscillatory activity was lower in the slowly growing children, resulting in a reduced spectral power. Estrogen treatment significantly increased the pulse amplitude but did not change the periodicity.

In study 2, the estrogen-treated Turner girls had a higher mean GH than the others, but the difference was not significant. Fourier analysis did not show significant differences between the 3 subgroups of patients over 12 years of age. There was no relationship between mean 24-hour GH levels and age. Linear regression analysis did not show a relationship between the height (standard deviation scores for Turner references and for bone age) and the mean 24-hour level of GH.

Thus, the authors point out that the regulation of GH pulse amplitude and frequency is normal in girls with Turner syndrome. This clearly shows that short stature in Turner syndrome is not related to insufficient or abnormal GH secretion. The findings agree with clinical therapeutic studies, which suggest that girls with Turner syndrome are relatively resistant to GH treatment and need high doses of GH for improving their growth rate.

WitJM, Massarano AA, Kamp GA, et al. *Acta Endocrinol* 1992;127:7-12.

Editor's comment: *The first trials of GH treatment in Turner syndrome were largely related to the reported finding by some groups of reduced release of GH, mainly after the age of 10 to 12 years. Further clinical experience clearly showed that the results of treatment with GH in these patients were in no way related to the results of any measurement of GH secretion. This sophisticated study gives clear confirmation that short stature in Turner syndrome, at least up to adolescence, does not result from abnormal or insufficient secretion of GH. This study may be of importance for future understanding of the short- and long-term effects of GH treatment in Turner syndrome, a very peculiar model of short stature with low biologic GH sensitivity but acceptable responses to supraphysiologic GH doses.*