

Growth Hormone and Tumour Recurrence

Ogilvy-Stuart et al report data on the recurrence of CNS tumors in children in the northwest region of England who were treated with human growth hormone (GH). Included in the analysis were 207 children with brain tumors between 0.5 and 14.4 years of age (median, 6.7 years); 47 of these (29 boys) received GH. The median length of time from diagnosis to initiation of therapy was 4.5 years, and the median duration of therapy was 3.2 years. Serving as a comparison group were 160 children who had not been treated with GH. Each child had received cranial irradiation (median dose, 3000 cGy); 36 children had received a tumor-site booster dose (median dose, 1500 cGy). All were evaluated for GH deficiency at approximately 2 years postradiotherapy, a time when tumor recurrence is most likely to occur and also a time at which tumor-induced GH deficiency may be readily identified. The dose of GH was 12 IU/wk prior to 1989 and 0.5 IU/kg/wk after 1989.

Five of the 47 children (11%) who were treated with GH had a clinical relapse associated with recurrence of brain tumor. In 2, relapse occurred after the completion of the GH treatment, while in 3 patients relapse occurred from 0.5 to 3.3 years after starting therapy. Forty-two of 160 children (26%) who did not receive GH

relapsed. Thus, the authors conclude that there is no association between GH and tumor recurrence.

Ogilvy-Stuart AL, Ryder WDJ, Gattamaneni HR, et al. *Br Med J* 1992;304:1601-1605.

Editor's comment: *This is another reassuring study for pediatric endocrinologists assessing children who have received cranial irradiation and who have growth retardation. It is noteworthy that 10 of 44 children with brain tumors who had computed tomography scans performed at the beginning of GH therapy showed residual tumor. Thus, there does not appear to be a clear association between tumor growth and GH treatment. The authors point out that as more children are successfully treated for CNS malignancy, more of these children will be presenting to pediatric endocrinology clinics for possible GH therapy. It is important that similar registry data be continued to ensure that children treated with GH do not show an increased risk of tumor recurrence.*

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Elevated Growth Hormone Secretory Rate in Premature Infants

Wright et al studied 5 premature infants (gestational age, 24 to 34 weeks) and 6 full-term infants for growth hormone (GH) secretory characteristics by drawing blood every 15 minutes for 6 hours for determination of GH concentrations. Deconvolution analyses were done to study the GH secretory characteristics in both groups.

The authors confirmed their own previous work that: premature infants have higher GH concentrations than full-term infants (18,100 ± 800 µg/L vs 10,200 ± 2700 µg/L; $P=0.067$); the half-life of circulating GH for both groups was similar to that reported for normal adult men (18.9 minutes); and premature infants had significantly higher secretory burst amplitudes than full-term infants, as well as higher production rates. The insulin-like growth factor 1 (IGF-1) values were lower in premature infants than in full-term infants.

When these data are interpreted in conjunction with other known data, eg, premature infants have lower levels of IGF-binding protein 3 and GH-binding protein than full-term infants, the authors conclude that the increased GH secretory activity in premature infants reflects an increase in hypothalamic GH-releasing hormone activity and/or reduced somatostatin tone.

Wright NM, Northington FJ, Miller JD, et al. Elevated growth hormone secretory rate in premature infants: deconvolution analysis of pulsatile growth hormone secretion in the neonate. *Pediatr Res* 1992; 32:286-290.

Editor's comment: *The authors are to be commended for performing a tedious task and deriving valuable data while drawing only 2.8 mL of blood. The findings provide further understanding of the pulsatile characteristics of GH secretion at a relative early gestational age (24 to 34 weeks).*

One must realize, however, that the characteristics of GH secretion are probably not related to fetal growth, as GH is not required for normal or near-normal fetal growth. The factors stimulating fetal growth are probably multiple (see GGH, 8[1]:1), but probably do not include GH, human chorionic somatomammotropin, prolactin, or IGF-1. Regardless, the data reported by Wright et al are valuable for the reasons stated above.

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