

## New Concepts of the Growth Spurt of Puberty

The authors emphasize that *final height attained is independent of the timing and intensity of the growth spurt*. Thus, those who enter puberty late have a relatively smaller growth velocity (GV) during the adolescent growth spurt, and they reach a final height identical to what it would have been had they entered puberty earlier and experienced a greater GV for a shorter period. A previous article by these investigators reported that short normal children and children with central precocious puberty (CPP) who had puberty arrested with a gonadotropin-releasing hormone agonist-analog (GnRHa) did not have an improved height prognosis.

The authors attribute the earlier onset of puberty in girls, compared with boys, to differences in luteinizing hormone (LH) pulsatility. Girls require lower doses of luteinizing hormone-releasing hormone (LHRH) to cause the release of LH than do boys, and LHRH agonist-analogs (LHRHa) block the release of LH more readily in girls than in boys. These observations may also help to explain why CPP is more common in girls than in boys and why the converse is true of constitutional growth delay.

The timing of the growth spurt is related to an increase in growth hormone (GH) production, particularly to the amplitude of the GH peaks, according to the authors. GV and GH secretion peaked when the testicular volume reached 12 mL in boys; in girls, this correlation was noted before any increase in serum estradiol or uterine size was detected by ultrasonography. The evidence suggests that changes in GH secretion are modulated by factors other than sex steroids, and the recent demonstration that inhibin can affect the GH response to growth hormone-releasing hormone (GHRH) (*J Endocrinol* 1988;116:301) may be relevant.

In discussing the effect of GnRH on growth in CPP, the authors state that GnRHa do *not* increase ultimate height. This phenomenon relates to the decreased GH production that occurs with the use of GnRHa. The researchers postulate that GH given concomitantly with GnRHa may play a beneficial role and that delay in the timing of puberty alone will probably not improve final height prognosis.

Stanhope R, Preece MA, Grant BD, et al. *Acta Paediatr Scand* 1988;347(suppl):30.

**Editor's comment**—*The studies reported and the concepts proposed are well worth reading in detail. The growth spurt of adolescence undoubtedly is attributable to increased GH production, as reported by Martha et al at the Society for Pediatric Research meetings in May 1989. Boys in stages 3 and 4 of sexual development have GH levels two to three times those found during stages 1 and 2 of puberty and after epiphyseal fusion occurs. There is a strong correlation among GV, GH production, and insulin-like growth factor-1 generation, as demonstrated in those studies. As for the lack of effectiveness of GnRHa in enhancing the ultimate height of girls with CPP, I disagree. Table 2 in the Boepple and Crowley article (Growth, Genetics, and Hormones March 1989; Vol. 5, No. 1) clearly indicates that the appropriate use of GnRHa prevents the loss of height that occurs in patients with untreated CPP. Among such children, those with a bone age greater than 13 years have an average predicted adult height of  $-3.7$  SD. Patients with treated CPP whose bone ages at the time of treatment are less than 10 years remain at essentially the same mean score ( $-1.1$  for bone age) after 3 years of GnRHa treatment.*

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