

Periodic Changes of Short-Term Growth Velocity ('Mini Growth Spurts')

Lower leg length was measured in 73 healthy children, ages 2.7 to 15.9 years, 18-106 times by the Valk machine once or twice a week. The standardized technique described in the abstract on knemometry (see p 13) was used. Straight lines covering three to four successive points were fitted to each 31-day period of each child. Rolling monthly average rates for each child were then computed (just as rolling annual velocities are calculated for stature when measurements are taken every three or six months). These rolling monthly rates were then plotted for each child on a day-by-day basis: the mean of the observations during days 1-31 was recorded, then the mean for days 2-32, and so on.

The deviations of the actual measurements from these individual curves showed significant clustering above or below the line in 38 of the 73 curves, and a characteristic up-and-down pattern of

growth velocity was found in 45 curves. The investigators referred to these variations as "mini growth spurts." The peaks have a velocity of about two to three times that of the troughs, and occur between 30 and 55 days apart. There was a significant correlation between the stature of the child and the frequency of his mini growth spurts.

Growth troughs coincided often with periods of intermittent infectious illnesses, but only a small percentage of the mini growth spurts could be explained as catch-up growth after this type of growth arrest. The reason for the majority of these spurts is unknown.

The authors conclude that "monitoring the pattern of short-term growth kinetics by multiple longitudinal knemometric measurements may provide an additional and very sensitive tool to control therapeutic regimes."

Hermanussen M, Geiger-Benoit K, Burmeister J, et al. *Ann Hum Biol* 1988;15:103-109.

Editor's comment—This paper

extends the observations noted in the abstract on knemometry. Many perfectly healthy children seem to have fluctuations in the growth rate of their tibial epiphyses, with peak-to-peak periods of approximately 30 to 50 days. Elucidation of the physiology of this phenomenon will be awaited with the greatest interest. Do such spurts occur in all the long bones at once, or do they alternate from one long bone to another (as was alleged by Godin and others in the early years of this century)? Rats have a tibial epiphyseal cell cycle time of two to three days; humans, about 20 days. Do mini growth spurts occur several days apart in the rat tibia? From a clinical point of view, the authors throw cold water (not for the first time) on the short-term use of short-term velocities. But they very correctly point out that the long-term use of short-term velocities—a daunting task for patient and anthropometrist alike—might yield very valuable information. This phenomenon seems to be analogous to the pulsatile secretion of hormones.

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Uniparental Disomy as a Mechanism in Human Diseases

Although an individual normally inherits one of each pair of chromosomes from mother and one from father, very rarely there is an individual who has inherited both chromosomes of a pair from only one parent. This phenomenon is known as uniparental disomy. Although it has been previously demonstrated in animals, the report by Spence et al marks the first time that uniparental disomy has been demonstrated by DNA studies in a human being with normal chromosomes. The affected individual, a girl with cystic fibrosis, inherited both of her number 7 chromosomes from her mother. Nonpaternity was confirmed by numerous other markers. Thus, in that particular family, the probabil-

ity of the proband's having a child affected with cystic fibrosis is reduced from the usual 25% to nearly zero.

Interestingly, this girl also had intrauterine growth retardation (IUGR). The observation of uniparental disomy suggests that it may occur for other chromosomes and for other diseases. The evaluation of families by studies in molecular genetics now makes it possible to recognize this type of mechanism. It seems likely that uniparental disomy is responsible for autosomal recessive diseases only rarely, probably accounting for less than 1% of individuals with

apparent autosomal recessive inheritance. However, the clue to such cases may be the presence of IUGR.

Spence JE, Perciaccante RG, Greig GM, et al. *Am J Hum Genet* 1988;42:217-226.

Editor's comment—Nothing is sacred anymore. Things as basic as Mendel's peas have exceptions. For the geneticist, however, exceptions serve as incentives to learn more about normal mechanisms of inheritance. At this point, we do not yet know how "normal" uniparental disomy is.

Judith G. Hall, M.D.

In Future Issues

Gonadotropin and Steroid Concentrations in the Fetus and Newborn, by Claude Migeon, M.D.

The Remarkable Catch-up Growth in American Slaves by Richard H. Steckel, Ph.D.

Growth in Late Adolescence, by Alex Roche, M.D.