

Growth of 519 SGA Infants

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below the tenth percentile) grow better than type I SGA infants. In many cases, infants with type II IUGR have some nutritional deficit during the late stages of gestation and their growth potential is thus not permanently affected. Other studies have confirmed the finding that prolonged IUGR (which would result in a normal PI) is associated with poor growth in infancy. Later growth, however, could be predicted by the degree of weight retardation. By the age of 2 years, one in every four SGA infants, regardless of PI, still had a weight below the tenth percentile. The authors also demonstrated that the risk factors most often related to poor intrauterine growth are maternal toxemia, maternal smoking of more than ten cigarettes a day, multiple pregnancies, and the birth of a previous SGA infant. Unfortunately, these data were not analyzed with respect to the type of IUGR.

Predictive Value of Minor Anomalies: Association With Major Malformations

The report is part of an ongoing study of congenital anomalies in white newborns. In this study, in which 4,305 babies were scored for 114 minor physical findings and for all major anomalies, these data confirm the previous hypothesis that infants with three or more minor anomalies are at increased risk of having a major anomaly. In this study, the risk for a major anomaly in the presence of multiple minor anomalies was only 20%; previous studies found a much higher incidence. Less than 4% (3.76%) of the 4,305 babies had a major malformation. Approximately five sixths of these major malformations were considered significant and required intervention; the remaining one sixth were

Tenovuo A, Kero P, Piekkala P et al. *Acta Paediatr Scand* 1987;76:636.

Editor's comment—*Although the article describing this study is somewhat difficult to read, the information presented is significant. The study demonstrates the difference between prolonged and short-term IUGR on future growth. Similar studies of infants from different populations should be carried out to confirm these findings. In addition, long-term follow-up studies of childhood growth and final adult height in these infants are required to provide better predictive information for pediatricians who counsel parents concerning their child's growth, and to help design studies directed at increasing our knowledge about growth reduction in these children. Parents of SGA infants should be advised that these infants will most likely remain smaller than average throughout the first two years of life.*

William L. Clarke, M.D.

thought to require no special care or treatment. The 3.2% incidence of major malformations requiring intervention is higher than previously reported. With regard to minor anomalies, 28% of the babies studied had one such anomaly, 8% had two, and 3.1% had three or more.

Leppig KA, Werler MM, Cann CI, et al. *J Pediatr* 1987;110:531-537.

Editor's comment—*This study indicates that the presence of multiple minor anomalies is a good predictor of a major malformation and that children with minor anomalies should be studied more intensively. It also indicates that minor anomalies are very common in the general population and that the presence of one or two minor anomalies should not cause great distress for the parents or the physician.*

Judith G. Hall, M.D.

Effects of Testosterone Therapy for Pubertal Delay

Wilson et al reviewed the charts of 50 adolescent boys treated with testosterone enanthate in oil to determine the long-term effect of testosterone therapy on growth and sexual development. Each boy received a total of four 200-mg injections, each given at three-week intervals. The authors also reviewed the charts of 38 adolescent boys who did not receive treatment.

Follow-up data were requested from subjects whose baseline visit was at least two years earlier. Nineteen (58%) of eligible treated subjects and 11 (52%) of eligible untreated subjects responded. A height Z score (the number of standard deviations away from the mean height for age) was calculated for each boy, and bone ages were obtained and read using the method of Greulich and Pyle. Adult height was predicted by a computer program based on the method of Bayley and Pinneau. The mean bone age delay, height Z score, average Tanner stage, predicted adult height, growth rate, serum testosterone, and somatomedin C concentrations, as well as midparental heights, were not significantly different between the treated and control groups.

Initial response to treatment at four months showed a significantly greater increase over baseline in the height Z score. At 12 months, however, only the mean increase in sexual maturation was significantly greater in the treated group. To minimize the statistically confounding effect of potential additional growth, data on final growth were obtained from subjects who were over 17 years of age. There was no significant difference in final absolute height Z scores between the treated and untreated groups, but the mean increase of final height Z scores from baseline was significantly greater among treated subjects because of dif-

ferences in the standard deviation of the final height Z scores between the two groups. Although not statistically significant, the actual mean height of the treated group was 4.9 cm greater than that of the untreated group. There was no significant correlation between baseline predicted adult heights and the actual heights at the time of the last visit. This study demonstrates that four courses of 200 mg testosterone enanthate at three-week intervals do not appear to compromise adult height in boys with delayed puberty.

Wilson DM, Kei J, Hintz R, et al. *Am J Dis Child* 1988;142:96-99.

Editor's comment—Although others have looked at the long-term effects of different androgen preparations, control populations with which to compare results are often not available. In addition, most other studies have utilized long-term (9- to 12-month) treatment regimens. Wilson et al also looked at patient satisfaction with therapy, and 95% of the treated subjects indicated that they believed the treatment had been helpful.

The authors correctly point out that patients who fail to show signs of pubertal regression a year after therapy should be carefully re-evaluated for hypogonadism. We have previously reviewed other reports in this publication (Vol. 3, No. 4 and Vol. 4, No. 2) of the long-term effects on height of testosterone injections for pubertal delay. The present study corroborates the findings of those studies and presents additional useful data for pediatric endocrinologists who treat this common problem.

William L. Clarke, M.D.

Letter to the Editor

I write in reference to "Catch-Up and Catch-Down Growth: A Review" by Dr. Tanner (*Growth, Genetics, and Hormones*, Volume 3, Number 4). I found the article provocative and enjoyable. I must say, though, that I do not like the term "catch-down growth" because it does not convey the appropriate meaning to students. The term seems to imply that the children get shorter and/or lose height potential, which of course is not the case.

The phenomenon is essentially another form of catch-up growth. Perhaps "catch-up growth, type II" would be a better term. What really happens is that height ages are trying to catch up with bone ages. When linear growth is abnormally stimulated, as by a virilizing disorder, and the disorder is then alleviated, the growth velocity drops for a while—but only for as long as it takes the height age to catch up to the bone age. Bone length tends to catch up with bone maturation. The result is restoration of height potential. Then normal growth resumes.

"Compensatory deceleration" is an accurate term for the phenomenon, but it does not convey the concept that the process is preserving rather than reducing height potential. Perhaps the term "catch-up growth, type II" better conveys this concept.

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Dr. Tanner's reply

I agree that "catch-down" growth is not a perfect term. In the formal setting, auxologists use the phrase "compensatory deceleration" or, preferably, "homeorrhetic deceleration." This expresses precisely what one is after; namely, a deceleration that restores the child to his programmed growth chart.

In the original paper on catch-up growth, Prader et al (1963) pointed out that this phenomenon was simply a special case of the principle of homeorrhesis described by Waddington in his classic book *The Strategy of the Genes* (London, 1957). Homeostasis is a well-known term and describes the tendency of an organism to return to a balanced position when pushed away from it. "Homeorrhesis" describes the same tendency, but in relation to an organism moving through time. Rthesis signifies flow as opposed to stasis.

I am not so sure that Dr. Rosenfield's explanation of the mechanism of homeorrhesis is correct in the general case. It is possible to advance both height growth and bone maturation, for example, by overfeeding (or even by providing a nice roomy uterus) and the stimulus is terminated when the animal slows down in both respects. We do not understand the mechanism of this at present, nor even whether the control is chiefly central or chiefly peripheral. There seems to be a size-for-age mismatch involved, and perhaps maturation-for-age and height-for-maturation mismatches as well.

I admit that the term "catch-down growth" has its disadvantages, but "catch-up growth, type II" sounds like a rare disease, probably with chromosomal deletion.

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