

Short Stature in Anorexia Nervosa Patients

In following 104 patients with anorexia nervosa, the authors found 85 suitable for comparison with 85 age-matched controls. As seen in the Table, a large percentage of the anorexic patients were short.

Information was available regarding parental heights for 35 patients. The mean actual height was at the 34th percentile, compared to a mean expected height at the 48th percentile, based on calculations of parental heights. Twenty-six females were postmenarchal, permitting comparison with the adjusted mid-parental height (Tanner scale). Nine had evidence of growth impairment and could not be classified under "familial short stature" by this method.

The patients' age at onset of anorexia ranged between 10 and 22 years. Symptoms first appeared an average of 12.9 months before seeking therapy, and the mean weight loss was 29 pounds (25% of total body weight). Of great importance in considering the etiology of the short stature is the fact that 80% developed anorexia after menarche, with symptoms of onset

Table Height-Related Statistics in Study Participants

Height percentiles	Anorexic patients		Controls		Expected, %
	n	%	n	%	
<5	12	14	1	1	4
5-9	3	4	4	5	5
10-24	28	33	16	19	15
24-49	22	26	28	33	25
>50	20	23	36	42	50

occurring more than one year postmenarche in 61%.

The conclusion is that some factor(s) other than malnutrition may account for the fairly high incidence of short stature. Possibly, there is a pathophysiologic factor producing short stature and, subsequently, anorexia. Patients with anorexia sometimes exhibit several indications of a hypothalamic abnormality affecting thyroid, gonadal, and adrenal function. The authors state that excessive somatostatin production cannot be excluded.

Nussbaum M, Baird D, Sonnenblick M, et al. *J Adolesc Health Care* 1985;6:453-455.

Editor's comment—These data are not only important but also provocative, since they are unexplained within the context of cur-

rent knowledge. Most patients with anorexia might be expected to have growth failure secondary to malnutrition. In the majority of these patients, growth retardation preceded malnutrition. Growth hormone levels are increased in most patients with anorexia, although IGF-I values are low, as is expected with starvation. We do not know whether GH and IGF-I levels are normal before the onset of anorexia. If available, these data might provide insight regarding the etiology of anorexia nervosa.

Furthermore, could these patients have hypercortisolism long before the anorexia begins? (See the review of the endocrine symposium on neuropsychiatric disorders, reported by Dr. Lifshitz in this issue.) If present, hypercortisolism could account for the growth retardation.

Hypercalciuria, Hyperphosphaturia, and Growth Retardation in Children With Diabetes Mellitus

The authors evaluated 157 diabetic children, 6 to 16 years of age, with insulin-dependent diabetes mellitus (IDDM) from 0.2 to 14 years. Eleven percent of the 157 subjects were shorter than would be anticipated, as assessed by comparison with the controls. Increments in height became smaller with the duration of IDDM and differed significantly from controls when IDDM had been present for more than seven years.

Growth retardation correlated with increased calcium and phosphorus excretion (as reflected by increased Ca/Cr and P/Cr ratios) and with poor control of IDDM (as evidenced by glycosylated hemo-

globin assays). Hypercalciuria was not correlated with increased serum calcium or other evidence of bone calcium mobilization. Hypercalciuria is reportedly caused by hypophosphatemia, and there was an inverse relationship between serum phosphorus and an increase of urinary P/Cr and Ca/Cr. Renal disease could not be demonstrated as a cause of increased Ca and P excretion when it occurred. The urinary loss of Ca also correlated inversely with plasma glucose at the time of urine collection. The increased urinary phosphorus appears to result from competition between glucose and both Ca and P for renal tubular reabsorption. There was some evidence of hypercalciuria as a renal response to functional phosphorus deficiency.

The authors conclude that the higher incidence of short children with IDDM is primarily associated with poor metabolic control, but the specific mechanism(s) of impaired growth is (are) not well defined and may not be due to a single cause.

Malone JI, Lowitt S, Duncan JA, et al. *Pediatrics* 1986;78:298.

Editor's comment—This study is very well done and carefully analyzed. The authors speculate that phosphorus supplementation might be beneficial. Further studies are certainly indicated to elucidate the causes and results of hypercalciuria and hyperphosphaturia, which are frequently seen in patients with poorly controlled IDDM. (See Harrison's article in Growth, Genetics, and Hormones, vol. 2, no. 2.)