tion, however, it prevents expression of maleness, producing an essentially female condition, but without reproductive function.

After this paper was submitted, Jacobs *et al.*(11) described bone marrow cell analysis from 4 patients with testicular feminization, all of whom had a 46/XY chromosomal constitution. Thus the correlation between XY sex chromosomes in females and the condition of testicular feminization is strengthened. Moreover, in one of these cases, as in the case of hemophilia discussed by us, a color blind male pseudohermaphrodite and a color blind normal male occurred in the same set of siblings. Therefore, since the gene responsible for testicular feminization is not closely linked to *either* of 2 different sex-linked genes, its autosomal location becomes highly probable.

Summary. A female patient with "male pseudohermaphroditism with feminizing testes" had a 46/XY chromosomal constitution, a fact confirming clinical and genetic deductions drawn by previous investigators largely on the basis of pedigree data, and in accord with recent brief reports on the chromosome condition in other such patients. A detailed chromosomal analysis is provided. Some previous cases having similar clinical manifestations had been recognized by previous workers as being caused either by an autosomal dominant or a sex-linked recessive genetic defect. Analysis of pedigrees from similar cases demonstrates the causative gene defect to be not strongly linked to either of 2 sex-linked genes, hemophilia and color blindness. Hence it is probably autosomal. Of particular importance is the finding that in the family of our patient, all female transmitters of the defect for whom data were available exhibited a delayed menarche of approximately 8 years. This situation has been rarely noted previously, and requires study as to its universal accompaniment of this condition. It is evident that in the present case the presence of this gene in heterozygous condition in an XX individual causes a delay of menarche, while in an XY individual it results in suppression of the male characteristics and a partial development of female traits. It is pointed out that more than one gene locus might be able to produce clinical effects like those here described. Study of the action of such genes appears highly important to problems of human development.

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Summation of Protein Anabolic Effects of Testosterone Propionate and Growth Hormone.* (25457)

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Simultaneous administration of testosterone propionate and a growth hormone preparation at their respective maximum effective

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TABLE I. Summation of Protein-Anabolic Effects of Growth Hormone (GH) and Testosterone Propionate (TP).

Treatment			Nitrogen retention						Max increase body wt		
			Maximum*			Total					
Rats, No./group	GH, u/day	TP, mg/day	GH	TP	GH + TP	GH	TP	GH + TP	GH	TP	GH + TP
			mg/day			mg			g		
4	1.1	.5	14	60	70	273	1050†	1239†	+ 4	+16	+21
4	1.1	.5	27	59	68	491	869	987	+11	+16	+25
3	2.2	.5	84		122	853		1455	+14		+23

* Avg of second, third and fourth periods (7 days) when nitrogen retention was at maximum.
† Retention was more prolonged.

doses to castrated mice resulted in a summation of their respective effects on body weight and protein deposition(1). These studies have been extended to nitrogen balance studies in adult castrated rats.

Procedure. The rats (Sprague-Dawley) were maintained in individual metal metabolism cages in air-conditioned room at 25.5-26.6° and with artificial light regulated at 12 hr/day. They were fed the previously described diet(2) in an amount that would maintain body weight and constant urinary nitrogen excretion. Some rats had been used in another similar experiment. They were approximately 1.5 years old at beginning of the first of these studies. Body weights were 320-480 g and nitrogen intakes were 291-388 mg/day. The rats were grouped to give a uniform distribution of both body weight and urinary nitrogen excretion. Growth hormone‡ and testosterone propionate§ were injected subcutaneously at the same time each day. The dose of testosterone propionate was known from previous studies(2) to produce a maximal protein anabolic effect. The initial dose (Exps. 1 and 2, Table I) of growth hormone also was expected to give a maximal response but the preparation proved to be less active than the previous material(3). Routine of feeding, urine collection and analysis was the same as previously described(2).

Results. Body weight. The androgen produced the expected maximal response in body weight(2). Simultaneous administration of growth hormone resulted in a summation of the effect of two hormones on body weight. Degree of summation was almost complete in the 2 experiments with the lower dose of growth hormone but was less than the sum at the higher dose level.

Nitrogen metabolism. The growth hormone at both dose levels produced a further increase in nitrogen retention both at maximal level and total amount. Summation of the effects of the 2 hormones was nearly complete in the first and third experiment but was intermediate in the second experiment.

Discussion. Ability of growth hormone to superimpose its protein-anabolic action on the maximal effect of testosterone propionate is in agreement with studies in mice. These data provide further support to the independent protein anabolic action of androgens and growth hormone(3,4). The action of these hormones not only is specific for certain tissues but very likely they stimulate synthesis of protein in the same tissue, *e.g.*, muscle, for different functions. These hormones, thus, function in a supplementary manner in growth and development of the various tissues of the body.

Summary. Simultaneous administration of testosterone propionate and growth hormone to castrated rats resulted in a summation of their respective effects on body weight and nitrogen retention even at maximal doses.

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‡ Growth hormone was provided by Parke, Davis and Co.

§ Testosterone propionate was provided as perandren, 10 mg/ml by Ciba Pharmaceutical Products.

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