coagulating glands than Δ^5-androstenediol, while the reverse is true of the post-pubertal castrates. The preputial glands, which are particularly sensitive to androstenediol at any age, do not show this change in sensitivity as clearly as the other accessory sex organs. However, even their responsiveness at puberty increases more markedly to androstenediol than to ethinyl testosterone.

Summary and Conclusions. Experiments on the rat indicate that in prepubertal castrates ethinyl testosterone stimulates the seminal vesicles, prostates and coagulating glands more markedly than an equivalent dose of Δ^5-androstenediol, while the reverse is true in post-pubertal castrates. It appears that at puberty the sensitivity of the accessory sex organs increases only with regard to steroid compounds of a certain chemical structure. This fact must be kept in mind when the hormonal potency of various testoid substances is to be compared.

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Pantothenic Acid Absorption in Pernicious Anemia.

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The most generally accepted view regarding the etiology of pernicious anemia is that the nervous manifestations of the syndrome result from the lack of some other factor than that required for hematopoiesis. The anemia and the neurological lesions frequently develop independently, since one may be present for long intervals before the other. The knowledge that this may occur has led to the acceptance, by a majority of observers, of the dual deficiency theory of the etiology of the disease.

Considerable attention, especially in recent years, has been directed toward the possibility that a deficiency of one or more vitamins might be responsible for the neural disturbances. These observations, together with the reported occurrence of the neuropathology of the spinal cord in chicks receiving a diet deficient in pantothenic acid,[1] prompted the work herein reported. Like thiamin,[2]

1 Phillips, P. H., and Engel, R. J., *J. Nutrition*, 1939, **18**, 227.

2 Field, H., Jr., Robinson, W. D., and Melnick, D., *Ann. Int. Med.*, 1940, **14**, 588.

pantothenic acid is alkali-labile and hence possibly might be destroyed in the intestinal tract of achlorhydric patients. Since study of the urinary excretion of the vitamins has proven of value in certain deficiency diseases, the following experiment was carried out. The daily pantothenic acid excretion of patients with pernicious anemia was compared to that of healthy subjects both with and without the previous administration of 100 mg of calcium pantothenate.

Experimental. From 2 to 8 individual 24-hour urine specimens from each of 7 patients with Addisonian pernicious anemia and one sample from each of 7 healthy medical students and older laboratory workers were collected and preserved with toluene. Both groups received nutritionally good diets during the course of the experiment although some of the patients did not eat normal quantities during the first few days. Pantothenic acid was determined by the method of Pennington, Snell and Williams,[3] with assays made at 4 levels. When a supplement of acid-autoclaved urine was added to the medium as recommended by these investigators, excellent agreement between the various assay levels was obtained.

In Table I are listed the values observed. The amounts found in the control specimens are somewhat greater than reported by Pelczar and Porter,[4] who observed an average excretion of 3.8 mg with a range of 1.46 to 6.79 mg. The average output of the patients, 4.4 mg per day, is not significantly lower than that noted for our control

TABLE I.
Pantothenic Acid Excretion.*

Pernicious anemia patients					Controls		
No.	Determinations†	Range, mg	Avg, mg	After 100 mg dose,‡ mg	No.	Daily excretion, mg	After 100 mg dose,‡ mg
1	8	4.9-8.0	6.8		1	5.7	16.5
2	2	4.2-4.9	4.5		2	4.4	
3	3	4.2-4.7	4.4		3	5.5	
4	5	2.7-5.1	3.9	14.0	4	5.9	13.5
5	5	2.5-7.3	4.9	15.4	5	7.1	30.6
6	5	2.7-5.7	3.8	9.7	6	8.1	20.8
7	4	2.0-3.4	2.7	13.5	7	5.9	10.9
	Avg		4.4	13.1		6.1	18.4

*Mg per 24-hour specimen.
†Each determination was made on a separate specimen.
‡100 mg of calcium pantothenate[1] was given orally.
[1] Merck and Co. generously supplied the calcium pantothenate.

[3] Pennington, D., Snell, E. E., and Williams, R. J., *J. Biol. Chem.*, 1940, **135**, 213.

[4] Pelczar, M. J., and Porter, J. R., PROC. SOC. EXP. BIOL. AND MED., 1941, **47**, 3.

subjects and certainly as high as others have reported.[4] That the lower excretions were probably due to a previously low intake of the compound is strongly suggested by the fact that in every case where there was an appreciable difference in the range of the daily excretion, the lower values were observed at admission of the patient, with a progressive rise while on the hospital diet. A typical case was patient No. 5 whose consecutive daily excretions were 2.5, 3.0, 5.6, 6.2, and 7.3 mg. This increase was not due to the parenteral liver extract, for analyses of the preparation indicated that he received less than one mg from this source during the whole course of treatment.

Further evidence that the excretion of pantothenic acid by the patients was not limited by impaired absorption was obtained by the oral administration of calcium pantothenate. Following 100 mg doses of the salt, the patients and controls excreted comparable amounts, as shown in the table.

This study is being extended to include other water-soluble vitamins.

Conclusions. In patients with pernicious anemia, the daily excretion of pantothenic acid both before and after administration of 100 mg of its calcium salt was slightly but probably not significantly less than that of healthy individuals studied in the same manner. This indicates there is no impairment of absorption of this compound in patients with pernicious anemia with which is always associated achlorhydria. The possibility must be considered, however, that the lack of hydrochloric acid may make the pantothenic acid in food less available for absorption. This is undergoing further study.

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Electrocardiographic Changes Associated with Acute Pancreatitis.

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We have had occasion to observe a series of cases of acute pancreatitis, in which the signs of upper abdominal peritoneal irritation were associated with elevated blood amylase and abnormal electro-