same sense, the behavior of the genitalia is not to be elucidated by the study of a simple reflex. The phenomena serve to define the properties of the pathways which mediate the activity of the genitalia.

The lack of the proprioceptive reflex from the segmental reflex of S_3 indicates that the striated musculature of the genitalia does not have a myotatic regulation with the same properties as that of the limb or tail muscles. Whether there are different types of muscle end organs, or any muscle end organs at all, must remain questionable until a histological examination has been made.

Summary. Activation of the third sacral and first caudal segment of the spinal cord evokes a segmental response on the ventral root resembling the cutaneous component of the segmental reflex of the more cranial parts of the spinal cord. A proprioceptive spike is absent in S_3, present in Ca_1. In each of these segments, there is a crossed reflex return resembling the cutaneous component and frequently somewhat later and smaller than the ipsilateral response. Stimulation of the penis or of the vulvar vestibule elicits the same response, though without the proprioceptive spike in the Ca_1 ventral root. Spinal transection in the lower thoracic region does not alter these reflexes under the experimental conditions.

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Thyroid Feeding and Vitamin B_6 Deprivation in the Rat.* (21120)

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In an earlier investigation(1) it was found that the basal metabolic rate of vit. B_6-deprived rats was not different from that of pair-fed control animals although lower than that of *ad libitum*-fed controls when expressed as oxygen consumption per unit of body weight. There is considerable evidence that the efficiency of energy production is altered by vit. B_6 deprivation. However, attempts to lower the basal metabolic rate by administration of thiouracil or by thyroidectomy did not cause definite alterations in the syndrome of vit. B_6 deprivation(1). It was felt advisable to study the effects of a hyperthyroid state on the deprivation syndrome in rats. The results of this study are presented.

Methods. Forty albino rats of the Wistar strain and of both sexes were divided into 4 groups equal with respect to number, sex distribution and an initial average body weight of 103 g. All animals were housed in individual, screen-bottomed cages with drinking water freely available. The basal diet employed was the 20% casein, 20% corn oil, vit. B_6-free diet previously described(2). Two groups were further provided with 50 μg pyridoxine hydrochloride per rat per day in their food and served as control animals. One vit. B_6-deprived and one control group were given one mg desiccated thyroid (U.S.P., Nutritional Biochemicals) per g of diet. To eliminate differences in analytical results between groups consequent to differences in the amount of food consumed, all groups were pair-fed with the vit. B_6-deprived group given desiccated thyroid such that the average daily food consumption of all groups was 13.4 g per rat. Following an experimental feeding period of 21 days, the animals were fasted for 18 hours and anesthetized with an intraperitoneal injection of 2% butylone in 0.9% saline. Blood was removed from the exposed hearts with the aid of a hypodermic syringe and was heparinized and pooled for each group. The following analyses were done in duplicate on

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TABLE I. Effects of Feeding Desiccated Thyroid to Vit. B_6-Deprived and Pair-Fed Control Rats (10 Rats/Group).

Group	Avg body wt gain, g	Carcass total crude fatty acids, %	Blood packed cell vol, %	Blood hemoglobin, g %	Blood urea, mg %	Blood sugar, mg %	Blood amino N., mg %
Control	89	13.3	42	12.1	24.0	106	11.9
Deprived	73	9.9	44	12.5	34.0	107	14.2
Control + thyroid*	70	7.8	44	11.3	23.8	80	14.6
Deprived + thyroid*	55	5.5	45	11.9	22.4	70	14.3

* Signifies provision of U.S.P. desiccated thyroid 0.1% in the diet.

the pooled blood samples by the procedures stated: hemoglobin, Collier(3); urea, Archibold(4); sugar, Nelson(5); amino nitrogen, Frame *et al.*(6); and packed cell volumes by the standard procedure. Carcasses were pooled for each group, frozen, passed through a power grinder, homogenized and analyzed for total crude fatty acids(7).

Results. The results of this study are set down in Table I. Following 21 days of experimental feeding, none of the animals exhibited acrodynia, the external manifestation of vit. B_6 deficiency in the rat although the deprived animals, particularly those fed desiccated thyroid, were in a poor, unkempt condition. As shown in the data, those animals not provided with pyridoxine had subnormal body weight gains on comparison with their comparable controls. As would be expected, desiccated thyroid-feeding *per se* reduced the body weight gains of both deprived and control rats. Carcass total crude fatty acid levels show the same pattern as body weight gains. Vit. B_6 restriction and thyroid-feeding each diminished carcass total crude fatty acids, the effect being greatest when both occurred concurrently. The customary difference(8) in total crude fatty acid levels of deprived and control animals was evident even when thyroid was administered.

The results of blood analyses as shown in Table I indicate that neither vit. B_6 deprivation nor thyroid-feeding markedly altered hemoconcentration. Slightly lower hemoglobin levels were noted in those animals fed thyroid but it cannot be stated that this necessarily represents anemia. A consistent observation in this laboratory has been an elevated fasting blood urea level in vit. B_6-deprived rats(2) which has been attributed, on the basis of *in vitro* studies, to an increased rate of production by liver tissue(9). In the present experiment this elevation is reconfirmed. However, with dietary provision of desiccated thyroid, the fasting blood urea level of deprived animals was restored to normal although that of controls was not altered. It is of interest that thiouracil administration and thyroidectomy did not eradicate this difference but rather elevated the fasting blood urea levels in both deprived and control groups(1). Further, thyroid-feeding depressed fasting blood sugar levels in both groups which might be indicative of the general catabolic state suggested by the data on body weight and carcass fat. No marked effect of vit. B_6 deprivation on fasting blood amino nitrogen levels was noted which is in accord with earlier observations(2). Feeding of desiccated thyroid was without apparent effect on blood amino nitrogen levels.

The dosage of desiccated thyroid used in this study was sufficient to cause decreased body weight gains and to markedly lower the amount of carcass fat in the experimental animals. Moreover, the dosage was similar to that which Houssay(10) demonstrated to be sufficient to counteract the effects of thyroidectomy and which Weiss *et al.*(11) showed to be non-toxic for rats. Cursory examination of the animals in handling showed them to be hyperexcitable which again suggests that a hyperthyroid state was attained under these experimental conditions.

The earlier investigation(1) of thiouracil administration and thyroidectomy demonstrated that the biochemical defects of vit. B_6 deprivation are not altered by hypothyroidism. The present study suggests the same conclusions for hyperthyroidism. Thus these

biochemical defects are apparently not caused or prevented by alterations in thyroid activity. This suggestion is supported by the finding of a normal basal metabolic rate in deprived animals on comparison with pair-fed controls(1).

Summary. On administration of desiccated thyroid in the diet, the syndrome of vit. B_6 deprivation in the rat was not markedly altered. One apparent effect of thyroid-feeding was to eliminate the customary elevation in fasting blood urea consequent to vit. B_6 restriction. This and an earlier study suggest that the biochemical defects of vit. B_6 deprivation in the rat are not caused or prevented by alterations in thyroid activity.

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Prolongation of Serum Prothrombin Time After Heparin. (21121)

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In the performance of the prothrombin consumption test the prothrombin time is determined 15, 30, 45, and 60 minutes following coagulation and centrifugation(1). The prothrombin time of *freshly* coagulated and centrifuged blood is not considered a true measure of prothrombin since considerable free thrombin occurs in the serum(1). The antithrombic effect of heparin was, therefore, expected to influence the prothrombin time of fresh serum. In order to confirm this hypothesis, a comparison of the prothrombin consumption tests was carried out before and after administration of heparin, with particular emphasis upon the prothrombin time of fresh serum. Further evidence of a thrombin effect as well as thromboplastin effect in fresh serum was obtained by the interaction of calcified serum upon plasma at various time intervals before and after administration of heparin.

Material and methods. Twenty normal individuals were divided into 2 groups of 10 each. Prothrombin consumption tests according to the procedure of Quick(1) were performed in all 20 individuals just before and one hour after the intravenous administration of heparin. The first group of 10 individuals received 50 mg of heparin while the second group received 75 mg of heparin each. Deprothrombinized oxalated plasma was obtained with barium sulfate according to Rosenfield and Tuft(2). In view of the emphasis in this study upon the prothrombin time of fresh serum the tests were not performed at intervals of 15, 30, 45, and 60 minutes following coagulation as described by Quick, but immediately after coagulation (zero-hour) as well as 30, 45, and 60 minutes later. A. *Prothrombin consumption test.* Five ml of venous blood were placed into a test tube in a water bath at 37°C. When coagulation was complete the tube was centrifuged for one minute at top speed. The prothrombin time of the