ment of adipose tissue for each hormone, were characterized by proportional increments of REP (Fig. 5).

Discussion. Previous reports have indicated that the increase of adipose tissue membrane resting electrical potential (REP) associated with insulin is a specific effect(1,2). The present data further confirm this conclusion. Other protein, including gelatin, human serum albumin, and insulin antibody exert no significant effect upon REP. Insulin antibody, in concentrations of 1/1000-1/4800, significantly blocks the effect of insulin, in concentrations of 100-1000 micro-units/ml, upon adipose tissue REP.

Epinephrine and norepinephrine, in at least one concentration between 0.1 and 10 gamma/l, significantly increased adipose tissue REP in each of 3 rats tested. There may be an important relationship between these hormones and intracellular substrate which, according to Maffly and Edelman(5), provide energy required to maintain membrane transport systems. It is possible that these hormones may operate in a similar manner to elevate REP. The fundamental interrelationships of electrical properties, transport mechanisms, and intracellular metabolism of the cell are emphasized by these studies.

Summary. Adipose tissue transmembrane or resting electrical potential (REP) is not altered by gelatin, serum albumin, or insulin antibody. Elevation of adipose tissue REP by insulin is blocked by insulin antibody. Epinephrine and norepinephrine significantly increase adipose tissue REP.

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Effect of Epinephrine on Metabolism of Thyroxine by Pituitary and Brain. (29150)

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After injection of I^{131}-labeled thyroxine (T4), triiodothyronine (T3) has been found in chromatograms of the pituitary gland (1-3) and of mouse pituitary tumors(4). Ford and Gross(5) reported the absence of T3 in the organs of the central nervous system (CNS) after injection of I^{131}-labeled T4. More recent publications have claimed, however, the presence of a compound with the same mobility as T3 in the brain, medulla, and spinal cord. The absence of T3 in the optic nerve was noted after intraperitoneal injection of I^{131}-labeled thyroxine(6).

The present study confirms the later findings(6), and provides additional information regarding the effects of epinephrine on the metabolism of I^{131}-labeled thyroxine in various organs. Radiothyroidectomized mice were injected with I^{131}-labeled T4 plus epinephrine, and chromatograms of the brains were made 30 minutes and 4 hours later. At 4 hours, a high percentage of a compound with the same mobility as T3 was found.

Materials and methods. Forty female Swiss white mice, body weight 20 $\pm$ 1 g, were kept at a constant room temperature of 22°C. For 10 days they were fed the Remington low-iodine diet and were given distilled water to drink. Then they were radiothyroidectomized by intraperitoneal in-

TABLE I. Percentage Distribution of Radioactivity in Brain, Pituitary, Kidney, and Serum 30 Minutes After Injection of I^{131}-Labeled T4.

	Group A—Without epinephrine				Group C—With epinephrine			
Tissue	O	I	T4	T3	O	I	T4	T3
Brain	19*	32.0	48.0	0	16	16	64.3	3.3
	0†	55.5	44.5	0	7	8.5	76.0	8.0
Pituitary	0	44.0	23.0	13	12	47.0	28.0	15.0
	12	53.0	21.0	12	4	39.0	39.0	17.0
Kidney					12	17	71	0
	0	52.0	43.7	0	3.5	18.0	76.5	0
Serum	0	25.0	75.0	0	0	20.0	80.0	0
	0	19.5	80.5	0	0	15.0	85.0	0

O, Origin; I, Iodine; T4, Thyroxine; T3, Triiodothyronine.
* Percentage observed in 1st exp.
† " " " 2nd "

jection of 50 μc of I^{131}, were placed on a regular diet, and were given tap water to drink.

One month later, the 40 mice were divided into 4 groups of 10 animals each, and every animal was injected intraperitoneally with 0.25 μg (1.3 μc) of I^{131}-labeled T4 as 0.1 ml of a 2.5 μg/ml solution freshly obtained from Abbott Laboratories, Oak Ridge, Tenn. Immediately afterward, every animal in groups C and D was also injected subcutaneously with 25 μg of epinephrine as 0.05 ml of a 500 μg/ml solution.

Thirty minutes later, the mice in groups A and C were anesthetized with pentobarbital, their abdomens were opened, and the animals were totally bled from the inferior vena cava by means of a heparinized syringe with an 18-gauge needle. The mice in groups B and D were handled in the same manner, but the bleeding was done 4 hours after the hormone injections, in view of the possibility that the time factor might influence the metabolism of thyroxine by the tissues. The brains, pituitaries, and kidneys were immediately removed and placed on dry ice. Total weight and radioactivity were recorded for the brains, and likewise for pituitaries and kidneys. The experiment was performed twice.

Sera were extracted according to the method of Block *et al*(7), and were chromatographed. The pooled tissues of each of the organs under study were separately chromatographed according to the method of Rogers and Woodhall(8), using a 5:1:4 mixture of *tert.*-amyl alcohol, 1 N ammonium hydroxide, and water as solvent. The standards were sprayed as in previous studies(4), and the principal peaks coincided closely with those of radioactive I^-, T4, and T3. Radioactivity was counted in a well-type scintillation counter for 5 minutes.

Results. Shortly after injection of epinephrine, the mice in groups B and D became restless, and had moderate tachycardia and some degree of pilar erection. The symptoms had almost completely subsided 30 minutes later, and were entirely gone at 4 hours. Such symptoms did not occur at all in groups A and C, which received no epinephrine.

At autopsy, no remarkable differences between any of the groups of mice were detected macroscopically in the organs under investigation. Chromatographic studies were conducted at 30 minutes and 4 hours after hormone injections. Peaks of radioactivity, which coincided with those for I^-, T4, and T3 on the standards, were located at 8-10 cm, 15-18 cm, and 20-23 cm from the origin, respectively.

Chromatographic studies at 30 minutes. In Group A, brains, kidneys, and sera showed I^- and T4, and pituitaries showed I^-, T4, and T3. In Group C, kidneys and sera showed I^- and T4, and pituitaries and brains showed I^-, T4, and T3. A small peak of radioactivity (3.7-8.0%) was present in the T3 area for Group C, but not for Group A (Table I). Fig. 1 shows a chromatogram of the brains and pituitaries from Group C. The ordinate shows counts per minute (cpm), and the abscissa centimeters from the origin (cm). Peaks of radioactivity corresponding

TABLE II. Percentage Distribution of Radioactivity in Brain, Pituitary, Kidney, and Serum 4 Hours After Injection of T4.

Tissue	Group B—Without epinephrine				Group C—With epinephrine			
	O	I	T4	T3	O	I	T4	T3
Brain	21.0*	28.0	32.0	1.7	18	12.0	27.0	43.0
	28.0†	39.0	24.0	7.5	0	19.0	22.0	52.0
Pituitary	11.0	24.0	43.0	6.0	10	39.0	39.0	12.0
	16.0	25.0	48.0	10.0	5	44.0	28.0	22.0
Kidney					20.0	27.0	63.0	0
	24.0	22.0	37.0	0	14.0	31.0	52.0	0
Serum	0	25.0	75.0	0	0	19.0	81.0	0
	0	30.0	70.0	0	0	15.0	85.0	0

O, Origin; I, Iodine; T4, Thyroxine; T3, Triiodothyronine.
* Percentage observed in 1st exp.
† " " " 2nd "

to I^-, T4, and T3 are well delimited, and coincide in both tissues.

Chromatographic studies at 4 hours. In group B, brains and pituitaries showed I^-, T4, and T3, and kidneys and sera showed I^- and T4. In Group D, brains and pituitaries showed I^-, T4, and T3, and kidneys and sera had peaks of radioactivity in the I^- and T4 areas (Table II). An interesting finding was a high percentage of a compound with the same mobility as T3 (43-52%) in Group D mice. The percentage of this compound was much less (1.7-7.5%) in the Group B mice, which did not receive epinephrine. Fig. 2 shows a chromatogram of pituitaries and brains from Group D. There is a high peak of radioactivity in the T3 area for the brain, more prominent than that for the pituitary.

Interpretation. Under the conditions adopted, a compound with the mobility of T3 was present in chromatograms of brains and pituitaries after injection of I^{131}-labeled thyroxine, and the injection of epinephrine markedly increased the percentage of the compound in brain tissue at 4 hours.

Discussion. The coincidence of the T3 peak of radioactivity for brain and pituitary in the present study confirms previous findings(6). Of greater importance, however, is the demonstration of an increase in such a peak 4 hours after injection of I^{131}-labeled thyroxine plus epinephrine.

The possibility that epinephrine might enhance the conversion of T4 to T3 in the brain, or favor the trapping of circulating T3 there, has not been explored before. The results of our study suggest that the relation-

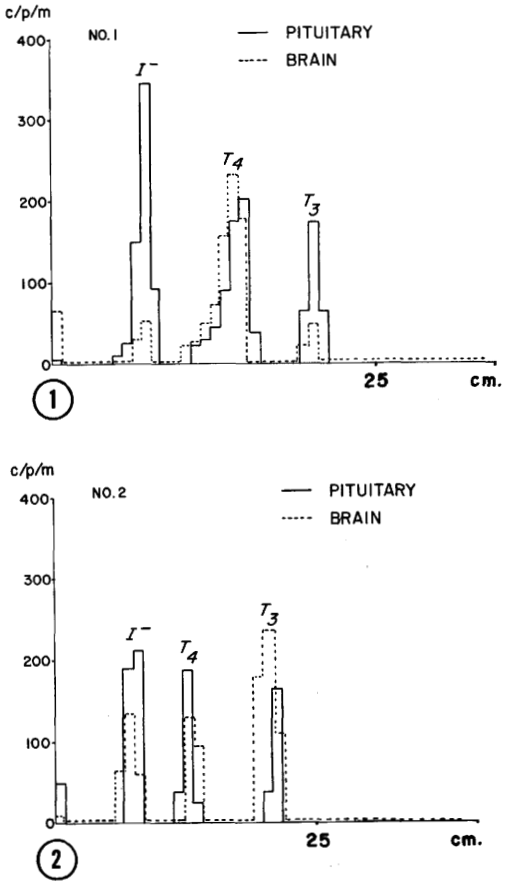

FIG. 1. Radioactivity measured by strip counting of chromatograms of brain and pituitary, 30 min after injection of I^{131}-labeled thyroxine plus epinephrine.

FIG. 2. Radioactivity measured by strip counting of chromatograms of brain and pituitary, 4 hr after injection of I^{131}-labeled thyroxine plus epinephrine.

ship of epinephrine to thyroid metabolism is different than it has previously been thought to be.

Brown-Grant(9,10) demonstrated that epinephrine affects the uptake and release of thyroxine by the thyroid gland. No clear explanation was provided, however, for the changes induced by epinephrine in thyroid function. A possible reciprocal balance between adrenocorticotropic hormone (ACTH) and thyrotropic hormone (TSH) has been postulated(11), but this hypothesis was soon abandoned, because similar changes were observed in the thyroid gland after administration of epinephrine to adrenalectomized rabbits(12).

On the basis of the present study, epinephrine apparently increases conversion of T4 to the faster-acting T3, which controls the release of TSH by inhibiting the hypothalamus (13), thus decreasing the levels of TSH and subsequently the uptake and release of iodine by the thyroid gland. Further experiments are under way to confirm present findings and to extend the study of the influence of epinephrine on the metabolism of T4 by various tissues.

Summary. The influence of epinephrine on the metabolism of thyroxine (T4) by brains, pituitaries, kidneys, and sera of I^{131}-radiothyroidectomized mice was studied with I^{131}-labeled T4. All mice received I^{131}-labeled T4, and half also received epinephrine. Several organs were then investigated chromatographically, each at the same two time intervals. Kidneys and sera indicated I^- and T4; brain and pituitaries indicated I^-, T4, and triiodothyronine (T3). These results confirm findings previously reported. This study has also provided evidence that epinephrine increases conversion of T4 to T3 in the brain. For this reason, a new mechanism is postulated to explain how epinephrine might affect uptake and release of I^{131} by the thyroid gland.

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Myocardial Transaminase Following Coarctation of Abdominal Aorta. (29151)

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Glutamic-oxalacetic Transaminase (GOT) has been found to be present in higher concentration in the left ventricle than in the right ventricle of the heart, with lesser amounts present in the left and right atria (1). Elevation of blood pressure in rats by use of desoxycorticosterone acetate (DOCA), resulted in an increased GOT concentration