

These observations make it still more convincing that polymorphonuclear cells are inactive in our experiments in which mononuclears have a marked protective effect.

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### Sympathetic Activity After Prolonged Administration of Thyroxin.

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Innumerable "acute" experiments have been reported in the literature on the effect of thyroid administration on the pressor response of laboratory animals to adrenaline. The results have been in the main ambiguous. In contrast to these experiments, in this work an effort was made to insure a hyperthyroid condition of the animal by the long-continued administration of thyroxin.

Large vigorous cats were selected. Some were first fed Squibb's thyroxin tablets daily and then received daily intravenous injections of synthetic thyroxin (Hoffmann-La Roche). Others received the intravenous injections alone from the beginning of the period of thyroxin treatment. The total amount of thyroxin given per cat varied from 40 to 80 mg. The length of period of thyroxin administration varied from 35 to 57 days. At the end of that time blood pressure tracings were made from the carotid artery in the usual manner before and after the intravenous injection of a constant dose of adrenaline chloride (0.3 cc. per kg. of a 1:100,000 solution in saline). Paraldehyde (1.5 cc. per kg.) given by tube on an empty stomach was used as an anesthetic, as suggested by the work of Luckhardt and Koppányi.<sup>1</sup> Sodium citrate (10% solution) served as an anticoagulant. Since some animals proved resistant to the action of thyroxin, only those were taken for the measurement of the pressor effect of adrenaline that showed a decided loss of body weight, 450 to 1000 gm. (1/3 to 1/7 of the original body weight). Five animals were finally chosen. Thirteen normal cats, treated exactly as those in the thyroxin series except for the administration of thyroxin, served as controls. Averages of the results for both series were as follows:

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<sup>1</sup> Luckhardt, A. B., and Koppányi, T., *Am. J. Physiol.*, 1927, lxxxii, 436.

TABLE I.

	Normal	Hyperthyroid
Average systolic blood pressure range before adrenaline	mm. 134-149	mm. 123-151
Average systolic blood pressure range after adrenaline	157-176	170-187
Average rise in systolic blood pressure after adrenaline	24	46
Average percentage rise in systolic blood pressure after adrenaline	17%	35%

The data clearly show that the same dose of adrenaline produces in the hyperthyroid cat a rise in blood pressure averaging 106% higher than in the normal control animals. Since no alkaline solution of thyroxin was injected into the animals during the measurement of the blood pressure and no artificial respiration was used, there is no reason for assuming that the greater vascular response was due to any increase in the pH of the blood. Neither could we observe any gradual increase of excitability of the autonomic nerves accompanying successive intravenous injections of constant doses of adrenaline as was reported by Lieb and Hyman<sup>2</sup> for pitthed, decerebrated cats maintained by artificial respiration.

A striking result of the faradic stimulation of the peripheral end of the divided splanchnic nerve was a marked dilatation of the pupils of the thyroxin-treated cats. In normal cats splanchnic stimulation produced no such reaction, in agreement with the results of Stewart, Rogoff and Gibson.<sup>3</sup> The hemodynamic response following splanchnic stimulation in thyroxin-treated animals was not markedly different from that shown by the normal controls. The insufficient number of observations on this point, however, does not permit of any definite conclusion.

Electrocardiographic studies before and after the administration of thyroxin seem to show definite changes in the cardiac response of hyperthyroid cats to adrenaline. These will be presented in a later report. Work is still in progress and is being extended to several phases of thyroid-adrenal inter-relationship.

<sup>2</sup> Lieb, C. C., and Hyman, H. T., *Am. J. Physiol.*, 1922-23, lxiii, 60.

<sup>3</sup> Stewart, G. N., Rogoff, J. M., and Gibson, F. S., *J. Pharm. and Exp. Therap.*, 1916, viii, 205.