sponsiveness is in marked contrast to the effect on chloride excretion. In application, the results also suggest that the validity of the standard radioactive iodide uptake studies of thyroid function is not affected by the administration of a mercurial diuretic.

Summary. 1. Meralluride sodium (Mercuhydrin®) had no effect upon the results of the 6 and 24 hour thyroid uptake procedure in 8 euthyroid patients. 2. Renal I^{131} clearances were determined before and after injection of meralluride sodium in 4 patients, and compared with simultaneous chloride excretion data. Radioiodide clearances remained unchanged in the face of a consistent and marked increase in chloride excretion.

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Effect of Colchicine on Eosinophil Count in the Hypophysectomized Rat.* (21136)

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In previous clinical studies(1) it has been observed that 3 mg of colchicine injected intravenously has an eosinopenic effect. On the other hand, it has been shown by various authors(2-4) as well as by our own investigations, that there is no demonstrable effect on the excretion of the urinary corticoids following colchicine administered orally or parenterally. Thus it appeared possible that this eosinopenic response to colchicine was not mediated through the pituitary-adrenal relationship. The purpose of this study has been to determine whether colchicine administration causes eosinopenia both in normal and in hypophysectomized rats.

Methods. The experiment was run with 22 young male rats of the Wistar strain. Eleven were hypophysectomized. Six of the hypophysectomized and 7 of the normal rats were injected subcutaneously with 1 ml of colchicine* in physiological saline solution in a concentration of 0.2 mg per ml. Control groups were similarly injected with 1 ml of physiological saline solution. For counting the eosinophils we employed the staining method of Speirs and Meyer(5). The stain contained 10 ml phloxine solution (1%), 1 ml diethylene glycol, 4 drops of alconox (0.5%), and 30 ml distilled water. To this solution 8 ml of acetone were added just before use. The eosinophils were counted before injecting colchicine or saline, and were again counted

TABLE I. Eosinophil Response to Colchicine and to Saline Injections in Intact Rats.

	Eosinophils/mm³ following No. of hr		
	0	4	48
	Saline inj.		
	182	200	500
	225	25	113
	225	138	578
	273	100	617
Mean value	226	116	452
	Colchicine inj.		
	475	38	250
	317	38	38
	13	13	100
	75	163	113
	100	57	113
	88	138	63
	1125	210	125
Mean value	313	94	115

* The colchicine used in these experiments was kindly provided by Dr. Glenn Ullyot, Smith, Kline and French Laboratories, Philadelphia.

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TABLE II. Eosinophil Response to Colchicine and to Saline Injections in Hypophysectomized Rats.

	Eosinophils/mm³ following No. of hr		
	0	4	24
		Saline inj.	
	140	Blood clotted	80
	168	325	200
	400	450	400
	350	385	125
	125	550	150
Mean value	237	427	191
		Colchicine inj.	
	225	125	—
	425	275	—
	140	25	—
	340	125	—
	150	125	—
	140	25	—
Mean value	237	117	

4 and 24 to 48 hours after the injections. Two series of animals were studied on different days but at the same hours of the day.

Results. In Table I are reported the results of injection of colchicine, 1 mg/kg in normal rats. The fall in eosinophil count after injection of colchicine is striking, with a difference of the means of minus 70%. The number of eosinophils continued low after 48 hours.

Intact rats injected with saline showed a decrease of 49%. This eosinopenic response to the stress of handling and tail clipping is well known, and is in agreement with the results of Speirs and Meyer(5).

The data of Table II show a 50% decrease in the number of circulating eosinophils in hypophysectomized animals 4 hours following the injection of colchicine (1.6 mg/kg of rat). All the hypophysectomized animals died from this dose of colchicine between 4 and 24 hours after injection. Since all animals were given 1 ml of the colchicine solution, the hypophysectomized rats received a relatively higher dose than did the intact rats because of their lesser body weight. We have noticed only a slight difference in the values of the initial eosinophil counts between hypophysectomized and normal intact male rats.

In hypophysectomized rats injected as controls with 1 ml saline, the eosinophils increased to an average of 81% above pre-treatment levels.

Discussion. The results of these studies show that the injection of colchicine is followed by a decrease in eosinophil count of 50% in hypophysectomized and 70% in normal intact male rats. It was interesting, on the other hand, to observe an increase of 81% in the eosinophil counts of hypophysectomized rats following saline injection in contrast to a decrease of 49% in normal intact rats. The decrease in eosinophil counts in the normal intact male rats following injection of either colchicine or saline is thus in contrast to the effect of these 2 injections in the hypophysectomized rats. The decrease in eosinophils following colchicine administration in the hypophysectomized rats suggests that the presence of the hypophysis is not essential to this pharmacological phenomenon. It is, perhaps, of even more interest to observe that the greater decrease in eosinophils following colchicine occurred in intact animals which, due to their relatively greater body weight, received 1.0 mg instead of 1.6 mg/kg of body weight.

Since, in these experiments, a 50% decrease in the circulating eosinophils occurred in hypophysectomized rats, it is evident that the decrease of eosinophils in the rat is not necessarily mediated by the pituitary gland. The eosinopenic effect of colchicine in the hypophysectomized male rat may be due to direct adrenal stimulation or to a peripheral effect not involving the pituitary-adrenal interrelationship. To clarify this question similar studies are being performed in adrenalectomized animals.

Summary. 1. Colchicine injected into normal male rats caused a 70% decrease in circulating eosinophils. Hypophysectomized rats similarly injected with colchicine showed a 50% diminution. 2. Saline injections in normal rats caused a 49% decrease in eosinophils, while in hypophysectomized rats there was an increase of 81%. 3. The eosinopenic response to colchicine appears, in the main, to be due to a direct adrenal stimulation or to a direct peripheral effect.

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Blood Clotting Time and Tissue Mast Cell Number of the Bat (*Myotis lucifugus*) in Different Physiological States. (21137)

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The bat is one of a number of mammals which in nature pass the winter in a state of dormancy characterized by low rates of respiration, heart beat, blood flow and oxygen consumption(1). Laboratory studies have shown that the resting bat is essentially poikilothermic(2,3) and that its rates of blood flow(3) and oxygen consumption(2,3) vary directly with the ambient temperature. At low temperatures (5-10°C) the red blood cells traverse the vessels in clumps and rouleaux, but thrombi and stoppage of flow are not seen(3). At high temperatures (20°C and upward) blood flow is much like that found in other mammals. Since intravascular clotting and thrombus formation do not accompany the sluggish circulation of the dormant bat, we have tried to establish whether different blood clotting times characterize the profoundly different physiological states which may be induced in the bat. We have already noted(4) that clotting time is markedly prolonged when the summer bat is exposed to cold. The present paper is concerned with the extension of these studies to winter bats and with the possibility that changes in clotting time might be accompanied by changes in the tissue mast cells which are thought to store and release the anti-coagulant, heparin(5,6).

Methods. *Small brown bats* (*Myotis lucifugus*) were collected in the winter from a cave in northern Illinois and in the summer from the attic of a farm house in southern Indiana. Blood clotting times were determined (in all cases at 23°C) by the capillary tube technic on samples obtained by puncturing a large vein in the web between the hind legs and from the cut carotid artery (the clotting time of the blood from each source was essentially the same). In each bat counts were made of the number of mast cells in single 12 μ thick sections of duodenum magnified 264x. The tissues were fixed in absolute alcohol and stained with toluidine blue. Studies were carried out at the time of collection and later in the laboratory at various times after placing summer bats in a refrigerator (5°C) and winter bats in an air conditioned room (23°C).

Results and discussion. In nature markedly different clotting times distinguish the summer bat from the winter bat (Table I). That of the former is short as in the homoiothermic mammals, while that of the latter is very long as in the aestivating ground squirrel(7) and in the hibernating hedgehog(8) and hamster(9). We interpret these data to indicate the existence of an adaptive mechanism which (a) prevents thrombus formation under the conditions of slow blood flow found in the dormant winter bat and (b) allows for rapid blood coagulation in the active summer bat. Table I also indicates that such a mechanism operates upon exposure of summer and winter bats to different ambient temperatures. Summer bats become dormant upon exposure to cold and show a marked prolongation of clotting time, while dormant winter bats awaken upon exposure to 23°C and manifest