as is usually the case in myelogenous leukemia. In the small number of cases studied the myeloid hyperplasia tends to return to "normal" when the thyrotoxicosis is removed.

Aspirated sternal marrow from individuals in a myxedematous state is markedly hypoplastic. Sufficient feeding of desiccated thyroid or injection of thyroxine causes an increase in the nucleated cells of this marrow to "normal".

These findings indicate that the thyroid secretion has a regulatory effect on quantity and quality of the bone marrow, as evidenced by differences found in the quality and quantity of aspirated sternal marrow.

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The Adrenals in Experimental Hypertension.

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In preliminary experiments by Goldblatt,[1] bilateral adrenalectomy in dogs appeared to interfere with the development or maintenance of the hypertension which follows experimental production of renal ischemia. Similar results were obtained by Blalock and Levy[2] and by Page.[3] On the other hand, Collins and Wood[4] concluded that, "It is unlikely that the adrenal cortex is involved specifically in the etiology of experimental renal hypertension other than in the sense that the cortex is important in the maintenance of blood pressure in normal as well as in hypertensive states."

Our experiments show that this form of experimental hypertension can exist in the complete absence of the adrenal glands, in untreated animals. Seven dogs were subjected to complete bilateral adrenalectomy in addition to constriction of the main renal arteries by means of Goldblatt clamps. Four of these animals were treated

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1 Goldblatt, Harry, *Ann. Int. Med.*, 1937, **11**, 69.

2 Blalock and Levy, *Ann. Surg.*, 1937, **106**, 826.

3 Page, *Am. J. Physiol.*, 1938, **122**, 352.

4 Collins and Wood, *Am. J. Physiol.*, 1938, **123**, 224.

by administration of salt (sodium chloride and citrate, occasionally also bicarbonate) alone or in addition to a commercial extract of adrenal cortex.† In 3 experiments no treatment whatever was employed and the use of salt for seasoning food was avoided. All the blood pressure observations were made by the carotid loop (Van Leersum) method. The cuff was adjusted by the same observer in every case and not less than 12 readings were recorded each time.

Following excision of the second adrenal, in hypertensive dogs, a decided fall of blood pressure sometimes occurs within a day. We have observed a similar fall of pressure in some hypertensive animals after operations other than adrenalectomy. Our experience has shown that return to hypertensive levels of pressure can occur in such adrenalectomized dogs without administration of salt or adrenal extract. The rise in pressure, therefore, cannot be attributed, with confidence, to treatment when given.

In the 3 untreated adrenalectomized dogs decisive hypertension was maintained in the complete absence of both adrenals. Two of these animals, whose average normal systolic blood pressure was approximately 130 mm and 135 mm Hg., respectively, were subjected to unilateral adrenalectomy and ipsilateral constriction of the renal artery at one operation; later, the same was done on the opposite side. Both dogs succumbed on the fourth day after the second operation, the blood pressure having reached 234 mm and 198 mm, respectively.

The average normal blood pressure in the third of the untreated adrenalectomized dogs ranged between 150 mm and 160 mm. One adrenal was excised and the corresponding renal artery was constricted. Later, the renal artery on the opposite side was constricted. Still later, while marked hypertension was present, the second adrenal was excised. The dog lived into the ninth day after the complete adrenalectomy. No significant change in the hypertensive level of blood pressure occurred until 2 days prior to the death of the animal. The pressure reached 264 mm Hg. on the second day after the adrenalectomy; it was still approximately 200 mm on the eighth day, when the dog was semi-comatose and obviously moribund.

Since the hypertension which follows production of renal ischemia was maintained in untreated, completely adrenalectomized animals, we are led to conclude that neither the cortex nor medulla of the adrenal plays a significant rôle in the etiology of this form of experimental hypertension.

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