themselves completely through swimming (resulting in contracted spleens). The total hemoglobin of the animals of the relaxed and contracted spleens were determined after having placed the extirpated organs into a measured volume of dilute adrenalin solution. Thus a quantitative designation of the function of the spleen as a reservoir for red blood cells was permitted. Our results are listed in Table I.

TABLE I.

Animal group	No. of animals	Hemoglobin in spleen in % of total hgb.	Hgb. of blood expelled from the extirpated spleen by adrenalin:	
			in % of total hgb.	in % of spleen hgb.
A. Chloralose poisoning	10	8.1	3.3	40.3
B. Ether narcosis	20	3.4	0.56	16.6
C. Exhaustion	10	1.7	0.2	11.5

Conclusions. 1. The spleen, though not alone in this function, is the most important blood reservoir in the dog. 2. Chloralose effects storage of red blood cells also in the spleen of the albino rat, while exhaustion leads to ejection of erythrocytes from this organ. The ability of the rat spleen to form a depot of red blood cells is very slight. 3. No generalizations concerning the function of the spleen as a reservoir of red blood cells can be permitted because of the great variability of its function in different animals.

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Influence of the Adrenal Glands on the Contractility of the Spleen.

E. HAAM AND H. S. THATCHER. (Introduced by R. Ashman.)

From the Department of Pathology, School of Medicine, University of Arkansas, Little Rock.

The purpose of the experiments was to determine the regulatory mechanism responsible for splenic contraction and dilatation. Tournade and Chabrol[1] believe in a combined nervous and hormonal control of the splenic contractions and Izquierdo and Cannon[2] observed no erythrocytosis after exposure to low oxygen tension in an

[1] Tournade and Chabrol, *Compt. rend. Soc. d. Biol.*, 1924, **90**, 835.

[2] Izquierdo and Cannon, *Am. J. Physiol.*, 1928, **86**, 545.

animal in which the medullary substance of the adrenal glands had been destroyed.

In 20 normal dogs under chloralose narcosis, the hematocrit curve of the peripheral blood was studied after the injection of adrenalin, after short asphyxia and after bleeding. The dogs were then adrenalectomized (2 stage operation) and on 11 of the animals which survived adrenalectomy for more than 2 days, the same tests were repeated and the results compared. It was found that the hematocrit curves after adrenalin injection show no or only a slight decrease in their maximal elevation after adrenalectomy. In contrast to this, the hematocrit values after brief asphyxia and after bleeding did not show the elevation found in normal dogs. In Fig. 1 the curves of hematocrit determinations of an adrenalectomized dog after adrenalin injection and after short asphyxia are shown. Fig. 2 records hematocrit curves obtained after bleeding of adrenalectomized and normal dogs.

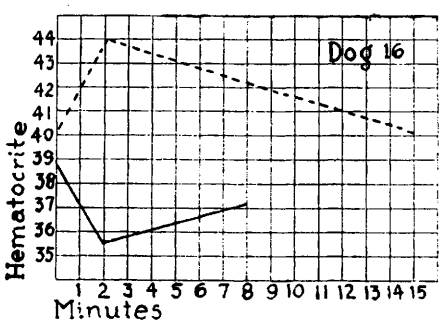

FIG. 1.

The solid line represents the hematocrit curve after brief asphyxia; the dotted line the hematocrit curve after adrenalin injection.

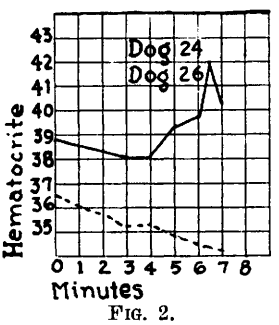

FIG. 2.

The solid line represents normal dog 26; the dotted line adrenalectomized dog 24.

The results seem to point to the assumption that adrenalin is the significant factor responsible for splenic contraction and the resulting erythrocytosis. As it is possible that the polycythemia and the change in the size of the red blood cells after adrenalectomy may be factors which influence the hematocrit curves, one of us is repeating the experiments and studying the contraction of the spleen of adrenalectomized animals *in vivo* by skiagraphic visualization methods.