

foreign protein. We believe these experiments furnish conclusive evidence of the existence of toxic hepatic products in the general circulation, at least during the initial stage of canine anaphylactic shock.

If blood is drawn from the carotid artery of an anaphylactic dog from two to five minutes after throwing the dog into anaphylactic shock, and this blood is immediately transfused into a partially exsanguinated normal dog, no recognizable anaphylactic phenomena usually occur. This finding is in line with the negative results previously reported by Weil.³ If, however, the foreign protein is injected directly into a mesenteric vein of the anaphylactic dog and shock blood is collected as it escapes from the liver, this blood, transfused into a normal dog, will reproduce all of the characteristic features of canine anaphylaxis. One hundred cc. of this hepatic blood, transfused into a 10 kg. normal dog, will cause a typical fall in arterial blood pressure lasting thirty minutes, typical contractions of the urinary bladder and of the gastro-intestinal tract, and a typical, usually complete loss of blood coagulability. The toxic hepatic products in themselves, therefore, are apparently sufficient to account for all of the observed anaphylactic phenomena.

The shock in normal dogs connected with an anaphylactic liver is less prolonged than the shock in intact anaphylactic controls. The toxic hepatic products, therefore, are presumably not the only factors operative in canine anaphylaxis.

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Changes in glycogen content of the liver in anaphylaxis.

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Changes in the glycogen content of the liver in canine anaphylactic shock were followed by routine histological and chemical methods.

³ Weil, R., *J. Immunol.*, 1917, ii, 399.

The normal canine liver has a fairly constant glycogen content. Stained by Best's carmine method, the parenchyma is usually seen to be fairly well filled with red granules. The granules are usually largest and most numerous in the central portions of the lobules. Quantitative determinations by the official method adopted by the American Agricultural Chemists show a glycogen content varying from 4 percent to 7 percent of the gross liver weight, an average of 5.3 percent in our series.

During typical canine anaphylactic shock (kymograph control), the hepatic glycogen practically disappears. The central half of each lobule often becomes free from stainable granules within three minutes. The whole liver becomes microscopically glycogen-free by the end of fifteen minutes. Less than 0.01 percent glycogen can usually be isolated from the liver at this stage.

No conclusion is as yet drawn as to the mechanism of this glycogen disappearance, nor as to its bearing on fundamental theories of anaphylaxis. Our findings, however, are in line with the initial hyperglycaemia in guinea pig anaphylaxis, recently reported by Zunz and La Barre.¹

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Preliminary note on the effect of a constant magnetic field on morphogenetic processes.

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It is generally supposed that a *constant* magnetic field has no effect upon life processes. Observers who have dealt with this question seem on the whole to be in quite general agreement that all attempts up to the present to demonstrate an effect have been complete failures, or else the conditions of the experiment have been too poorly defined to demonstrate an effect. In view of such a situation it is clear that any contrary statement of value must be supported by results obtained under clearly defined experimental conditions.

¹ Zunz, E., and LaBarre, J., *Compt. Rend. Soc. Biol.*, 1924, *xc*, 121.