Conclusions. It appears that the nervous control of the renal vascular bed acts to protect that organ to some degree at least from certain noxious agents and removal of this nervous control exposes the kidney to greater injury when toxic materials are injected, and allowing them to reach the kidney in marked concentration.

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A Biological Reaction for Hyperthyroidism.

D. A. WILLIS AND J. M. MORA. (Introduced by W. F. Petersen.)

From the Departments of Surgery and Pathology, University of Illinois College of Medicine.

A few years ago, Dresel[1] described a new biological reaction for hyperthyroidism based on the observation that the serum of toxic goitre patients when injected into mice rendered their livers glycogen poor. It is upon this reaction that we wish to present this preliminary report. Our observations are in accord with those of Dresel, though we have modified the technique of the test somewhat.

Dresel injects the mice, feeds them oats and water for 48 hours, then starves them for one hour before examining the livers for the presence of glycogen. We have observed, however, that even after several hours of starvation with this method, all the mice may still possess glycogen rich livers, or else the results may vary according to the amount of food which the animals have consumed prior to this starvation period. We, therefore, inject the serum, feed carbohydrates and water for 30 hours, starve the mice 16 hours, inject 1.5 cc. of 10% glucose solution and examine the livers 3 hours later. By this method the amount of food ingested by the mice becomes an unimportant factor, since after this starvation period all the mice have glycogen free livers and each is then supplied with a known quantity of glycogen-forming material. The liver, quickly removed from the animal which has been killed by breaking the neck, is ground with sea sand and 5 cc. of hot water, mixed well with 3 cc. of 20% trichloracetic acid, centrifuged and filtered. To 20 drops of the filtrate, one drop of Lugol's solution is added. The presence of glycogen is indicated by a mahogany brown color, while a yellow colored solution indicates little or no glycogen.

[1] Dresel, K., and Goldner, M., *Verhandl. d. deutsch. Gesellsch. f. inn. Med.*, 1929, Kong. 41, 534. Dresel, K., *Deutsch. Med Wnschr.*, 1929, **55**, 259.

We have tested the serum of 20 toxic goitre cases, 10 conditions other than thyroid disease, and 2 cases of "thyroid constitution", having experimented with some hundred and seventy-five mice. We have noted that the toxic serum generally renders the liver of the mouse glycogen poor, while the normal serum leaves the liver rich in glycogen. Serum from patients with so-called "hyperthyroid constitution" or possessing the "Basedow stigma" as Dresel terms it, and presenting rapid pulse, sweating and nervousness but normal basal metabolic rates, may give a similar reaction. We are inclined to believe, however, that by the use of smaller amounts of serum such reactions may become fewer. Some of our experiments tend to indicate that iodides diminish the activity of the serum. There also seems to be some indication that the livers of mice injected with toxic serum form glycogen more rapidly than normally, but as stated above, also lose the glycogen more quickly.

Hyperthyroid serum evidently contains some substance which has the power of driving the glycogen from the liver. Whether this is a substance foreign to the normal organism or only increased in amount in the toxic goitre patient we are not at this time prepared to state, though we feel it is probably the latter.

Because he finds that the action of the thyroxin-like acting substance in the serum is 50 to 100 times as effective as the estimated thyroxin per cubic centimeter of blood serum, Dresel believes that some other substance perhaps tyrosin-like enhances the reaction. We have noted that the liver may become glycogen poor many hours sooner than would be expected from thyroxin action, and feel that the reaction may perhaps be dependent upon a substance derived from some other organ of internal secretion, most likely the suprarenal glands. It is even probable that the toxic serum drives the glycogen from the livers not only through increased oxidation but also through altered permeability.

This biological reaction, so simple and yet so delicate, at least offers the possibilities of: (a) a biological test for borderline cases of hyperthyroidism; (b) another method of approaching the study of hyperthyroidism; (c) a reaction as an aid in controlling experimental hyperthyroidism.