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Renal Denervation. II. Vascular Changes Induced in the Intact and Denervated Kidney by Rattle Snake Venom.*

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In a recent report[1] we described a method of antimortem injection to demonstrate in detail the renal vascular bed by roentgenograms and demonstrated the changes seen when the method was applied to the denervated kidney. We will now show the effect of rattle snake venom on the normal and denervated kidneys as demonstrated by the same means. Noguchi[2] described rattle snake venom as a vascular poison resulting pathologically in vascular paralysis and capillary hemorrhages into the tissues. Anuria is a frequent clinical symptom.

We used young dogs throughout. The technique already described[1] was employed in denervating one kidney. After periods exceeding 2 weeks rattle snake venom was injected intravenously. It was found that the minimum lethal dose was above 0.5 of the dry venom per kilo body weight. The kidneys were injected with bismuth oxychloride at intervals up to 48 hours after the administration of the snake venom. The resulting effect varied in degree depending on the dose and the interval elapsing between the injection of the venom and sacrificing of the animal. From the standpoint of the x-ray findings there was profound vascular damage as indicated by beading of the vessels and obliteration of a large portion of the vascular bed varying in degree with the size of the dose. The effect was uniformly more profound in the denervated kidney.

Microscopically the kidney tissue displayed congestion of the capillary tufts, dilatation of the interstitial blood spaces and in a few instances extravasation of blood. Varying degrees of tubular degeneration were noted. The changes seen in the histological section were patchy in distribution with intervening areas of relatively normal tissue. More or less marked endothelial swelling of the arterioles was common. Again the evidences of the action of the venom were more pronounced in the denervated than in the normal kidney.

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1 In press.

2 Noguchi, Carnegie Institute of Washington, 1903, 106-141.

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.

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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.