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Renal and Vascular Responses to Epinephrine Injections in Glomerular and Aglomerular Fish.

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In a previous publication[1] it was reported that epinephrine infusions in the dog resulted in either an oliguria or a polyuria, depending on the rate of infusion. From the work of Richards and Plant[2] and Winton[3] the hypothesis was advanced that the dilute epinephrine in the blood stream resulting from a slow infusion constricted the efferent arterioles, causing a polyuria; but with a more rapid infusion and a resulting higher concentration the afferent, or both the afferent and efferent, arterioles were constricted, causing an oliguria. The present experiments were designed to determine the rôle of the glomerulus in the urinary response to epinephrine.

Seventeen experiments were carried out on 15 toadfish *(Opsanus tau)*, an animal possessing aglomerular kidneys.[4] Twenty-eight experiments were performed on 24 puffers *(Spheriodes maculatus)* which possess glomerular kidneys. The animal was anesthetized with urethane in sea water and after anesthesia had occurred it was transferred to a more dilute urethane solution. A slow stream of oxygen bubbled through the mouth and out the gill slits to prevent asphyxia. One ureter was cannulated and the urine flow was determined by frequent observations of the movement of the meniscus in the cannula. The arterial blood pressure was determined in a number of experiments by means of a Hg manometer, 10% sodium citrate being used as the anticoagulant. The gonadal vein was cannulated for the injection of the solution. After determining a normal urine rate dilute sea water equivalent in volume to that of the epinephrine solution to be injected was given by vein for the control injection. The volume never exceeded 1 cc.

In the toadfish, doses of epinephrine varying from 67 to 6 micrograms per 100 gm. body weight had no specific effect on the urinary rate. The blood pressure increased, in one experiment the increase

1 Toth, L. A., *Am. J. Physiol.*, 1937, **119**, 140.

2 Richards, A. N., and Plant, O. H., *Am. J. Physiol.*, 1922, **59**, 191.

3 Winton, F. R., *J. Physiol.*, 1931, **73**, 151.

4 Marshall, E. K., *Bull. Johns Hopk. Hosp.*, 1929, **45**, 95.

was from 12 to 18 mm. Hg and lasted for 40 minutes. The heart rate usually showed a slight increase after both the epinephrine and the control solutions, the increase not exceeding 10 beats per minute. In contrast, the glomerular kidney responded with a tremendous polyuria (as great as 500× the control rate) to doses of epinephrine that ranged from 242 to 3 micrograms per 100 gm. body weight. The larger increases in urinary rate after epinephrine occurred in those experiments in which the control rate was low. A significant and prolonged rise in the blood pressure occurred after the epinephrine, the rise in one experiment being from 6 to 14 mm. Hg and lasting for approximately 80 minutes. The heart rate fell markedly after the injection of the drug (from 120 to 60 beats per minute in the above experiment) but gradually rose to the control level. This fall in heart rate was interpreted by Lutz[5] as the response of an unbalanced parasympathetic mechanism in an organ lacking a sympathetic accelerator innervation. Control injections did not change the heart rate or blood pressure significantly.

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On the Nature of Insulin Convulsions.

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With the injection of doses of insulin just adequate to induce convulsions, the animal will usually exhibit only clonic convulsions, but if twice that dosage be given to this same animal, tetanic convulsions usually result. If the low sugar level be the sole cause of the convulsions, then why would their character change with the increased dose? With doses of insulin adequate to render an animal convulsive, or even sub-convulsive, animals, as well as man, exhibit evidence of a functionally modified nervous system long before the true blood sugar level has fallen to zero and before any convulsions appear. Some of the evidences of this functional modification are marked salivation, profuse sweating, pupillary changes (dilation just before the onset of the convulsions and hippus often accompanied by nystagmus during the seizure), marked restlessness and increased sensitivity to stimuli such as light or noise.

[5] Lutz, B. R., *Am. J. Physiol.*, 1930, **94**, 135.