The conclusion seems warranted that inclusion blennorrhea and swimming-bath conjunctivitis are essentially the same disease, the former representing the conjunctival response of the infant, the latter the conjunctival response of the adult to the same etiological agent. Thus, the studies reported on this aspect of the disease by Thygeson receive confirmation from the present observation.

9333 P

Responses of the Kidney to Reflex and Direct Stimulation of the Splanchnic Nerve.

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The intravenous administration of epinephrine, as is well known, results in reduction of the kidney volume by reason of the vasoconstrictor action of the drug. Reversal of this reaction following the administration of ergotamine tartrate in small doses has been amply demonstrated.[1, 2] The experimental results here reported demonstrate the reactions of the kidney to reflex stimulation of its vasomotor nerves by means of other agents and to direct splanchnic stimulation both before and after the administration of ergotamine tartrate.

The experiments have been carried out on dogs under morphine-urethane and cats under nembutal anesthesia. Acetyl-beta methylcholine chloride was administered to the dogs by means of iontophoresis. In the cats all the drugs used were administered intravenously. In the experiments carried out on the dogs, the changes in kidney volume, blood pressure and urine output were recorded; in those carried out on the cats the records included changes in kidney volume, blood pressure and limb volume.

The typical effects of acetyl-beta methylcholine chloride administered by iontophoresis in dogs is a very short preliminary rise in kidney volume, followed by a fall corresponding to the fall in general blood pressure, and reduction in the urine output until after a few minutes it consists of an occasional drop. The duration of these

[1] Hamet, R., *Compt. Rend. Acad. Sci.*, 1926, **182**, 1045.

[2] Wulp, G. A., and Nelson, E. E., *J. Pharm. Exp. Therap.*, 1931, **42**, 143.

experiments was about one hour. A current of 40 milliamperes was applied for 6 minutes. The output of urine is restored as soon as the blood pressure commences to rise toward the normal. When the blood pressure has returned to normal, the kidney volume increases until it rises above that recorded initially and returns to the initial level at the end of 5 to 10 minutes.

According to Hamet,[3] the initial rise in kidney volume and the secondary rise after the return of the systemic blood pressure to normal are both definite vasodilator responses of the kidney caused by acetylcholine. Acetyl-beta methylcholine chloride was used in these experiments because of its prolonged action and stability. Our results agree with those of Hamet, but if there is a definite vasodilator response to acetylcholine, there probably should be vasodilator fibers to the kidney vessels.

In the experiments carried out on cats similar results were obtained by the intravenous injection of 0.2 cc. of 1-1,000 solution of acetyl-beta methylcholine chloride. The initial rise in kidney volume was not evident, but there was a definite decrease in limb volume, subsequently followed by a slight increase. Kidney volume in the cats reacted in the same manner as it did in the dog.

Stimulation of the distal end of the cut splanchnic nerve causes a rise in blood pressure, a decrease of kidney volume and a rise in limb volume. After ergotamine tartrate the kidney response is reversed. One-half cubic centimeter of a 1-2000 solution of ergotamine tartrate was found to be sufficient to cause the reversal effect upon the kidney vessels but not upon the systemic blood pressure. This dosage corresponds to that found necessary by Hamet,[1] Wulp and Nelson,[2] and Van Dyke,[4] to elicit this reaction on the renal vessels. The minimum dosage necessary to cause the reversal effect on the renal vessels varies widely, from 0.0054 mg. to 0.11 mg. per kilo body weight, but is rarely as high as the latter. Stimulation of the splanchnic nerve after the administration of ergotamine causes a definite increase in kidney volume, but also a slight rise in systemic blood pressure and an increase in limb volume.

With the adrenal glands intact it is almost impossible to produce the reversal effect of epinephrine upon the blood pressure no matter how much ergotamine is injected; consequently, the right adrenal was ligated and the left either ligated or removed. Even after this has been done it is difficult to produce a reversal effect when the splanchnic nerve is stimulated at 12 cm. for 10 seconds.

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[3] Hamet, R., *Compt. Rend. Soc. de Biol.*, 1926, **94**, 727.

[4] Van Dyke, H. B., *J. Pharm. Exp. Therap.*, 1926, **27**, 299.

Our results indicate that by the time enough ergotamine has been administered to prevent the rise in blood pressure or to produce the blood pressure reversal effect by stimulation of the splanchnic nerve after ergotamine, the kidney vessels no longer react and are paralyzed so that stimulation produces only a small response which corresponds to the changes in blood pressure.

The reaction of the kidney vessels after small doses of ergotamine cannot be explained at present. Possibly the constrictor fibers to the kidney vessels are more sensitive to ergotamine than the other vessels, or their control over these vessels is less complete, consequently, they require less of this alkaloid to paralyze the endings, thus allowing the kidney vessels to react to adrenalin in the same manner that they do after section of the splanchnic nerve, *i. e.*, by dilatation.

The only direct response of the renal vessels seems to be constriction to adrenalin; all the other responses evidently are passive. The kidney volume increases or decreases as the blood pressure rises or falls.

The results of these experiments seem to indicate that the renal vessels are without vasodilator fibers and that the splanchno-peripheral balance probably plays the most important rôle in governing the vasomotor activities of the kidney and, consequently, kidney volume.

9334 P

Treatment of Serum Serving to Remove Its Serum-Sickness-Causing Activity.

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We have reported previously concerning a reaction appearing on the ears of rabbits[1] subsequent to an injection of either whole horse serum or fractions thereof, which may be considered as analogous to serum-sickness in human beings.[2] In addition, studies concerning the serum-sickness-causing activity of the various serum proteins led to the suggestion that the more important substance concerned in causing serum-sickness may be some agent other than

[1] Fleisher, M. S., and Jones, L., *J. Exp. Med.*, 1931, **54**, 597.

[2] Fleisher, M. S., and Jones, L., *J. Immun.*, 1933, **24**, 383.