THE BLOOD SUPPLY OF THE KIDNEY. V. THE INFLUENCE OF THE VAGUS NERVE UPON THE VASCULARITY OF THE LEFT ORGAN.*

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It has been demonstrated by direct measurements of the blood-flow[1] that the kidneys receive a very effective vaso-motor supply by way of the greater splanchnics. The question whether the blood-vessels of these organs are also innervated by the vagus nerve remains, however, still rather unsettled, in spite of the fact that several investigations pertaining to it have been made. Thus Cohnheim and Roy[2] concluded upon the basis of oncometric determinations that the nerves just mentioned are devoid of vaso-motor fibers for this particular circuit. The problem was again dealt with plethysmographically by Bradford[3] and Walravens.[4] These authors also deny the existence of such fibers within the vagal pathway.

Masius,[5] on the other hand, holds that fibers of this type are present, because when chloral or atropin are injected, the stimulation of the peripheral stump of the vagus does not produce an inhibition of the urinary secretion; and, furthermore, vaso-constrictory influences cannot well be excluded, because the inhibition, whenever present, continued for a longer time than the fall in arterial blood-pressure. Arthaud and Butte[6] also observed, during the inhibition of urinary secretion, a decrease in the flow of blood from the opened renal vein. It happened at times that long-con-

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[1] See preceding papers of this series in *Arch. f. d. ges. Physiol.*, 1908, cxxiii, 553; 1908, cxxv, 221; 1909, cxxvii, 143, 148.

[2] *Virchows Arch. f. path. Anat.*, 1883, xcii, 424.

[3] *Jour. Physiol.*, 1889, x, 358.

[4] *Arch. ital. de biol.*, 1896, xxv, 169.

[5] *Bull. Acad. roy. d. sc. de Belg.*, 1888, xv, 528; 1888, xvi, 60.

[6] *Arch. de physiol. norm. et path.*, 1890, series 5, ii, 379.

tinued stimulation of the vagus produced a decrease in the flow of urine, while, at the same time, the blood-pressure increased. Schneider and Spiro[7] observed a cessation of urinary secretion without a reduction of the blood-pressure; and again if the vagus was stimulated slowly and the renal nerves were then torn, a belated increase in urinary secretion appeared, such as might have been incited by a vaso-dilatory impulse.

In view of this diversity of opinion, and because an oncometric tracing cannot be regarded at all times as a true indicator of the vascularity of an organ, we have sought to approach the above problem by determining the renal blood-flow directly by means of the stromuhr. This instrument[8] was connected with the left renal vein of larger dogs, in the manner described in detail in the preceding communication.[9] Chloroform and ether were employed for the narcosis.

On account of its effect upon the general blood pressure, excitation of the cervical portion of the vagi could not be resorted to even after the administration of atropin, since a certain disturbance of the vaso-motor mechanism by this drug seems not improbable. We decided, therefore, to stimulate the vagal fibers below the heart, either directly above the diaphragm and centrally to the gastric plexus, or distally to the plexus between the stomach and the supra-renal capsule.

Having opened the cavity of the thorax in the region of the ensiform cartilage, both vagi were divided and their distal ends placed separately in covered electrodes, which were protected, in addition, by a rubber membrane. In stimulating the nervous pathway between the gastric plexus and the ganglia in the vicinity of the left adrenal body, an exact mode of procedure was impossible on account of the great diversity of the fibers found in this locality. The excitation of the vagi was repeated several times in the course of each determination of the renal blood-flow. Various strengths of a tetanic current were employed.

[7] *Ergebn. d. Physiol.*, 1902, i, Abt. 1, 419.

[8] The recording stromuhr of Burton-Opitz was employed. See *Arch. f. d. ges. Physiol.*, 1908, cxxi, 150.

[9] *Arch. f d. ges. Physiol.*, 1908, cxxiii, 553.

The records of the stromuhr in all of the five experiments performed by us do not exhibit variations in the flow greater than those occurring normally, and hence we must accept the view that the vagi do not embrace nerve fibers by means of which the vascularity of the kidney may be altered. Negative results were obtained not only on stimulation of the intra-thoracic portions of the vagi, but also on stimulation of the communicating ramus between the gastric and supra-renal plexuses. In the latter case, to be sure, it is not established that this bridge is formed solely by vagal fibers or their postganglionic terminations, because this pathway might also be used by sympathetic fibers in their passage from the ramifications in the neighborhood of the adrenal body to those upon the ventral and dorsal aspects of the stomach. Two facts, however, seem to speak against such a manner of distribution. In the first place, it has been shown by Burton-Opitz[10] that the vaso-motor nerves of the stomach, derived, apparently, solely from the splanchnic system, ascend by way of the plexus gastro-lienalis. To be sure, stimulation of the peripheral ends of the vagi were followed by changes in the vascularity of the stomach as well as of the intestine, but these alterations, which at times bear a striking resemblance to those of vaso-motor origin, were proved to be dependent upon musculo-motor reactions incited with the help of these nerves. Next, we have the present finding, namely, that this bridge appears to be devoid of vaso-motor fibers for the kidney.

Quite different results were obtained with reference to the afferent character of the vagi. When central excitation of the vagi was resorted to at any one of the points indicated above, we obtained, of course, the well-known inhibition of respiratory activity, as well as very conspicuous rises in arterial blood-pressure. Hence the familiar deduction that the vagi represent largely afferent channels, must also be extended to those fibers which pass between the gastric and supra-renal complexes of ganglia. This route, therefore, seems to be set aside for the reception of those impulses from various intestinal organs which are destined to enter central structures by way of the vagi.

Another question presents itself at this time. Is it possible to

[10] *Arch. f. d ges. Physiol.*, 1910, cxxxv, 205.

produce an active variation in the renal blood-flow reflexly by means of central excitation of the vagi? Both Roy and Bradford have taken oncometric records of the kidney during the stimulation of the central ends of the vagus and sciatic nerves, and both authors agree that the excitations are usually followed by well-marked contractions of the kidney. Bradford characterizes this contraction as sudden and persistent. As the paper in question deals with the innervation of the renal blood-vessels, it may be inferred that the changes in the volume of the kidney serve here as an index for the blood-flow through this organ and are therefore indicative of vasomotor reactions.

With the intention of altering the renal blood-flow reflexly, we have made use of central stimulation of the vagi at the points previously designated and also of central excitation of the cervical portion of this nerve, the nerve on the opposite side being left intact. To test the effects further, we have also performed several experiments in which stimulation of the central end of the divided sciatic nerve was resorted to, the right nerve being the one most frequently tested. In some of the experiments curare was employed in addition to the ether.

In spite of the fact that the stimulations aforesaid have never incited significant reductions in the blood-flow, such as would conform to the well-marked decrease in the volume of the entire organ, described by Bradford, mention must nevertheless be made of certain minor observations. Thus, it seemed remarkable that by far the largest number of stimulations did not produce a change in the volume of flow, even at a time when the very pronounced rises in arterial blood-pressure had fully developed. In the absence of a greater blood-flow through the kidney during the period of increased blood-pressure, we are forced to the conclusion that the renal blood-vessels retain the normal size of the blood-bed tonically. The high blood-pressure does not produce an active or passive enlargement of the blood-bed to accommodate a greater inflow, but causes the blood-vessels to become tonically set, so that the blood-flow remains practically unaltered.

In a previous paper we have proven that profound reductions in the renal blood-flow occur whenever the greater splanchnic nerve is

TABLE I.

Blood-Flow in Left Renal Vein (Experiment IV).

Phase of stromuhr.	Duration of phase in seconds.	Total quantity of blood in c.c.	Quantity of blood per second in c.c.	Blood pressure in mm. Hg.		Procedure.
				Renal vein.	Femoral artery.	
1	11.1	18.6	1.67	12.0	112.6	None.
2	11.4	18.6	1.63	—	—	
3	11.8	18.5	1.56	—	—	Stimulation of central
4	11.4	18.5	1.62	—	118.5	end, right sciatic, 12 cm., 20 seconds.
5	10.6	18.5	1.74	—	131.6	None.
6	11.6	18.5	1.60	—	130.5	
7	10.8	18.6	1.72	—	—	
8	11.4	18.6	1.63	—	116.8	
9	12.0	18.9	1.57	—	—	
10	12.9	19.0	1.53	—	113.2	
11a	4.9	8.6	1.55	—	—	Stimulation of central
11b	9.8	10.9	1.11	10.0	118.0	end, right sciatic,
12a	8.1	10.0	1.23	—	—	8 cm., 18 seconds.
12b	8.0	10.8	1.35	12.0	140.5	None.
13	13 2	20.2	1.53	—	—	
14	13.1	20.0	1.52	—	131.5	
15	13.0	19.8	1.52	—	—	
16	13.2	19.8	1.50	—	113.0	
17a	4.2	6.4	1.52	—	—	Stimulation of central
17b	9.5	12.6	1.32	12.0–	119.5	end, right sciatic,
18a	9.0	12.4	1.37	—	—	8 cm., 20 seconds.
18b	4.8	7.2	1.50	12.0	136.5	None.
19	12.9	19.6	1.52	—	—	
20	13.1	19.8	1.51	—	116.0	

stimulated,[11] and that the decrease appears in spite of the high systemic blood-pressure. However, when, the renal plexus is destroyed beforehand, these stimulations cause an increase in the flow in harmony with the high blood-pressure. In a similar manner, we have produced an increased renal blood-flow in the denerved left kidney, on stimulation of the central stump of the right sciatic nerve. Hence, after removal of the central nervous influences, the renal blood-flow becomes subject to general factors and may then be readily altered passively.

Under certain conditions, the stimulations were followed now

[11] *Loc. cit.*

and then by a decrease in the volume of flow, but the reduction never assumed a significant value. At best we have obtained only such quantitative changes as are given in the accompanying record (table I) of a part of experiment IV. In view of the fact that stimulation of direct vaso-constrictory paths, such as the greater splanchnics or the nerves of the renal plexus, readily leads to a cessation of the blood-flow through this organ, some doubt must arise as to the true nature of the reduction observed in these experiments. Without desiring to deny the existence of a reflex vaso-motor mechanism for the kidney, we are nevertheless forced to admit that we have not been able to influence the renal blood-flow reflexly in a measure which would render the vaso-motor origin of these vascular changes unquestionable.

It must be regarded as certain that the changes here noted are not of sufficient magnitude to be recognized as true vaso-motor reactions. This view seems the more plausible in that the reduction appeared altogether too abruptly and showed too great a constancy. If produced actually by vaso-constrictory means, other than mere variations in arterial tonicity, these vascular reactions should have exhibited a slow and gradual onset and should have continued wave-like until some time after the cessation of the stimulation. Hence, as these decreases in the blood-flow do not display the usual characteristics of vaso-constrictory reactions, we are inclined to regard them as being due to a tonic retention of the renal blood-bed.

When the systemic blood-pressure rises in consequence of central stimulation of the vagus or sciatic nerve, the kidney endeavors to preserve a normal blood supply by keeping the caliber of its blood-vessels unchanged. The latter end is accomplished by a tonic setting of its vascular channels, which at times may go somewhat beyond its normal limit and cause such slight reductions in the blood-flow as have been observed in these experiments. It may also be regarded as proved that the kidney plays a very unimportant part in the production of those pronounced rises in arterial pressure which usually appear in the course of the excitations of the central end of the divided sciatic or vagus nerves.