Section of the right vagus in the mid-cervical region of the rabbits produced no Wallerian degeneration in the thoracic sympathetic trunk and hence none in the splanchnic nerves.

In three cats (right vagus cut in two and left vagus in one) results were similarly negative. In a fourth cat, however, with right vagus cut above the anastomosis with the inferior cervical sympathetic ganglion, two degenerated medullated fibers were clearly identified in the right thoracic sympathetic trunk above the origin of the splanchnic nerve. One degenerated fiber was found in the right splanchnic and one in the right lumbar sympathetic trunk. Evidently one of the two vagus fibers coursed via the splanchnic and the other passed farther down the sympathetic trunk. Serial longitudinal sections and teased glycerol specimens agreed absolutely with each other. The glycerol specimens had never been in any fat solvent. Control material from corresponding nerves on the opposite side were run through the same reagents with the experimental specimens.

We do not believe these results are due to faulty technique, since one of us (A. T. R.) has used the Marchi method more or less continuously for over ten years and should realize its limitations. Hence we conclude that the number of medullated fibers from the vagus that course into the abdominal viscera via the splanchnic nerves in both rabbits and cats must be very small and therefore insignificant functionally.

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Inhibition of renal secretion following injury in the neighborhood of the colliculi.

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Desiring to obtain some data on the output of certain urinary constituents in dogs during short intervals of time, we thought we could get rid of the influence of anesthetics by decerebration. However, we were greatly surprised to find that on decerebration, the flow of urine ceased, although there had been a good flow

previously. There was no material alteration of blood pressure and respiration was kept up. Subsequent investigation showed this to be a constant phenomenon. Under normal conditions, after decerebration, there are a few drops of urine at the previous rate, and then a marked slowing in the rate amounting in most cases to a stoppage, and lasting as long as the experiment, in one case, seven hours after decerebration.

In our experiments, dogs have been used exclusively. They were given morphine and ether. Cannulae were inserted into the ureters close to bladder and the flow of urine recorded by allowing the drops of urine to fall on a lever which closed a circuit and thus recorded the flow by means of an electromagnet. Blood pressure was recorded from the left carotid, and the right carotid and the two vertebrals were usually ligated or clamped. The skull was trephined and the urine output recorded this way for a time. A few results may be given. Thus one kidney which produced 75 drops of urine in the thirty minute period previous to decerebration, produced 9 drops in the following thirty minutes. Another kidney which produced 138 drops in 30 minutes previous to decerebration, produced only 24 drops during 30 minutes following decerebration. Another kidney which formed 49 drops in 15 minutes previous to decerebration produced only 9 drops in the following 35 minute period.

Altogether, over thirty experiments have, so far, been done with similar results.

We believe the few drops of urine coming after decerebration at the previous rate were already in the ureter.

That this stoppage of renal secretion is a nervous phenomenon can readily be shown by sectioning the nerves going to one kidney before the operation. Decerebration then has little effect on the denervated kidney. In our experiments we found the flow of urine from the denervated kidney to be irregular and coming more or less in gushes. The results of one experiment may be given. Left kidney denervated.

Before decerebration:

Average drops per min. (20 min.) from left kidney.....2.2
Average drops per min. (20 min.) from right kidney.....2.3

After decerebration:

Average drops per min. (45 min.) from left kidney.....4.2
Average drops per min. (45 min.) from right kidney..... .5

It is noticed that the denervated kidney increased in activity while the kidney with uncut nerves was markedly slower than before the operation.

Section of one splanchnic nerve has a similar effect on the flow from the kidney of that side as section of the nerves to the kidney itself.

When the kidneys stop secreting as a result of decerebration, one can cause them to secrete by large doses of urea, or in most cases, by large doses of physiological saline intravenously, but one has to inject more saline to produce a diuresis than in normal animals. In a number of cases, injection of large amounts of saline produced only one or two drops of urine.

A study of the blood showed that the saline did leave the blood for the tissues but much more slowly than normal. After a dose of 20 cc. per kilo the blood of a normal animal returns to its original condition in from 30 to 35 minutes, but after decerebration little more than half has left at that time.

If one produces a diuresis before decerebration, either by intravenous injection of saline or by giving water by the mouth, then decerebration usually causes only a temporary slowing for a few minutes and then the secretion goes on at its former rate for a time and then ceases when the excess of fluid is removed.

The two things best known to affect the rate of kidney secretion are blood pressure and lack of oxygen. These two factors may be ruled out in these experiments. On decerebration there is, as a rule, immediately, either a slight rise or fall of pressure, lasting for a few minutes, and then a return to normal. There is usually increased pulse pressure after decerebration. Lack of oxygen cannot be the cause, because of the sudden onset of the effect. Experiments to test this point show a gradual slowing as asphyxia comes on. We believe the cause of this stoppage on decerebration lies in the altered permeability of the capillaries.

Various authors (Ashner, Camus and Roussy, Bailey and Bremer Curtis[1]) have described how injury to the brain stem in the region of the hypophysis (*Corpora mammilaria* Bourquin[2]) causes an increased output of urine. Destruction around the colliculi has the opposite effect. Our experiments show that one cannot get very far away from the colliculi and get this effect.

[1] Curtis, G. M., *Arch. Int. Med.*, 1924, xxxiv, 801. (Gives earlier literature).

[2] Bourquin, H., *Am. J. Physiol.*, 1926, lxxvi, 181.

We hope soon to be able to give a more exact location of the center.

We wish to call attention to a very similar phenomenon connected with the *nervi erigens* described by Martin and Tainter.[3] After decerebration, stimulation of the *nervi erigens* failed to produce its usual effect. Section of the nerves did not abolish the inhibition. If one sectioned the nerve before decerebration there was no effect on the action of the nerve as a result of decerebration. We know of no studies dealing with the permeability of the capillaries during stimulation of the *nervi erigens,* but the probabilities are that erection would be accompanied by outpouring of fluid to the tissue spaces. Our results and those of Martin and Tainter seem to indicate that injury in the neighborhood of the colliculi throws the capillary cells into some kind of a fixed condition of lessened permeability. We believe this mechanism is involved in those conditions of anuria seen when calculi are passing down the ureters. We have not been able to remove this condition by stimulation of nerves, but our experiments along this line have been very few.

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Further observations on relation of glomerular function to phenolsulphonephthalein excretion in frog's kidney.

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Since Rowntree's Phenolsulphonephthalein Test[1] of renal function is used widely in clinical practice, it is important to determine whether Phenolsulphonephthalein and similar dyes are excreted through the glomeruli, through the tubules, or through both.

In previous communications, we,[2] and also Richards and

[3] Martin, E. G., and Tainter, M. L., *Am. J. Physiol.*, 1923, lxv, 139.

[1] Rowntree, L. G., and Geraghty, J. T., *J. Pharmacol. and Exp. Therap.*, 1910, i, 579; *Arch. Int. Med.*, 1912, ix, 284.