THE INFLUENCE OF UNILATERAL SPLANCHNIC NERVE DIVISION ON THE DAILY EXCRETION OF WATER, SODIUM AND POTASSIUM

by

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INTRODUCTION

The influence of the nervous system on renal function has been studied for many years. Partial or complete interruption of the renal nerves has been produced by splanchnic nerve section, thoracolumbar sympathectomy, and stripping of the renal pedicle. Many investigators have shown that in the anesthetized animal renal denervation is followed by increased urine flow, increased electrolyte excretion, and usually, increased glomerular filtration rate and renal plasma flow. Two major questions remain unanswered. First, is denervation effective in changing the excretion rates of electrolyte and water only under the stresses of anesthesia and laparotomy? Second, are the effects of nerve section on salt and water excretion entirely secondary to changes in renal hemodynamics?

If electrolyte and water excretion are subject to control by the nervous system, this control might be concerned with either conservation when the animal is deprived of water and electrolyte, or with elimination when the animal is loaded with water and electrolyte, or both.

Teleologically, one would expect activity to be concerned with conservation in mammals, since they are more often exposed to water and electrolyte deprivation (at least in the primitive state) than they are to excessive loading. Furthermore, it would seem likely that these nervous influences, if they exist, could be more readily demonstrated under conditions of deprivation or excessive administration.

Heretofore, investigation in this field has been limited almost exclusively to experiments on animals variously loaded with water and

solutes in order to obtain urine flows adequate for the estimation of excretion rates and clearances. Frequently there is considerable variation in excretion in consecutive samples of urine from one kidney when collection periods are short. These unilateral variations may often be greater than the variations in excretion between the two kidneys. When long collection periods are used or when the excretion rates of several consecutive short collection periods are averaged, these unilateral fluctuations tend to be minimized.

This study was undertaken to circumvent the difficulties produced by anesthesia, excessive water and salt loading, and short collection periods by using long collection periods (24 hours) in unanesthetized, normally active dogs under conditions of moderate salt intake and severely restricted salt intake. It was hoped that by the use of these procedures additional insight into the presence and importance of nervous influences on renal function might be obtained.

REVIEW OF LITERATURE

In 1859 Claude Bernard (1) first described the phenomenon of "denervation diuresis". He found that unilateral section of the splanchnic nerve is followed by increased urine flow from the homolateral kidney. This diuresis he attributed to increased renal blood flow. Cohnheim and Roy (1) in 1883 utilized the oncometer to show that renal volume is increased upon section of the splanchnic nerves and is decreased on stimulating the splanchnic nerve. They attributed these volume changes to increased and decreased renal blood flow. Bradford (2) showed, through stimulation studies in 1889, that renal nerves are present in the sympathetic nerves of the fifth thoracic to the second lumbar segments. He also showed that these renal nerves are most abundant in thoracic segments ten, eleven, and twelve. In 1908 Burton-Opitz and Lucas (3), using the Ludwig stromuhr on the renal vein, confirmed the results of oncometer studies. They further demonstrated (4) that the renal innervation is unilateral. All of these experiments were on anesthetized laparotomized animals.

More recent studies can be placed in two groups: Experiments on anesthetized animals, and experiments on unanesthetized animals.

A. Experiments on anesthetized animals.

In 1919 Marshall and Kolls (5,6) and in 1922 Marshall and Crane (7) reported the results of experiments involving acute and chronic unilateral splanchnectomy in anesthetized dogs. They observed higher excretion rates of water and chloride from the denervated kidney as compared with the control kidney. These increases were frequently accompanied by lower excretion rates of creatinine from the denervated kidney. In a series

of related experiments (6) involving occlusion of the renal artery they observed changes in water and chloride excretion opposite in direction from those changes observed in splanchnicotomised animals. From this they concluded that splanchnic section results in increased water and chloride excretion solely because of increased renal blood flow. They found essentially no difference with respect to excretion between acute and chronic denervation.

In 1948 Kriss Futcher and Goldman (8) utilised clearance techniques in dogs to study the effects of splanchnic section. They found that the difference in output of water and chloride between the denervated and normal kidney could not always be accounted for by increased filtration rate (and hence increased load of water and solute) in the denervated kidney.

Kaplan and others (9,10) studying the effects of splanchnic section in hydropenic dogs with sodium chloride diuresis and again with mannitol diuresis, again found that section is followed by increased water and electrolyte excretion which cannot be entirely accounted for by increased load delivered to the renal tubule. They postulated that denervation removes the kidney from the influence of specific neural control of electrolyte excretion.

In two experiments on acutely denervated dogs (renal pedicle stripped) Berne (11) found higher excretion rates of sodium and water from the denervated kidney as compared with the control kidney. In 19 of 25 experiments on chronically denervated dogs (with diuresis produced by various methods) he found sodium excretion higher from the denervated kidney than from the control kidney. However, in those experiments in which the filtration rate on the denervated side was lower or essentially

equal to that on the control side he found that sodium excretion showed the same left to right relationship as the filtration rates. He concluded that the effect of denervation was to increase the filtration rate and thus increase solute load to the tubule which is followed by increased solute excretion and urinary volume.

Page et al (12) reporting the results of splanchnic section in anesthetized dogs also concluded that increased sodium excretion from the denervated kidney was the result of increased filtration rate.

B. Experiments on unanesthetized animals

In 1934 Rhoads (13) et al studied a dog with a single explanted kidney. They concluded that renal blood flow was not effected either by local block of the renal nerves with procaine or by renal denervation. Hiatt (14), using a unilaterally sympathectomized dog, found no difference in creatinine and diodrast clearances between the denervated and normal kidney. Maluf (15) studied the effects of denervation in a dog with a transplanted kidney. He concluded that transplantation with consequent complete interruption of the renal nerves is followed by no significant change in renal function.

In 1952 Berne (10), using dogs denervated by stripping the renal pedicle, reported results obtained in unanesthetized animals. He concluded that there is essentially no difference between the two kidneys in urine flow and sodium excretion except when the animal is subjected to emotional stress or anesthesia.

Surtshin and others (16,17) used dogs with explanted bladder trigones. They reported that unilateral sympathectomy is followed by no significant difference in urine flow, sodium excretion or filtration rate. In 2 of 5 animals they demonstrated that emotional stress is

followed by reduction in filtration rate and sodium and water excretion from the normal kidney without change in the denervated kidney. They also showed that deep pentobarbital anesthesia and ether anesthesia are followed by decreased filtration rate and decreased water and sodium excretion from the normal kidney and no change in the function of the denervated kidney.

Kaplan, West and Foman (18) in 1953 published the results of experiments on 4 splanchnicotomized, unanesthetized dogs. In 5 experiments carried out on 3 of the dogs before splanchnic section they found that spontaneous fluctuations in function between the 2 kidneys were about the same as others have observed. In 20 of 21 experiments on the 4 dogs after splanchnic section they found considerably greater excretion of sodium and chloride from the denervated kidney. They also found that excretion was reduced from the normal kidney.

Meilman and Winer (19) reported in 1953 that in 8 normally-hydrated, unanesthetized, unilaterally splanchnicotomized dogs there was greater sodium excretion from the denervated kidney. They state that urine flows were equal bilaterally.

In 1954 Page (12) et al stated that "there was little or no difference between the two kidneys in urine flow or sodium excretion in the unanesthetized state 24 hours after (unilateral) splanchnicectomy". These observations were made in 3 animals.

In summary, it appears that most investigators who have worked in this field are in agreement that renal denervation in the anesthetized animal is followed by increased excretion of salt and water. There is no agreement as to whether or not the increased water and solute excretion is due entirely to increased solute load delivered to the renal

tubule. Results and interpretations thereof, obtained in unanesthetized animals, have been conflicting.

METHODS

Three adult female mongrel dogs weighing 36, 25 and 27 pounds were used in this study. Separate collections of urine from the kidneys was made possible by splitting the bladder sagittally and re-forming each half into an individual bladder for each ureter and kidney. Mushroom-shaped nylon cannulas (see Fig. 1) were inserted through the bladder and abdominal walls. Urine was collected in polyethylene bags, which were attached to the cannulas. See the appendix for a complete description of the surgical technique and description of the cannulas and bags.

Renal denervation was accomplished by transpleural resection of the splanchnic nerve through an incision in the tenth intercostal space. The nerve was resected immediately above the diaphragm and a three to four centimeter segment removed.[1]

The animals were maintained on a diet of casein proteinate,[2] lard, corn oil and sucrose mixed in the proportions shown in table I. The mixed food contained 4.65 calories per gram. Sodium content of the food was less than 0.5 millequivalents per hundred grams. Potassium content was negligible. Moderate salt intake consisted of 86 mEq. (5 gm.) of sodium chloride and 13.5 mEq. (1 gm.) of potassium chloride added to the food daily. When electrolyte was restricted, no salt was added to the diet. A vitamin supplement was added to the diet daily.[3]

The animals were fed once daily. Dog G usually received 200 grams food per day. Dog B usually received 250 grams daily. Dog J received

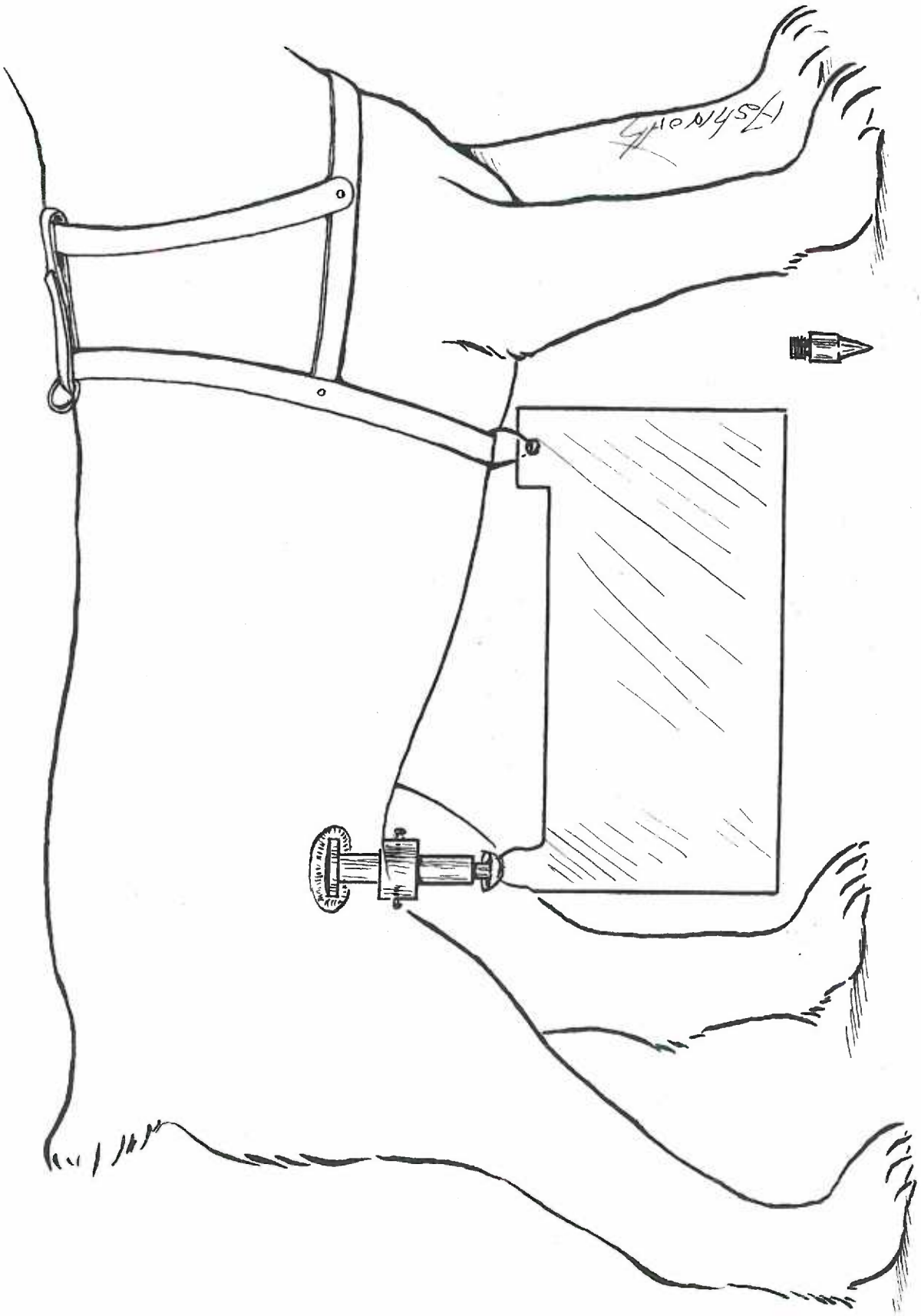
[1]This procedure was suggested by Dr. A. C. Barger of the Harvard University Department of Physiology.

[2]Casec, Mead Johnson & Co.

[3]Zymacaps, Upjohn Co.

Figure I

Diagram showing cannulae and collection bags in relation to the animal. For clarity only one cannula and bag are shown. Inset at lower right shows puncture bullet.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175-250 grams daily. The food was mixed with 500 ml. of warm water. Water was also allowed ad libitum. Intake was estimated by filling the water cans with one liter of water daily and measuring the residual on the following day. Daily urinary volumes were measured by emptying the collection bags into a 500 ml. graduated cylinder. Aliquots were taken from each sample for the determination of sodium and potassium. The urine was refrigerated until the determinations were made. Blood was taken by venipuncture every other day for plasma, sodium and potassium determinations. Heparin was used to prevent clotting. All samples were collected just before the animals were fed. Samples were collected within half an hour of the same time every day.

Sodium and potassium concentrations were determined with the Barclay Flame Photometer in appropriately diluted samples. Endogenous urinary creatinine was determined by the Bonsnes and Tausky (20) modification of Folin's (21) adaptation of the Jaffe reaction between creatinine and alkaline picrate. The Coleman Universal Spectrophotometer Model 14 was used in these determinations.

At the conclusion of the experiments the animals were sacrificed. Inspection of the site of splanchnic section revealed complete interruption of the nerve above the diaphragm in all three animals. Gross and microscopic examinations of bladders, ureters and kidneys were made. On the day previous to her demise dog G ceased to excrete urine from the control (left) cannula. On autopsy the cannula was found to be herniated through the rectus abdominus. The sinus left in the path of the cannula was completely closed. The bladder on the left contained about 300 ml. of urine and the ureter was slightly dilated. The bladder on the denervated (right) side showed moderate inflammation, but no dilatation. The ureter

on the right was not dilated. There were small calculi in both renal pelves. Microscopic inspection of the kidney of dog G revealed mild subacute pyelitis with subacute ureteritis on the right (denervated kidney). The left (control) kidney showed minimal subacute pyelonephritis.

The kidneys of dog B showed minimal acute necrotizing papillitis with intratubal calculi on the right (control) and subacute pyelitis on both sides. The ureters were microscopically normal on both sides. Gross examination of both bladders showed inflammation. Dog J became ill during the post-denervation studies. On the last day of salt depletion following nerve section she developed anorexia, vomiting and diarrhea. She was sacrificed after two days of illness. Gross examination of kidneys, ureters and bladders revealed no pathology except inflammation of the bladders. Microscopic examination of kidneys and ureters revealed only minimal pyelitis on the right.

RESULTS

Table 2 indicates daily food, water and salt intakes for each animal. Daily urine volumes as well as urinary concentration of sodium and potassium are included. Twenty four hour urinary excretions of sodium and potassium are also included. Table 2 also contains plasma concentrations of sodium and potassium.

Table 3 shows mean values for each animal of urine flow, sodium excretory rate and potassium excretory rate obtained under similar experimental conditions. Mean urinary concentrations of sodium and potassium are also included. On 3/27 dog G began to show signs of infection on the left side. Sulfadiazine therapy was instituted on 3/27. Since sulfa drugs are known to be carbonic anhydrase inhibitors, it was thought that the drug might bring about altered function in the dog's kidneys. Consequently, all urine flows and electrolyte excretory rates obtained from dog G after 3/27 were excluded from Table 3. Excretory rates from dog J post-splanchnicotomy were left out of Table 3 also because an inadequate number of control samples were obtained.

The columns marked D-I (meaning denervated minus innervated) in Table 3 show the mean numerical differences between denervated and innervated kidneys. The columns marked D/I (meaning denervated divided by innervated) give the mean ratios of urine flow and sodium and potassium concentrations and excretory rates. The D/N ratio can be used to compare percentage difference between the two kidneys using the innervated kidney for reference.

As shown in Tables 2 and 3, dog G was studied under conditions of moderate salt intake and sodium deprivation before splanchnic section. After section, she was studied under conditions of moderate load, sodium deprivation, potassium deprivation, and deprivation of both sodium and

and potassium. Dogs B and J were studied under conditions of moderate load and deprivation of both sodium and potassium before splanchnic section as well as after.

Urine Flow

There was considerable daily variation in total urine volume excreted by the two kidneys of any one animal as Table 2 shows. However, the D/I ratio remained fairly constant day to day for each dog. The differences between flows from the two kidneys also remained fairly constant in any one animal.

Before splanchnic section two of the animals (dogs G & B) had D/I ratios for urine flow slightly greater than one. Dog J had gross differences in urine flow from the two kidneys before section. In Dog J the splanchnic nerve was sectioned on the same side as the kidney which secreted the lesser quantities of urine.

Following nerve section urine D/I was less than one in both dog G and Dog B. D-N was significantly different after splanchnic section for both dogs G and B. Post-splanchnicotomy studies were begun six days in post-operative dogs G and three days post-operative in Dog B. Post-splanchnic section mean values for Dog J were omitted from Table 3 because studies were started immediately post-operatively and an inadequate number of samples of control observations were obtained. However, the data was included in Table 2. Table 2 shows that on the first post-operative day Dog J excreted considerably more urine from the denervated kidney than from the control kidney. On the second post-operative day urine flows were almost equal and from the third day on, urine flow was somewhat lower from the denervated kidney than from the normal kidney. Although the urine

flow from the denervated kidney was lower than that from the control kidney, there was less difference between the flows from the two kidneys after splanchnic section than there was before section. In other words urine flow was more nearly equal bilaterally after splanchnic section. Although salt deprivation appeared to be followed by decreased water intake and urinary output, there appeared to be no significant change in the differences in outputs of the two kidneys in any of the three animals.

Creatinine

Values obtained for urine creatinine concentration and excretion were excluded from Tables 2 and 3. There were gross variations in the values obtained for creatinine excretion in urine from each kidney in all animals. Serial determinations on single urine samples on successive days revealed that creatinine concentration was reduced in the urine on standing. Since not all determinations were made on the day of sample collection and no preservative was added to the urine, it was thought that the values obtained for urinary creatinine excretion were unreliable.

Sodium

Sodium concentrations were nearly equal in urine from the two kidneys of both dogs G and B before splanchnic section. Daily sodium excretion from the two kidneys of these animals had roughly the same relationship as did urine flows. In the third dog (J), while on moderate salt load before splanchnic section, sodium concentration was considerably higher in the urine from the kidney with the lower urinary flow. Daily sodium excretions were generally lower from the kidney with lower urine flow in dog J before splanchnic section.

Following splanchnic section and while receiving moderate amounts of sodium and potassium both dogs G and B had D/I ratios for sodium concentration considerably greater than one. Since urine flow was lower from the denervated kidney, there was no significant difference in daily sodium excretion from the two kidneys in either animal. When sodium and potassium were restricted, the concentration differences were more marked. In spite of lower urine flows from the denervated kidneys the denervated kidneys excreted significantly greater quantities of sodium than did the control kidneys. Dog G was also subjected to restriction of sodium while potassium was supplied and vice versa. There appeared to be no significant difference in sodium excretion from the two kidneys under these conditions.

As mentioned previously mean values for Dog J were not included in Table 3. However, daily excretions are shown in Table 2. On the day following splanchnic section urine collection was started immediately following surgery. Dog J excreted considerably more sodium from the denervated kidney than from the normal kidney. Urine sodium concentrations were about the same bilaterally. On the second post-operative day D/I for sodium concentrations began to rise. On the second and third post-operative days both kidneys excreted about the same quantity of sodium. On the fourth and fifth post-operative days D/I for sodium concentration was considerably greater. Sodium excretion was also somewhat higher from the denervated kidney. Dog J had received moderate amounts of sodium and potassium through the fifth post-operative day. On the sixth post-operative day sodium and potassium were withheld from the diet. Sodium concentration and excretion in the urine from the denervated kidney dropped more than they did from the control kidney. This experiment was discontinued at the end of the ninth post-operative day because the dog refused to eat and was vomiting.

Plasma sodium concentrations as shown in Table 2 did not seem to be consistently changed during either salt depletion or administration either before or after splanchnic nerve section in dogs G and B or in dog J before splanchnic section. After splanchnic section in dog J plasma sodium concentration progressively decreased.

Potassium

There were no consistent changes in the differences between the two kidneys in urine potassium concentration or excretion following splanchnic section in dogs B and J. Potassium concentrations were not obtained in dog G prior to splanchnic section. In the experiments in which potassium alone was restricted considerably more potassium was lost in the urine than when both sodium and potassium were restricted. When sodium alone was restricted, there appeared to be less potassium excreted than when sodium is not restricted.

Plasma concentrations of potassium were not consistently changed during any of the experiments on any of the animals.

In summary following splanchnic section two of three dogs showed decreased water excretion and increased sodium concentration in the urine from the denervated kidney as compared with the urine from the normal kidney. Sodium excretion was greater from the denervated kidney than from the normal kidney only when both sodium and potassium were withdrawn from the diet. Potassium excretion appeared to be unchanged by splanchnic section.

DISCUSSION

Results obtained in previous investigation in this field are conflicting. There is no unanimity of opinion as to the presence of neural influences on renal function. At present two major issues remain undecided. First, are nervous influences on renal function evident only when the organism is subjected to the stresses of anesthesia and laparotomy? Second, does the nervous system exert control over renal tubular activity?

Most investigators are agreed that anesthesia and laparotomy are accompanied by neurogenically mediated renal vasoconstriction. Many have concluded that the nervous system does not exert a tonic influence on the renal vasculature in resting, unanesthetized animals. Others have concluded that the renal vascular system is under the control of the nervous system in unanesthetized animals. Rhoads et al (13) studied dogs with single explanted kidneys. They stated that they found no change in the function of those kidneys when the renal nerves were blocked with novocain. However, inspection of their data reveals that renal blood flow was increased following nerve blockade in four of the five experiments for which data is given. Maluf (15) reported studies on a single unanesthetized dog with one kidney transplanted to the thigh. It is doubtful that his results are reliable since there was always pus and albumin present in the urine from the transplanted kidney. Kaplan et al (18) found increased urine flow, sodium excretion and filtration rate in unanesthetized unilaterally splanchnicotomized dogs.

The differences of opinion on the presence of neural influences on the renal vasculature are not necessarily incompatible. Berne (11) and Surtshin et al (17) found that emotional and painful stimuli may be followed by decreased renal plasma flow and glomerular filtration rate

in the innervated kidney, but not in the denervated kidney. It is conceivable that those investigators who found evidence of neural influences on the renal vasculature may have been working with animals subjected to emotional stress.

The question of nervous influences on renal tubular function has been argued for many years. As mentioned previously, Marshall and others (5,6,7) attributed greater urine flow and chloride excretion from the denervated kidney to altered renal hemodynamics. However, their protocols show that creatinine excretion was not always greater from the denervated kidney. In fact, creatinine excretion was frequently lower from the denervated kidney than from the innervated kidney. Kriss et al (8) and Kaplan et al (9,10) found that the difference in amounts of sodium excreted by the denervated and normal kidneys was frequently less than the difference in amount of sodium filtered through the glomeruli of the two kidneys. Berne (11) and Surtshin et al (16,17) found that greater sodium excretion from the denervated kidney was almost always accompanied by increased filtration rate. They maintain that the greater excretion of sodium from the denervated kidney is due only to increased filtration of sodium whether differences in filtration rate can be detected or not.

Further evidence for the existence of nervous influences on tubular function is provided by the findings of Blake (22) that in unanesthetized animals emotional stress is followed by reduced excretion of sodium without concomitant changes in filtration rate.

In evaluating the conflicting evidence in this field several differences in technique should be noted. In the first place denervation has been produced by at least three different methods. The least extensive

denervation, that of Berne (11), involved stripping the renal pedicle. Surtshin et al (16,17) and Hiatt (14) utilized the most radical denervation procedure. They performed complete unilateral thoraco-lumbar sympathectomy. Most other investigators have utilized unilateral splanchnic nerve resections. Cow (23) in 1914 described an adreno-renal venous anastomosis. He postulated that nervous influences on the adrenal medulla may affect homolateral renal function through the direct circulation of adrenaline from the adrenal medulla to the renal parenchyma. This hypothesis was tested by Marshall and Kolls (5) and Kriss et al (8) who concluded that the effects of unilateral adrenalectomy are due to injury to the renal nerves during the procedure. Recently Dempster and Graber (24) repeated unilateral adrenalectomy in dogs taking great care to avoid the renal nerves. They found increased excretion of water and sodium from the homolateral kidney. It is conceivable that the studies with splanchnic section and sympathectomy might, by depriving the adrenal of its nerve supply, influence homolateral renal function differently than does "simple" renal denervation.

Another possibility is that in performing unilateral sympathectomy or in stripping the renal pedicle damage to the kidney might result. This difficulty would presumably be avoided by supra-diaphragmatic splanchnic section in which procedure the kidney is not disturbed.

Berne (11) has suggested that there may be a difference in renal function in acutely and chronically denervated animals. In two experiments on acutely denervated, anesthetized animals urine flow and sodium excretion were greater from the denervated than from the normal kidney while renal plasma flow and glomerular filtration rate were lower from the denervated kidney. A third animal which responded in the same manner to denervation was found at autopsy to have a large renal infarct.

Berne also suggested that the results obtained in the other two animals might have been due to temporary injury to the operated kidneys. Marshall and Kolls (5,6,7) used both acutely and chronically denervated animals in their studies and found no differences in function between the two groups.

Another consideration in evaluating results of experiments in which short collection periods were used is the variability in sodium excretion from the individual kidney. Berne (11) observed unilateral variations in sodium excretion up to 100% in successive 10 to 15 minute collection periods. The variation in excretory rates from a single kidney was frequently greater than the variation between the two kidneys. Mueller et al (25) using 3-hour collection periods found much lower unilateral variation.

Nearly all of the previous studies have utilized diuretic agents in order to obtain adequate urine flows. Agents most commonly used have been water or saline by stomach tube and intravenous saline or mannitol solutions. If neural influences are concerned with conserving sodium when sodium is deprived, it is not surprising that some investigators have failed to demonstrate these nervous influences when the renal tubules are overloaded with salt in an effort to stimulate urine flow.

Many investigators in this field have apparently failed to take cognizance of the fact that the function of the two kidneys in any one animal may not be identical. Mueller et al (25) found differences in sodium excretory rates between the two normal kidneys as great as 35%.

Considering the experimental evidence brought forth to date the following conclusions would seem most reasonable. First, the renal vasculature is probably not subject to significant tonic sympathetic nervous

activity in the resting unanesthetized animal. Second renal function may be influenced by the nervous system in unanesthetized animals subjected to emotion stress. Third, sodium excretion may be influenced by the nervous system.

Most studies on unanesthetized animals have been performed in dogs lying quietly in slings or on dog boards. The "normal" life of dogs living in animal quarters presumably entails considerably more activity and trauma than are present in most acute experiments on unanesthetized dogs. This study was undertaken in order to study the normally active animal rather than the animal in the quiet or "basal" state.

In the present study two of three unanesthetized, normally active dogs had decreased urine flow from the denervated kidney as compared with the innervated kidney following unilateral splanchnic nerve section. D/I ratios for sodium concentration were increased in the same animals, but D/I ratios for sodium excretion were increased only when sodium and potassium intake were restricted.

In the absence of information about renal hemodynamics in these animals one can only speculate on the possible mechanisms responsible for these changes.

The rate of urine excretion is dependent upon the relationship between the amount of water delivered to the renal tubule and the amount of water reabsorbed by the renal tubule. The amount of water delivered to the renal tubule is dependent upon the rate of glomerular filtration.

Water reabsorption may be divided into two portions, the quantity of water osmotically obligated to solute reabsorbed in the proximal portions of the renal tubule (obligatory reabsorption) and the quantity of water actively reabsorbed in the distal tubule under the influence of the antidiuretic hormone (facultative reabsorption).

Therefore, there are three possibilities to consider in explaining the lower urinary volumes excreted from the denervated kidney as compared with the innervated kidney. One possibility is that the denervated kidney is more sensitive to the anti-diuretic hormone than is the innervated kidney. This seems unlikely in view of the observation of Klisieke et al (26) that the denervated and innervated kidneys respond in a parallel manner to the diuretic stimulus of water, and that water diuresis is inhibited to the same degree in the denervated and innervated kidney by the antidiuretic hormone.

A second possibility is that the denervated kidney reabsorbs greater quantities of osmotically active solute in the proximal tubule, thus increasing the obligatory reabsorption of water. That this is unlikely is indicated by the fact that sodium, which is the predominant osmotically active constituent of the glomerular filtrate, appeared in the urine of both innervated and denervated kidneys in similar quantities. The third and most likely cause for the lower urine flow from the denervated kidney would seem then, to be lower glomerular filtration rate. Possibly decreased filtration rate from the denervated kidney might be produced by increased sensitivity of the renal vasculature to circulating hormonal agents such as adrenalin.

Sodium excretion is dependent upon the relationship between glomerular and tubular activity. The amount of sodium delivered to the renal tubules depends upon the glomerular filtration rate and the plasma sodium concentration. Approximately 85% to 90% of the filtered load of sodium is reabsorbed in the proximal tubule, and this is apparently dependent upon the load of sodium delivered to the tubule. That is, when filtration rate or plasma sodium concentration is elevated, greater amounts of sodium are

delivered to the distal tubule. When sodium load is decreased by lowered filtration rate or plasma sodium concentration, lesser amounts of sodium are delivered to the distal tubule.

Very little is known about the mechanisms of distal tubular sodium reabsorption. The best evidence available at present indicates that sodium - potassium and sodium-hydrogen exchange mechanisms are involved.

There are two possibilities to consider in explaining the results obtained in this study. First, greater sodium excretion from the denervated kidney than from the innervated kidney when sodium and potassium were restricted, may have resulted from greater load of sodium delivered to the tubule. Greater load of sodium can result only from increased plasma sodium concentration and increased glomerular filtration rate. Obviously the plasma sodium concentration in the blood perfusing the two kidneys was the same. It is unlikely that filtration rate was greater in the denervated kidney as previously pointed out. If anything, it is more likely that filtration rate was lower in the denervated kidney. Therefore, it seems unlikely that increased sodium load explains the greater excretion of sodium when sodium and potassium were withheld from the diet. The most likely explanation remaining, then, is that sodium reabsorption was decreased in the denervated kidney.

It is not possible to state with certainty which portion of the renal tubule might be involved in producing the results obtained in this study. However, the fact that sodium excretion was greater from the denervated than from the innervated kidney only when both sodium and potassium were withheld might indicate that the distal tubule is involved.

Another interpretation of these results might be made if the filtration rate was lower in the denervated than in the normal kidney. Then,

presumably there would have been lower amounts of sodium delivered to the tubule in the denervated kidney. Since sodium excretion was usually about the same from the two kidneys following unilateral denervation, the fraction of filtered sodium excreted must have been greater from the denervated kidney. Therefore, it is conceivable that splanchnic section resulted in loss of nervous control of sodium excretion from the denervated kidney. Increased excretion of sodium by the denervated kidney might have been obscured by concomitant decrease in filtration rate. The differences in excretion of sodium might have become apparent only when sodium reabsorption was further decreased by potassium restriction.

SUMMARY

The influence of unilateral splanchnic nerve section was studied in three normally active, unanesthetized dogs. In two of the animals unilateral splanchnic section was followed by decreased excretion of urine from the homolateral kidney as compared with that from the normal kidney. Sodium concentration was greater in the urine from the denervated kidney in the same animals. Sodium excretion was greater from the denervated kidney only when sodium and potassium were withheld from the diet.

It was concluded that the results obtained in this study are compatible with the hypothesis that renal tubular function is under the control of nervous influences.

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APPENDIX

Preparation of Animals

Under sodium pentobarbital anesthesia (32 mg./kg.) the abdomen was prepared by clipping and shaving the hair and by application of 70% alcohol and tincture of zephiran. The abdomen was opened low in the midline. The bladder, which is entirely intraperitoneal in the dog, was pulled through the incision. Gauze was packed between the bladder and the abdominal wall. The urethra was isolated and sectioned. The distal stump was closed with catgut. The anterior bladder wall was incised in the mid-sagittal plane from fundus to urethra. Bleeding from the bladder was controlled through the use of Duval lung clamps. The ureteral orifices were located and small polyethylene catheters were inserted into the ureters to aid in locating the ureters in the paravesicle tissue. Bladder resection was completed by incising the posterior vesicle wall in the sagittal plane midway between the ureteral orifices. This left each half of the bladder approximately egg-shaped with the ureteral orifices located near the smaller apex. The cannulas (described below) with their puncture bullets attached were forced through the larger apex of the bladders. The cannulas were then forced through the parietal peritoneum and the rectus abdominus muscles about four centimeters above the symphysis pubis and three centimeters lateral to the midline. Small skin incisions were made to admit the tips of the puncture bullets and the cannulas were forced through the skin. The bladders were then folded in the line of the shorter axis of the ellipse. One suture of No. 2 medium chromic catgut was placed through the approximated ends of the long axis of each bladder. Bladder closure was then completed by continuous suture with No. 2 medium chromic catgut through the bladder muscularis

and visceral peritoneum. Care was taken to exclude bladder mucosa from the suture line. The abdominal wall was then closed in layers with interrupted sutures. Retaining rings were placed on the cannulas and the puncture bullets were removed. The animals were returned to the animal quarters and allowed to recover for several days before they were used for experimentation.

The cannulas were obtained from L. W. Carson, 515 N. Main St., Wheaton, Ill. They were originally designed for use in intestinal pouches. They were mushroom-shaped 2 3/4 inches long, $\frac{1}{2}$ inch outside diameter in the shaft 1 1/8 inch diameter at the head and had a 5/16 inch lumen. The distal ends of the cannulas were tapped to receive threaded puncture bullets and nipples. Retaining rings were 3/4 inch long and 1 1/8 inch outside diameter. The retaining rings fit snuggly on the cannulas and were held in place by one or two 10/24 brass machine screws. Twenty gauge polyethylene bags with welded seams were used for sample collection. The bags were nine inches long and five inches wide. Their capacity was about 900 ml. each. The bags had an open neck at one end. At the other end was a tab with a grommet through it. The threaded nipples ($1\frac{1}{2}$ inches long) were held in place on the necks of the bags by two turns of a No. 18 B & S gauge nichrome wire twisted tightly to insure against leakage. The bags were tested by filling with water and air. The ends of the nipples were then occluded and considerable manual pressure was exerted on the bags. If there was any evidence of leakage of air or water, the bags were discarded.

The bags were suspended on the dogs as shown in the figure. The bags were supported on one end by the nipples screwed into the indwelling cannulas. At the other end string or wire clips inserted through the

eyelets in the tabs fastened the bags to a harness. Ordinary dog harnesses were obtained from a local variety store.

TABLE I

Diet Composition

Constituent	Amount Gms.	Carbo-hydrate	Protein	Fat	Calories	Sodium Content Mg.	Potassium Content Mg.
Casec	28		25		100	14.00	—
Lard	10			10	90	.03	.02
Corn oil	5			5	25	.01	.005
Sucrose	55	55			220	—	—
Totals	98				435	14.04	.025
Content / Gm.					4.65	0.144	.00026

TABLE 2A

Date	Daily Intake Casec	Na	K	Cl	H2O	Wgt.	Water Output ml./24 hr.			Sodium Conc. mEq/L.		Sodium Output mEq/24 hr.			Potassium Conc. mEq/L.		Potassium Output mEq/24 hr.			Plasma Na.	Plasma K.
	Gm	Gm	Cl Gm	ml.	Lb.	Rt.	Lt.	D-I	Rt.	Lt.	Rt.	Lt.	D-I	Rt.	Lt.	Rt.	Lt.	D-I	mEq/L.	
12/13	109	5	1		36	364	338	+26	110	117	40.0	39.5	0.5							
12/15	109	0	1			230	196	+34	32.0	30.5	7.4	6.0	1.4							
12/16	109	0	1			181	165	+16	29.1	28.7	5.3	4.7	0.6							
12/17	109	0	1			238	215	+23	19.5	18.5	4.6	4.0	0.6							
12/19	109	5	1			295	268	+27	59.2	62.0	17.5	16.6	0.9							
12/20	109	5	1			337	306	+31	127	141	42.9	43.1	-0.2							
12/31	109	5	1			356	319	+37	152	165	54.3	52.6	1.7							
	Right splanchnic nerve sectioned 2/9/55																			
4/5	200	5	1	1375	33½	520	584	-64	27.4	24.7	14.3	14.4	-0.1	14.8	11.9	7.7	7.0	0.7	146	3.9
4/8	200	5	1	1200	34	405	508	-103	85.6	81.3	34.7	41.3	-6.6	20.7	11.7	8.4	5.9	2.5	150	4.0
4/9	200	5	1	1075		444	418	26	46.6	43.1	20.7	18.0	2.7	20.4	13.2	9.1	5.5	3.6		
4/10	200	0	0	750	34	368	360	8	40.0	30.0	14.7	10.8	3.9	13.9	7.5	5.1	2.7	2.4	153	4.1
4/11	200	0	0	675		364	316	48	31.0	22.7	11.3	7.2	4.1	8.5	6.7	3.1	2.1	1.0		
4/12	200	0	0	825	34	381	346	35	20.3	16.7	7.7	5.8	1.9	6.1	4.8	2.3	1.7	0.6	150	4.1
4/13	200	0	0	750		431	393	38	21.0	13.0	9.1	5.1	4.0	4.0	3.7	1.7	1.5	0.2		
4/14	200	0	0	925	34	410	365	45	15.7	10.2	6.4	3.7	2.7	2.5	2.4	1.0	0.9	0.1	152	4.4
4/15	200	5	1	1500		546	481	65	24.2	19.0	13.2	9.1	4.1	6.2	6.3	3.4	3.0	0.4		
4/16	200	5	1	1600	35	766	732	34	41.3	38.3	31.6	28.0	3.6	10.7	15.5	8.2	11.4	-3.2	148	3.9
4/17	200	5	1	1375		631	630	1	65.9	58.7	41.6	37.0	4.6	9.1	11.2	5.7	7.1	-1.4		
4/18	200	5	1	1200	35	592	535	57	68.3	63.3	40.4	33.9	6.5	11.5	13.5	6.8	7.2	-0.4	156	4.2
4/19	200	5	1	975		506	426	80	93.9	87.9	47.5	37.5	10.0	10.2	13.7	5.2	5.8	-0.6		

Dog - G
Sulfadiazine 1 Gm. daily 4/5-4/17

TABLE 23

Date	Daily Intake: Cases	Na Cl Gm	K Cl Gm	H2O ml.	Wgt. Lb.	Water Output ml./24 hr.: Rt.	Lt.	D-I	Sodium Conc. mEq/L.: Rt.	Lt.	Sodium Output mEq/24 hr.: Rt.	Lt.	D-I	Potassium Conc. mEq/L.: Rt.	Lt.	Potassium Output mEq/24 hr.: Rt.	Lt.	D-I	Plasma Na. mEq/L.	Plasma K. mEq/L.
2/15	135	5	1		32	373	382	-9	144	141	53.7	53.8	-.1	30.7	34.9	11.5	13.4	-1.9	158	4.1
2/17	135	5	1			290	342	-52	151	127	43.9	43.4	0.6	26.3	20.0	7.7	6.9	0.8		
2/18	135	5	1		31½	385	395	-10	127	111	48.8	45.0	3.8	26.9	21.8	10.4	8.6	1.8	152	3.8
2/19	135	0	1	435	31	221	259	-38	44	34	9.7	8.7	1.0	11.2	9.0	2.5	2.4	0.1		
2/20	200	0	1	800		263	310	-47	31	23	8.0	7.0	1.0	16.0	15.8	4.2	4.9	-0.7		
2/21	200	0	1	620	31	252	291	-39	18	19	4.5	5.5	-1.0	11.0	11.7	2.8	3.4	-0.6		
2/22	200	0	1	600		310	348	-38	16	15	5.0	5.2	-0.2	16.9	17.5	5.3	6.1	-0.8	154	4.5
2/23	200	0	1	700	31	373	431	-58	12	10	4.4	4.4	0	16.7	16.7	6.3	7.2	-0.9		
2/24	200	5	1	1050	30½	312	360	-48	25	29	7.9	10.4	-2.5	46.7	43.2	14.6	15.6	-1.0	155	7.0
2/25	200	5	1	1155	31	460	460	0	62	55	28.7	25.3	3.4	28.4	28.1	13.1	12.9	0.2		
2/26	200	5	1	795	31	284	363	-79	138	136	39.3	49.4	-10.1	20.5	21.4	5.8	7.8	-2.0	155	3.3
2/28	200	5	1	620	31	250	315	-65	160	150	40.0	47.1	-7.1	17.6	17.4	4.4	5.5	-1.1		
3/1	200	5	1	760	31½	238	279	-41	164	159	39.0	44.3	-5.3	14.1	19.8	4.6	7.5	-2.9		
3/2	200	5	1	560	31½	311	332	-21	143	141	44.4	46.8	-2.4	17.1	17.7	5.3	5.9	-0.6		
3/3	200	5	1	745	32	339	330	+0	138	132	46.6	43.6	3.0	15.1	16.5	5.1	5.5	-0.4		
3/4	200	0	0	400	31½	239	226	-27	36	21	8.7	5.5	3.2	3.4	3.1	0.8	0.8	0	171	2.3
3/5	200	0	0	500	31	155	192	-37	37	25	5.8	4.9	0.9	2.5	2.3	0.4	0.4	0		
3/6	200	0	0	600	31½	213	248	-35	40	17	8.4	4.2	4.2	2.2	1.1	0.5	0.3	0.2	150	4.3
3/7	200	0	0		31	307	388	-81	20	12	6.2	4.5	1.7	1.1	0.9	0.3	0.4	-0.1		
3/8	200	0	0	625	31	254	314	-60	16	10	4.0	3.2	0.8	0.9	0.9	0.2	0.3	-0.1	147	3.9
3/9	200	5	1	1035	31½	261	340	-79	25	19	6.6	6.3	0.3	7.2	8.4	1.9	2.8	-0.9		
3/10	200	5	1	875	31½	377	437	-60	110	100	41.6	43.9	-2.3	10.6	11.6	4.0	5.1	-1.1	161	4.2
3/11	200	5	1	825		270	342	-72	158	142	42.8	48.8	-6.0	17.0	17.3	4.6	5.9	-1.3		
3/12	200	5	1	625	32	248	295	-47	173	154	42.8	45.4	-2.6	26.5	26.8	6.6	7.9	-1.3	155	4.6
3/13	200	5	1	700	32	258	327	-69	175	154	45.2	50.2	-5.0	31.6	31.6	8.2	10.3	-2.1		

Dog - Gertrude
Right splanchnic nerve sectioned 2/9/55

TABLE 2C

Date	Daily Intake Casec Gm	Na Cl Gm	K Cl Gm	H2O ml.	Wgt. Lb.	Water Output ml./24 hr. Rt.	Lt.	D-I	Sodium Conc. mEq/L. Rt.	Lt.	Sodium Output mEq/24 hr. Rt.	Lt.	D-I	Potassium Conc. mEq/L. Rt.	Lt.	Potassium Output mEq/24 hr. Rt.	Lt.	D-I	Plasma Na. mEq/L.	K.
3/18	200	5	1	575	33	200	195	-5	179	162	35.8	31.5	4.3	48.2	46.9	9.6	9.1	.5		
3/19	200	5	1	625		253	283	-30	155	142	39.3	40.2	-1.9	29.1	27.9	7.4	7.9	-.5		
3/20	200	5	1	600	32½	235	268	-33	188	171	44.2	45.9	-1.7	30.7	29.2	7.2	7.8	-.6	150	5.2
3/21	200	5	1	625		210	239	-29	191	173	40.2	41.4	-1.2	34.3	30.0	7.2	7.2	.0		
3/22	145	5	1	425	32½	215	228	-13	200	191	43.0	43.6	-.6	46.0	46.5	9.9	10.6	-.7	141	4.3
3/23	200	5	0	550		212	239	-27	182	149	38.7	35.6	3.1	18.5	16.2	3.9	3.9	.0		
3/24	200	5	0	425	32½	166	174	-8	232	218	38.5	37.9	.6	15.0	15.8	2.5	2.8	-.3	148	3.9
3/25	200	5	0	650		261	370	-59	143	119	37.3	38.1	-.9	8.0	5.8	2.1	1.9	.2		
3/26	200	5	0	850	32½	346	408	-62	129	111	44.7	45.4	-.7	6.8	5.0	2.4	2.0	.4		
3/27	200	5	0	1250		483	513	-30	96.0	45.2	46.4	48.8	-2.5	4.9	4.2	2.4	2.2	.2	142	3.9
3/28	200	5	1	975	33	466	516	-50	76.4	75.6	39.0	39.0	-3.4	8.0	8.0	3.7	4.1	-.6		
3/29	200	5	1	1250		466	544	-78	90.4	85.2	42.1	46.4	-4.2	8.5	8.2	4.0	4.5	-.5	151	3.9
3/30	200	5	1	1175		534	591	-57	73.2	69.6	39.1	41.1	-2.0	11.5	10.9	6.1	6.4	-.3		
3/31	200	5	1	1400	33	632	699	-67	59.2	42.4	37.4	29.6	7.8	12.9	13.8	8.4	9.7	-1.5	149	4.1

Dog - G
Right splanchnic nerve sectioned 2/9/55
Sulfadiazine 1 Gm. daily started on 3/26/55

TABLE 2 D

Date	Daily Intake					Water Output ml./24 hr.			Sodium					Potassium					Plasma	
	Casec Gm	Na Cl Gm	K Cl Gm	H2O ml.	Wgt. Lb.	Rt.	Lt.	D-I	Conc. mEq/L. Rt.	Conc. mEq/L. Lt.	Output mEq/24 hr. Rt.	Output mEq/24 hr. Lt.	Output mEq/24 hr. D-I	Conc. mEq/L. Rt.	Conc. mEq/L. Lt.	Output mEq/24 hr. Rt.	Output mEq/24 hr. Lt.	Output mEq/24 hr. D-I	Na. mEq/L.	K. mEq/L.
3/3	180	5	1	1240	25	361	465	+104	77.2	69.2	27.9	32.2	4.3	30.5	25.5	11.0	11.9	0.9		
3/4	180	5	1	665	24	260	320	+60	165	178	43.0	56.8	13.8	24.0	22.7	6.2	7.3	1.1		
3/5	180	5	1	800	24	303	381	78	130	124	39.3	47.2	7.9	19.4	16.6	5.9	6.3	0.4		
3/6	180	5	1	825	24	253	336	83	148	144	37.4	48.4	11.0	20.9	17.6	5.3	5.9	0.6		
3/7	250	5	1	775	24	255	327	72	112	106	28.5	34.8	6.3	18.7	15.2	4.8	5.0	0.2	164	4.5
3/8	250	0	0	625	24	238	278	40	24.3	24.3	5.8	6.8	1.0	5.3	4.2	1.3	1.2	-0.1		
3/9	250	0	0	625	24	209	251	42	18.7	13.7	3.9	3.4	-0.5	3.0	2.7	0.6	0.7	0.1	150	4.1
3/10	250	0	0	825	24	304	360	56	12.3	10.7	3.7	3.9	0.2	2.4	1.7	0.7	0.6	-0.1		
3/11	250	0	0	750		328	385	57	11.3	10.7	3.7	4.1	0.4	1.4	1.6	0.4	0.6	0.2	150	4.7
3/12	250	0	0	1125	24	475	520	45	8.2	8.2	3.9	4.3	0.4	1.2	1.2	0.5	0.6	0.1		
3/13	250	5	1	1200	24	467	502	35	28.4	31.7	13.3	15.4	0.6	7.2	7.3	3.4	3.7	0.3	153	3.9
3/14	250	5	1	1050		404	440	36	86.6	87.9	35.0	38.7	3.7	8.0	7.4	3.2	3.3	0.1		
3/15	250	5	1	1200		522	548	26	74.4	74.4	38.8	40.8	2.0	6.4	5.8	3.3	3.2	-0.1		
	Left splanchnic nerve sectioned 4/6/55																			
4/9	250	5	1	1075		447	345	-102	42.0	65.0	18.8	22.4	3.6	31.4	54.0	14.0	18.6	4.6		
4/10	250	5	1	1200	28½	564	366	-198	54.4	67.4	30.7	24.7	-6.0	14.3	28.6	8.1	10.5	2.4	150	4.0
4/11	250	5	1	900		518	342	-176	76.0	133	39.4	45.6	6.2	14.6	27.3	7.6	9.3	1.7		
4/12	200	5	1	700	28	336	250	- 86	109	139	36.7	34.7	-2.0	20.0	32.0	6.7	8.0	1.3	155	3.9
4/13	200	5	1	550		225	221	- 4	100	128	22.5	28.2	5.7	28.0	27.6	6.3	6.1	-0.2		
4/14	200	0	0	500	27½	255	203	- 52	32.6	49.5	8.3	10.1	1.8	14.5	21.3	3.7	4.3	0.6	161	4.4
4/16	200	0	0	100	27	332	324	- 8	18.0	29.9	6.0	9.7	3.7	5.9	6.1	2.0	2.0	0.0	150	4.0
4/17	200	0	0	800		372	362	-10	12.3	21.5	4.6	7.8	3.2	3.3	3.3	1.2	1.2	0.0		
4/18	200	0	0	525	27	246	243	-3	16.7	31.2	4.1	7.6	3.5	2.5	2.8	0.6	0.7	0.1	149	3.9
4/19	200	0	0	500		247	240	-7	13.3	20.9	3.3	5.0	1.7	1.8	2.3	0.4	0.6	0.2		
4/20	200	5	1	1400	27½	204	188	-16	32.0	40.8	6.5	7.7	1.2	21.5	25.8	4.4	4.9	0.5	156	4.1
4/21	200	5	1	700		293	262	-31	104	126	30.5	32.9	2.4	20.2	27.0	5.9	7.1	1.2		
4/22	200	5	1	650	28	330	318	-12	122	161	40.1	51.1	11.0	11.1	16.5	3.7	5.3	1.6	156	3.7
4/23	200	5	1	500		250	227	-23	171	218	42.8	49.6	6.8	14.2	22.9	3.5	5.2	1.7		
4/24	200	5	1	?	27½	197	182	-15	228	238	44.8	43.4	-1.4	19.1	26.5	3.8	4.8	1.0	156	4.1

Dog - B

TABLE 2E

Date	Daily Intake Casec Gm	Na Cl Gm	K Cl Gm	H2O ml.	Wgt. Lb.	Water Output ml./24 hr. Rt.	Lt.	D-I	Sodium Conc. mEq/L. Rt.	Lt.	Sodium Output mEq/24 hr. Rt.	Lt.	D-I	Potassium Conc. mEq/L. Rt.	Lt.	Potassium Output mEq/24 hr. Rt.	Lt.	D-I	Plasma Na. mEq/L.	K.
4/18	175	5	1	750	26	262	168	- 94	148	164	38.8	27.6	-11.2	41.2	57.6	10.8	9.7	-1.1	148	3.9
4/19	175	5	1	500		441	212	-229	138	216	60.8	45.7	-15.1	22.0	38.4	9.7	8.1	-1.6		
4/20	175	5	1	650	26½	431	188	-243	116	229	50.0	43.0	- 7.0	18.9	41.5	8.2	7.8	-0.4	149	4.2
4/21	175	5	1	775		381	202	-179	120	198	45.7	40.1	- 5.6	23.6	45.5	9.0	9.2	0.2		
4/22	175	5	1	550	26	363	216	-147	128	203	46.5	43.9	- 2.6	21.8	36.8	7.9	8.0	0.1	154	4.1
4/23	175	5	1	700		382	221	-161	122	209	46.6	46.1	-0.5	16.5	28.8	6.3	6.4	0.1		
4/24	175	0	0	?		343	154	-189	19.4	24.0	6.7	3.7	-3.0	6.3	13.5	2.2	2.1	-0.1		
4/25	175	0	0	550		300	144	-156	16.6	17.3	5.0	2.5	-2.5	5.5	10.9	1.7	1.6	-0.1		
4/26	175	0	0	500	26	287	170	-117	25.0	25.6	7.2	4.4	-2.8	7.7	12.3	2.2	2.1	-0.1	147	3.8
4/27	175	0	0	500		282	166	-116	20.0	15.6	5.6	2.6	-3.0	9.6	13.9	2.7	2.3	-0.4		
4/28	175	5	1	750	26	345	184	-161	109	146	37.5	26.9	-10.6	24.3	40.3	8.4	7.4	-1.0	148	3.4
4/29	175	5	1	500		258	175	-83	170	199	43.9	34.8	-9.1	31.9	41.5	8.2	7.3	-0.9		
4/30	175	5	1	600	26	234	159	-75	172	218	40.3	34.6	-5.7	28.3	34.6	6.6	5.5	-1.1	150	3.8
5/1	175	5	1	?		314	204	-110	147	180	46.1	36.7	-9.4	21.8	29.3	6.8	6.0	-0.8		
	Left splanchnic nerve sectioned 5/2																			
5/3						350	468	+118	143	146	50.2	68.3	+18.1	37.6	31.2	13.2	14.6	+1.4		
5/4	250	5	1	750		270	268	-2	67	75	18.1	20.1	+2.0	30.8	35.2	8.3	9.4	+1.1		
5/5	200	5	1	450	25½	182	144	-38	102	132	18.6	19.0	+0.4	26.4	34.0	4.8	4.9	+0.1	146	3.6
5/6	200	5	1	725		333	307	-26	143	172	47.7	52.8	+5.1	12.2	14.8	4.1	4.5	+0.4		
5/7	200	5	1	725	25½	290	283	-7	134	175	38.8	49.6	+10.8	14.6	16.5	4.2	4.7	+0.5	152	4.1
5/8	200	0	0	525		228	199	-29	27.3	25.4	6.2	5.1	-0.9	5.2	5.2	1.2	1.0	-0.2		
5/9	250	0	0	525	25½	219	180	-39	24.3	17.0	5.3	3.1	-2.2	4.0	3.6	0.9	0.6	-0.3	145	3.9
5/10	250	0	0	525		240	194	-46	26.4	12.8	6.3	2.5	-3.8	3.6	3.4	0.9	0.6	-0.3		
5/11	250	0	0	525	25½	270	224	-46	20.3	10.0	5.5	2.2	-3.3	2.3	2.0	0.6	0.4	-0.2	143	3.6

Dog J

TABLE 3

Mean Values

Dog	Experimental Conditions		Urine Flow				Sodium Concentration			Sodium Excretion				Potassium Concentration				Potassium Excretion			
			Rt.	Lt.	D-I	D/I	Rt.	Lt.	D/I	Rt.	Lt.	D-I	D/I	Rt.	Lt.	D/I	Rt.	Lt.	D-I	D/I
G	Before Section	Control Na	329	298	31	1.11	116	126	.92	38.2	37.4	0.8	1.02							
		Restrict.	216	192	24	1.12	26.9	25.5	1.05	5.8	4.9	0.9	1.18							
	Splanchnic sectioned on right	Control Na	310	349	-39	0.89	123	115	1.07	38.1	40.2	-2.2	0.95	23.2	23.2	1.00	7.2	8.1	-0.9	.89
		Restrict. Na & K	284	328	-44	0.87	26.1	18.9	1.38	6.3	6.2	0.1	1.02	14.8	14.6	1.01	4.2	4.8	-0.6	.88
		Restrict. K	234	282	-48	0.83	28.2	15.6	1.81	6.6	4.4	2.2	1.50	1.7	1.4	1.21	0.4	0.4	0.0	1.00
		Restrict.	294	331	-37	0.89	140	124	1.29	41.1	41.2	-0.1	1.00	8.8	7.6	1.16	2.6	2.5	0.1	1.04
B	Before Section	Control Na & K	353	415	62	1.18	93.2	94.9	1.02	32.9	39.4	6.5	1.20	15.3	14.0	0.92	5.4	5.8	0.4	1.07
		Restrict.	311	359	48	1.15	13.5	12.5	0.93	4.2	4.5	0.3	1.07	2.3	1.9	0.83	0.7	0.7	0.0	1.00
	Splanchnic sectioned on left	Control Na & K	336	270	-66	.80	93.2	126	1.35	31.3	34.0	2.7	1.09	19.0	29.6	1.56	6.4	8.0	1.6	1.25
		Restrict.	290	274	-16	.94	17.9	29.2	1.63	5.2	8.0	2.8	1.54	5.5	6.2	1.27	1.6	1.7	0.1	1.66
J	Before section	Control Na & K	341	191	-150	0.56	134	198	1.48	45.6	37.9	-7.7	0.83	24.0	39.2	1.63	8.2	7.5	-0.7	0.92
		Restrict.	303	184	-129	0.61	20.1	17.9	0.89	6.1	3.3	-2.8	0.54	7.3	10.9	1.49	2.2	2.0	-0.2	0.91