REPORT

of

Committee on Thesis

The undersigned, acting as a Committee of the Graduate School, have read the accompanying thesis submitted by Stuart William Harrington for the degree of Master of Science in Surgery. They approve it as a thesis meeting the requirements of the Graduate School of the University of Minnesota, and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Science.

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THE UNIVERSITY OF MINNESOTA

GRADUATE SCHOOL

Report

of

Committee on Examination

This is to certify that we the undersigned, as a committee of the Graduate School, have given Stuart William Harrington final oral examination for the degree of Master of Science in Surgery. We recommend that the degree of Master of Science in Surgery be conferred upon the candidate.

Minneapolis, Minnesota

June 1920

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T H E S I S

THE EFFECT UPON THE KIDNEY OF VARIOUS SURGICAL PROCEDURES UPON ITS URETER, BLOOD SUPPLY AND CAPSULE.

Stuart William Harrington.

Submitted to the Graduate Faculty of the University of Minnesota in partial fulfillment of the requirements for the Degree of Master of Science in Surgery.

May, 1920.

Introduction.

This experimental study upon the kidney and ureter was undertaken to determine the effect produced upon these structures by some of the common surgical procedures or by accidents occurring to them during operations upon other organs.

The problems studied have been classified into four general series, and the phases investigated have been arranged in groups as follows:

SERIES I: The Effect Produced Upon the Kidney and Ureter by Complete Sudden Occlusion of the Ureter or the Collateral Venous Circulation

Group I: Ligation of the Ureter

Group II: Ligation of the Collateral Venous Circulation and the Ureter

SERIES II: The Effect of Occlusion of the Various Vascular Radicals of the Kidney

Group I: Ligation of the Renal Artery

Group II: Ligation of One or More Branches of the Renal Artery

Group III: Ligation of the Renal Vein

Group IV: Ligation of One or More Branches of the Renal Vein

SERIES III: The Effect Upon the Kidney and Ureter of Trauma to the Ureter

Group I: Ureter Clamped

Group II: Ureter Stripped

Group III: The Methods of Anastomosing the Ureter

SERIES IV: The Effect of Decapsulating the Kidney

Group I: Kidney Decapsulated and Wrapped in Omentum

Group II: Kidney Decapsulated and Wrapped in Omentum; Renal Artery or Branches of the Renal Artery Ligated.

The different points in the embryology, anatomy, and physiology are taken up and will be referred to as they have a bearing on the subject at hand.

Embryology.

The process of development of the kidney is more complex than that of any other organ of the human body. A definite understanding of the embryology is most essential to a clear conception of many pathologic and anomalous conditions of the kidney which are due to defective and abnormal development, such as dystopic kidney, supernumerary ureters, double pelves, aberrant blood vessels causing hydronephrosis, and many other conditions.

Following the impregnation and segmentation of the ovum, there is the formation of the three primary germ layers of the body; the ectoderm, the mesoderm, and endoderm. It is from the differentiation and grouping of the cells of these layers that the anlagen of the various developing organs of the embryo are formed.

In the early stages of embryonic growth, certain cells of the mesoderm multiply and group themselves into two lateral plates which lie parallel with the medullary groove, the anlagen of the central nervous system. The lateral plates are divided by a longitudinal groove into the mesial portion and the postero-lateral portion. The mesial portion, called the ventral mesoderm is later divided transversely into 38 segments called mesodermic somites. The cells lying below this longitudinal groove are called the intermediate cell masses and it is from these cells that the kidneys are developed, thus they are of mesodermic origin and are not typical secretory glands, as the ordinary secretory glands are developed from the endoderm or epiderm.

The cells of the intermediate cell masses become thickened and project into the posterior part of the celom and are called the Wolffian ridges which are concerned in the development of the excretory organs. The process directly concerned with the formation of the kidney begins with the

differentiation and rearrangement of the cells in the mesodermic germ layer, and three distinct sets of organs appear in succession, pronephros, mesonephros, and metanephros.

The pronephros develops from the cephalic end of the Wolffian ridge. Corresponding with the process of transverse division of the ventral mesoderm into 38 mesodermic somites, there is a division of the intermediate cell masses down as far as the 10th segment. Posterior to this the Wolffian ridge continues as a column called the nephrogenic cord. Each of the anterior segmental masses of the Wolffian ridge develops a lumen beginning anterior to the cervical region, and by a process of fusion forms the pronephrotic duct which runs backward to open into the cloaca. The pronephrotic tubules are being formed at the same time by invaginations of the intermediate cell masses. These communicate with the pronephric duct and the celomic cavity. The openings into the celomic cavity are called the nephrostomes. Near these openings, branches of the aorta evaginate the celomic epithelium to form external glomeruli. The anterior tubules begin to degenerate as the posterior ones are being formed, all having disappeared in the human embryo of 5 mm. The pronephric duct is the only part remaining, and is now called the Wolffian duct. The mesodermic cells of the nephrogenic cord finally arrange themselves in small solid cords with one end in contact with the celomic epithelium and the other with the Wolffian duct. These cords develop a lumen at their outer ends which communicate with the Wolffian duct. There is an accumulation of the mesodermic cells at the opposite end into which arterial branches grow forming internal glomeruli. This process begins at the 5th or 6th cervical segment and extends to the third or fourth lumbar segment. A process of degeneration takes place in the anterior tubules as the posterior tubules are being formed, in the same manner as in the pronephros. At the 16th week, only the ducts and rudimentary parts of the tubules remain to form accessory parts of the reproductive apparatus.

The development of the metanephros or permanent kidney is the third and final stage and begins before degenerative changes in the mesonephros have reached the lumbar region. At about the fourth week a bud-like growth (renal blastema) appears upon the dorsal surface of the Wolffian duct near its entrance into the cloaca. It grows backward and upward into the posterior portion of the nephrogenic cord into a mass of mesodermic cells called the metanephric blastema. The tubular outgrowth thus formed becomes the ureter and the dilated germinal portion, divided into the cephalic and caudal branches, forms the primary renal pelvis. Each branch is capped with a mass of cells of the metanephric blastema, in which there are two zones consisting of different types of cells, an inner epithelial and outer mesenchymatous. The former becomes the excretory part of the kidney, and the latter forms the supporting structures. The major and minor calices and collecting tubules are formed by outgrowths of the primary renal pelvis. As each outgrowth occurs there is a simultaneous division of the cells of both zones of the metanephric blastema forming a cap of metanephric tissue over each ampulla so that finally each collecting tubule is surrounded by its own cap of inner zone cells called the renal vesicle, which becomes attached to its corresponding terminal tubule and later acquires a lumen continuous with that of the tubule. It then elongates into an S-shaped tube, and its closed end is invaginated by a branch of the renal artery, thus forming a glomerulus. The tubular structure thins out forming two layers of tubular epithelium which almost completely envelop the glomerulus, called Bowman's capsule. The remaining portions of the adult tubule are developed from the curves of this primary S-shaped tubule. The formation of tubulo-glomerular units begins in embryos of about 30 mm. and continues until birth.

During the process of division of the primary pelvis the mesenchyme lying between the evaginations acts as septa which divide the developing kidney into lobe-like pyramids. These divisions may be seen upon

the surface of the fully formed kidney.

As has been shown, the kidney has its origin in the pelvis, in the posterior portion of the Wolffian ridge, and by continued growth it gradually ascends from the pelvis to reach a permanent location about the eighth or ninth week. During its development in the pelvis its transverse axis is antero-posterior with the pelvis anterior, and during its migration from the pelvis it rotates on its axis from this antero-posterior position to a lateral position with the pelvis directed inward and downward.

During the migratory period it is supplied with capillary blood vessels from the neighboring tissues. The lower ones disappear as new ones come into the organ at higher levels until the transposition is completed. The final renal arteries are those enlarged capillaries which were most advantageously placed to supply the renal parenchyma when the kidney had reached its final position.

The renal capsule and supporting connective tissue of the parenchyma are the remains of the lobular septa and are derived from the outer zone of the metanephric blastema comprised of mesenchymatous cells.

Anatomy of the Kidney.

It is unnecessary to go into a detailed description of the anatomy of the kidney as this can be found in any of the textbooks. Only a brief resume' of the main points to be remembered in connection with this work will be outlined.

The kidneys lie in the lumbar region under the lower portion of the thoracic wall. They are behind the peritoneum and surrounded by a mass of fat and loose areolar tissue which constitutes the fatty capsule. The true capsule is thin, smooth, and glistening, and consists of two layers,

an outer fibrous layer and an inner layer of smooth muscle and delicate fibrous tissue which communicates with the kidney reticulum. The outer fibrous layer can be easily removed from the normal kidney leaving the inner layer which is adherent to the kidney tissue. This true capsule completely envelops the kidney and is carried over the hilum to cover the wall of the sinus which is the central cavity of the kidney.

The blood vessels and excretory ducts pass through the hilum of the kidney, the ducts being posterior, the renal vein anterior with the renal artery between. The ureter dilates at the hilum into a funnel-shaped sac called the renal pelvis. It is divided into three large tubular divisions called major calices, which in turn are partitioned off into eight smaller tubular divisions called minor calices, the upper and lower of which have double papillae. The remaining six minor calices form a double row anterior and posterior to the median sagittal line of the kidney. The kidney parenchyma is divided into two parts, an outer glandular portion called the cortex, and an inner medullary portion consisting of the pyramids, the bases of which rest upon the cortex, the apices projecting into the kidney pelvis. Prolongations of the cortical substance containing the blood vessels, nerves and lymphatics lie between the pyramids and are called the columns of Bertin.

The renal parenchyma is composed of a large number of units each of which consists of a long branched tube closed at one end and running a complex course through the cortex and medulla to terminate with other tubules in a collecting tubule which empties into the kidney pelvis. This unit or uriniferous tubule takes its origin in Bowman's capsule, a hollow sphere of delicate flattened epithelium, one side of which is invaginated by a tuft of capillaries and arterioles until the cavity is almost obliterated. This structure is called the malpighian body. These malpighian bodies are situated in the cortical substance of the kidney. The glomerular capsule empties into the

tubule proper by a narrow neck lined with cuboidal epithelium. The first portion of the tubule runs a tortuous spiral course in the neighborhood of the glomerulus and is called the proximal convoluted tubule. It is lined by a high cuboidal epithelium having distinct basement membrane containing many granules arranged in a striated manner in their outer zone. The tubule passes in a straight course towards the pelvis of the kidney, then doubles back forming the loop of Henle and again passes the region of the capsule. This loop portion of the tubule is smaller than the rest but its lumen is not diminished, the difference in the size being due to the type of epithelium with which it is lined - a thin pavement variety with large nuclei and few or no granules. The next portion runs a tortuous course in the region of the capsule and is called the distal convoluted tubule. It is lined with epithelium similar to that in the proximal convoluted tubule except that it has few granules. The last portion, called the collecting tubule, is lined with low columnar epithelium and passes through the medulla emptying into the pelvis through the papilla. The number of kidney units or uriniferous tubules contained in the kidney of a dog of 11 kg. has been estimated at 142000, and the number in the human kidney, 2000000.

The lymph vessels form a capillary network around the tubules, then unite to make larger trunks which leave the kidney in two ways: The majority pass from the hilus with the renal vein. The others leave the kidney with the veins which perforate its capsule.

The nerve supply of the kidney comes from the sympathetic system, being derived mainly from the semilunar ganglion, a few branches from the plexuses around the suprarenal and aorta, and as a rule, one twig directly from the splanchnic plexus.

The blood supply of the kidney is carried to it by the renal artery which comes off the aorta opposite the first lumbar vertebra. The right renal artery is a little longer and higher than the left and passes beneath

the vena cava, head of the pancreas, and second portion of the duodenum to reach the hilus of the kidney. The left renal artery passes beneath the head of the pancreas before reaching the hilus. The renal artery sends branches to the fatty capsule and suprarenal before entering the hilus. At the hilus, this artery divides into four, and sometimes five branches. Of these, three and sometimes four carrying two-thirds to three-fourths of the blood, pass into the kidney anterior to the pelvis; one, sometimes two of the branches carrying one-fourth to one-third of the blood, pass posterior to the pelvis. These branches hug the respective walls of the sinus very closely. Just before entering the kidney parenchyma they break up into a number of branches which completely surround the papillae in a fan-like arrangement. Their course through the kidney corresponding in position to the original tracts of connective tissue that separated the primary divisions of the fetal kidney. They are called the interlobar arteries. When they reach the base of the pyramids at the juncture of the cortex and the medulla, they pass in a direction parallel to the surface of the kidney, and are termed the arcuate arteries. From the convex side of these arches, branches are given off at an acute angle which run towards the periphery and subdivide into smaller vessels all of which terminate in fine branches running a straight course outward between the lobules, and are called the interlobular arteries. From the under surface of the arcuate arteries there are a few small branches running a downward course and which terminate in interlobular arteries. From these interlobular branches there arise at all levels through the cortical substance afferent glomerular branches, thus the terminal branches of each interlobular artery as it reaches the periphery of the cortex divides into afferent glomerular vessels. The terminal branches of the arcuate arteries ultimately form interlobular arteries which in turn end in afferent glomerular vessels. It is thus seen that the afferent glomerular vessels arise from all branches of the renal artery beginning with the arcuate arteries, and with few exceptions each afferent

glomerular vessel terminates in a glomerulus. The main exceptions are the A. Nutriciae Pelvic-renalis and the A. Recurrentes within the sinus of the kidney. The kidney vessels are end-arteries in the strictest sense of the word, there being no anastomoses between the primary branches of the renal artery. Each glomerulus constitutes a retemirabile, the branches of which unite to form a single efferent vessel regarded as an arterial and not a venous vessel. This efferent vessel after leaving the glomerulus soon divides to form a capillary network, the disposition of which differs in different portions of the kidney. The efferent branches of the glomeruli, the afferent branches of which arise from the arcuate arteries, and also efferent branches of many of the glomeruli the afferent branches of which spring from the lowermost portions of the interlobular arteries, divide into bundles of long slender branches which pass into the medulla and are called the Arteriolae Rectae or Arteriolae Rectae Spuriae. The efferent vessels of the remaining glomeruli divide to form capillary plexuses which surround the segments of the renal tubules of the cortex. There is not a difference of kind of capillary plexuses but a difference in arrangement due to the character of the tubular structure of the various portions of the kidney. There are terminal arterial branches described which end in capillaries without the interposition of a glomerulus, supplying the kidney tubule with arterial blood which has not passed through a glomerulus. These vessels are called Arteriolae Rectae Verae in the medullary portion of the kidney. They are the end branches of the interlobular arteries in the periphery of the cortex, but according to Huber, "There is evidence to show that such Arteriae Recti-Verae are at one time in their development efferent branches of a glomerulus which later with the uriniferous tubule, degenerate during the development of the kidney". He also states that these vessels are more numerous in the dog than other mammals. The practical significance of this arrangement of the blood supply is that all nutrient blood coming to the kidney tubules first passes

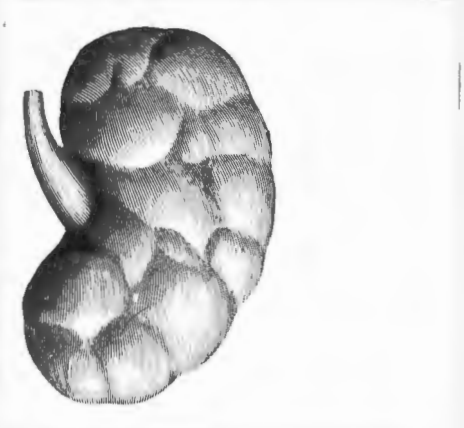
through the glomerulus and its concentration is more or less altered by the entrance of substances into the kidney tubule.

The glomerulus is so constructed that an enormous amount of blood comes in contact with the epithelium of Bowman's capsule. The afferent vessel of the glomerulus is larger than the efferent vessel. After entering the capsule of Bowman, the afferent vessel subdivides into five diverging branches which form numerous subdivisions and communicate very freely, finally uniting into one vessel, the efferent vessel of the glomerulus, thus forming practically a spherical tuft of blood channels.

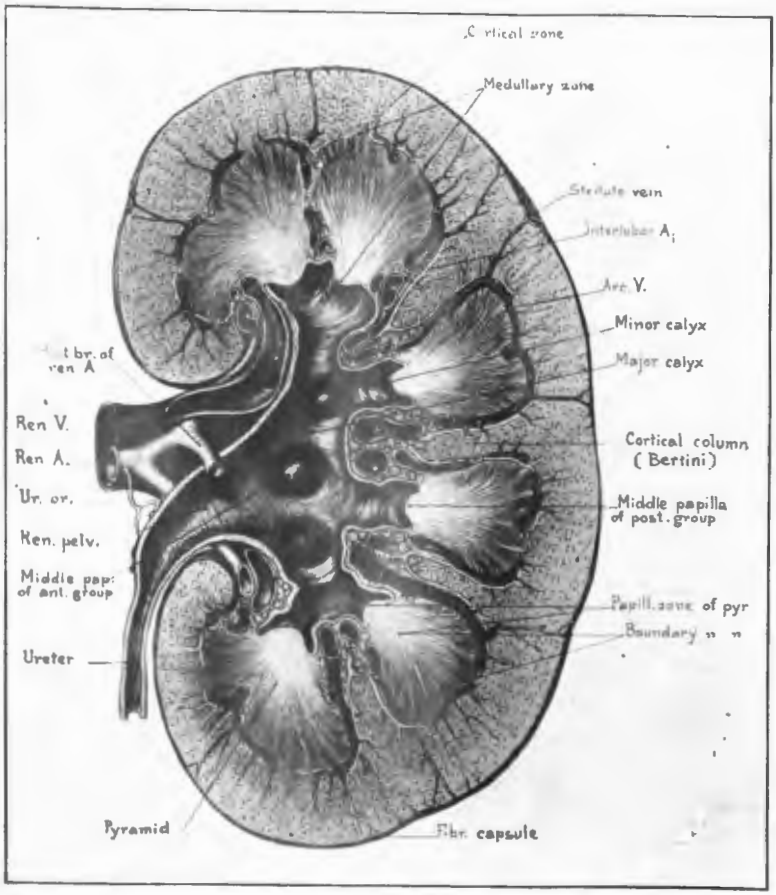
The arterial system is fundamentally the same in kidneys of various mammals, but the venous system, especially the larger venous branches of the kidney vary a great deal in different animals. In the dog relatively large veins are formed immediately under the capsule which receive venous radicals draining the outer half of the cortex, uniting to form relatively large veins which pass through the cortex to end in arcuate veins. These latter also receive short venous branches from the cortex and numerous branches formed by the anastomosis of Venulae Rectae. According to Huber, this is the probable arrangement in the human kidney. In the guinea pig and rabbit the veins begin in the cortex as interlobular venous branches similar to the interlobular arterial divisions and end in the arcuate veins, which also receive the Venulae Rectae. In the cat, large veins are formed immediately under the capsule which converge toward the hilum receiving all along their course radicals from the outer half of the cortex. The arcuate veins in the periphery of the medulla receive the Venulae Rectae and short radicals which drain the lower portion of the cortex. The subcapsular and arcuate veins unite at the hilum to form the renal vein.

In the human, the arcuate arteries are formed by the anastomosis of the larger trunks from the renal cortex at the base of the

pyramids. These arcuate veins in turn unite to form the larger trunks called the interlobar veins which run between the sides of the pyramid and the columns of Bertini until they reach the lower portion of the pyramid where they lie between the arterial branches and the pyramid. Here they form a second system of anastomoses both anteriorly and posteriorly, making a collar around the calyx. The veins about the posterior calices run forward, usually in two large trunks which pass anterior to the pelvis of the kidney and unite in the renal sinus with large vessels from the anterior calices to form the renal vein, which empties into the vena cava. It is generally stated that the large veins at the hilum are in front of the artery. However, this is usually not the case, as at the hilum as well as throughout the kidney, the veins are situated between the arteries and the pelvis, while at the vena cava, the vein is in front of the artery.



Fetal Kidney



-Longitudinal Section Through Human Kidney, Showing Gross Anatomy.

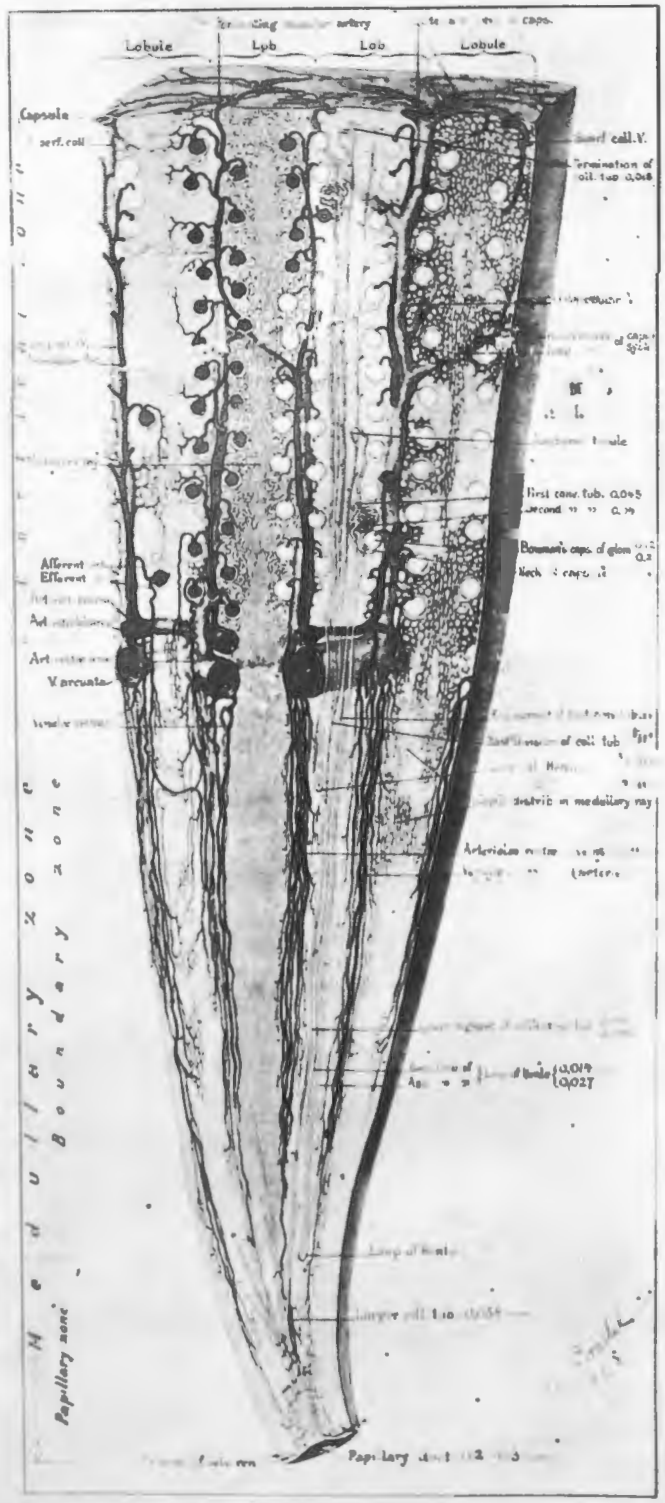


FIG. 101.—A LOW-POWER MAGNIFICATION OF A WEDGE OF KIDNEY SUBSTANCE EXTENDING FROM PAPILLA TO SURFACE. The arteries are shown black, the veins gray. Four lobules are included.

... principle of the vascularization; note the efferent branches of the

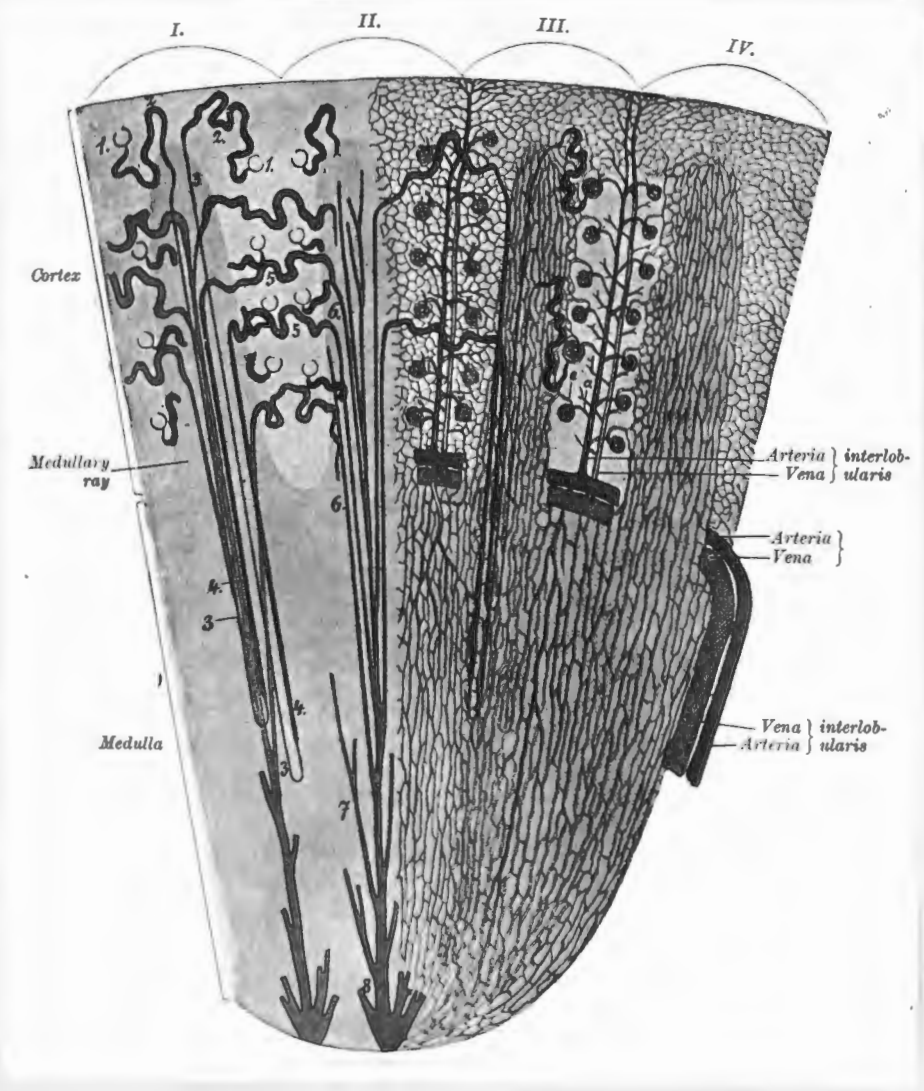


Diagram of a longitudinal section of the human kidney showing the microscopic structure of the cortex and medulla.

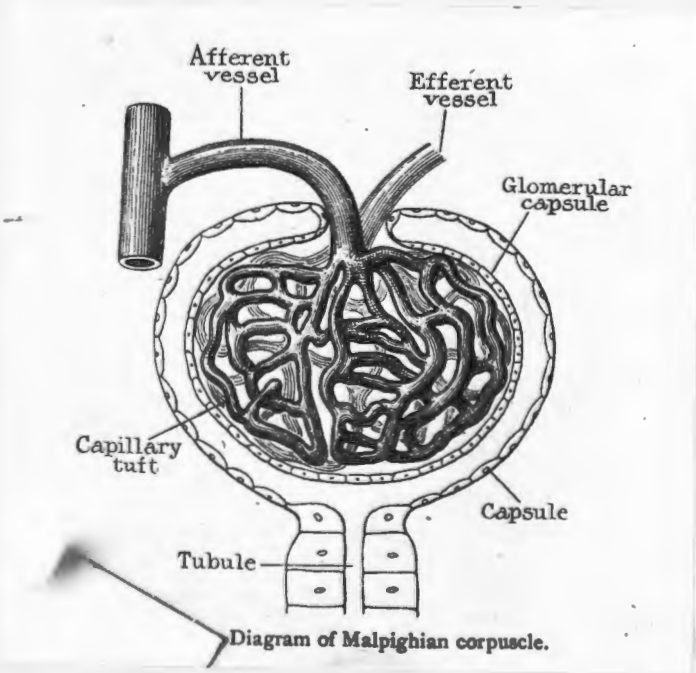
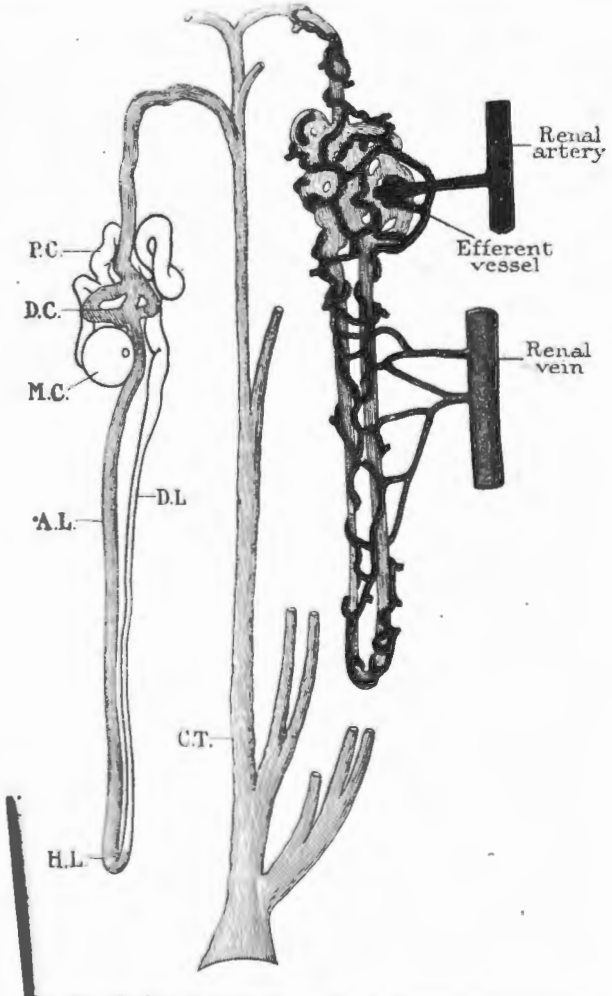
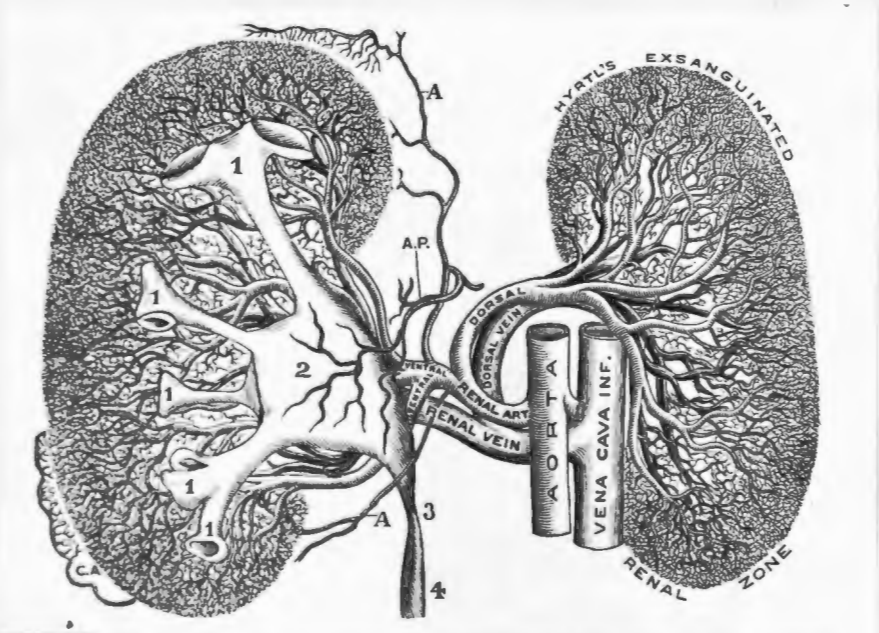


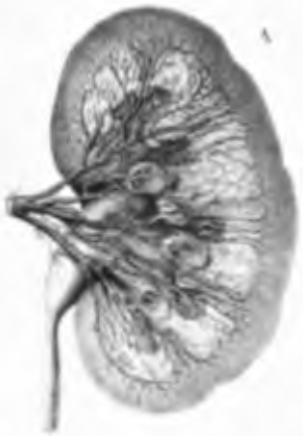
Diagram of Malpighian corpuscle.



On the left the tubule is drawn after a diagram of G. C. Huber's. The tubule is outlined from the capsule to the loop of Henle and is shaded from that point to the end of the collecting tubule. On the right a diagram of the circulation is added : M.C., Malpighian corpuscle ; P.C., proximal convoluted tubule ; D.L., descending limb ; H.L., loop of Henle ; A.L., ascending limb ; D.C., distal convoluted tubule ; C.T., collecting tubule.

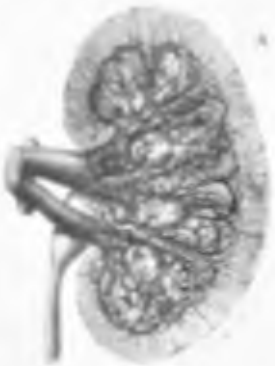


Corrosion anatomy. The renal vascular blades opened like a book. (Byron Robinson.)



A

B



A

B

PHYSIOLOGY

The first theory as to the formation of the urine was put forth in 1842 by Bowman, who based his ideas of the mode of renal activity upon the anatomical characteristics of the kidney. The relation of the glomerulus to the kidney tubule suggested to him that the water was secreted in the glomerulus and the solids by the tubules and were washed out into the pelvis by the water from the glomerulus. This view was contradicted two years later by Ludwig who thought that the secretion of urine was due to a process of filtration. He held that the capsule was a simple filter which allowed the passage of all the non-protein constituents of the blood plasma to pass through it into the tubules, where by a process of diffusion, much of the fluid was returned into the blood, the remainder of the filtrate being eliminated as the urine. This theory was also advocated upon an anatomical basis but later examined experimentally. These two views had about an equal number of followers until Heidenhain, in 1874, supported and elaborated upon Bowman's theory by two important investigations, which was the first experimental work done to substantiate it. The Bowman-Heidenhain theory, as it is now called, attributes the secretion of a dilute fluid not far removed from the proteinized lymph to the glomerulus. This fluid as it passes down the tubule carries with it the products of the secretion of the tubular walls such as urea, uric acid, and salts, thus forming the normal urine. The theory, briefly stated, attributes the secretion of the urine to the vital activity of the tubular cells. This work entirely overshadowed Ludwig's theory which is no longer held as more recent advances in physical chemistry has completely disproven his view by showing that the known physical forces are

inadequate to form from the plasma a fluid of greater osmotic pressure (Cushny). The recent or modern theory embodies both views in some respects, and consists of two distinct processes differing in site and nature. The filtration of the non-colloidal constituents of the plasma takes place through the glomerular capsule and by active absorption the fluid best adapted for the tissues is re-absorbed through the tubular cells; the remainder passes into the pelvis as normal urine. These processes are independent but are co-ordinated to a degree by the common blood supply, as its augmentation favors filtration through the capsule increasing the nutrition of the cells of the tubules, and in this way enhancing their activity. This theory therefore embodies both the process of filtration and the vital activity of cells.

Review of the Literature on Hydronephrosis.

The literature upon experimental hydronephrosis is very voluminous. The problem has been attacked from many angles and therehas been a great variation in the results obtained. Only a brief resume' will be given here. The following workers have produced hydronephrosis by complete, sudden occlusion of the ureter; but most of the recent observers have been working upon the problem of the restoration of function in the hydronephrotic kidney:

B. S. Amos ligated the ureter of ten rabbits and six guinea pigs, producing dilatation of the ureter and pelvis of the kidney, resulting in the death of each animal. The rabbits lived from twentyeight to 130 days, with an average of fifty-two days. The guinea pigs lived from five to twenty-three days, with an average of nineteen and one-half days. She concluded that in every instance, ligation of the ureter will cause a hydronephrosis, but that ligation of the ureter does not stop the secretion of urine, and assumes that death is caused by absorption of toxic constituents of the urine from the hydronephrotic sac.

E. R. Bradford, in twelve two-stage experimental operations upon dogs (first ligating the ureter from eleven to forty days, then bringing the ureter to the skin and producing a urinary fistula), found upon sacrificing the dogs in from seven to fifty-one days, that ligation of the ureter caused a hydronephrosis in all of the animals; three of the dogs had developed a pyonephrosis. After the second operation atrophy ensued; the kidney retained its normal shape but was reduced from one-third to one-fourth its normal size.

R. Pearce, cited by G. D. Scott, reported a series of experiments upon five rabbits and one rat, in which he produced a hydronephrosis by ligating the ureter, the degree of the hydronephrosis depending upon the duration of the obstruction. He concluded that "ligation of the ureter or vessels of the kidney does not produce changes which differ in any way from those which follow unilateral nephrectomy and that under normal physiological conditions, degeneration of the renal cells does not lead to the production of auto-nephro-toxins".

F. A. Bainbridge produced a hydronephrosis in experiments upon cats by ligating one of the ureters. After varying periods of dilatation, he measured the pressure of the fluid in the sac and analyzed the fluid. He then emptied the pelvis and gave a diuretic, then collected the urine from the two sides, and found a steady decrease in the amount of water and solids excreted by the hydronephrotic kidney, decreasing directly with the duration of the obstruction. He found that the kidney was still capable of secreting water and nitrogenous substances at the end of two months. Injecting potassium iodid into the dilated ureter at a pressure of fifty to eighty mm. of mercury, he obtained no evidence of absorption from the renal pelvis.

E. H. Weld, in experiments upon dogs, produced a hydronephrosis by ligating the ureter and showed that hydronephrotic kidneys allow absorption from their sacs in inverse proportion to the size of the sac or the amount of kidney destruction, that the absorption takes place through the medullary portion

of the kidney which is the portion first destroyed by hydronephrotic pressure atrophy.

T. Sollman, W. W. Williams, C. E. Briggs, in a series of experiments upon dogs to determine the possibility of re-establishing the kidney function after temporary ligation of the ureter, produced a hydronephrosis by ligating the ureter of four dogs. Two of the dogs died. The remaining two dogs were re-operated upon in 107 days and a urinary fistula established with the hydronephrotic kidney. The operated kidneys were found to contain from eighty-eight to 250 c.c. of fluid and weighed one-third to one-half as much as the opposite normal kidney when the fluid was removed. From these two experiments they concluded that the kidney function could not be restored if the ureter had been occluded for any considerable length of time.

G. B. Scott, in a series of thirty experiments upon dogs, of three to 198 days duration, in which he produced a complete obstruction by ligating the ureter, concluded that sudden complete obstruction of the ureter causes hydronephrosis, the degree depending upon the duration of the obstruction, and that the compensatory circulation by anastomosis of the renal vessels with the vessels entering the kidney through the capsule, probably plays no part in determining whether a primary atrophy or a hydronephrosis will develop. In twenty experiments from one to 163 days duration, in which he produced an incomplete obstruction of one ureter, he found a varying degree of hydronephrosis, depending upon the duration and upon the pressure required to force the urine past the constriction. The dilatation is less rapid than in complete obstruction of the ureter, and that complete obstruction of the ureter never produces atrophy of the kidney. Only one of his cases of incomplete obstruction resulted in primary atrophy.

R. A. Johnson, in a series of experiments upon rabbits to study the function of the hydronephrotic kidney by means of the phenosulpho-

nephthalein test of Rowntree and Geraghty, produced a hydronephrosis in all the animals by doubly ligating and sectioning the ureter. The studies upon the re-establishment of function of the kidney were carried out by completely obstructing the ureter for three, seven, fourteen, seventeen, nineteen, and twenty-one days, at which time a uretero-cysto-neostomy was done. The opposite kidney was removed five, sixteen, thirty-five, fourteen, eighteen, three, and twenty-one days later respectively. The last four rabbits died of renal insufficiency, in nine, three, four, and nine days respectively, after nephrectomy. In the first three, the phenosulphonephthalein test became normal in seventeen, forty, and 152 days respectively after the opposite kidney was removed. From these experiments he concludes that "complete obstruction of the ureter causes atrophy of the renal parenchyma, especially marked in the portions lateral to the renal sinus, the longer the duration of obstruction the greater the degree of atrophy, and that if the obstruction is removed within two weeks, the kidney may regain its normal structure except for a varying amount of atrophy in the lateral portions. Kidneys obstructed for two weeks or less, may regain their normal function as rated by the phthalein test. The longer the period of obstruction the slower the rate of recovery."

Frank Hinman, in a series of experiments to study the repair after hydronephrosis in white rats, shows that in rats, the findings are almost constant after complete obstruction of the ureter, which is the production of a hydronephrosis, the degree depending upon the duration of the obstruction, and that atrophy is never seen without hydronephrosis. From his functional studies he concluded that the recovery and hypertrophy of already atrophic structures and their renewed susceptibility and even hypersusceptibility to intravital staining, is evidence of the ability of the hydronephrotic kidneys up to a certain stage (sixty days) to undergo considerable anatomic and functional restoration. That, in a hydronephrosis of ninety-five days or

longer, recovery either anatomic or functional of the secretory elements cannot be demonstrated.

J. D. Barney, in a series of experiments, ligated one ureter in thirty-three different animals, seven rabbits and twenty-six dogs. Duration of the experiments two to 258 days, an average of thirty-seven days. He produced a hydronephrosis in all except one experiment and concluded that the production of hydronephrosis depends entirely upon the anastomosis of the capsular vessels with the vessels of the general venous circulation and that the degree of the hydronephrosis is dependent upon the extent of the anastomosis. In the one exception he produced an atrophy in the kidney by ligating its ureter. In this case he did not observe a dilatation of the collateral blood supply and concluded that because of the lack of development of this collateral venous circulation, hydronephrosis did not develop and atrophy ensued. In a later series of experiments upon four dogs, he ligated the collateral capsular veins at the same time that he ligated the ureter. Two of these dogs died of sepsis. The remaining two dogs were sacrificed in fifty days. At autopsy he found atrophy of the kidney in each instance. His conclusions are, "if the venous collaterals of the kidney do not develop or if their formation can be prevented after occluding the ureter, hydronephrosis will not develop and renal atrophy will take place".

W. Lindemann, in six experiments upon dogs, ligated one ureter. In three of these primary atrophy of the kidney resulted; in the remaining three a simple hydronephrosis developed. He concluded that whether a primary atrophy of the kidney or a hydronephrosis resulted depended upon the development of the compensatory anastomosis through the capsular vessels. He states that the increased intrapelvic pressure following ligation of the ureter blocks the renal vessels coming into the hilus and the further effect of the ligature depends upon the amount of blood the kidney substance can obtain through

anastomosis between the renal vessels and its capsular vessels. Kidney atrophy means a primary insufficiency of compensation through the blood supply of the capsule, but if the capsular blood supply is good, hydronephrosis will develop.

Geo. D. Stewart and W. H. Barber, in a series of experiments upon dogs, stripped the ureter of all its blood vessels and nerves, from the pelvis of the kidney to the bladder, producing a ureteral paralysis. In another series, the uretero-vesical valve was also cut. A cubital foreign body infected with autogenous colon bacilli and other organisms was placed in the bladder. Their conclusions were that 66 percent of cases of parlysis of the ureter are followed by urinary stasis and kidney distention and that the pathological changes in hydronephrosis of functional origin correspond to the age of the adynamic ureter. In one case they stripped both ureters, ligating the ureter on one side. There was a hydronephrosis of both kidneys, that upon the side of the ligated ureter being twice as great. They believe that complete sudden obstruction of the ureter brings about rapid atrophy following a transitory distention.

Method of Experimentation.

Dogs were used in all experiments because of the similarity of the anatomy, more especially the physiology and blood supply, to that of the human. Each surgical procedure was carried out with the strictest observance of aseptic technic identical with that used upon the human. The dogs were anesthetized with ether, the abdomen shaved, thoroughly cleansed with alcohol and benzine and two applications of 10 percent iodine. A straight upper right rectus incision was made permitting an abdominal exploration and transperitoneal exploration of both kidneys and ureters. In many instances surgical procedures were carried out other than those upon the kidney and ureter as gastroenterostomy, partial resection of the stomach, cholecystectomy, fat transplantations, etc., for the study of other problems, but, in no instance was any procedure undertaken which would in any way influence the results of the problem at hand. The peritoneum and fascia were closed with catgut and the skin with linen. The wound was sealed with collodion. The technic of the various surgical procedures will be stated briefly under each series of experiments. The dogs were kept under the most favorable conditions for health.

The first problem investigated was the effect produced upon the kidney by sudden complete occlusion of the ureter and the factors concerned in the end results. This problem has a practical significance as it is frequently necessary to ligate one of the ureters during the course of various surgical procedures upon the human, such as the removal of malignancy of the bladder or pelvis where it is impossible or does not seem advisable to make other disposition of the ureter. The ureter has been ligated almost with impunity in the human although many textbooks upon surgery differ as

to the effect produced upon the kidney.

Watson and Cunningham state that complete total obstruction of the ureter does not cause hydronephrosis. Morris states that sudden complete stoppage of the outflow of urine leads to rapid atrophy and ultimate disappearance of the affected kidney. Keyes states that sudden complete obstruction of the ureter causes damming back of the urine upon the kidney with acute renal congestion and a diminished secretion of the urine and this causes an increased intrarenal pressure. The congestion is exchanged for atrophy. Adami and Nicholls state that complete obstruction to the outflow of urine leads to atrophy of the affected kidney.

Series No. 1.

The Effect Produced Upon the Kidney by Complete Sudden Occlusion of the Ureter.

Group 1.

The operative procedure in this group of twenty dogs was practically the same in each instance. The appearance and relative size of the kidneys and ureters were determined by transperitoneal exploration; the intestines were packed off and the lower third of the ureters exposed. If normal, one ureter was doubly ligated with linen and sectioned, then replaced behind the peritoneum. The immediate recovery of the animals from the operation and their general health during the experiment was good in all cases. Eleven of the dogs were sacrificed at desired intervals; of the remaining nine, five died of distemper, two of sepsis from other operative procedures, and two from accident. A complete autopsy was made on each animal.

In this group there are two experiments in which the ureter was doubly ligated with catgut and sectioned. I have also included two in which the ureter was ligated with catgut but not sectioned, because they

indicate the necessity of using permanent suture material to tie off the ureter when complete occlusion is desired.

The primary effect upon the kidney, resulting from ligation of the ureter, is shown in Experiment No. 20. There is a marked congestion and edema from the increased intrarenal pressure which partially obstructs the venous return by compression of the smaller capillaries. This pressure is at its maximum in about twenty-four hours and gradually decreases with the duration of the obstruction. Rosow, cited by Barney, found an intrarenal pressure of 90 mm. in twenty-four hours, and 6 mm. at the end of 279 days. The increased pressure soon produces a dilatation of the pelvis and ureter. The sequence of events is very regular but not uniform in all the animals of the same duration of obstruction.

The increased intrarenal pressure produced by obstruction to the outflow of urine, next causes a dilatation of the kidney pelvis and flattening of the renal papillae and compression of the medulla. This process is well-marked in seventy-two hours. It is progressive and fairly uniform in the sequence with which the portions of the kidney are affected. There is first, a definite thinning out of the renal parenchyma in the lateral portions of the kidney within ten days. The poles are next involved, showing a rather marked change in fourteen to eighteen days. The median sagittal portion is the most resistant and at twenty-one days considerable renal parenchyma remains, while at four weeks and over there is only a very thin layer of renal parenchyma in scattered patches lining the sac. The interior of the kidney is partitioned off by columns of fibrous tissue radiating in a fan-like arrangement from the ureteral outlet. These bands or columns contain the blood vessels, which with their fibrous sheaths, are relatively unaffected by the intrarenal pressure that causes atrophy of the renal parenchyma lying between them. It is these bands of fibrous tissue that cause the lobulated appearance of the surface of the kidney.

The renal vessels can be seen running through the lobular grooves.

The pelvis and ureter are markedly dilated and there is an accompanying dilatation of the capsular veins, radiating from the pelvis and passing over the surface of the sac. The degree of dilatation seems to depend upon the amount of hydronephrosis present. The changes in the renal parenchyma follow the same progressive course as the gross changes. There is dilatation of the whole tubule (most marked in the collecting tubule and Bowman's capsule) with compression of the convoluted tubule and loop of Henle. Atrophy is noted first in the convoluted tubules but is soon followed by the same process in all of the tubular structures. The glomerular tufts like the larger blood-vessels in the medulla are much more resistant than the renal parenchyma and remain for a long time after the tubular structure has disappeared completely.

A kidney which has been obstructed for twenty-one days had the power to secrete urine as shown in Experiment No. 6, in which the dog was re-operated on the twenty-first day and the contents of the hydronephrotic sac removed. The dog was sacrificed on the fortieth day and the sac was refilled with fluid. In Experiments No. 7 and 8, the hydronephrotic sac was evacuated on the fourteenth and seventh day after the ureteral ligation. The sacs had refilled when the dogs were sacrificed on the twenty-eighth and twentieth days respectively.

The contents of the hydronephrotic sac are acid in reaction with a specific gravity which is fairly constant, ranging from 1008 to 1010. The fluid contains albumin and urea. There is usually sediment containing blood, probably the result of the early congestion of the kidney. In five experiments there was a marked infection (pyonephrosis) present at autopsy. The accompanying charts and photographs arranged in the order of the duration of the experiments give a very graphic idea of the progressive development of the hydronephrosis.

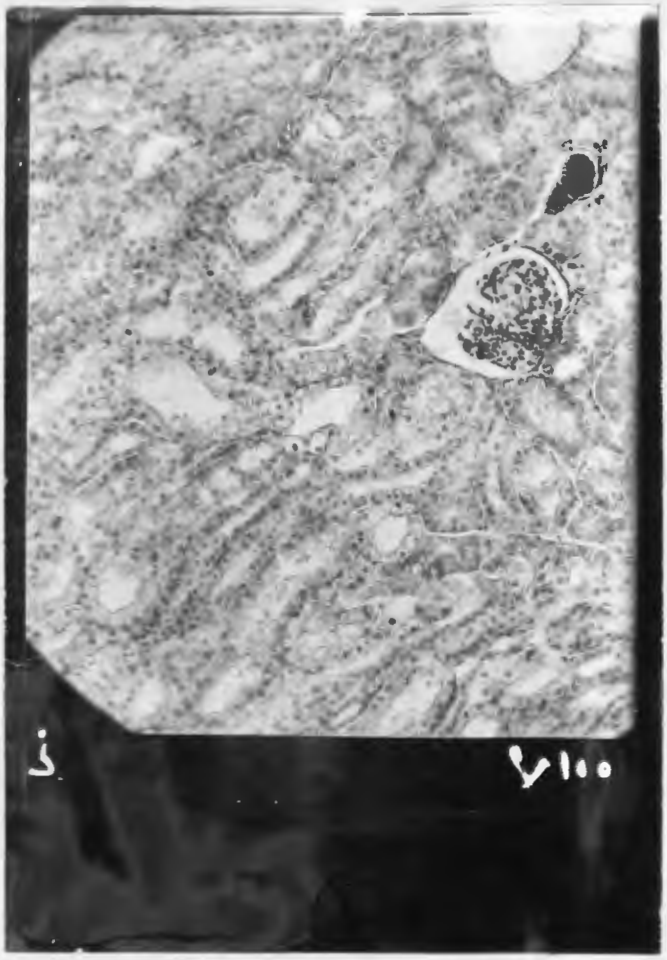
In Experiments No. 1 and 12, the ureters were ligated with #1 plain catgut. In the former, the dog died on the sixteenth day of peritonitis from leakage of the ureter, a pyonephrosis having developed in the kidney. In the Experiment No. 12, the dog was sacrificed on the eleventh day. The capsular veins were dilated and the ureter and pelvis enlarged but collapsed, showing evidence of a previous hydronephrosis, the contents of the sac having leaked out through the ureter. In both these experiments there had been an absorption of the catgut ligature with leakage of the contents of the hydronephrotic sac. In Experiments No. 21 and 22, the ureters were ligated with #1 plain catgut but not sectioned. In No. 21, the dog died 223 days after operation. On examination of the ureter, the site of the catgut ligature could not be found. There was no dilatation of the ureter and the kidney was normal. In Experiment No. 22, the dog was sacrificed in 239 days because of an infection resulting from another operation. Upon examining the ureter a slight constriction was found at the point of ligation with some dilatation of the ureter and pelvis above the constriction. There was slight atrophy of the kidney, which weighed 33 grams, while the opposite kidney weighed 37 grams.

These two experiments show the necessity of using permanent ligating material when complete obstruction of the ureter is desired, as absorbable ligatures may produce only partial constriction, and atrophy of the kidney ensue as shown in the accompanying photographs.

Series I

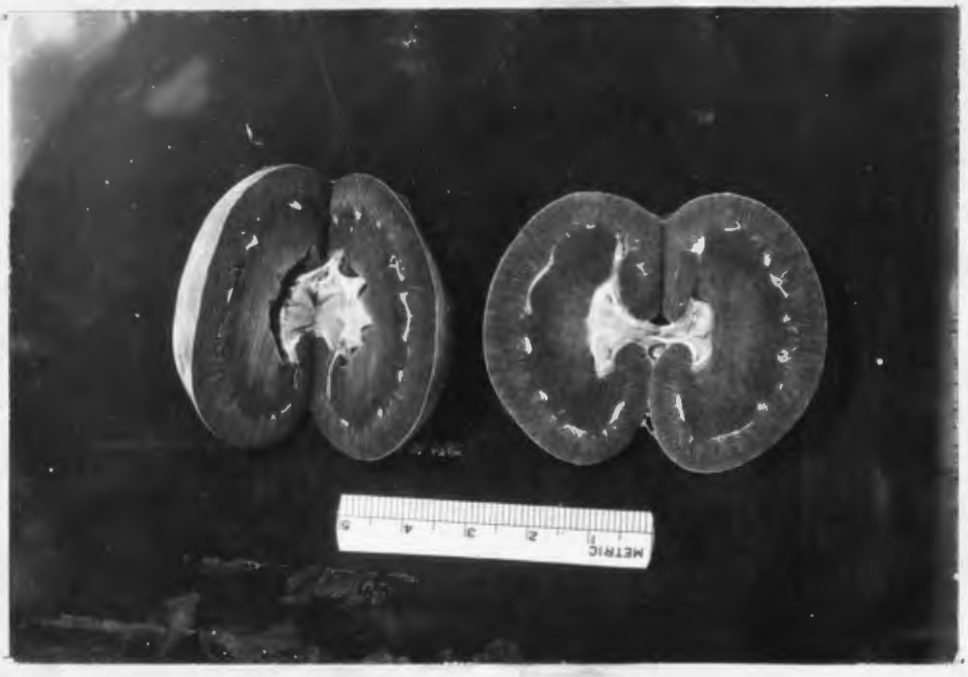
GROUP I - DOUBLE LIGATION AND SECTION OF ONE URETER.

Exper. Number	Animal Number	Series Number	Date of Operation	Duration of Experiment in days	Ureter Ligated Right or Left R L	Double Ligation and Section of Ureter	Autopsy Number	Pyonephrosis	Hydronephrosis	Small	Moderate	Large	Dilatation of Capsular Veins	Condition of Unoperated Kidney	Reoperation; Contents of Sac Removed	Sac Refilled
			1916													
723	B678	1	12-23	447	R	+	121		+			+	+	hyper.		
			1917													
566	C 95	2	6-10	217	RR	++	119		+			+	+	hyper.		
			1917													
43	B732	3	1-20	153	R	++	386		+			+	+	norm.		
			1917													
290	B901	4	3-26	102	R	+	407		+			+	+	norm.		
			1916													
724	B655	5	12-23	47	R	+	54		+			+	+	norm.		
			1916												after	
727	B679	6	12-23	40	R	+	38		+			+	+	hyper.	21 days	+
			1917												after	
31	B720	7	1-3	28	L	+	62		+			+	+	norm.	14 days	+
			1917												after	
97	B767	8	2-10	20	L	+	105		+			+	+	norm.	7 days	+
					plain catgut											
			1917												after	
631	C136	9	9-10	16	R	+	530	+			+		+	norm.	7 das.	+
			1917													
17	B706	10	1-9	13	L	+	16		+		+		+	norm.		
			1917													
436	C 12	11	6-4	12	R	+	377		+		+		+	norm.		
					plain catgut											
			1917													
592	C115	12	8-24	11	R	+	506		+		+		+	norm.		
			1917													
51	B740	13	1-27	10	L	+	50		+		+		+	hyper.		
			1917													
18	B707	14	1-9	9	L	+	15		+		+		+	norm.		
			1917													
14	B703	15	1-6	7	L	+	13		+	+			+	norm.		
442	C 18		1917													
442	C 18	16	6-8	6	R	+	370		+	+			+	hyper.		
			1917						Dilatation of pel-							
519	C129	17	8-31	4	R	+	511		vis and ureter				+	norm.		
			1916						Dilatation of pel-							
725	B528	18	12-23	3	R	+	577		vis and ureter				+	norm.		
			1917						Dilatation of pel-							
284	B872	19	3-17	3	L	+	154		vis and ureter				+	norm.		
			1916						Congestion & edema							
726	B600	20	12-25	1	R	+	576		of entire kidney				+	norm.		
					plain catgut									both		
			1917											both		
252	B874	21	3-17	223	L	0	49		0		Ureter normal			norm.		
					plain catgut						Ureter partial ly constricted					
			1917													
250	B873	22	3-17	239	L	0	48		0		Slt.atrophy of kidney.			norm.		



Experiment No. 9
B 600

Ligation of the ureter- twenty-four hours duration. Dilatation of the renal tubules and glomeruli. Cloudy swelling and beginning degeneration of the renal parenchyma.

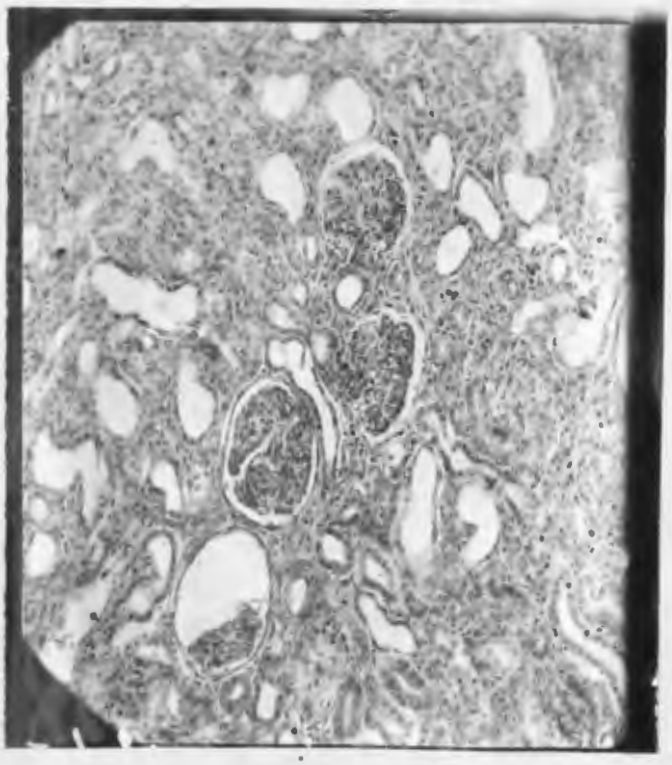


Experiment No. 18
B 528

Ureter ligated- three days duration. Beginning hydronephrosis. Flattening of the renal papilla with depression and narrowing of the medulla.

Experiment No.8
B 767

Ligation of the ureter- twenty days duration. Hydronephrosis. Marked dilatation of the capsular vessels over surface of hydronephrotic sac.

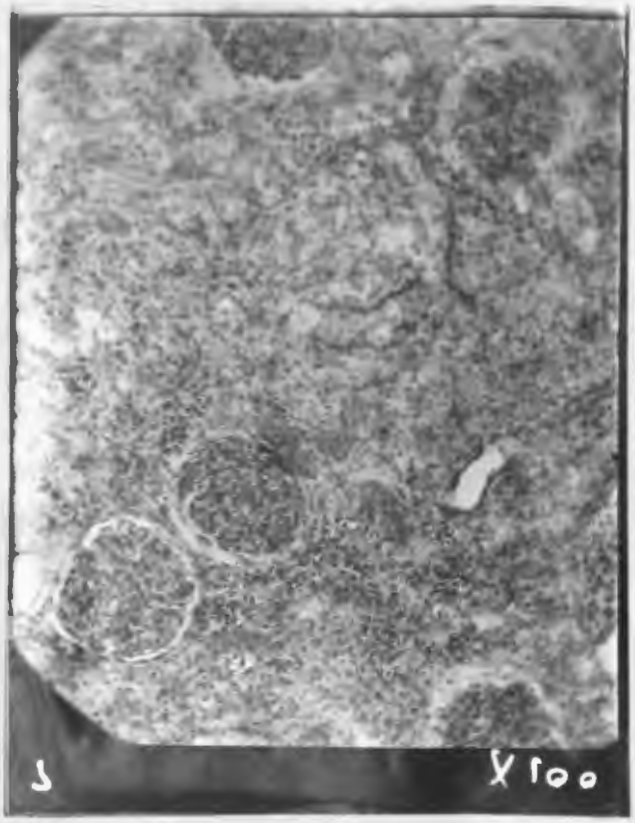


Marked dilatation of renal tubules and degeneration of the renal parenchyma.

Experiment No.7
B 720

Ligation of the ureter- twenty-eight days duration. Hydronephrosis. Marked destruction of the renal parenchyma in the lateral portions, considerable tissue remaining in the median sagittal portion.

Experiment No. 5 B 655 — Ligation of the ureter- forty-seven days duration. Large hydronephrotic sac with marked dilatation of the capsular vessels.

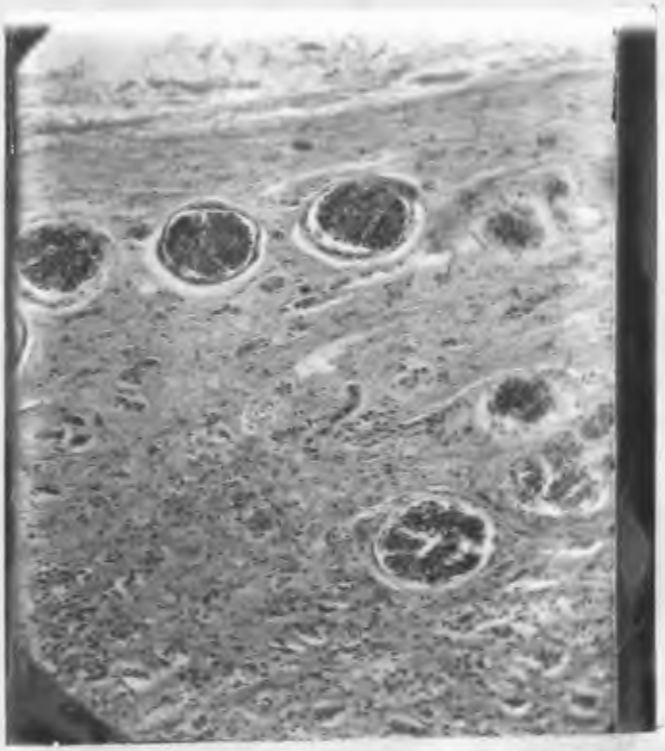


Marked degeneration of the renal parenchyma.

Experiment No. 4
B 901

Ligation of the ureter- 102 days duration. Complete destruction of the renal parenchyma and large monolocular hydronephrotic sac.

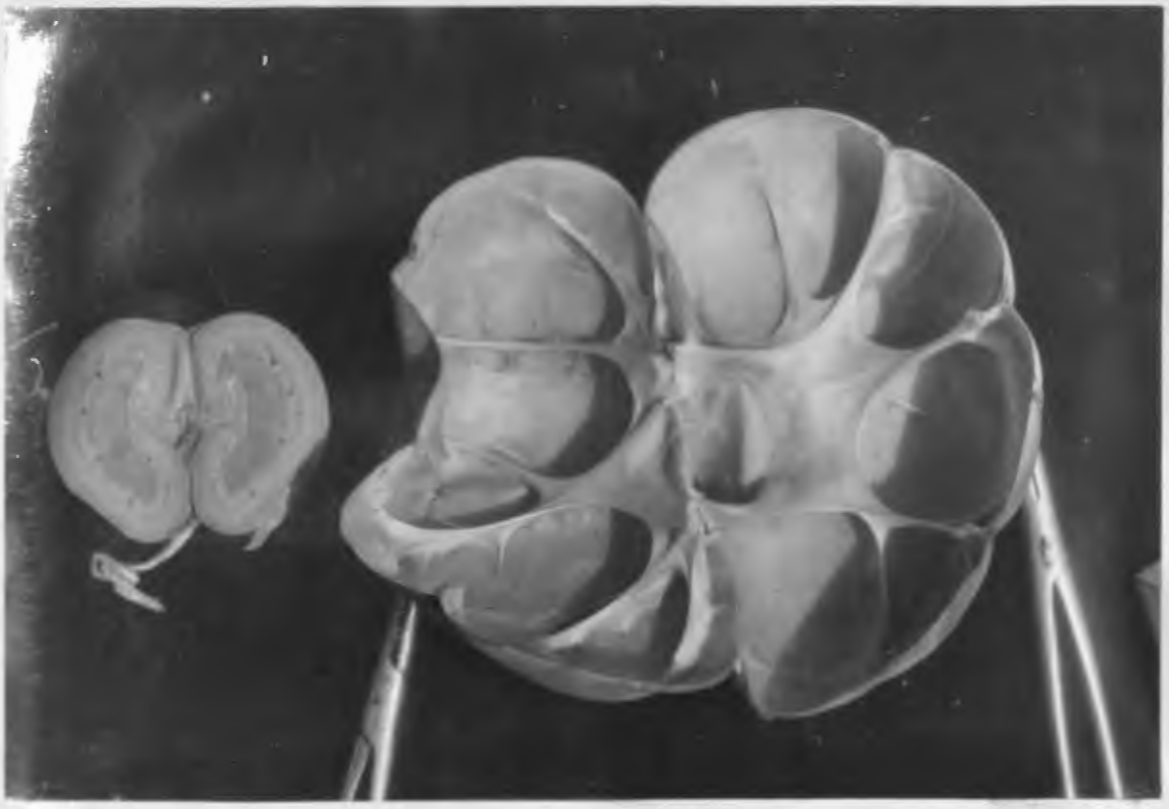
Experiment No. 3 Ligation of the ureter- 153 days duration.
B 732 Complete destruction of the renal parenchyma, connective tissue bands radiating from the pelvis.



Marked atrophy of the renal parenchyma. Glomeruli fairly well preserved.

Experiment No.1
B676

Ligation of the ureter- 447 days duration. Enormous lobulated hydronephrotic sac. Complete destruction of the renal parenchyma- loculated surface due to connective tissue septa.



Experiment No. 1 B676 — Ligation of the ureter- 447 days duration. Enormous lobulated hydronephrotic sac. Complete destruction of the renal parenchyma- loculated surface due to connective tissue septa.

From the results of the experiments upon this group of dogs it is conclusive,

1, That sudden complete occlusion of the ureter produces a hydronephrosis in every instance, the degree depending upon the duration of the obstruction.

2, That the hydronephrotic cavity is developed by dilatation of the pelvis and atrophy of the renal parenchyma and the walls of the kidney.

3, That the atrophic changes are quite uniform in distribution but vary in different animals.

4, That the kidney is capable of secreting after thirty-six days complete ureteral obstruction.

5, That the renal blood vessels are the last structures to be affected by the increased intra-renal pressure.

6, That in every instance, there is dilatation of the capsular veins, the enlargement depending upon the amount of hydronephrosis present.

Group II.

Ligation of the Collateral Venous Circulation and the Ureter

The consistency with which a dilatation of the capsular veins was found accompanying hydronephrosis led to the experiments in Group II, of this series.

The collateral veins are ordinarily too small to be noticed in the normal kidney. The dilatation of these veins during the development of hydronephrosis and the observation that their enlargement seemed to be dependent upon the amount of renal distention brought up the question of whether they were one of the causative factors in the production of the hydronephrosis or the result of the hydronephrosis. In order to determine the point, the capsular blood-supply was ligated at the same time as the ureter. As cited by Barney, Tuffier and Lejars, working upon the collateral circulation ligated the renal vein and found that the renal venous blood was returned to the general circulation by four routes, 1, the inferior diaphragmatic and suprarenal vessels, 2, ureteric and spermatic, 3, the subcutaneous plexus of the lumbar region, 4, the plexus which surrounds the last intercostal nerve, the ileoinguinal nerve and the ileohypogastric nerve.

The technic of the operative procedure in this group differs from that in the former in that after the collateral circulation was carefully dissected out and the ureter freed, both were doubly ligated and sectioned so that the only blood entering or leaving the kidney was that which passed through the renal artery and vein. A definite hydronephrosis was produced in each instance and its progress was essentially the same as that in the former group except that it developed more slowly and to a less degree than when the collateral circulation was left intact. Frequently the

kidney and its hydronephrotic sac were found to be smaller than the opposite normal kidney in spite of the distinct hydronephrotic cavity produced by the progressive atrophy of the renal parenchyma.

The following chart of thirty-one experiments shows the results obtained:

GROUP II - DOUBLE LIGATION AND SECTION OF ONE URETER AND COLLATERAL BLOOD SUPPLY

Exper. Number	Animal Number	Series Number	Date of Operation	Duration of Experiment in Days	Ureter Ligated Right or Left R L	Double Ligation & Section of Ureter	Ligation of Collateral Blood Supply	Autopsy Number	Pyonephrosis	Hydronephrosis	Small	Moderate	Large	Dilatation of Capsular Veins	Adhesions Around Operated Kidney	Condition of Unoperated Kidney
581	C697	1	1918 8-12	453	L	+	+	530		+			+	0	0	norm.
636	C730	2	1918 8-26	422	L	+	+	493		+		+		0	0	norm.
35	B724	3	1917 1-13	337	L	+	+	658		+		+		0	+	hyper.
34	B723	4	1917 1-13	297	L	+	+	592	+				+		+	hyper.
430	C 6	5	1917 5-28	217	R	+	+	677		+		+		0	+	hyper.
582	C106	6	1917 8-28	200	L	+	+	105	+				+	0	+ omen.	hyper.
40	B729	7	1917 1-20	159	L	+	+	396		+			+	0	+	norm.
421	B997	8	1917 5-25	150	R	+	+	555		+			+	0	0	norm.
32	B721	9	1917 1-13	149	L	+	+	363		+			+	0	0 omen.	norm.
637	C731	10	1918 8-26	115	L	+	+	702		+			+	0	+	hyper.
435	C 11	11	1917 6-1	68	R	+	+	462	+				+		+	norm.
422	B996	12	1917 5-25	59	R	+	+	436		+		+		0	0	norm.
410	B990	13	1917 5-14	35	R	+	+	380		+		+		0	0	norm.
580	C104	14	1917 8-20	34	L	+	+	527	+ rupt			+			+ omen.	norm.
563	C 92	15	1917 8-10	32	L	+	+	518	+				+		+ omen.	norm.
522	C 70	16	1917 6-27	27	L	+	+	475		+		+		0	+	norm.
33	B722	17	1917 1-3	24	L	+	+	52		+			+	0	0	hyper.
483	C608	18	1918 7-15	23	L	+	+	428		+		+		0	0	norm.
20	B709	19	1917 1-9	23	L	+	+	37		‡			+	0	+	norm.
525	C 72	20	1917 7-27	22	L	+	+	490		+			+	0	0	norm.
420	B996	21	1917 5-25	19	R	+	+	368		+				0	+	norm.
41	B730	22	1917 1-20	17	L	+	+	51		+		+		0	0	norm.
502	C627	23	1918 7-22	17	L	+	+	434		+	+			0	0	norm.

GROUP II - DOUBLE LIGATION AND SECTION OF ONE URETER AND COLLATERAL BLOOD SUPPLY. (Continued)

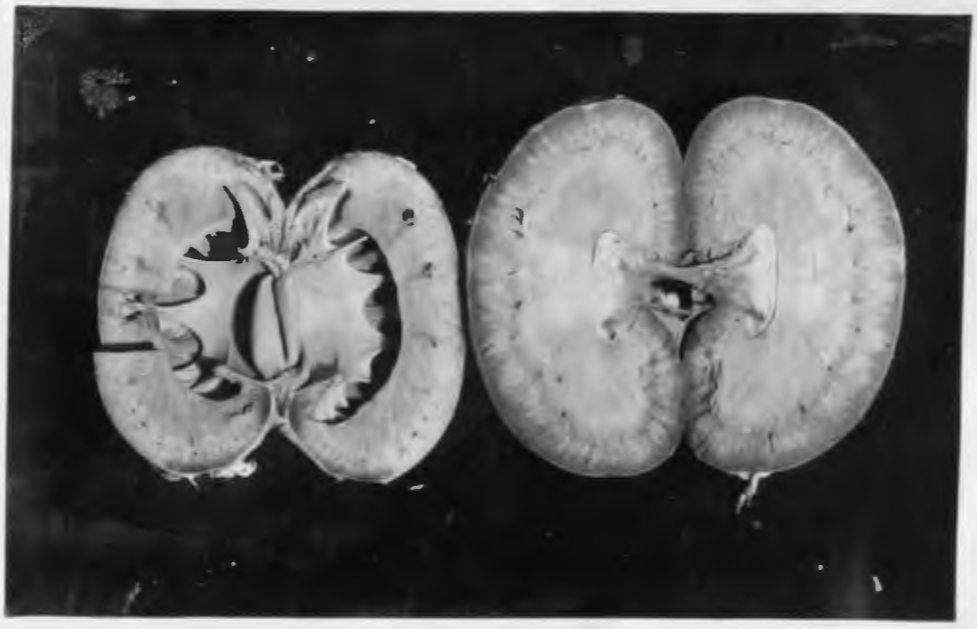
Exper. Number	Animal Number	Series Number	Date of Operation	Duration of Experiment in Days	Ureter Ligated Right and Left R L	Double Ligation and Section of Ureter	Ligation of Collateral Blood Supply	Autopsy Number	Pyonephrosis	Hydronephrosis	Small	Moderate	Large	Dilatation of Capsular Veins	Adhesions Around Operated Kidney	Condition of Unoperated Kidney
19	B708	24	1917 1-9	16	L	+	+	40		+	+			0	+	norm.
411	B991	25	1917 5-14	10	R	+	+	336		+	+			0	+	norm.
545	C666	26	1918 8-5	10	R	+	+	457		+	+			0	0	norm.
545	C 84	27	1917 8-3	9	L	+	+	468		+	+			0	0	norm.
522	C646	28	1917 7-29	8	L	+	+	419		+	+			0	0 omen.	norm.
580	C696	29	1918 8-12	5	R	+	+	463		+	+			0	+	norm.
722	C161	30	1917 10-26	5	R	+	+	582		+	+			0	0	norm.
503	C628	31	1918 7-22	4	L	+	+	393		+	+			0	0	norm.

Experiment No.29
C 696

Ligation of the ureter and collateral blood supply- five days duration. Small hydronephrosis. Flattening of the renal papillae and thinning of the medulla.

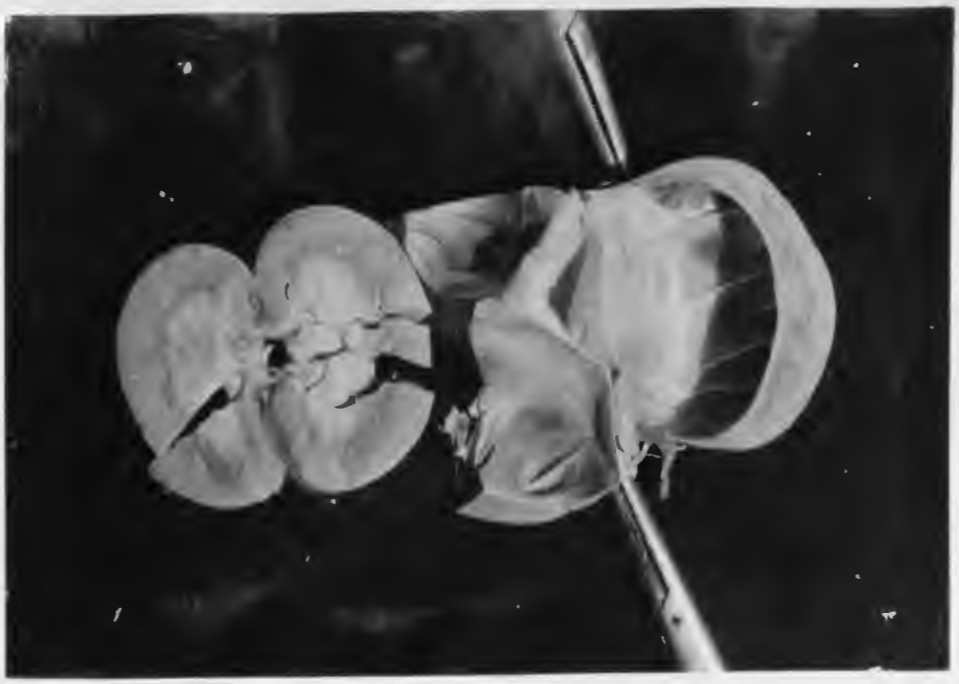
Experiment No.25 B 991 Ureter and collateral blood supply ligated- ten days duration. Small hydronephrosis. Destruction of renal parenchyma in lateral portions. Median sagittal portion still preserved. Opposite kidney normal.

Experiment No.23 Ureter and collateral blood supply ligated-
C 627 seventeen days duration. Small hydronephrosis.
Opposite kidney normal.



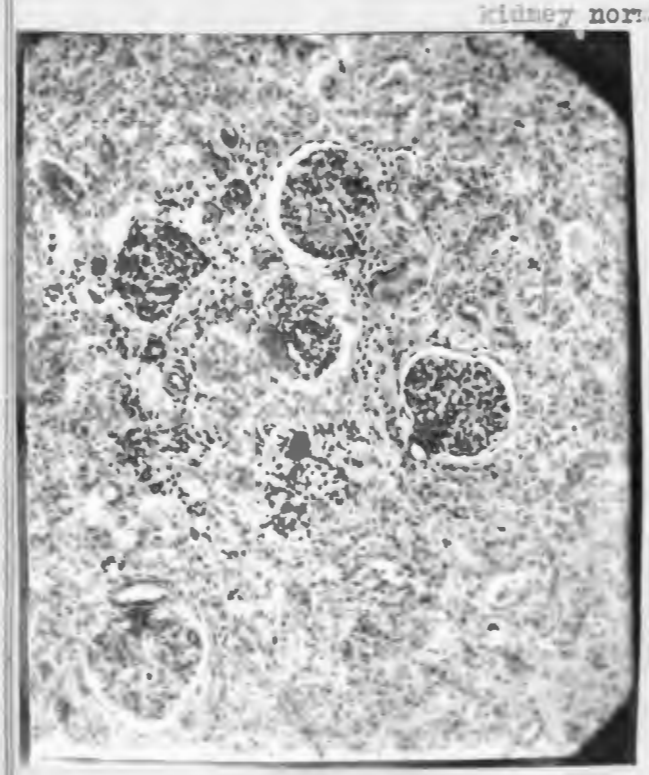
Experiment No. 18
B 606

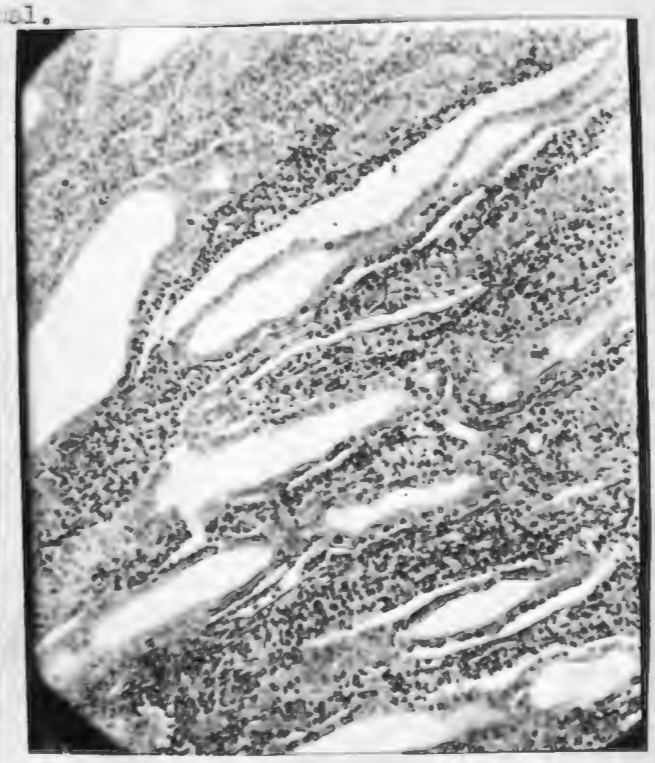
Ureter and collateral blood supply ligated-twenty-three days duration. Large hydronephrotic cavity with atrophy of the kidney. Opposite kidney normal.



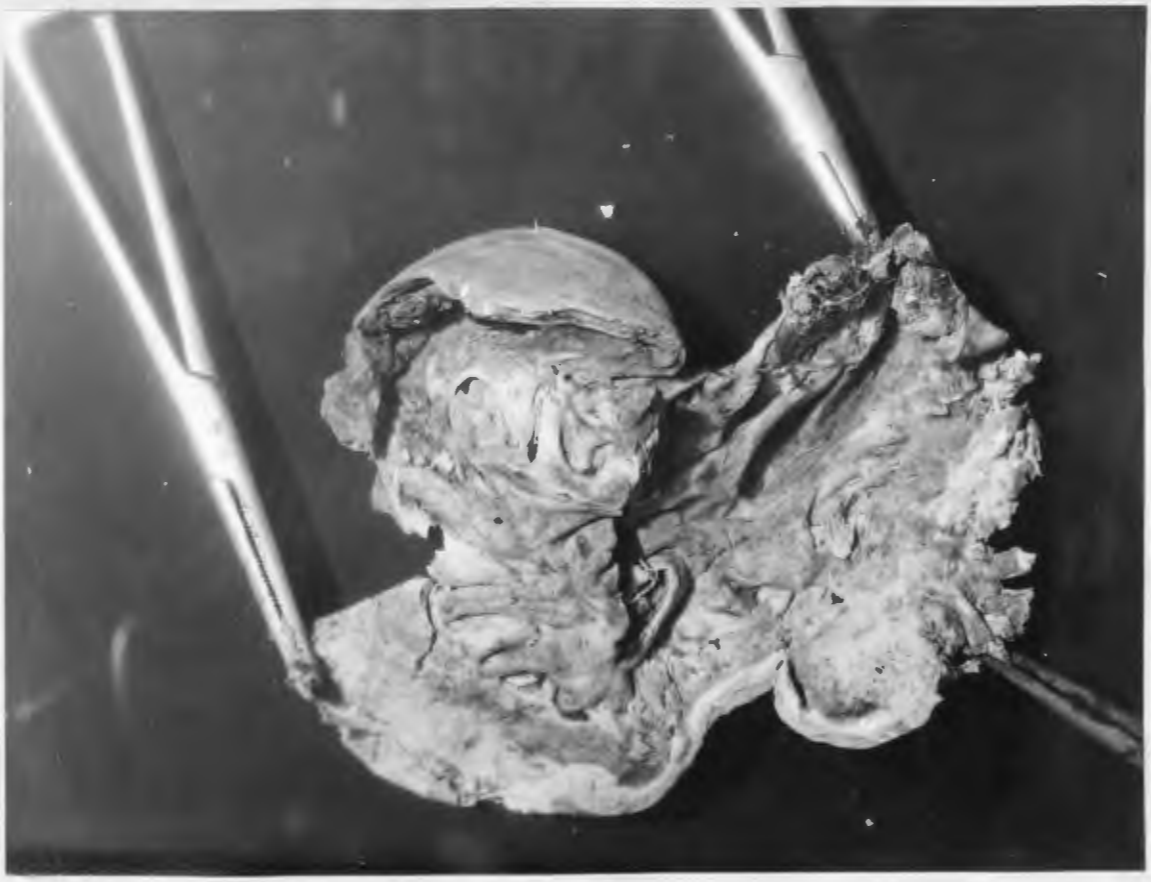
Experiment No.17
B 722

Ureter and collateral blood supply ligated-twenty-four days duration. Large hydronephrotic sac. Atrophy of the kidney, particularly in the lateral portions. Opposite kidney normal.



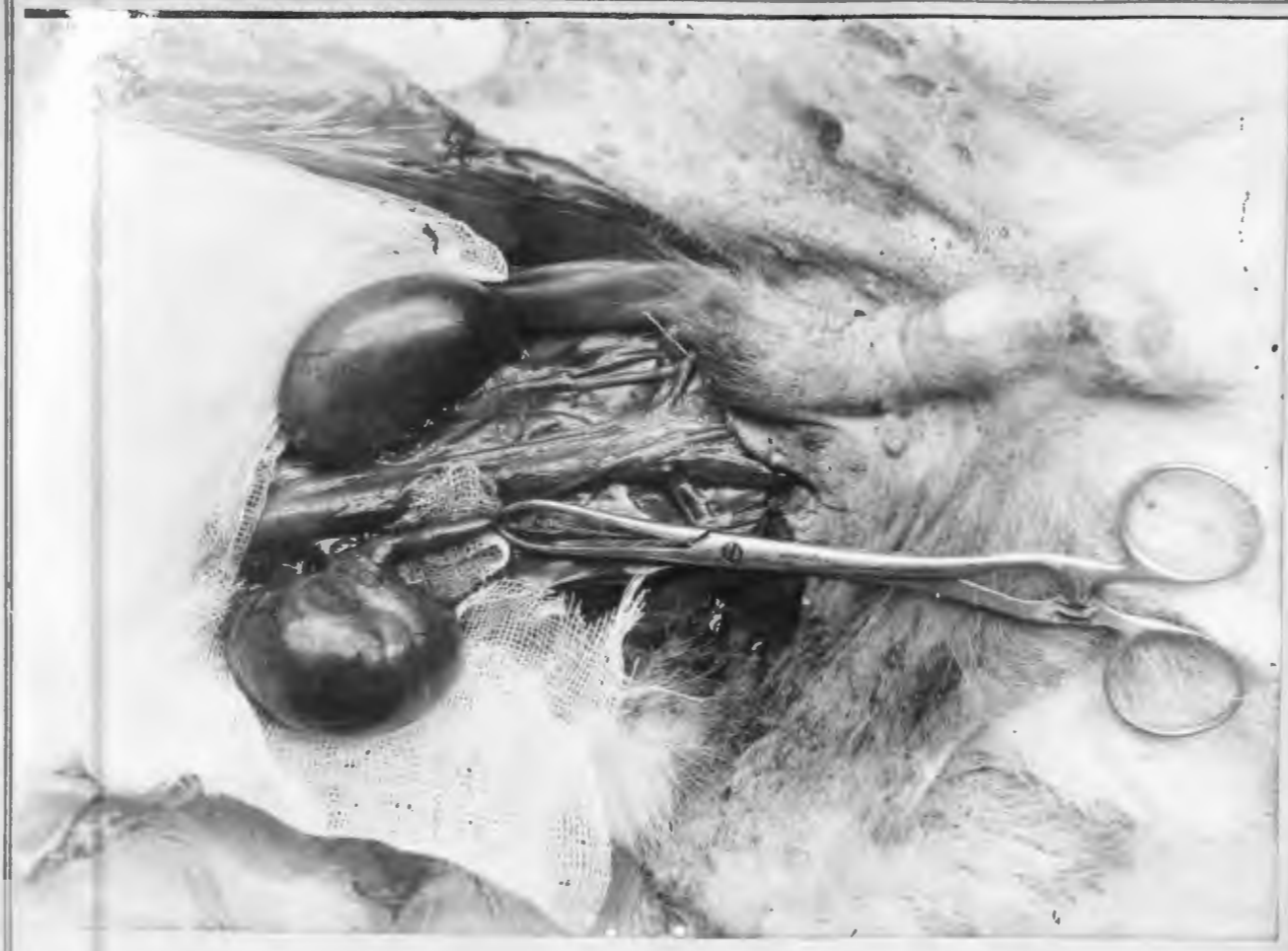


Degeneration of the convoluted tubule. Dilatation of the collecting tubules and glomeruli.



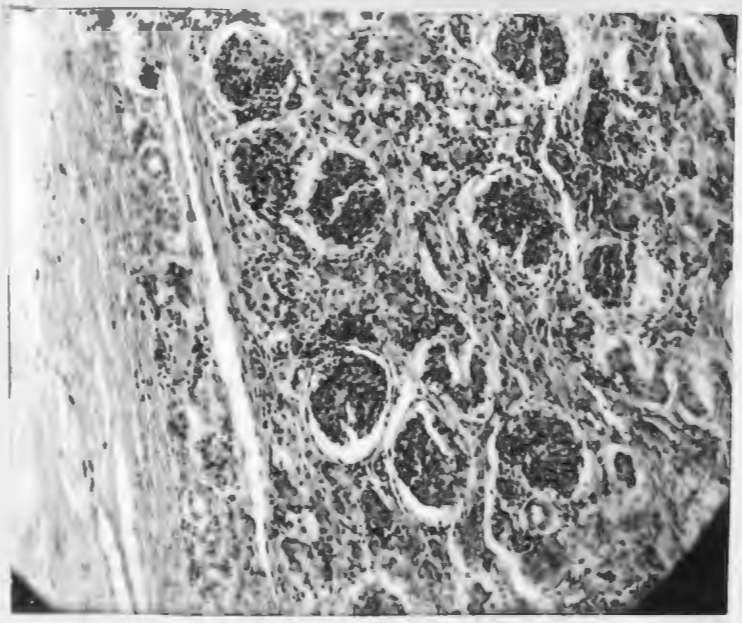
Experiment No. 15
C92

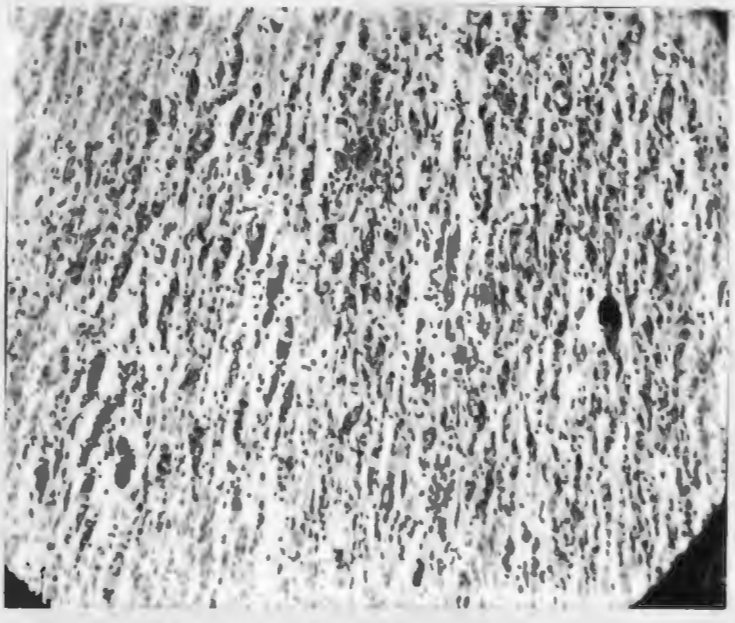
Ureter and collateral blood supply ligated-thirty-two days duration. Large pyonephrosis which ruptured causing peritonitis and death.



Experiment No. 13
B 990

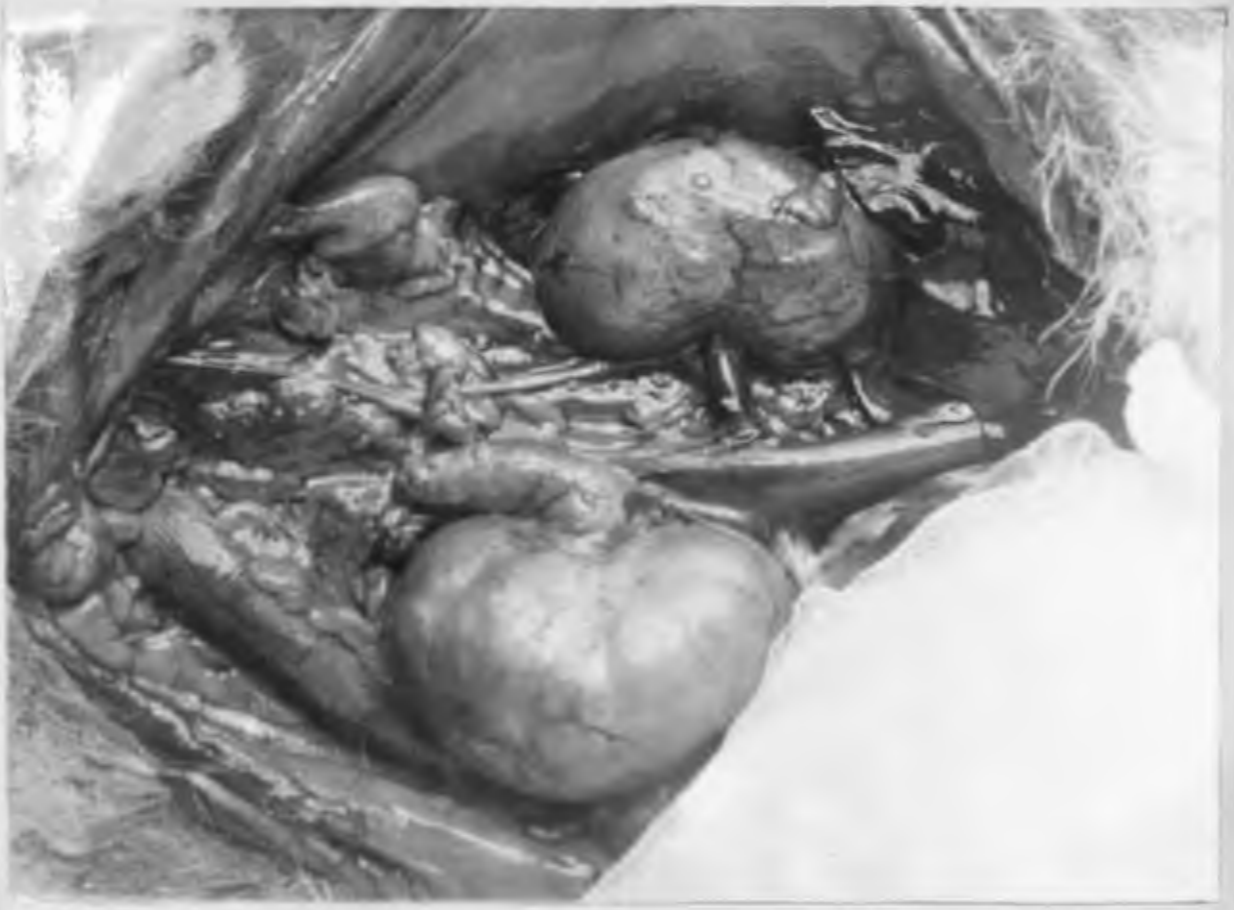
Ureter and collateral blood supply ligated-thirty-five days duration. No dilatation of capsular vessels. Large hydronephrotic cavity with atrophy of the kidney. Kidney and hydronephrotic sac smaller than opposite normal kidney.





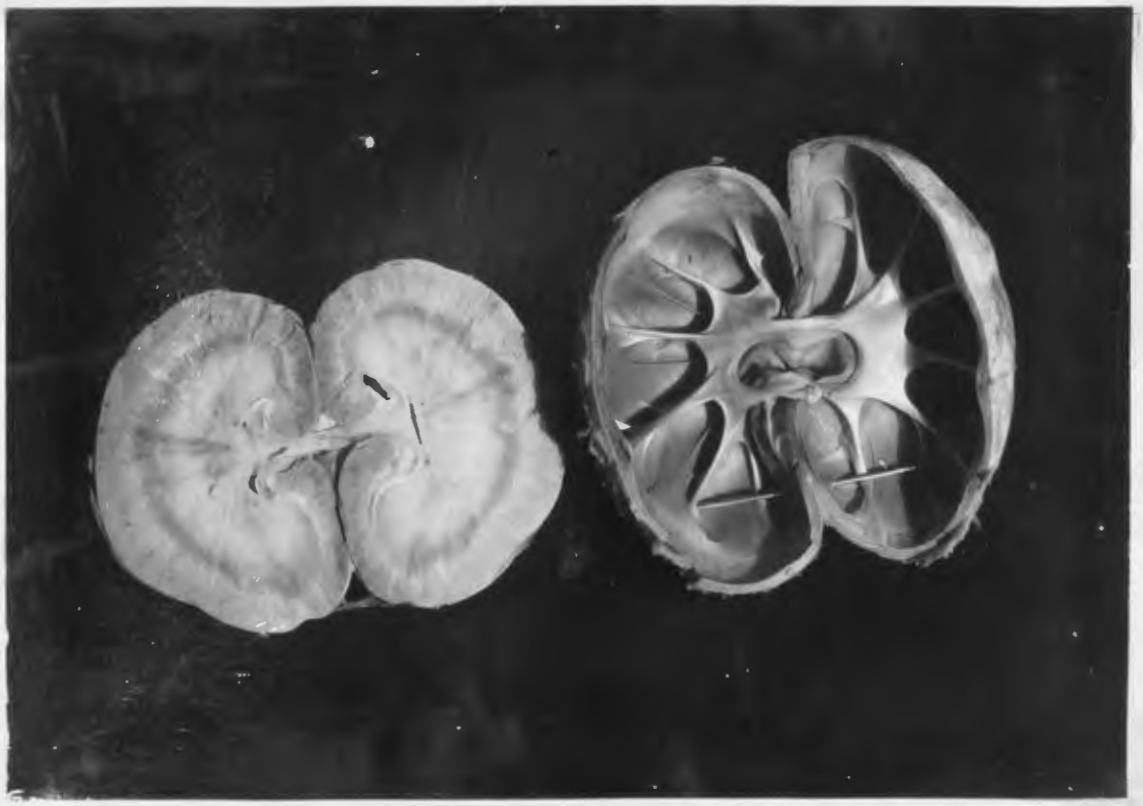
Experiment No. 13
B 990

Ligation of the ureter and collateral blood supply- thirty-five days duration. Marked degeneration of kidney tubules of both cortex and medulla. Glomeruli fairly well preserved in cortical portion proximal to the capsule.



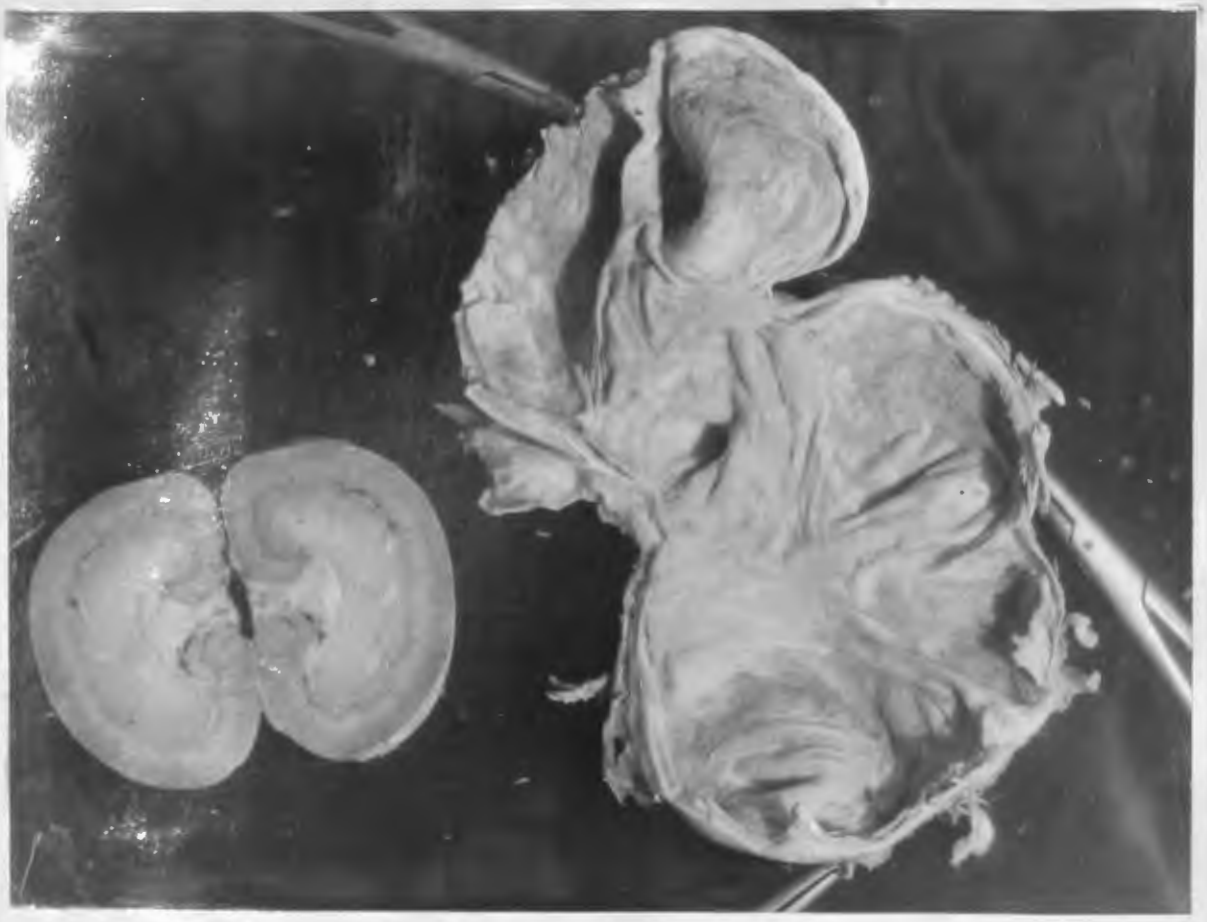
Experiment No. 9
B 721

Ureter and collateral blood supply ligated-
149 days duration. No dilatation of the capsular veins. Large hydronephrotic kidney with dilated tortuous ureter. Kidney lower than opposite normal kidney.



Experiment No. 8
B 997

Ureter and collateral blood supply ligated-150 days duration. Large hydronephrotic sac. Complete destruction of renal parenchyma with persistence of connective tissue bands between the lobules. Opposite kidney normal.

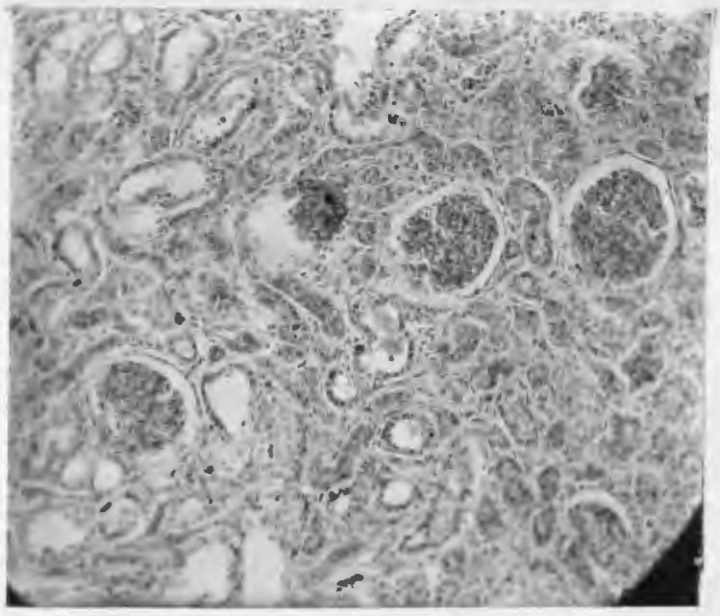


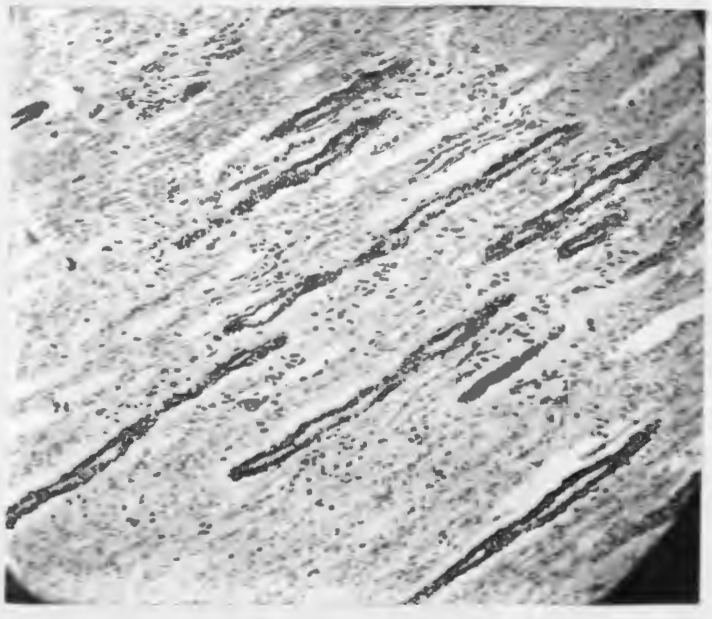
Experiment No. 4
B 723

Ureter and collateral blood supply ligated-
297 days duration. Large pyonephrosis.
Opposite kidney normal.

Experiment No. 2
C730

Ureter and collateral blood supply ligated-422 days duration. Large hydronephrotic sac with almost complete destruction of the renal parenchyma except in the median saggital portion. Opposite kidney normal.





Experiment No. 2
C730

Ligation of ureter and collateral blood supply-422 days duration. Marked degeneration of the renal parenchyma. Glomeruli fairly well preserved.

In Experiments No. 1 and 4, 453 and 297 days obstruction respectively, the hydronephrotic sacs were the largest in the group but not as large as those in the preceding group in which the ureter alone was ligated. In Experiment No. 4 (297 days obstruction), the animal developed the largest hydronephrotic sac of the series. The kidney weighed 540 grams, and the sac contained 250 c.c. of purulent fluid. There were many adhesions about this pyonephrotic kidney. In Experiment No. 1, of 453 days ureteral obstruction, the kidney weighed 382 grams, and the sac contained 190 c.c. of clear fluid. There was no dilatation of the capsular vessels.

In Experiments No. 2 and 3, of 422 and 337 days duration respectively, a moderate degree of hydronephrosis was developed, the sacs containing 40 c.c. and 30 c.c. of fluid respectively. No dilatation of the capsular vessels could be detected. In Experiment No. 2, the hydronephrotic kidney was smaller than the opposite normal kidney; as shown in the photograph, there was complete destruction of the kidney parenchyma.

Experiment No. 15 was of thirty-two days duration. The animal appeared in good health at night but was found dead on the following morning. At autopsy there was a large pyonephrosis which had ruptured and produced a peritonitis, the cause of death. Many adhesions were found about the pyonephrotic sac.

The animals in Experiments No. 4, 6, 11, 14, and 15 also developed pyonephrosis in the operated kidney, which makes its incidence much higher in this group than the preceding in which the collateral circulation was not interfered with.

In many of the animalsof this group, there were adhesions of the omentum and of the adjacent structures to the operated kidney, but not

sufficient to have any bearing upon tbe blood supply of the kidney.

It seems quite conclusive from these experiments that;

1. Hydronephrosis will develop in the kidney of a dog following the ligation of one ureter and the collateral blood supply.

2. The hydronephrotic sac will be smaller and develop more slowly when the collateral circulation is intact.

3. Pyonephrosis is more common when the collateral circulation has been ligated.

4. Progressive atrophic changes occur in the kidney in the same sequence as then the ureter alone is ligated.

5. Ligation of the ureter does not affect the general health of the dog, as ten animals lived ower 100 days and two over 400 days before being sacrificed.

In summarizing the observations made on the two groups of Series I, it is important to note that after obstructing the outflow of urine by ligating the ureter, the normal secretion of urine continues until the ureter and pelvis of the kidney are completely filled; after that, urinary secretion takes place under the abnormal condition of back-pressure from the damming up of urine into the pelvis of the kidney, which immediately dilates because its walls are elastic and not capable of resisting this increased intra-pelvic pressure. The secreting power of the kidney is greatest at the time of ligation of the ureter, gradually diminishing in direct proportion to the duration of the obstruction, as the increased intrapelvic pressure causes a compression of the renal parenchyma by flattening of the pyramids and thinning of the medulla and cortex and a damming back of urine in the tubules, which exerts pressure directly upon the cells lining the tubules and results in decreased nutrition and atrophy of the renal parenchyma. The increased intra-pelvic and intra-renal pressures cause a compression of the small venous capillaries where only low pressure obtains, thus partially obstructing the venous return. This produces a congestion and edema of the kidney but does not completely obstruct the venous return, and blood continues to pass from the kidney through the renal vein although the amount is greatly diminished, and the glomerulus is the last kidney structure to be destroyed. The partial obstruction of the venous return affects the renal parenchyma and secretory power of the kidney in two ways: It decreases the amount of blood passing through the kidney and consequently the amount of secretion possible. Likewise the amount of nutrition carried to the cells of the parenchyma must be diminished. Partial obstruction of the venous return is relieved in two ways, first, by dilatation of the capsular veins, and second, by absorption from the medullary portion of the kidney, which is in inverse proportion to the destruction of the renal parenchyma. Dilatation of the capsular veins is caused by increased intra-

renal pressure and is secondary to the production of hydronephrosis as a hydronephrotic sac develops when the collateral circulation is ligated. The partial compensation of the venous return by the dilatation of the capsular vessels permits a greater secretory activity of the kidney because of the increased blood supply and nutrition and results in a larger and more rapidly developing hydronephrosis, which in turn causes more speedy destruction of the parenchyma than is found when the collaterals are ligated.

I conclude that the production of hydronephrosis is dependent upon the amount of resistance afforded by the pelvis to the increased intra-pelvic pressure incident to the secretory activity, which is variable, but greatest at the time of obstruction, and gradually diminishes so that in four weeks no secretion is demonstrable. It is possible that this may be the explanation of the difference in the results obtained in experimental investigations in animals and the clinical observations noted in the human, i. e., in the human the collateral venous circulation is practically negligible and the kidney pelvis and capsule are more resistant. It would seem that if it were possible to enclose the kidney pelvis and ureter in a plaster Paris jacket and completely obstruct the outflow of urine, the increased intra-pelvic pressure would become so great that it would stop all secretory activity and result in atrophy of the kidney.

Literature on the Blood Supply of the Kidney

Brodel studied the vascular system of the human kidney by colloidal injections and by Schieferdecker's corrosion method. He describes in detail and illustrates the intrinsic blood vessels of the kidney, also the course of the larger blood vessels, their relation to the pelvis of the kidney and to surgical operations.

Carrel in association with Janeway ligated branches of the renal artery to obtain insufficiency of renal function and studied its effect upon the blood pressure.

Bradford states that ligation of the renal artery is followed by a gradual shrinkage of the kidney with necrosis of the epithelium and without any noticeable overgrowth of fibrous tissue.

MacNider in a series of experiments upon twenty-two cats, ligated the posterior branch of the renal artery for periods ranging from two hours to ninety days and observed the following sequence of events: 1. Following the ligation of the posterior branch of the renal artery there is an ischemia and resulting atrophy of the portions supplied by the branch. 2. A more or less imperfect collateral circulation develops in this zone, which first appears in the medulla and later invades the cortex. 3. With the development of the anastomosis, there is at first an ingrowth of connective tissue cells into the necrotic zone followed by an ingrowth of renal epithelium. 4. Some of the glomeruli are regenerated by an ingrowth of capillary buds which later become canalized and contain blood. 5. A secondary fibrosis is inaugurated and with its development there is an atrophy of the renal tubule, fibrosis of the glomeruli, and obliterative changes in the vessels. 6. The resulting picture is comparable to a chronic interstitial nephritis.

Gross studied the blood vessels of normal and pathological kidneys by means of colored injections, frozen sections and skiagraphs. In nephritis he found that the blood vessels were well injected by the colored material while in the normal kidney the medullary vessels were not injected. Further studies were made after placing normal kidneys in formalin for a short time to constrict the cortical tissue before injecting. The result was that only those portions of the columns of Bertini were injected which had not been thoroughly fixed; the pyramids were not injected. He then concluded that the reason for the injection of the pyramids in the nephritic kidney was partly the obliteration and loss of cortical vessels, partly a different anatomical course and arrangement of the blood vessels in the medulla. In another series of experiments, skiagraphs were taken of normal and pathologic kidneys after injecting the blood vessels with a barium suspension in gelatin. In normal kidneys he observed that the renal arterial architecture resolves itself into a simple tree-like dichotomous arrangement of the branches of the principal afferent artery; that there is no anastomosis of the arcuate arterial branches, but that the vessels running between the cortex and the medulla break up rather abruptly into small branches which give rise to the high pressure in the latter.

Series No. 2.

The Effect of Occlusion of the Vascular Radicals of the Kidney.

This study of the ligation of the various vascular radicals of the kidney was undertaken with a view of determining the ultimate effect upon the renal parenchyma and whether a collateral venous circulation would be established. It was shown by Bowman in 1842 that the arterial radicals of the kidney were end arteries, but Hürl in 1882, was first to point out that there was no anastomosis between the branches of the renal arteries. These facts are often lost sight of by surgeons in doing various surgical procedures upon the kidney, as mentioned by Brödel in 1901.

Series No. 2 comprises four groups. The experiments in the first two groups pertain to the arterial blood supply, and those in the third and fourth, to the venous supply. The operative procedures were essentially the same in the four groups except that different vessels were ligated.

Group I.

Effects Produced by Ligation of the Renal Artery.

My purpose in this study was to determine, first, the results produced upon the kidney, second, whether a collateral blood supply would be established, third, whether the omentum when wrapped about a decapsulated kidney would furnish sufficient blood to continue the function of the kidney.

After exposing the kidney the renal artery was carefully dissected from the perirenal fat and doubly ligated and sectioned. As shown in the accompanying chart, the renal artery alone was ligated in three dogs; in

two, the artery was ligated and the kidney decapsulated and wrapped in omentum.

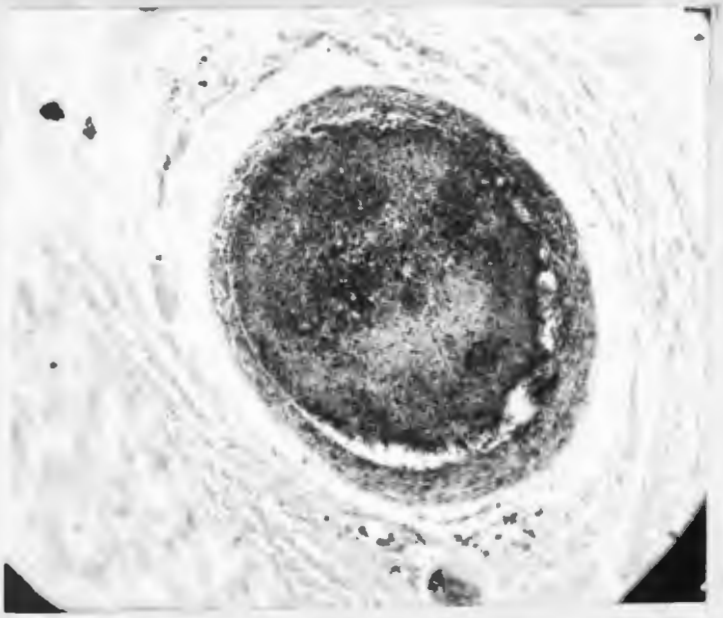
Series 2 - EFFECTS OF OCCLUSION OF THE VASCULAR RADICALS OF THE KIDNEY

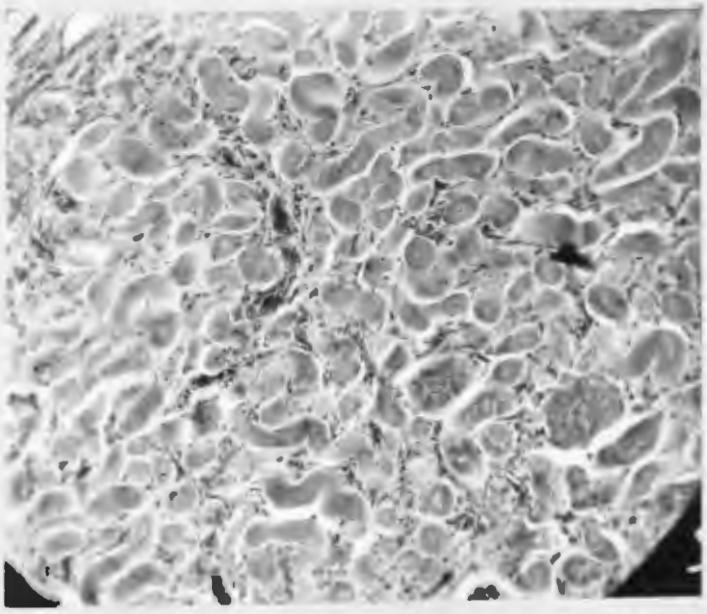
GROUP I - LIGATION OF RENAL ARTERY

Experiment Number	Animal Number	Series Number	Date of Operation	Duration of Experiment in Days.	Renal Artery Ligated Right or Left	Decapsulation of Kidney and Wrapped in Omentum	Autopsy Number	Kidney Tissue Pale and Degenerating	Collateral Arterial Circulation Formed	Atrophy of Operated Kidn	Weight of Operated Kidney	Adhesions Around Operated Kidney	Weight of Unoperated Kidney
190	B828	1	1917 3-5	31	L		214	+	0	+	10 gm.		40 gm.
99	B769	2	1917 2-10	16	L	+	90	+	0	+	18 gm.	oment.	27 gm.
53	B742	3	1917 1-27	10	L	+	53	+	0	+	14 gm.	oment.	26 gm.
242	B869	4	1917 3-16	8	L		173	+	0	+	30 gm.		45 gm.
490	C 47	5	1917 7-9	4	R		419	+	0	+	38 gm.		42 gm.

Experiment No. 2
B 769

Ligation left renal artery; kidney wrapped in omentum- sixteen days duration. Atrophy of the entire kidney, weighing ten grams. Not affected by wrapping kidney in omentum.





Experiment No. 2
B 769

Ligation of the renal artery; kidney wrapped in omentum- sixteen days duration. Amyloid degeneration of the kidney parenchyma of cortex and medulla. Thrombosis of kidney vessels.

Experiment No. 1
B 828

Ligation of the left renal artery- thirty-one days duration. Marked atrophy of left kidney, weighing ten grams. Right kidney normal, weighing forty grams.

Sudden complete occlusion of the renal artery by ligation resulted in a gradual symmetrical atrophy of the entire kidney as shown in the photographs. In Experiments No. 1, 4, and 5, no collateral arterial anastomosis was formed. In Experiments No. 2 and 3 (sixteen and ten days duration), the kidney was wrapped in the omentum, but this had no effect upon the degree of atrophy present in the kidney.

From these five experiments it is evident, 1, that the renal artery is the only source of blood supply to the kidney, furnishing both nutritional and functional blood, and is a strict end artery, 2, that a collateral arterial anastomosis is not formed after ligating the renal artery, 3, that wrapping the kidney in omentum after its decapsulation and ligation of the renal artery does not produce arterial anastomoses supplying the kidney parenchyma and therefore has no effect upon the development of atrophy.

Group II

Ligation of One or More Branches of the Renal Artery.

In the human it is frequently found necessary to ligate an aberrant vessel passing to the lower pole of the kidney, causing an intermittent obstruction to the outflow of urine and producing a hydronephrosis.

In sixteen animals after exposing the kidney the branches of the renal artery were carefully dissected free from perirenal fat and one or more doubly ligated and sectioned (three to 526 days duration) with the view of determining as accurately as possible what may be expected in the human if for any reason the same procedure is found necessary. In the dog there are usually four branches of the renal artery, two to the anterior and two to the posterior surface. The anterior vessels are the larger, supplying the anterior two-thirds of the kidney, the smaller posterior branches serving approximately the posterior third. They usually divide just inside the hilum into two smaller branches, one to each pole. For convenience of description these vessels have been termed anterior-superior and inferior, and, posterior superior and inferior, the areas of kidney to which they pass being designated quadrants named according to the vessel. This division on the basis of the distribution of the arterial branches is not absolutely accurate as the posterior branches supply less kidney tissue than the anterior vessels, and there is also a variation in the size of the superior and inferior branches, the size of the vessel being in accordance with the area supplied.

Immediately upon ligating a branch of the renal artery there is marked congestion of the portion of kidney supplied by that vessel, pro-

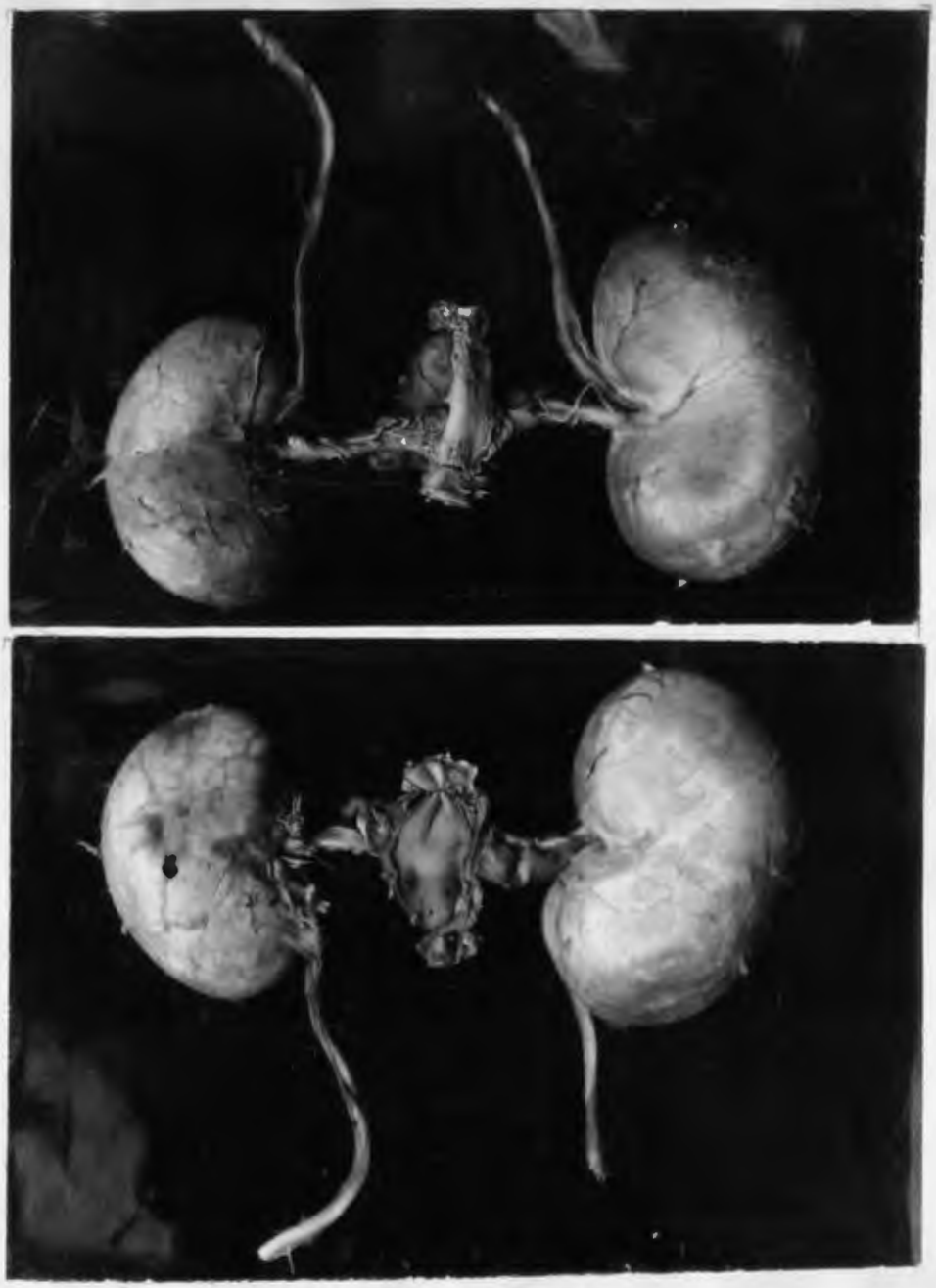
ducing a bluish discoloration of the cortex which has a sharp line of demarcation. A linen thread was sutured accurately around the margin of this area so that it would be possible to determine whether the circulation in any part of the area was later restored and the ultimate fate of this portion of the renal parenchyma. The accompanying photographs indicate the results obtained.

GROUP II - LIGATION OF ONE OR MORE BRANCHES OF THE RENAL ARTERY

Experiment Number	Animal Number	Series Number	Date of Operation	Renal Artery Right or Left R L	Branches of Renal Artery Ligated: Anterior Superior	Anterior Inferior	Posterior Superior	Posterior Inferior	Duration of Experiment in Days	Decapsulation of Kidney and Wrapped in Omentum.	Autopsy Number	Atrophy of Kidney: Anterior Superior Quadrant	Anterior Inferior Quadrant	Posterior Superior Quadrant	Posterior Inferior Quadrant
			1918												
273	C444	1	4-29	R	+			+	526	+	464	+			+
			1918												
442	C569	2	7-1	R				+	464		465				+
			1918	R			+	+						+	+
398	C529	3	6-17	L	++		+		348	+	261	+		+	
			1918												
265	C438	4	4-22	R			+	+	325	+	93			+	+
			1918			+							+		
331	C477	5	5-20	R		+	+	+	43		326			+	+
			1918	R	+		+							+	
364	C505	6	6-3	L		+	+	+	38	+	347		+	+	+
			1918	R	+	+		+				+	+	+	+
274	C445	7	4-29	L			+		23		261			+	
			1918				+	+						+	++
235	C412	8	4-4	R					15	+	220				
			1918	R	+	+						+	+		
291	C456	9	5-6	L	+	+			14		258	+	+		
			1918					+							+
236	C413	10	4-8	R					11		218				
			1918		+	+						+	+		
257	C430	11	4-15	R					10		230				
			1918												
329	C476	12	5-20	L	+	+			7		271	+	+		
			1918				+	+						+	+
256	C429	13	4-15	R					4		219				
			1918		+	+						+	+		
225	C402	14	4-4	R					4		180				
			1918				+	+						+	+
224	C401	15	4-4	R					4		179				
			1918	R			+	+						+	+
363	C504	16	6-3	L			+		3		283			+	

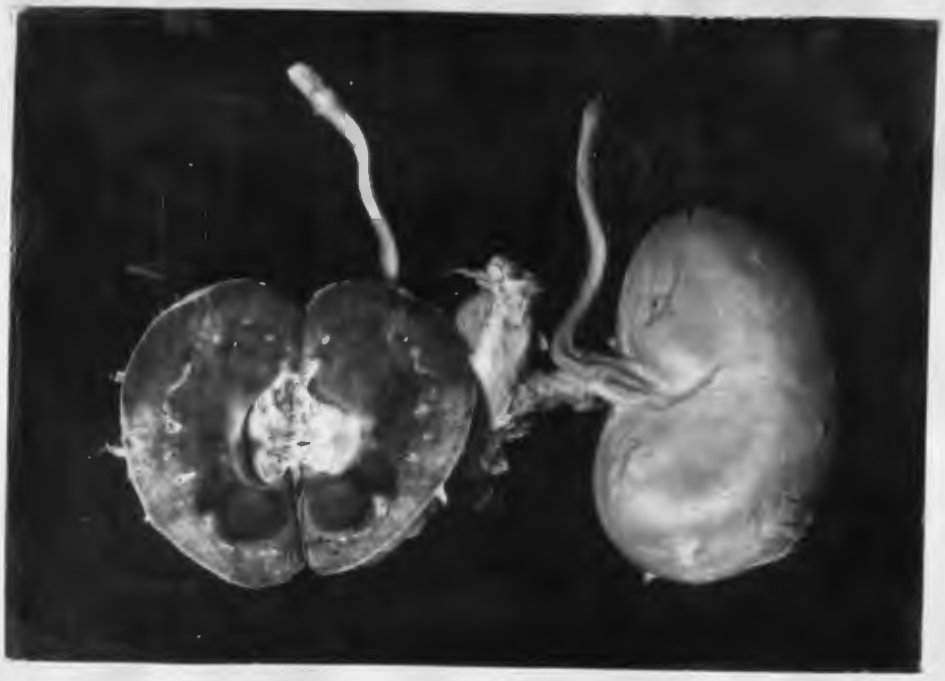
Experiment No. 16
C 504

Ligation of post. sup. branch of left and post. sup. and inferior branches of right renal artery- three days duration, showing degeneration of the posterior poles and areas supplied by these branches.



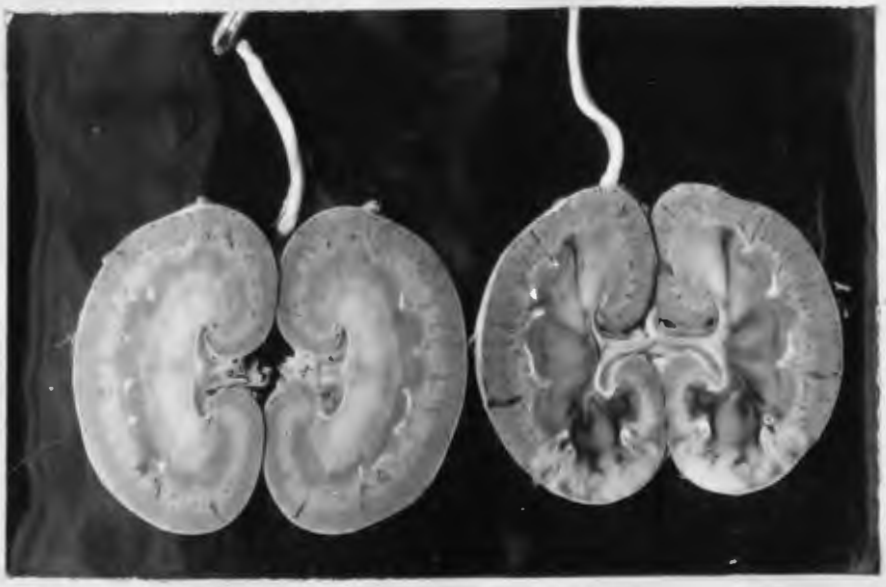
Experiment No. 11
C 430

Ligation of the anterior, sup. and inf. branches right renal artery- ten days duration. Atrophy of the anterior pole right kidney. Left kidney normal.



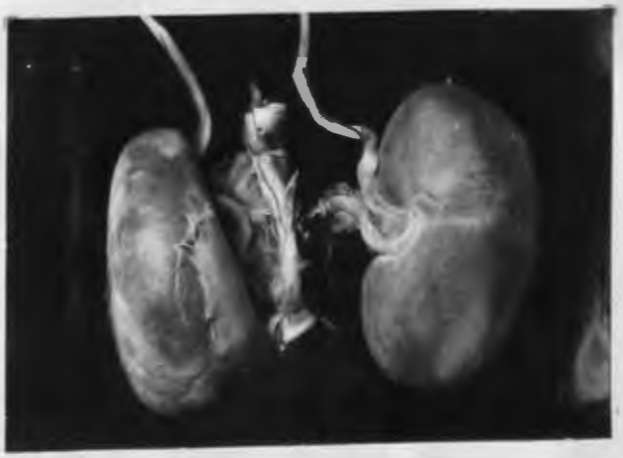
Experiment No. 11
C 430

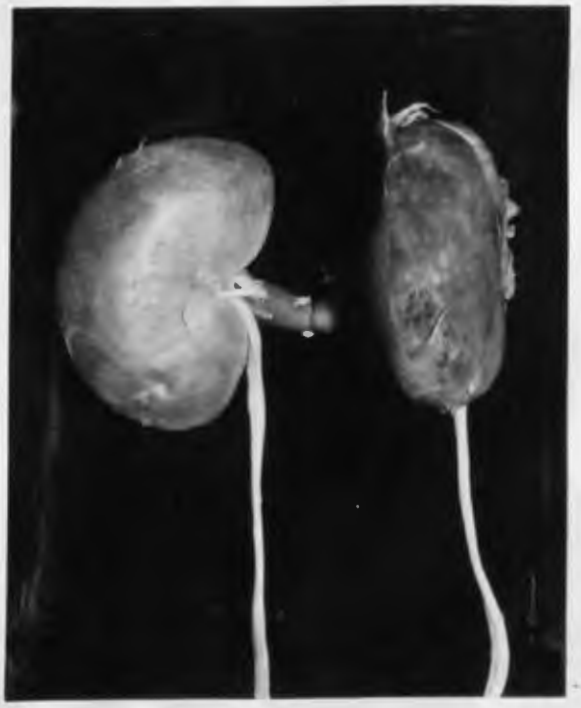
Ligation of the ant. sup. and inf. branches of the right renal artery- ten days duration. Atrophy of the anterior pole of the right kidney. Left kidney normal.



Experiment No. 10
C413

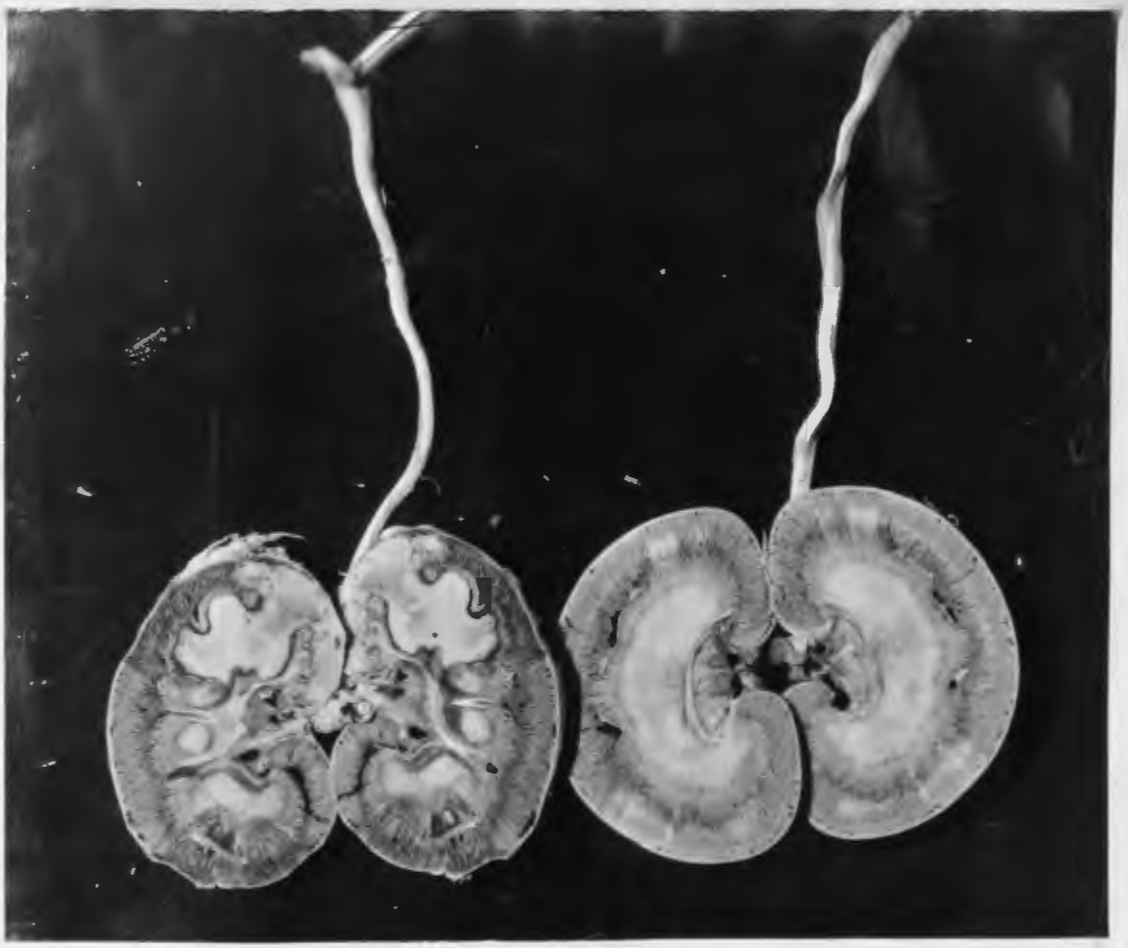
Ligation of the post. inf. branch of the right renal artery- eleven days duration. Atrophy of the renal tissue supplied by this branch. Left kidney normal.





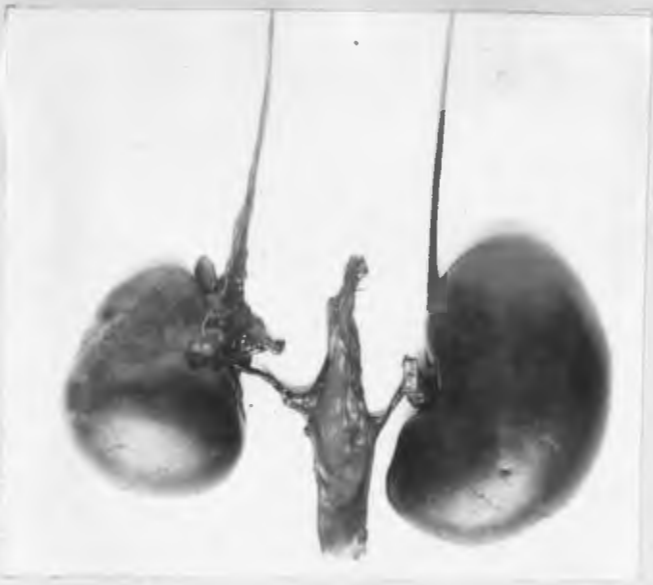
Experiment No. 8
C 412

Ligation of the post. sup. and inf. branches of the right renal artery. Decapsulation and wrapped in omentum- fifteen days duration. Atrophy of the lower pole of the right kidney supplied by the posterior branch, unaffected by decapsulation and wrapping in omentum. New capsule formed right kidney.



Experiment No. 8
0412

Ligation of the post. sup. and inf. branches of the right renal artery. Decapsulation and wrapped in omentum- fifteen days duration. Atrophy of the lower pole of the right kidney supplied by the posterior branch, unaffected by decapsulation and wrapping in omentum. New capsule formed right kidney.

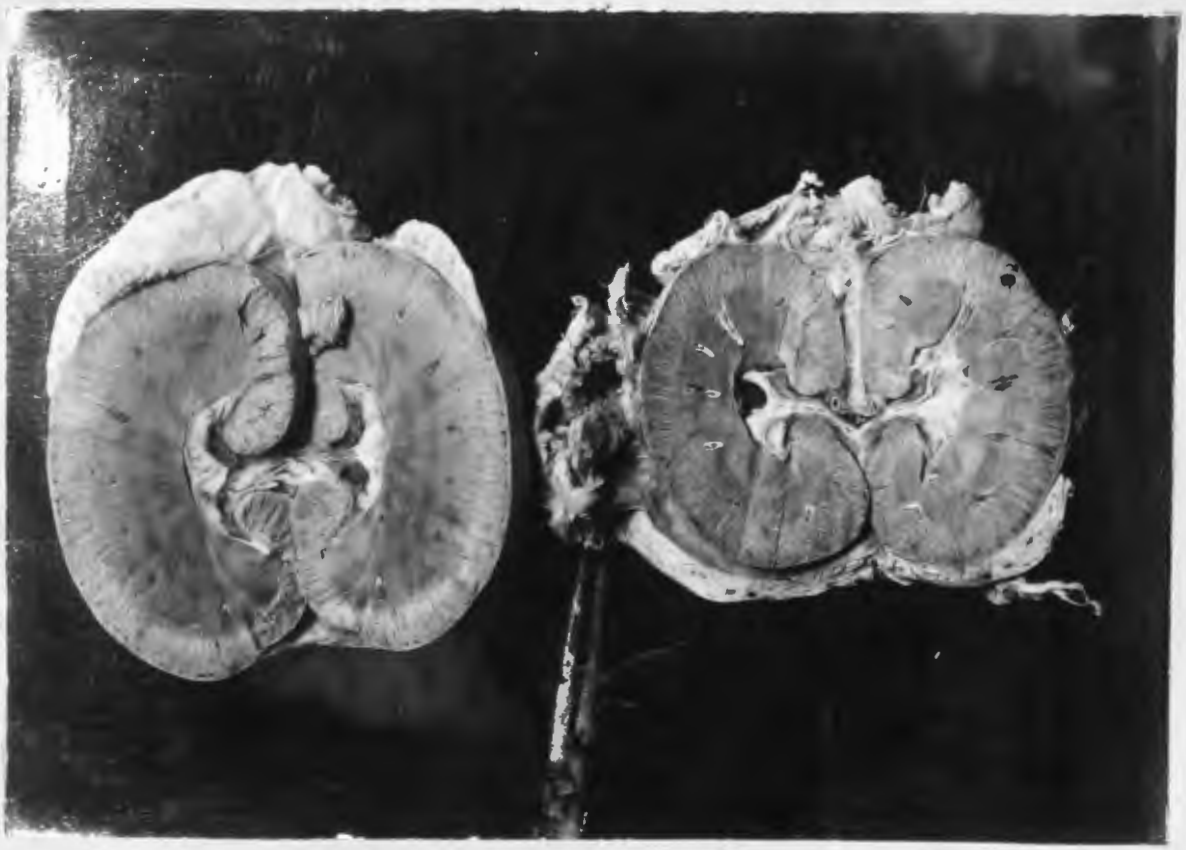


Experiment No. 6
C 505

Ligation of the ant. super. and post. super. branches of the right and ant. inf. and post. inf. branches of the left renal artery. Both kidneys decapsulated and wrapped in omentum. thirty-eight days duration. Atrophy of area supplied by these vessels, unaffected by wrapping kidney in omentum. New capsule formed both kidneys.

Experiment No. 6
C 505

Ligation of the ant. super. and post. super. branches of the right and ant. inf. and post. inf. branches of the left renal artery. Both kidneys decapsulated and wrapped in omentum- thirty-eight days duration. Atrophy of area supplied by these vessels, unaffected by wrapping kidney in omentum. New capsule formed both kidneys.



Experiment No. 4
C 436

Ligation, posterior superior and inferior branches of the right renal artery; decapsulation and wrapped in omentum- 325 days duration. Atrophy of the posterior half of the kidney- not affected by decapsulation and wrapping kidney in omentum.

These experiments show quite conclusively that, (1), ligation of any branch of the renal artery causes atrophy of the area of renal parenchyma supplied by it; (2) each branch entering the kidney carries functional and nutritional blood to its respective area; (3) there is no anastomosis between any of the branches of the renal artery.

Group III

Effects produced by the Ligation of the Renal Vein.

My purpose in studying this phase of the problem was to determine, (1), the results produced upon the kidney, (2), whether a collateral venous circulation would be established and what veins comprise it, (3) whether this collateral venous circulation is capable of assuming the function of the renal vein and maintaining a functionating kidney.

Lindemann, in 1898, stated that after obstruction of the renal vein there is a collateral circulation developed sufficient to maintain the function of the kidney. Testut, cited by Barney, in the Annals of Surgery, 1917, states that the collateral circulation is capable of maintaining the renal function in part. Tuffier and Lejars, cited by Barney, state that obliteration of the renal vein results in the formation of a collateral circulation whereby venous blood from the kidney will be carried back to the general circulation through this plexus by four routes, (1), suprarenal and inferior diaphragmatic, (2), ureteric, and spermatic or ovarian, (3), subcutaneous plexuses of the lumbar veins, (4), a plexus which surrounds the last intercostal, ileoinguinal, and ilechypogastric nerves.

After exposing the kidney the renal vein was carefully dissected from the perirenal fat at the hilum and doubly ligated and sectioned. Eight experiments were performed in this group, as charted below.

Immediately after ligating the renal vein there is a marked enlargement and bluish discoloration of the kidney from venous congestion. The organ becomes very tense from the engorgement of blood. This is followed almost immediately by dilatation of the capsular vessels, especially the suprarenal, the ovarian or spermatic and branches to the lumbar veins. In a few instances a vein was found passing directly from the capsule of the kidney to the renal vein. Two or more of these dilated capsular vessels were found in most instances.

In Experiments No. 4, 5, 6, 7, and 8, and two days duration respectively, there was a marked congestion and enlargement of the kidney with beginning degenerative changes.

In Experiment No. 6, the dog died on the fourth day. At autopsy it was found that death was caused by hemorrhage from the operated kidney, which had ruptured in several places, as shown in the accompanying photograph, allowing the blood to escape and infiltrate the surrounding tissues. This kidney weighed 200 grams and showed enormous congestion throughout the parenchyma.

Experiments No. 3 and 4, of twelve and ten days duration respectively, showed a beginning atrophy of the entire kidney. In both instances there was a pronounced dilatation of the capsular veins.

In Experiments No. 1 and 2, of 132 and 345 days duration respectively, there was a marked atrophy of the entire kidney. In Experiment No. 1, only a small nodular of kidney tissue was left. In No. 2, the remaining kidney tissue was symmetrically atrophied and weighed four grams at the end of 345 days.

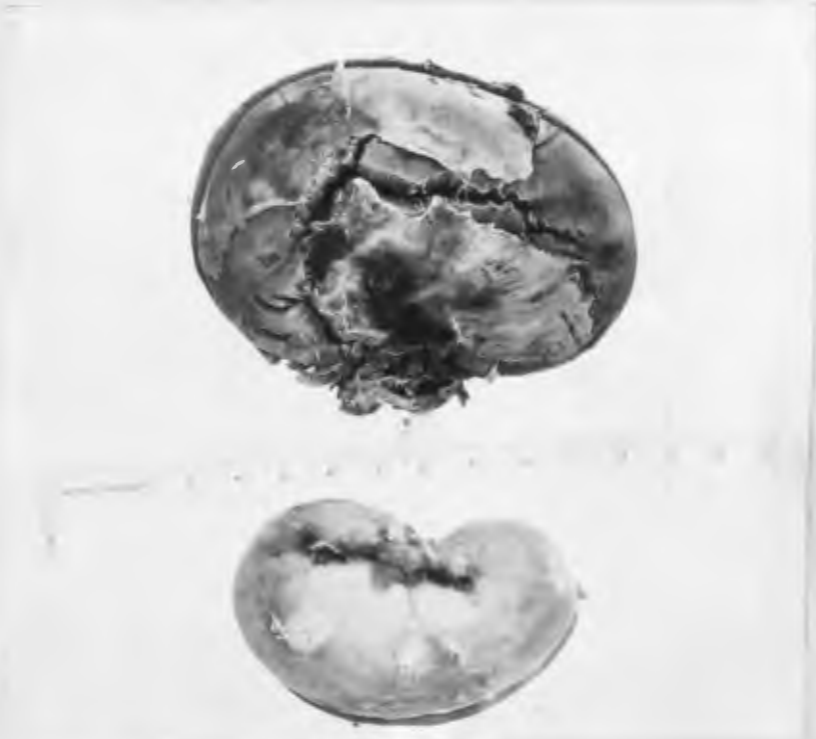
Series 2 - EFFECTS OF OCCLUSION OF THE VASCULAR RADICALS OF THE KIDNEY

GROUP III - LIGATION OF THE RENAL VEIN.

Exper. Number	Animal Number	Series Number	Date of Operation	Duration of Experiment in Days	Renal Vein Ligated Right or Left	One Branch of Renal Vein Ligated	Autopsy Number	Dilatation of Capsular Veins	Suprarenal	Ovarian or Spermatic	Lumbar	Congestion of Operated Kidney	Atrophy of Operated Kidney	Weight of Operated Kidney	Weight of Unoperated Kidney
156	B804	1	1917 2-26	345	L		49	+	+	+	+		+	5 gm.	30 gm.
291	B902	2	1917 3-26	132	R		453	+	+	+			+	4 gm.	20 gm.
50	B739	3	1917 1-27	12	L		55	+		+	+	+	+	24 gm.	30 gm.
418	C548	4	1918 6-24	10	L		336	+	+	+		+	+	25 gm.	37 gm.
126	B795	5	1917 2-19	6	L		87	+	+	+	+	+		34 gm.	30 gm.
246	B871	6	1917 3-17	4	L		164	(Kidney ruptured in several places; hemorrhage cause of death)						200 gm.	57 gm.
240	B868	7	1917 3-16	4	L		152	+	+	+		+		30 gm.	20 gm.
304	B913	8	1917 3-28	2	L		190	+	+	+		+		32 gm.	25 gm.

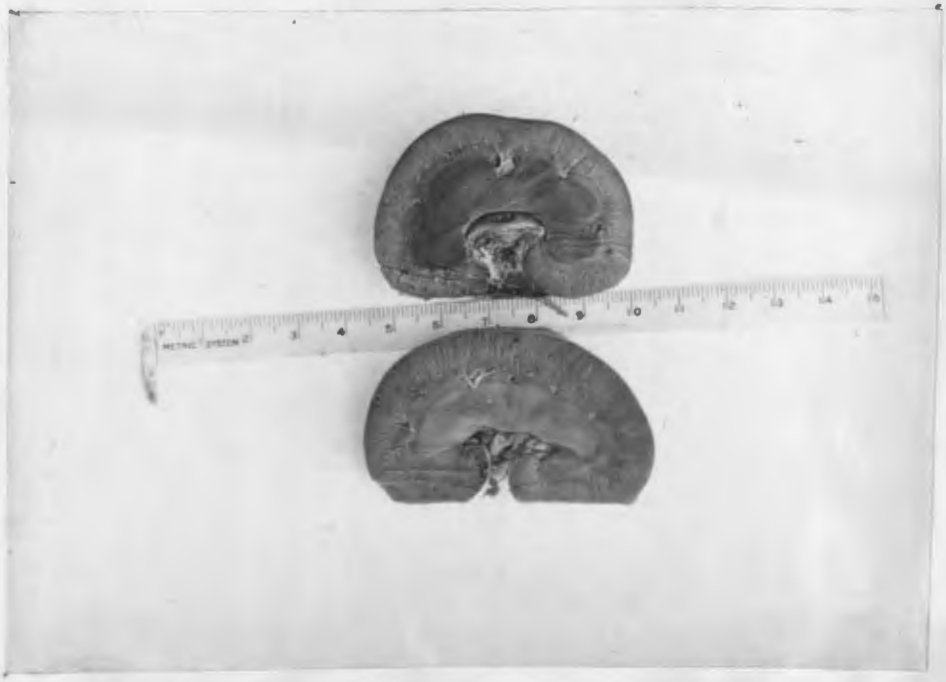
GROUP IV - LIGATION OF ONE BRANCH OF THE RENAL VEIN.

416	C546	1	1918 6-24	359	R	Post.	283					0	0	32 gm. R	34 gm. L
393	C524	2	1918 6-10	37	R L	Ant. Post.	572					0	0	34 gm.	37 gm.



Experiment No. 6
B 871

Ligation of the left renal vein- four days duration. Rupture of left kidney from engorgement of blood, causing death from hemorrhage. Right kidney normal.



Experiment No. 5
B 795

Ligation of the left renal vein-six days duration. Marked congestion of left kidney, weighing thirty-four grams. Right kidney normal, weighing thirty-six grams.

Experiment No. 2
B 902

Ligation of the right renal vein- 132 days duration. Atrophy of entire kidney, weighing four grams. Left kidney normal, weighing twenty grams.

From this group of experiments it is quite conclusive that, (1), there is symmetrical atrophy of the kidney after complete sudden occlusion of the renal veins, (2), that a partial venous collateral circulation to the kidney is established comprising chiefly the ovarian or spermatic, the suprarenal, and branches of the lumar veins, (3), that this collateral circulation of the kidney is not capable of assuming the function of the renal vein and maintaining a functionating kidney, (4), ligation of the renal vein in a dog may cause the kidney to rupture.

GROUP IV

Ligation of One of the Larger Branches of the Renal Vein

I undertook this part of the experimental work with a view of determining whether there would be a dilatation of the capsular vessels after partial obstruction of the venous return from the kidney, and whether there an anastomosis between the venous radicals of the kidney would occur.

The surgical procedure was the same as in the former groups except that after exposing the kidney, the larger of the two main branches of the renal vein was dissected free from the perirenal fat and doubly ligated and sectioned. There were two experiments made.

In Experiment No. 1, the dog was sacrificed in 359 days. At autopsy it was found that the kidney was normal in size and no change had taken place in the renal parenchyma but that slight dilatation of the capsular vessels could be demonstrated.

In Experiment No. 2, the animal died on the thirty-seventh day. At autopsy, the operated kidney was normal in size and there was no change in the renal parenchyma, but a moderate dilatation of the capsular vessels.

Experiment No. 2
C 524

Ligation of one branch of the right renal vein-duration thirty-seven days. Right kidney normal.

Series No. III

Trauma to the Ureter

Group I

Ureter clamped with an Artery Forcep

The frequency with which the ureter is accidentally and unavoidably traumatized during the course of various surgical precedures is very hard to determine. It is known that the ureter is sometimes accidentally clamped with an artery forcep during operations in the vicinity. And my purpose in doing the following experiments was to determine the ultimate effect upon the ureter and kidney. I do not know of any other instance in which this study has been made.

The operative procedure is essentially the same as that used in the former series. The ureter was exposed and crushed with a Kocher artery forcep for from one to thirty minutes and then replaced behind the peritoneum.

The following group of eleven experiments of from four to 251 days duration does not complete the investigation but the accompanying charts indicate the results obtained to date.

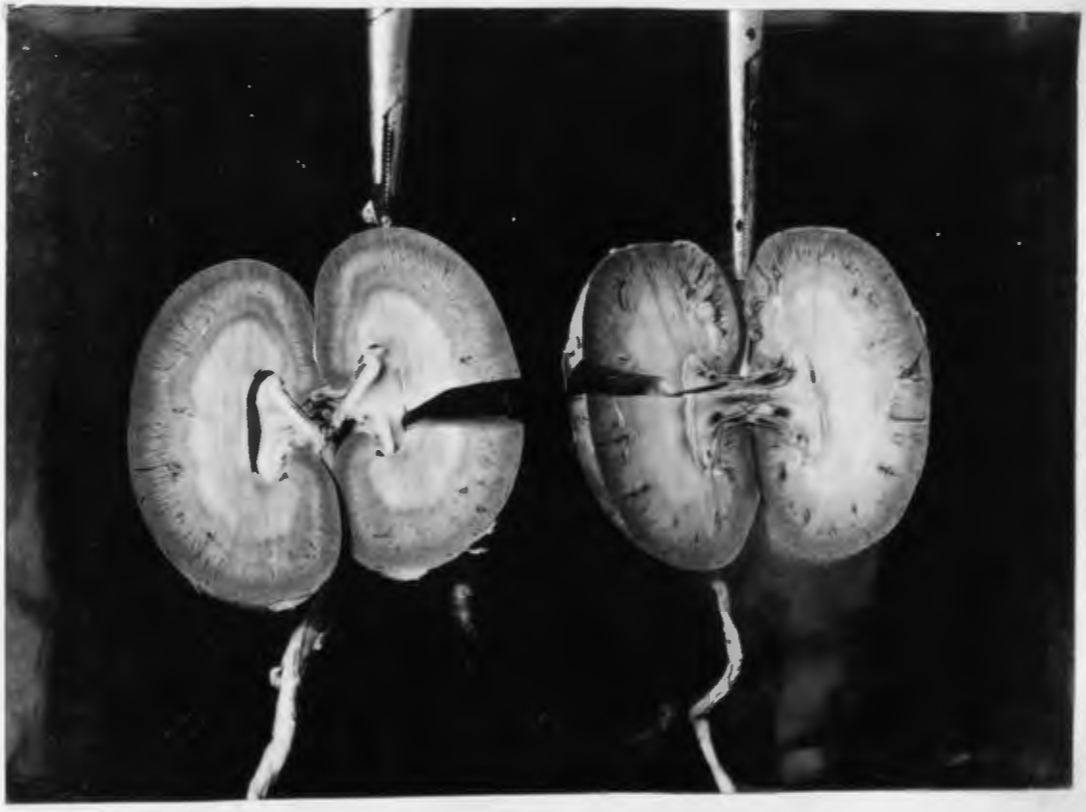
Series 3 - EFFECTS OF TRAUMA TO THE URETER

GROUP I - EFFECTS OF CLAMPING THE URETER.

Experiment Number	Animal Number	Series Number	Date of Operation	Duration of Experiment in Days.	Clamp on Ureter	Minutes Ureter Clamped	Notch of Kocher Clamp	Autopsy Number	Condition of Clamped Ureter: Degenerating Area	Condition of Clamped Ureter: Partial Constriction	Condition of Clamped Ureter: Dilated	Condition of Kidney of Clamped Ureter: Normal	Condition of Kidney of Clamped Ureter: Hydronephrotic	Dilatation of Capsular Veins.
			1917										Mkd.	
349	B953	1	4-16	251	R	10	3rd	619		+	+ 7-8x	+ 34 gm.	18 gm.	0
			1917								+			
262	B883	2	3-19	36	R	30	4th	278		+ Slt	Slt	+	Small	0
			1917											
348	B952	3	4-16	23	R	30	3rd	318		+	+	+	n	0
			1917											
191	B829	4	3-5	20	R	5	2nd	194		+	0	+		0
			1917											
192	B830	5	3-5	10	R	10	2nd	144	+	+	0	+		0
			1917		2x									
260	B881	6	3-19	7	R	3	2nd	181	+ Slt	+	0	+		0
			1917		4x									
225	B799	7	3-13	6	L	1	2nd	148	+		0	+		0
			1918											
664	B746	8	9-9	5	L	45	2nd	523	+	+	+ 2x	+		0
			1917											
333	B937	9	4-7	5	R	30	3rd	253	+	+	+ 4x	+		0
			1917											
318	B923	10	4-2	4	R	30	4th	215		+	+ Slt	+	Slt	0
			1917											
379	B974	11	4-30	4	L	30	3rd	417	+		+	+		0

Experiment No. 10
B 923

Ureter clamped for thirty-minutes. Duration of experiment four days. Slight constriction of the ureter which was dilated to four times normal. Slight hydronephrosis of the kidney. Opposite kidney normal.



Experiment No. 2
B 883

Thirty-six days duration. Ureter clamped thirty minutes. Partial constriction of the ureter. Ureter dilated seven to eight times normal. Slight hydronephrosis.

Experiment No. 1
B 953

Duration 251 days. Ureter clamped ten minutes.
Partial constriction with dilatation of the ureter.
Large hydronephrotic sac with atrophy of the kidney.
Opposite kidney normal.

Experiment No. 1 demonstrates the end-results upon the kidney. The right ureter was crushed with a Kocher clamp to the third notch for ten minutes. The animal was sacrificed in 251 days. The ureter showed a partial constriction which was almost complete but water could be forced through the constricted area with a pistin syringe. Both the ureter and pelvis were moderately dilated above the point of constriction. The kidney was small with a large hydronephrotic cavity as shown in the accompanying photographs, the parenchyma being almost entirely destroyed by pressure atrophy, the entire organ weighing eighteen grams. There was no dilatation of the capsular veins, but there was compensatory hypertrophy of the opposite kidney which weighed fifty grams. The condition was very similar to that found in the series where the ureter and the collateral veins were doubly ligated and sectioned.

In Experiment No. 2, the right ureter was clamped with a Kochar forcep to the fourth notch for thirty minutes. The dog died in thirty-six days. At autopsy it was found that there was partial constriction of the ureter at the point of application of the clamp. The ureter was enormously dilated seven to eight times its normal size above the point of constriction, and three times normal below it. There was a moderate degree of dilatation of the pelvis, a small hydronephrotic cavity, and slight atrophy of the renal parenchyma. The kidney weighed thirty grams. There was no dilatation of the capsular veins. The opposite kidney weighed thirty-seven grams.

The remaining experiments, Nos. 3 to 11 inclusive (four to twenty three days duration showed a degenerating area at the point of application of the clamp which gradually formed into scar tissue depending upon the duration

of the experiment. This scar tissue produced a stricture of the ureter with varying degrees of dilatation of the ureter above it.

From this particular study, it is quite evident, (1), that injury to the ureter by crushing with an arterial forcep causes degenerative changes at the point of application resulting in the formation of scar tissue which produces a partial constriction that may later progress to complete stricture, (2), that there is dilatation of the ureter and pelvis in most instances which may progress to the point of hydronephrosis and degeneration of the kidney, with no dilatation of the capsular veins, (3), that there is no leakage of urine following the application of a clamp to the ureter.

Group No. 2

Effects Produced by Stripping the Ureter.

During the course of various surgical procedures upon the kidney, ureter, and bladder as well as during other operations in their vicinity, it is often necessary to strip the ureter for a distance from the surrounding tissue. The practical significance of the extent to which this may be done is of utmost importance to the surgeon because of its bearing upon the end-results of various surgical procedures. Stewart and Barber, in a series of nine experiments upon dogs produced ureteral paralysis by stripping the ureter of all nerve, vessel, and lymphatic connections from the pelvis of the kidney to the bladder. This ureteral paralysis was accompanied by urinary stasis and distention of the kidney in 66 per cent of the cases.

My purpose in doing the following experiments was to determine the result produced upon the ureter and kidney by freeing the ureter from the surrounding tissues. In the following group there are six experiments ranging from fourteen to seventy-four days in duration. The operative procedure is essentially the same in each instance. After exposing the ureter it was lifted from its bed and stripped with a piece of gauze for a distance of from two inches to its entire length and dropped back into the abdominal cavity using care to preserve as much of the blood supply as possible.

It was my purpose in attempting this feature of the work, to determine what effect the procedure would have upon the kidney and the ureter. In the following six experiments, ranging from fourteen to seventy-four days in duration, the ureter was exposed, lifted up from its bed,

stripped with a piece of gauze for a distance of from two inches to its entire length, and then dropped back into the abdominal cavity, excercising care to preserve as much of the blood supply as possible.

A study of the results obtained indicate that the ureter may be stripped of its surrounding tissues without permanent injury or deleterius effect upon the kidney providing its blood supply is preserved, as in no instance was there any pathologic change found in the kidney or ureter.

Series 3 - EFFECTS OF TRAUMA TO THE URETER

GROUP II - EFFECTS OF STRIPPING THE URETER

Exper. Number	Animal Number	Series Number	Date of Operation	Duration of Experiment in Days	Ureter Stripped with Gauze	Blood Supply Preserved	Distance Ureter was Stripped	Autopsy Number	No Change in Ureter	Kidney Normal
583	C107	1	1917 8-20	74	L	+	entire length	317	+	+
536	C 75	2	1917 7-30	41	R	+	4 in.	372	+	+
285	B899	3	1917 3-24	31	R	+	3 in.	277	++	+
191	B829	4	1917 3-5	26	L	+	2 in.	194	+	+
525	C 72	5	1917 7-27	22	L	+	4 in.	490	+	+
					R	+	entire length		+	+
224	B783	5	1917 3-13	14	L	+	"	186	+	+

Series III. Group 5.

Methods of Anastomosing Ureters

During the course of various surgical procedure in the vicinity of the ureters, they are sometimes cut, and not infrequently the ureter is severed during accidents, fractures, etc. It was to determine the best method of anastomosing these injured ureters that I did the following twenty-two experiments. The operative procedure was the same as previously described up to the point of exposing the ureter. The ureter was completely cut across in each instance.

In five experiments (Method A) the cut ends of the ureter were anastomosed, end-to-end, with interrupted silk sutures. In thirteen experiments designated (Method B) the anastomosis was made by drawing the proximal end into the distal end for a distance of one-fourth inch, using three interrupted silk sutures, one at the lower end and two at the upper end of the anastomosis. In the remaining four experiments (Method C) the proximal end of the ureter was anastomosed into the side of the opposite ureter and sutured behind the peritoneum. The accompanying chart arranged according to the method used, indicates the results obtained.

GROUP III - METHODS OF ANASTOMOSING THE URETER.

Experiment Number	Animal Number	Series Number	Date of Operation	Duration of Experiment in Days	End to End Anastomosis	Cuff Anastomosis	Uretero-ureterostomy	Autopsy Number	Slight Constriction at Anastomosis	Marked Obstruction at Anastomosis	Ureter Dilated Above Anastomosis	Urinary Leakage	Operated Kidney Normal	Operated Kidney Showed Hydronephrosis	Atrophy of Operated Kidney
313	B920	A1	1917 3-31	3	R			199				+			
443	C 19	A2	1917 6-11	3	R			371				+			
483	C 43	A3	1917 7-6	3	R			413				+			
314	B921	A4	1917 3-31	19	R			233				+			
334	B938	A5	1917 4-9	56	R			344			Slt	0		Slt	
452	C 28	B6	1917 6-22	5		R		397				+			
617	C127	B7	1917 8-31	14		R		522	+	+		0			+
600	C119	B8	1917 8-27	18		R		520	+		Slt	0	+		
469	C 36	B9	1917 7-2	36		R		460	+		0	0	+		
489	C 46	B10	1917 7-9	43		R		493	+	+	+	0			+
454	C 30	B11	1917 6-25	48		R		469	0		Slt	0	+	0	
589	C112	B12	1917 8-24	49		R		538	+		Slt	0		Slt	
630	C135	B13	1917 9-10	60		R		597	0		Slt	0	+	0	
336	B940	B14	1917 4-9	67		R		373	+		Slt	0		+	
625	C131	B15	1917 9-7	81		R		623	0		Slt	0	+	0	
599	C118	B16	1917 8-27	95		R		633	+		Slt	0		+	
453	C 29	B17	1917 6-22	187		R		611	0		Slt	0	+	0	
455	C 31	B18	1917 6-25	223		R		8	+		+	0		+	
470	C 37	C19	1917 7-2	2			R into L	402				+			
498	C 55	C20	1917 7-13	3			L into R	420				+			
499	C 56	C21	1917 7-13	4			L into R	423		L		+	R 0 L +		
491	C 48	C22	1917 7-9	25			L into R	449		+		0	L Slt	L +	

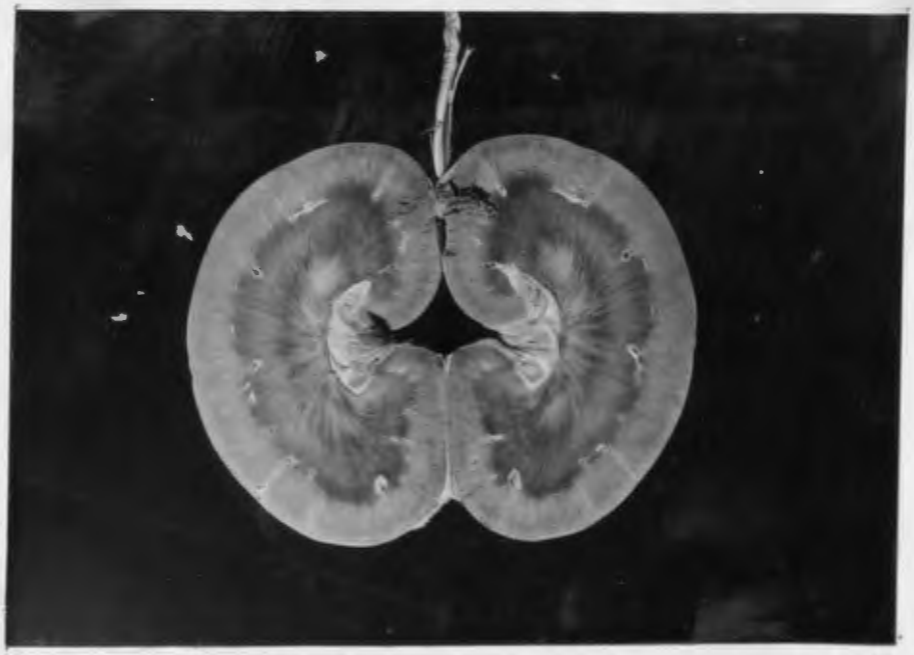
Experiment No. A 5
B 936

Fifty-six days duration. End-to-end anastomosis of the right ureter. Slight constriction and dilatation of the ureter; slight hydronephrosis of the right kidney. Opposite kidney normal.

Experiment No. B 15 C 131 Eighty-one days duration. Cuff anastomosis of the right ureter. Slight dilatation of the ureter. No stricture. Right kidney normal. Opposite kidney normal.

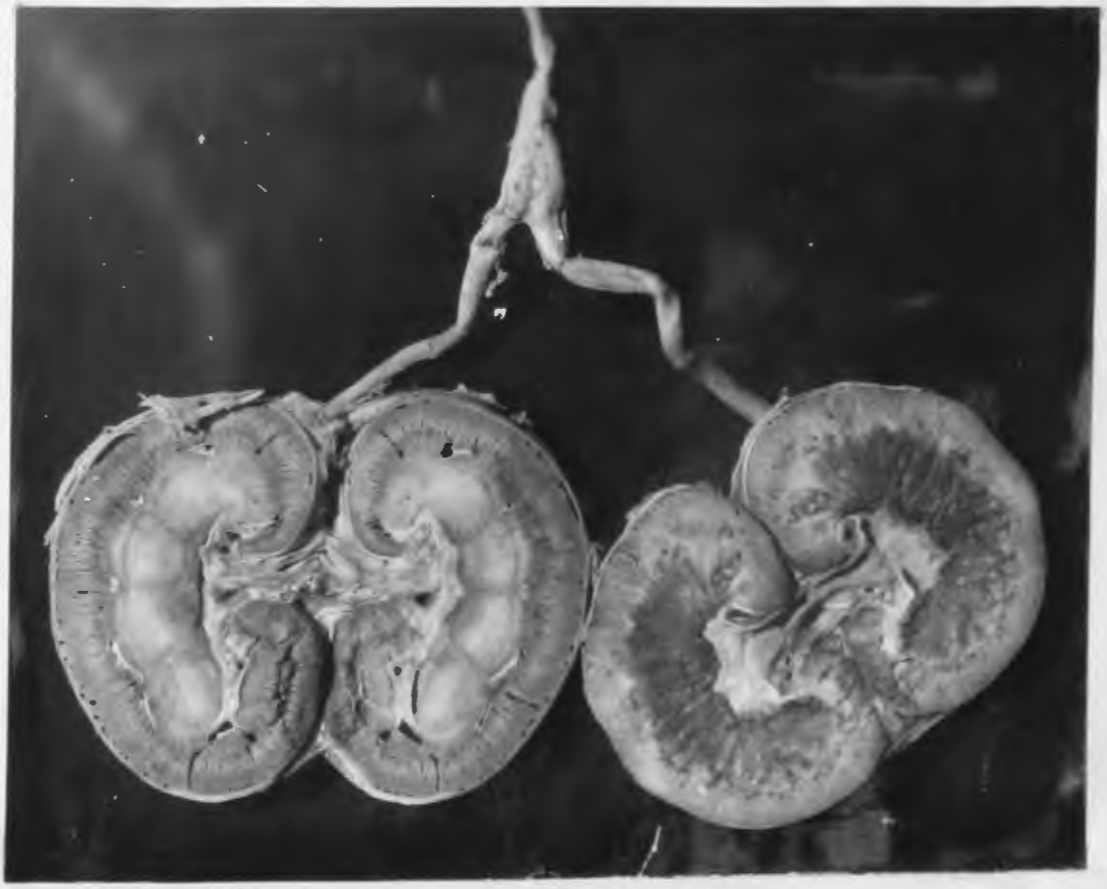
Experiment No. B 10
C 46

Forty-three days duration. Cuff anastomosis of the right ureter. Partial constriction and dilatation of the right ureter. Slight hydronephrosis with atrophy of the right kidney. Opposite kidney normal.



Experiment No. B 8
C 119

Eighteen days duration. Cuff anastomosis of the right ureter. Right ureter slightly constricted and dilated. Kidney normal.



Experiment C 22
C 48

Twenty-five days duration. Left ureter transplanted into the right. Partial constriction and dilatation of left ureter with slight hydronephrosis and atrophy of the left kidney. Right ureter dilated above the anastomosis. Right kidney normal.

Method A: <u>End-to-end anastomosis</u>. Three of the five animals, (Experiments 1, 2, and 3), died upon the third day of peritonitis from urinary leakage. In Experiment No. 4, the animal died upon the nineteenth day of sepsis, resulting from an abscess which had formed at the site of the anastomosis, burrowing into the pelvis forming a large pocket of pus. In Experiment No. 5, the animal was sacrificed upon the fifty-sixth day. At autopsy a slight constriction and dilatation was found and a slight hydronephrosis as shown in the accompanying photograph.

Method B: <u>Cuff anastomosis</u>. This group consists of thirteen experiments of from five to 223 days duration. In Experiment No. 6, the animal died upon the fifth day of peritonitis from urinary leakage, the only case of urinary leakage in this group. In Experiments Nos. 7 and 10, there was a marked narrowing of the ureters at the point of anastomosis causing a partial constriction and dilatation of the ureter above this point with atrophy of the kidney and a moderate degree of hydronephrosis as shown in the accompanying photographs. In the remaining ten experiments, there was a slight constriction of the ureter at the point of anastomosis, in most cases producing a slight dilatation of the ureter. There was little or no effect produced upon the kidney in five cases, and a slight hydronephrosis in five cases. The accompanying photograph shows the usual appearance of the kidneys.

Method C: Uretero- Ureterostomy. Three of the four animals in this group, Experiments No. 19, 20, and 21 died upon the second, third and fourth days respectively after operation, of peritonitis from urinary leakage. In Experiment No. 22, the animal was sacrificed upon the twenty-fifth day. At autopsy it was found that the anastomosis had healed perfectly, apparently without urinary leakage. The anastomosed ureter was dilated from partial constriction at the point of implantation and its kidney slightly atrophied. The recipient ureter was slightly dilated above the point of anastomosis, but the kidney appeared normal. The accompanying photograph shows the result obtained very nicely.

From these experiments it would seem that in a dog, (1), cuff anastomosis is the method of choice as in one case only did leakage of urine occur, (2), peritonitis resulting from urinary leakage is the most frequent cause of failure and death in anastomosing ureters.

Series IV

Group I.

Effects of Decapsulation of the Kidney

Group II.

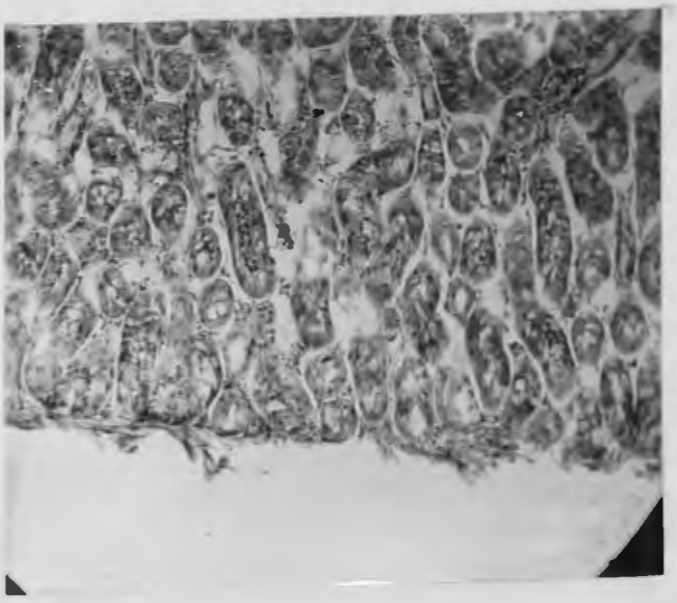
Effects of Decapsulation of the Kidney
and Ligation of the Arterial Blood Supply of the Kidney.

There has been a great variation in the results obtained by experimental decapsulation of the normal kidney, particularly, the distribution of the blood vessels that are formed to repair the injury. The results of practically all observers demonstrate that a new dense fibrous capsule is rapidly formed with an increase in the connective tissue of the cortex. There is still some controversy over the derivation of the new capsule formed and the distribution of the new blood supply. Emerson, in 1903, after twenty experiments upon dogs, concluded that the kidney formed a new capsule with marked vascular connections with the surrounding viscera but not with the fatty capsule of the kidney and also that connective tissue bands penetrate the renal parenchyma simulating a diffuse nephritis. Johnson, in 1903, from experiments upon fifteen dogs, concluded that the kidney formed a new capsule and that there was round-cell infiltration of the cortex but no anastomosis between the renal and peritoneal blood channels. Clifford, in 1904, from experiments upon the normal kidney of rabbits, found that a new very vascular capsule was formed after decapsulation but that these new vessels do not anastomose with the branches of the renal artery of the cortex. Hall and Herxheimer, working with rabbits observed the formation of a new vascular fibrous capsule, but very few new blood channels between the kidney and the adherent tissues. Stier, in 1913, from two series of ex-

periments upon cats and dogs, in two stages, found that a new capsule was formed from the omentum and that a collateral circulation is established in ten days which is capable of maintaining the kidney function properly when the renal vessels are tied off.

My purpose in undertaking the following series of thirteen experiments upon the kidneys of dogs, was to determine, (1), whether a new capsule would form, (2), whether the capsular circulation would be increased or decreased, (3), whether there would be an anastomosis between the branches of the renal artery and the arterial capsular blood vessels, (4), whether there are changes produced in the renal parenchyma. There are other points to determine in relation to the decapsulation of pathologic kidneys which have not been completed.

The operative procedure was the same as in the other series up to the point of exposing the kidney. Both layers of the fibrous capsule of the kidney were removed in the majority of instances, using care to injure the renal parenchyma as little as possible. It is usually difficult to remove the inner, thin, reticular layer of the capsule because of its intimate association with the connective tissue surrounding the tubules of the cortex. There is considerable oozing of blood from the kidney surface after removal of its capsule. This was controlled by compression with gauze for about four or five minutes. The decapsulated kidney was then wrapped in omentum and replaced in the abdomen. In Series II, seven experiments upon the blood supply were done in connection with this problem, two in which the renal artery was ligated and the decapsulated kidney wrapped in omentum, and five in which one or more branches of the renal artery were ligated and the kidney decapsulated and wrapped in omentum. The following charts indicate the results obtained:

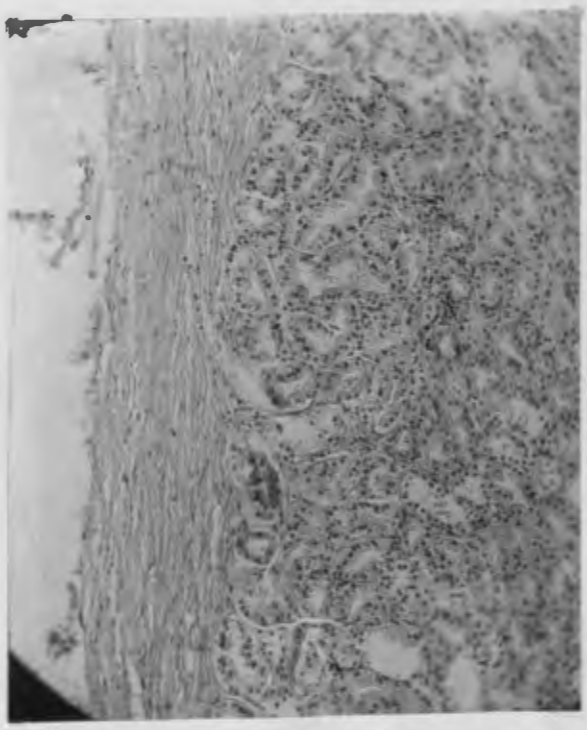


Experiment No. 11
B 768

Decapsulation of the left kidney- seven days duration. Beginning formation of new capsule from fibroblasts of the intra-tubular connective tissue.

Experiment No. 7
B 985

Decapsulation of the right kidney, wrapped in omentum- forty-six days duration. Formation new dense, thick, fibrous capsule.

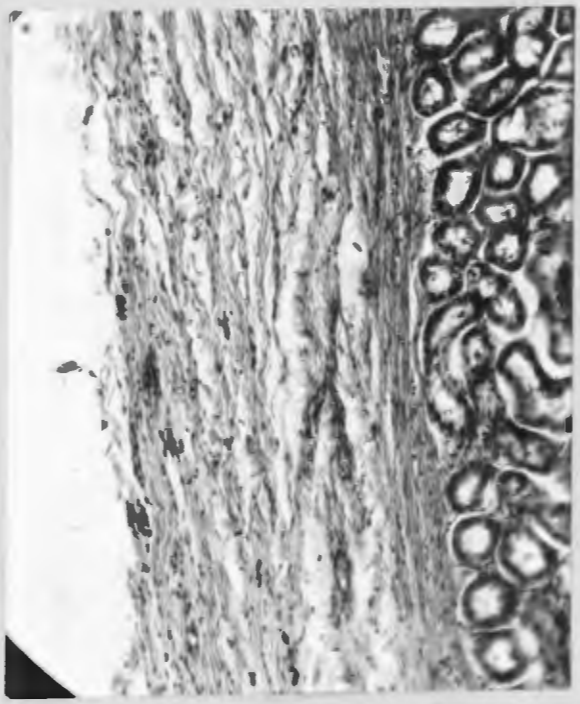


Experiment No. 5
B 855

Decapsulation of the right kidney, seventy days duration. Formation new, thick, dense, fibrous capsule.

Experiment No. 4
C 82

Decapsulation of right kidney- 115 days duration. Formation of new fibrous capsule closely associated with connective tissue of tubules.



Experiment No. 1
B 793

Decapsulation of left kidney. Wrapped in omentum, 383 days duration. Formation of a thick, fibrous capsule associated with the intratubular connective tissue.

From these experiments upon the normal kidney of the dog, it seems conclusive that after decapsulation, (1), the kidney rapidly forms a new capsule which is well marked in seventeen days, (2), there is an increase in the interstitial tissue of the cortex, (3), the newly formed capsule is more dense and fibrous and contains fewer (new) blood vessels than the normal capsule.

From the experiments of Series II, upon the blood supply, the kidney being decapsulated and wrapped in omentum after ligating the renal artery or one or more branches of the renal artery, it was found that these procedures had no effect upon the production of atrophy, which indicates that these capsular vessels do not anastomose with the branches of the renal artery.

Series 4.

GROUP I - EFFECTS OF DECAPSULATION OF THE KIDNEY.

Experiment Number	Animal Number	Series Number	Date of Operation	Duration of Experiment in days.	Decapsulation and Kidney Wrapped in Omentum	Decapsulation of Kidney	Autopsy Number	Congestion of Cortex	New Capsule Formed	Macroscopic Increase in Capsular Veins	Macroscopic Decrease in Capsular Veins	Increase in Connective Tissue in Cortex	Ligation Renal Artery, Decapsulation of Kidney and Wrapped in Omentum	Ligation of One Branch of Renal Artery and Decapsulation of Kidney and Wrapped in Omentum.	Anastomosis of Renal and Capsular Vessels.
124	B793	1	1917 2-19	353	L		53		+		+	+			
286	B900	2	1917 3-24	203		R	539		+		+	+			
591	C114	3	1917 8-24	169		R&L	58		+		+	+			
543	C 82	4	1917 8-3	113	R	L	675		both	+		+			
220	B855	5	1917 3-12	70		R	333		+		+	+			
367	B984	6	1917 5-7	51		R	398		+		+	+			
398	B985	7	1917 5-7	46		R	387		+		+	+			
261	B882	8	1917 3-19	42		R	293		+	+		+			
542	C 81	9	1917 8-3	17	R	L	489		R		+				
538	C 77	10	1917 7-30	17	R	L	480		+	+		+			
98	B768	11	1917 2-10	7	L		77	+							
535	C 74	12	1917 7-30	3	R	L	447	L + R O							
537	C 76	13	1917 7-30	2	R	L	446	R-slt L +							

GROUP II - EFFECTS OF DECAPSULATION AND LIGATION OF ARTERIAL RADICALS OF KIDNEY

273	C444	1	1918 4-29	526	R		464		+					R	O
99	B769	2	1917 2-10	16	L		90		+				L		O
53	B742	3	1917 1-27	10	L		53		+				L	R	O
398	C529	4	1918 6-17	348	R L		261		+					L	O
265	C438	5	1918 4-22	325	R		93		+					R R	O
364	C505	6	1918 6-3	38	R L		347		+					L	O
235	C412	7	1918 4-4	15	R		220		+					R	O

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