THE HYDRODYNAMICS OF THE UPPER URINARY TRACT (URODYNAMICS)*

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IT HAS BEEN customary to think of the urinary tract as divided into two zones of influence. The urologist has been held responsible for stones, for enlarged prostates and other obstructions and, in fact, everything connected with the tubes and reservoirs which conduct the urine to the exterior, but he is usually *persona non grata* when the renal parenchyma is concerned, except when neoplasm or tuberculosis is present, or after hydronephrotic atrophy has nibbled away most or all of the parenchyma. Yet our knowledge of obstruction and its handmaiden, infection, has been increasing to a point where one wonders whether this dividing line between medical and surgical diseases of the kidney has been drawn with complete accuracy, and, if not, just where it should be drawn.

No definitive pathological judgment may be rendered until we know so much about the normal function of any organ or set of organs that we can state positively and accurately what the criteria of normality are. Any departure from these criteria means disease. As far as the bladder is concerned, we have approached very close to that point. With the help of cystoscopy, urethroscopy, cystography, cystometry and uroflometry,[4] we remain in little doubt as to what the bladder can and will do.

Above the bladder, however, is a region about which we remain in comparative ignorance. We know that urine is propelled through normal ureters by peristaltic waves, but how are these waves initiated? What is the pressure existing under normal circumstances in the renal pelvis? Is there a regular cycle of rhythmic muscular contraction in the renal pelvis, and if so what are the systolic and diastolic pressures? How does urine travel from kidney to bladder after the ureter is dilated and peristalsis abolished? How high can the pressure in the renal pelvis go before the secretion of urine is impaired in any way? What are the differing degrees and stages of renal impairment brought about by increasing degrees of increased intrapelvic pressure, or, if you like, pelvic hypertension? How great a degree of pelvic hypertension must there be to produce clinically demonstrable hydronephrosis, and how long must it act in order to do so? These questions lead up to the more fundamental and more important ones. What is the effect on the renal parenchyma of prolonged pelvic hypertension, lasting for weeks, months or years, but of a degree not quite sufficient to produce hydronephrotic atrophy? And still further; is it possible that changes so produced might be very like, or even in fact identical with some of those conditions we have been accustomed to think of as primary diseases of the parenchyma?

These questions have occurred to relatively few investigators who have made studies, often ingenious, almost always crude, and amazingly few in number and unsatisfying as to results. I shall try to outline briefly the development of such knowledge as we now have.

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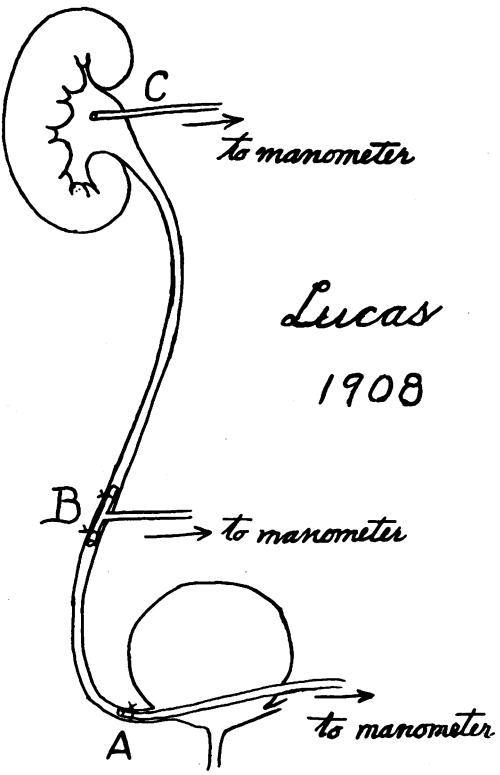

FIG. 1. Experimental set-up of Lucas.

In 1905,[13] Henderson found that ureteral peristalsis ceased when the pressure (he does not state whether of the upper or lower ureter) had risen to 26 to 32 mm. Hg in the dog, and 8 to 16 mm. Hg in the rabbit. Above this point the pressure varied with the blood pressure.

In 1908, Lucas[18] performed carefully planned experiments. He placed cannulas in the pelvis, in the mid-ureter, and in the lower end of the ureter, as shown in Figure 1, and determined renal blood flow by measuring the venous output. He found that if the ureter is obstructed below a cannula, the pressure is raised and the number of peristaltic contractions increases. This is the normal response to ureteral obstruction, and is the underlying basis of ureteral colic. Increasing the bladder pressure has no such effect. As the pressure in the ureter went up, pelvic pressure rose, but less rapidly. He says "the amount of pressure necessary to almost absolutely suppress the blood flow through the kidney when exerted in the ureter varied directly with the nearness of (the point of) insertion of the cannula to the pelvis of the kidney." The pressures necessary to do this in the rabbits used as experimental animals were noted as varying from 10 mm. Hg in the upper ureter to 50 mm. Hg in the lower ureter. Isolated ureters were able to exert, at their lower ends, pressure equal to that of 92 cm. of Ringer's solution. In general, the critical pressure in the lower ureter appeared to be about 15 mm. Hg. Below this point, pressure in the pelvis remained at approximately zero, while pressures above 15 mm. Hg, if continued long enough, brought about an eventual increase in pelvic pressure.

It further interested Lucas that ureteral peristalsis carried away all urine from the pelvis even when the pelvic pressure was recorded at zero. Possibly Lucas' manometric apparatus was not sufficiently sensitive to record the low pressures adequate to initiate ureteral peristalsis. However, artificially induced elevation in pelvic pressure increased peristaltic activity at once. If this increase was sudden the blood flow through the kidney was slowed more than if the increase was gradual.

Lucas says, in summary, "ureteral peristalsis is surprisingly strong and efficient when called upon to maintain or restore normal intra-pelvic pressure." Altogether Lucas' work is an important milestone in the study of the ureter. It illustrates the amazing power of the ureter to protect the kidney against back pressure.

In 1919, Satani[22, 23] published two papers entitled *Experimental Studies of the Ureter.* He varied intrapelvic pressure by injecting fluids through a pelvic cannula, as shown in Figure 2. With low pelvic pressure peristaltic waves were slow, as pressure increased they became more frequent and, finally, when the pressure became so great that the maximum peristaltic activity could not pre-

vent it from increasing, the ureter dilated and the fluid flowed through it as through a hose. Urea solutions appeared to stimulate the ureter more than salt solution, and an admixture of sand caused vigorous contractions and even anti-peristalsis. He also studied the effects on the ureter of the stimula-

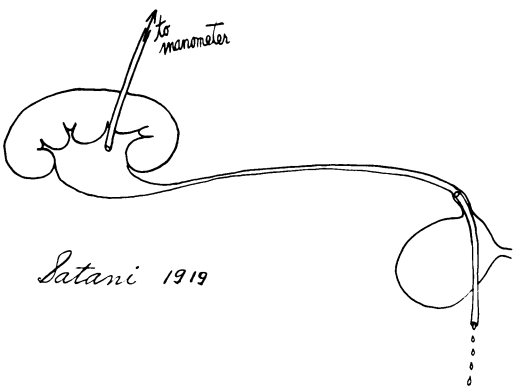

FIG. 2. Experimental set-up of Satani. The drops indicate that the rate of flow of the urine was determined.

tion of various nerves, and the effects of drugs on rings cut from the ureter.

From 1931 to 1940, numerous papers were published by F. R. Winton and his coworkers.[6, 29, 30, 31] They note that Ludwig, Cushny, and Brodie and Cullis observed that if the "ureter pressure" is raised, the flow of urine is reduced. These early workers apparently did not realize that the pressures in different parts of the ureter could vary greatly, or that it was the intrapelvic pressure alone which influenced the secretion of urine. Their opinions as to the intraparenchymal mechanisms involved are scarcely more than guesses.

Winton tried to study these relations, his favorite medium being a kidney-heart-lung preparation. Unfortunately the value of his results is lessened by the fact that he records the "ureteral pressure" without stating at what point in the pelvis or along the ureter the pressure was measured. In general, he was the first to note that elevation of pressure—presumably intrapelvic—up to about 10 mm. Hg had no effect on the amount or the composition of the urine secreted. This has been confirmed by many observers since, although the critical pressure varies with different species of animals, and is still not accurately known for the human being.

If the urinary flow is decreased in one kidney by increasing intra-pelvic pressure, and to an exactly equal extent in the other kidney by decreasing the arterial pressure, the changes in the composition of the urine are the same on the two sides.

In other experiments the Winton group kept the amount of urine secreted constant by raising the intra-pelvic and arterial pressures at the same time. This produced no change in chloride clearance, a slight depression in urea clearance, and a substantial depression in creatinine clearance. From these and other results they conclude that the main effect of increased intra-pelvic pressure is to reduce glomerular filtration, although they do not exclude effects on tubular resorption entirely. The question as to whether the effects of pelvic hypertension on urine composition results mostly from decreased glomerular filtration, or from changes in tubular resorption, or from both together has been discussed back and forth since then until the present day without receiving a positive, categorical answer. It has absorbed the renal physiologists to a very great extent, but is really of minor interest to the clinical urologist, who cares more about knowing exactly what changes do occur, and exactly what degree of dysfunction of the conducting part of the urinary tract is necessary to bring them about.

In 1937, Levy, Mason, Harrison and Blalock[16] studied the effects of ureteral occlusion on the blood flow and oxygen consumption of the kidneys. The hand of the future great cardiovascular surgeon can be seen in the ingenious experimental setup shown in Figure 3. A double balloon cannula inserted into the vena cava through the jugular vein isolated the caval orifices of the two renal veins, and allowed the venous outflow of the

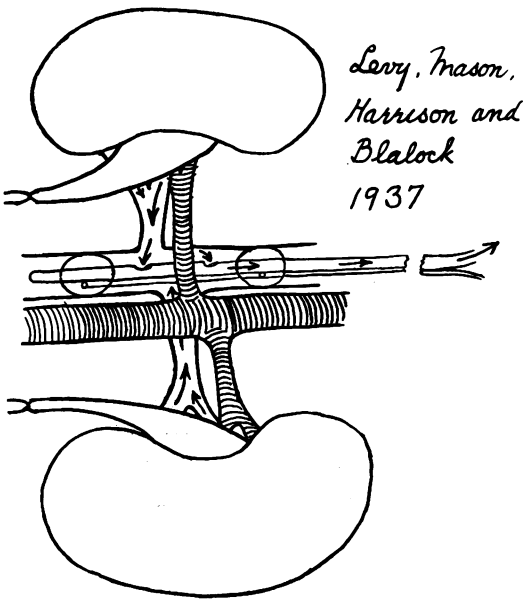

FIG. 3. Experimental set-up of Levy, Mason, Harrison and Blalock. The tube was passed down through the vena cava superior and right auricle into the vena cava inferior. The two balloons isolated the renal veins and the blood from them was collected through the tube.

two kidneys to be collected. The ureters were completely occluded. The result showed a diminution of blood flow, as a consequence of which the oxygen consumption was reduced, although there was no change in the arteriovenous oxygen difference. Simultaneous determinations of intrapelvic pressure would have increased the value of these experiments.

Also in 1937, Pilcher, Bollman and Mann[21] added some important data. They collected urine and determined pressures through a cannula placed in the ureteral meatus through the open bladder. At pressures up to 20 to 30 cm. of water the ureter functioned well and kept the pelvis practically empty. Above 40 cm. of water hydroureter and hydronephrosis began. At 30 cm. of water and below the urinary composition is unchanged. At 40 cm. of water the quantity is diminished, and there is a fall in chloride output, both absolutely and percentagewise. Up to 60 cm. of water creatinine and urea were unchanged, since as the quantity went down, the concentration went up. Above 60 cm. there was a diminution in the excretion of these nitrogenous substances. Phenolsulphonephthalein excretion began to diminish at 35 cm. of water. It must be remembered that these are lower ureteral pressures, not intra-pelvic pressures.

Perhaps the most important observation of these authors was that the onset of infection altered greatly the effects of increased ureteral pressure. After the kidneys became infected, water and chloride were excreted in larger amounts at the same pressures, while the excretion of urea, creatinine and phenolsulphonephthalein was markedly diminished.

In 1942, Lich[17] performed inulin and phenol-red clearances on normal humans and on others with chronic prostatic obstruction, and concluded that the renal dysfunction in chronic obstruction is primarily of the tubules. I confess my inability to judge whether this conclusion is justified or not.

In 1948, Lapides[14] made careful studies in humans by means of catheters placed in the lower ureters by cystoscope. He confirmed the fact that peristalsis varies in frequency with the amount of urine secreted, and therefore, almost surely, is initiated by an increase in the intra-pelvic pressure. He also concluded that ureteral peristalsis is not influenced by any drugs, including morphine and Depropanex. Results in the field of the pharmacology of the ureter are still somewhat contradictory, so perhaps we should reserve judgment.

In 1949, Selkurt and his co-workers[11, 24, 25] began to publish a series of papers covering many aspects of renal function. They showed that any decrease in the difference between arterial and venous pressure impairs the excretion of nitrogenous substances and causes particularly a retention of sodium and water. Since pelvic hypertension produces a similar change in the urine, it is interesting to speculate whether increased venous pressure brought about by the pressure

of dilated tubules upon peri-tubular capillary networks may play a part in this type of renal impairment.

In 1950, Pitts and Duggan[20] performed experiments which made them suspect that failure to excrete sodium and water properly may be due to excessive resorption in the convoluted tubules owing to prolonged contact with the tubular cells. Since back pressure may slow up the flow of urine through the tubules, it is possible that this helps to explain the failure to excrete sodium and water in urinary obstruction. Blake, Wegria, Ward and Frank[2] lend support to this idea by concluding that changes in the rate of sodium excretion cannot be explained as due to alterations in the glomerular filtration rate.

Also in 1950, Gottschalk[10] investigated the renal interstitial pressure, that is, the pressure in the intercellular spaces of the kidney which are entirely outside vessels, tubules or glomeruli. Needles were thrust into the tissues, and it was found that the normal pressures in guinea pigs, rabbits and cats averaged about 10 mm. Hg, and in dogs 16 mm. Hg. It is interesting that Winton in 1933 thought this intra-renal pressure was from 4 to 14 mm. Hg. When Gottschalk increased the venous pressure, there was no effect until the venous pressure equalled the pre-existing intra-renal pressure; then the two pressures rose together. When "ureteral pressure" was increased, intra-renal pressure rose, apparently, according to Gottschalk, owing to compression of the intra-renal veins.

In 1951, Goodyer and Glenn[9] showed that the pulsatory character of the arterial pressure was not essential to normal renal function. If the mean arterial pressure was maintained, without pulsation, there was no change in the excretion of electrolytes, water, inulin, or para-amino-hippuric acid.

Also in 1951, Wilson, Reisman and Moyer[28] showed, in addition to the well-known fact that sodium and water are retained during chronic prostatic obstruction with back pressure, that these two substances are rapidly excreted after relief of the obstruction, and that this increased excretion may persist long after all excessive

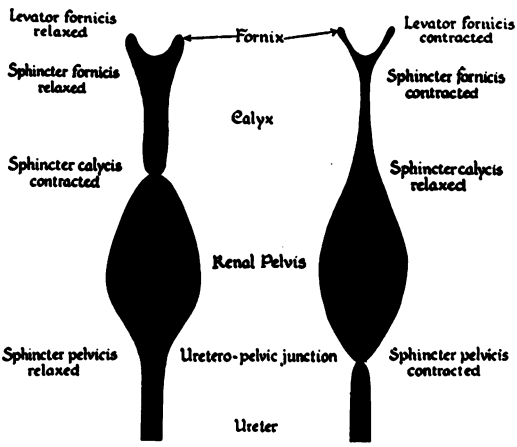

FIG. 4. Diagrams showing on the left systole and on the right diastole of the renal pelvis. During systole the infundibulum is closed off from the pelvis so that the flow from the collecting tubules can continue. From P. Narath, *Renal Pelvis and Ureter*, New York, Grune and Stratton, 1951.

amounts of water and sodium have been removed, so that dehydration and sodium deficit of serious degree may eventually result. This sodium deficit is not shown by the ordinary tests for nitrogen, chlorides, CO_2 combining power, plasma proteins, etc. Physical signs are much more important in the detection of this condition, which may easily be fatal if not relieved.

Peter Narath's[19] remarkable book on the renal pelvis and ureter was published in 1951. A trained investigator, as well as a uniquely expert radiological technician, equipped with the latest and best of German apparatus, he showed by his studies and pictures that there is a real pelvic systole, and that the calyces are actually closed during this systolic phase by sphincter-like muscles (Fig. 4). He also noted that the ureter and pelvis were better filled and in some cases moderately dilated if the bladder were very full. These pictures are very con-

clusive as to certain disputed points, particularly the muscular apparatus of the infundibula and calyces. Unfortunately they tell us nothing about the pelvic and ureteral pressures involved.

In 1952, Share[26] again studied the effect of "ureter pressure" on renal secretion. He collected urine at pressures of 15, 20, 25 and 30 mm. Hg. Up to 15 mm. there was no discernible effect. Above this pressure, excretion of sodium and water were progressively reduced, that of potassium to a lesser degree. While crude, these experiments confirm the results of others as to the immediate or acute effects of pelvic hypertension upon renal secretion. At almost the same time, Selkurt, Brandfonbrenner and Geller[25] found that if the "ureteral pressure" was increased to 52 cm. of water, the excretion of sodium and water was reduced 55 per cent, potassium 80 per cent, plasma flow 95 per cent, and glomerular filtration 80 per cent. All this fits together very well, but we have to regret that the increased pressure is called "ureteral pressure" so that we cannot know whether the figures given represent actual intra-pelvic pressures or not.

Beautifully organized experiments were performed by Durand and Descotes[5] in 1952 to test the effect on the ureter of the stimulation of various nerves. In general, they found that extrinsic nervous control of ureteral movements is practically nonexistent. In addition, the usual intra-pelvic pressure, 13 to 18 cm. of water in the dog, remains unchanged. This conforms with the observation that the most extensive sympathectomies for other conditions never impair the function of the ureters.

In 1953, Lapides[15] summarized what little we know or guess about renal impairment due to chronic prostatism. That we are dealing with many different degrees and different stages of kidney damage is shown by the great variation in the nature of the retentions and deficits found in clinical cases. For the present we must, as Lapides states, individualize the treatment of each patient, but we may hope that in the future we shall be able to separate definite types and degrees of renal dysfunction.

Baker,[1] in 1953, studied the electromyography of the ureter. These studies again show the remarkable autonomy of the ureter. Medication, anesthesia, respiration, motion and chilling have no effect on the orderly sequence of peristalsis, or the electrical waves which mark the progress of the muscular contraction along the ureter. Only if some form of obstruction is present are the duration and voltages increased, obviously a sign of increased effort. Other studies, such as those reported by Hanley[12] in 1949, are made with a large catheter in the ureter, and therefore scarcely represent normal or even natural function, whether the catheter is placed in a normal or in an abnormal ureter.

Altogether, these studies have supplied only a small store of knowledge in comparison with other fields of investigation, as, for example, hemodynamics. Unfortunately the student of ureteral function soon realizes that much of the enormous quantity of highly detailed work which has been done on renal function must be regarded with reservations because so frequently no account has been taken as to the presence or absence of pelvic hypertension. In addition, all of the experimental work on the ureter has been in the form of acute experiments. In a word, we know full well that severe and protracted pelvic hypertension damages the kidney severely, even to complete hydronephrotic atrophy, but we do not know just how much pelvic hypertension is necessary to cause kidney damage, nor what its effects are on the parenchyma, nor what the relations are among the factors of, first, degree of pelvic hypertension; second, the duration of pelvic hypertension; and third, the character of the functional and structural changes produced in the parenchyma. In other words, we do not know what the difference will be between a kidney which has been exposed to an intra-pelvic pressure of

15 mm. Hg for six months and another kidney which has been exposed to an intra-pelvic pressure of 20 mm. Hg for six months. Might some such kidneys look very like what are described as chronic pyelonephritis, glomerulonephritis, or arteriosclerotic kidney?

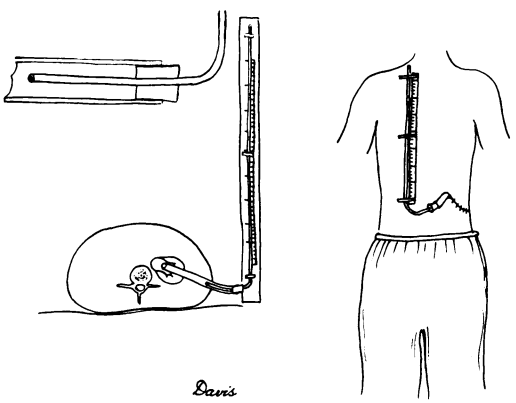

FIG. 5. Measurements of intra-pelvic pressure in the human. Left, above, method of attaching small polyethylene tube to nephrostomy tube. Left, below, arrangement with patient lying in bed. Right, arrangement with patient standing or sitting.

These important deficiencies in our knowledge are due in very large part to the fact that we have had no suitable method for measuring the intra-pelvic pressure either experimentally or clinically. Tubes inserted through the ureters are valueless, since they impair the function of the ureter, upon which the intra-pelvic pressure depends entirely. Tubes inserted through the parenchyma may indeed be attached to manometric devices, but cannot be worn long enough to give a true picture of what is going on in the pelvis. In addition, fluid manometers, no matter how ingeniously constructed, are of poor sensitivity for the magnitude of the pressure changes involved, and they produce serious artefacts by reason of the necessary movements and inertia of the fluid contents. If one reduces the caliber with a view to minimizing the movements of the fluid, capillarity introduces new and different artefacts. Besides, it is very difficult to produce graphs with such manometers.

My own interest in these matters led me to try to measure intra-pelvic pressure in a few patients who happened to be wearing nephrostomy tubes. The set-up was much as is shown in Figure 5. A small polyethylene tube, 1.5 mm. in internal diameter, was attached in water-tight fashion to the nephrostomy tube, and the height to which the urine rose in the tube was observed directly. With the patient in bed, the tube was attached to an upright pole stand. When the patient was up, the tube was fastened to the skin of his back with adhesive plaster. In both cases a centimeter scale was placed alongside the tube.

In one case a nephrostomy tube was kept in a hydronephrotic pelvis, after an intubated ureterotomy operation[3] had been performed on a uretero-pelvic obstruction, because the pelvis still failed to empty promptly. Observation showed that all of the urine was transported down the ureter, without pain or fever, at a pressure of 15 cm. of water. The nephrostomy tube was eventually removed, and the sinus closed promptly. The patient has remained clinically well since, a period of about four years.

In another case the hydronephrotic kidney, which was in addition the patient's only kidney, held 360 ml. An intubated ureterotomy had been performed, which permitted the urine to flow freely into the bladder, as seen by fluoroscopy. Intra-pelvic pressure was measured at 12 to 15 cm. water with the patient lying in bed. Here again the tube was eventually removed, and the patient has remained clinically well for a period of over three years.

In a third case, a nephrostomy tube had to be placed in a slightly dilated left pelvis on account of pain and fever resulting from obstruction of the lower ureter by a spreading carcinoma of the bladder. Shortly afterward bilateral uretero-enterostomy was performed. Two or three weeks later, prepara-

tory to removing the nephrostomy tube, intra-pelvic pressures were measured. It was noted that clamping of the tube caused pain, and that the pressure at which all of the urine was carried away by the ureter increased slowly and progressively from day to day. Above about 25 cm. of water pain prevented further measurements, and it became obvious that the nephrostomy tube could not be removed. The patient died after a short and rapid down-hill course, and autopsy showed that a metastatic nodule in the ureteral wall just above the ureteroenterostomy had gradually produced complete occlusion. The increasing degree of obstruction had been accompanied and demonstrated by the progressive rise in the intra-pelvic pressure.

In a fourth case a small stone had been removed from the renal pelvis, and a slight uretero-pelvic obstruction treated by intubated ureterotomy. With the nephrostomy tube clamped, the patient was perfectly comfortable, with normal temperature and no evidence of urinary tract infection. Careful observations showed that when he was lying quietly in bed the intra-pelvic pressure remained at eight to ten cm. of water; when he sat quietly in a chair it rose to 12 to 15 cm. of water; when he stood erect or walked slowly it registered at 17 to 18 cm. of water, and when he exerted himself by lifting a heavy chair it rose to 23 cm. of water. In each case all of the urine was passing down the ureter. Respiratory waves with an amplitude of about 1 cm. were present at all times, but I could make out no evidence of rhythmic contractions of the pelvic muscles, probably because my improvised manometer was not sensitive enough. Obviously the pressure was markedly influenced by the intra-abdominal pressure, yet a pressure of 8 cm. of water sufficed to initiate ureteral peristalsis and to keep the pelvis practically empty.

There seems to be a good deal of evidence that the pelvis can compensate against obstruction just as the bladder does. The walls of hydronephrotic pelves, particularly the small and moderate sized ones, are markedly thickened due to muscular hypertrophy. The degree of muscular hypertrophy no doubt varies in different individuals, just as in the bladder wall. We cannot, therefore, make even a rough guess as to the intra-pelvic pressure by studying pyelograms or even by observing the kidney directly. Direct manometric studies by thrusting a needle into the exposed but unopened pelvis might help, but this would give us records only of cases far advanced enough to require operation.

Apparently the parenchyma is prepared to deal with a certain degree of pressure increase—probably about enough to account for the elevations of intra-abdominal pressure occurring in ordinary life. Above this point we remain in comparative ignorance until we reach those extreme degrees of pelvic hypertension which give rise to unmistakable hydronephrotic atrophy.

Altogether I believe that the proper exploration of this very important field must begin with a thoroughly planned experimental study in animals. The stage is set for such a program. In preparing the plan one must first see clearly what questions need to be answered. Methods of studying renal function are well known, so that the study can be concentrated upon an investigation of pressure conditions at all points. Pressures, indeed, are vital to the entire process of urine secretion, conduction, storage and expulsion from the body.

In Figure 6 I have attempted to show the various localities at which the pressures existing can influence function markedly. Proceeding from below upward, No. 1 indicates the bladder. Pressure varies in the normal bladder, but remains little higher than intra-abdominal pressure except during the comparatively short period preceding and during the act of voiding. If obstruction is present below the bladder, bladder pressures may rise very high, and voiding, or attempting to void, may come to occupy a large part of the time. Nevertheless, the ureters

must be able to discharge into the bladder all urine as rapidly as it is formed if back pressure on the kidneys, that is, pelvic hypertension, is to be avoided. No. 2 is the lower ureter, No. 3 the mid-ureter, No. 4 the upper ureter. The adequacy of the ureteral function determines the pressure in the renal pelvis, No. 5. For this reason it cannot be measured with instruments passed through the ureter. Narath's studies indicate that the pressure in the calyces, No. 6, is not always the same as in the pelvis. The renal tubule, or nephron, Nos. 7, 8, 9, 10 and 11, has no muscle in its walls. The pressures in the nephron, therefore, must depend on (a) the filtration pressure in the glomerulus, the pressures on the exterior of the tubule exerted by (b) the interstitial pressure, No. 20, (c) the pressure in the peritubular capillary networks, No. 17, (d) the resistance offered by the pressure in the calyces, Nos. 5 and 6, and (e) the relation between the size of the tubule and the quantity of fluid which must be transported through it. The filtration pressure varies with changes in the arterial pressure, No. 13; the venous pressure, No. 19; the interstitial pressure, No. 20; and the pressure exerted from within outward upon the peritubular capillary networks, No. 17, by the tubule of the nephron, Nos. 7, 8, 9 and 10, more or less dilated as it may be. Surely a complicated situation, but not discouragingly so, because the most important and significant pressures are subject to measurement by experimental if not by clinical means. To specify: Bladder pressure, No. 1, can be accurately determined. The adequacy of ureteral function, Nos. 2, 3 and 4, is best estimated by determining the intra-pelvic pressure, No. 5. Pressure in the renal artery, No. 13, can be assumed to be the same as the general arterial pressure. Venous pressure, No. 19, and blood flow can be determined experimentally, as can interstitial pressure, No. 20.

The key measurement, however, remains that of intra-pelvic pressure. For this purpose new and fascinating instrumental facilities are available. Pressure gauges as small as 2.5 mm. in diameter have been developed by Gauer[7, 8] along the lines suggested by Wetterer.[27] The moving parts are

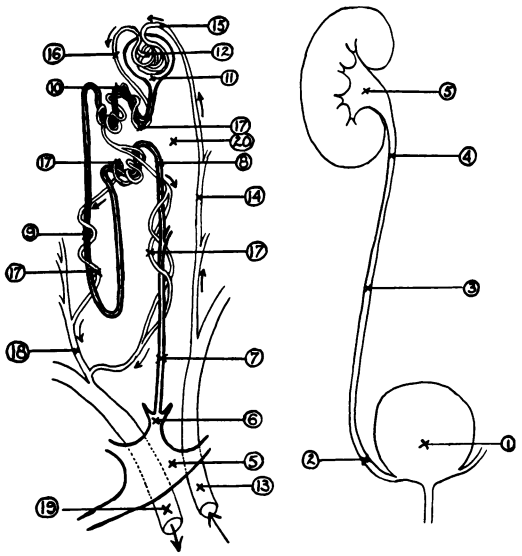

FIG. 6. Diagram of bladder, ureter, pelvis and nephron, showing the 20 points at which pressures may vary with consequent effect on the secretion of urine. Explanation of figures in text.

small, and the excursion almost infinitesimal. It is enough, however, to generate a tiny electrical current, which is conducted out of the body by a wire and then amplified electronically so that it can be recorded as a graph. This arrangement does away with all artefacts, and can be made as sensitive as desired. If a tube is kept in the pelvis, similar accurate pressure measurements may be made at any time with a strain gauge.

Ureteral peristalsis will soon be made visible in the intact body by amplified fluoroscopy, and such observations can be permanently preserved by means of motion picture photography. The value of these studies will be enormously multiplied if they are made along with simultaneous recording of intra-pelvic pressure.

All of the highly developed methods of studying renal physiology will equally gain

in value, and, I personally believe, will become much more easily comprehensible if they are coördinated with determinations of the intra-pelvic pressures in the experimental animals or the patients under investigation.

The use of these novel modalities will mark a new and brilliant era in the study of the physiology of the urinary tract.

At the present moment surgical means of removing practically any form of obstruction at any point in the urinary tract are available. It remains only to determine how to detect abnormal pressure conditions before irreparable damage is done.

Many presumedly primary diseases of the renal parenchyma are of completely unknown origin. Wherever we may find that pelvic hypertension is a factor in the etiology of such diseases, we can apply radical and dependable surgical therapy. We are obligated, therefore, to explore the possibilities, with some fair reason to hope that we may learn not only to cure infection and prevent hydronephrosis, but perhaps also that conditions, now thought to be diseases primary in the parenchyma, are really the result of chronically increased intra-pelvic pressure.

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