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Anuria

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INDEX

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William A. O'Brien, M.D.

I. LAST WEEK

Date: March 11, 1937

Place: Nurses' Hall
Recreation Room

Time: 12:15 to 1:25 P.M.

Program: Movie: Heart of the Sierras

Abstract: Giant Cell
Tumors of Bone

Case Report: 1

Present: 118

Discussion: R. W. Koucky
L. G. Rigler
C. W. Waldron
R. M. Johnson
W. T. Peyton

II. MOVIE

Title: Pacing the Thoroughbreds

Released by: Fox Film Corp.

III. NO MEETING

Next Week on account of
Spring Vacation.

IV. ABSTRACT

ANURIA

A. K. Doss

Definition

Anuria is a term used to designate absent elimination of urine from the body resulting from any condition located at any point along the urinary tract from the preglomerular arterioles to the urethra. In this discussion, the term will refer only to conditions in which the disease is located in the kidney and ureter.

Classification

Various investigators have commented on anuria from the point of view of the etiology, location of the pathology, etc., however the classification given by Hinman seems to be as inclusive and comprehensive as any.

I. Mechanical obstruction at some portion of the tract

A. Extrarenal

1. Obstruction in the lower tract.
2. Obstruction in the upper tract.

B. Intrarenal

1. Usually regarded as secretory form--an obstruction of multiple renal tubules such as is caused by albumin, hemoglobin crystals precipitated after excretion in some forms of hemoglobinurias (improperly grouped transfusions, burns, malaria, infections as with B. welchii and intoxifications as with potassium chlorate), etc.

II. Secretory forms of oliguria and anuria may be classified according to causation into

A. A primary diminished intake (oligodypsia).

B. The dehydration form (in diarrhea, excessive vomiting, sweating, etc.).

C. The retention form (non-obstructive).

1. Retention of water (during insulin therapy and often associated with renal insufficiency).

2. Retention of salt (injury of nerves at the base of the 3d ventricle, Jungmann).

3. With renal insufficiency caused by local pathological changes (nephropathic):

   a. of glomerulonephritis

   b. of nephrosis (especially mercury and lipoid).

D. The circulatory form (mainly vasomotor and mechanical).

   1. The inhibitory form (vascular spasm of main vessels following splanchnic stimulation, or of capillaries following peripheral irritation (the urethrorenal, vesicorenal, ureterorenal, and renorenal reflexes). The anuria which follows ureteral catheterization (and pyelography) is of this type.

   2. Hysteria.

   3. Low blood pressure (as in shock).

   4. Massive disturbance of circulation as in thrombosis, embolism and tumors.

E. The endocrine form (pituitary, oftenest with dystrophia adiposogenitalis; thyroid, the oliguria, incontinence and nocturia of hypothyroidism; adrenal, possibly some of paroxysmal forms similar to paroxysmal hypertension).

F. The allergic form.

G. The hepatic form (an oliguria, anuria or even pure uremia may be caused by injury to the liver).

H. The renal form (an oliguria the counterpart of the renal polyuria of diabetes insipidus).

I. The malingering form. (The patient hides urine in hot water bottles, flower pots, etc.)

III. A combination of obstructive and secretory factors occurs in many forms of oliguria and anuria.

IV. Mechanical obstruction at some portion of the tract:

A. Extrarenal

1. Extrinsic of ureter

a. Anuria has been known to appear following surgical procedures in the lower abdomen and pelvis.

One such case is reported in the literature in which catheterization of the bladder showed absence of urine on repeated examinations. Bilateral ligation of the ureters was suspected. Bilateral nephrostomy was immediately established upon realizing the circumstances. Sufficient time was allowed for dissolution of the suture material used, ureteral catheters were then passed readily into the renal pelves and the normal flow of urine was uneventfully established.

Not infrequently neoplasms, especially in the instance of involvement of the lower abdomen, may cause complete bilateral obstruction of the ureters. Myers reports such a case in which anuria persisted for 30 days with little increase in the blood pressure (not over 140 mm. Hg.), absence of frank coma, with a blood urea nitrogen of 169 mg. % on the 27th day following the onset of complete anuria.

2. Intrinsic of ureter

a. Ureteral stones, either by their appearance bilaterally causing obstruction or as a result of reflex stimulation in association with a unilateral stone, are responsible for the majority of cases of anuria. Eisendrath gives the following conditions under which anuria develops in association with stones:

(1) When the ureter of a solitary kidney is blocked.

(2) When both ureters are simultaneously blocked.

(3) When the ureter is blocked and the other kidney is not developed.

(4) When the fused ureter of two kidneys or the single ureter of a fused kidney is blocked.

(5) When one ureter is blocked and the opposite kidney is reflexly suppressed.

b. Blood clots, resulting from hemorrhage into the renal pelvis, or detritious material resulting from severe pyelonephritis may pass into the ureters and produce obstruction. Occlusion as a result of stricture of the ureters and edema at the uretero-pelvic juncture are also to be considered.

B. Intrarenal

a. Of the intrarenal forms of anuria that associated with transfusion reactions is probably the most interesting and certainly the most distressing as this almost invariably means the addition of a complication which readily becomes of more importance than the original picture which indicated the need of transfusion. Bordely describes the mechanism of this type of anuria as follows:

(1) By mechanical blockage of their tubules, the kidneys have been rendered functionless as excretory organs.

(2) The kidneys are sensitive to certain bodies contained in the injected blood, and functional decline results from a local reaction which is of the nature of an anaphylactic shock.

(3) The immediate transfusion reaction brings about a metabolic disturbance that affects renal function.

(4) By the action of toxic substances set free in the blood at the time of transfusion, the functioning renal tissue is so severely damaged that it is unable to perform its duties.

The above mentioned possibilities certainly include most, if not all, of the theories concerning the mechanism of oliguria and anuria following transfusion reactions.

V. Secretory form of oliguria and anuria

A. Primary diminished fluid intake or dehydration from excessive fluid loss as in diarrhea, excessive vomiting, sweating, and the like are obviously associated with a certain degree of temporary anuria, persisting only so long as the unbalanced water metabolism exists. These types of anuria may be readily dismissed but must surely be remembered when confronted with a case of anuria.

B. Retention or nonobstructive type

Retention of water during insulin therapy.

"It is well known, for instance, that institution of insulin treatment is often accompanied by edema formation. It is not yet possible to give an explanation of this, but Kulin has observed a considerable decrease in the colloid osmotic pressure of the blood

in the cases which develop edema and in some cases he saw this before edema developed." It is rather unlikely that this factor would become so prominent that complete anuria would be established.

Retention of salt.

A lesion involving the region of the tuber cinerum is said to result in salt retention followed by fluid retention, when the "salt regulating center" is involved; however, cases of this sort are relatively rare.

Renal insufficiency.

Simple nephrosis due to mercurial poisoning is rather commonly encountered. Richards investigated bichloride poisoning in frogs. He found that the bichloride kidney during life displays the same disabilities as those exhibited by the surviving kidney acutely injured, that is loss of water extruding power and loss of selective impermeability. The part of the tubule largely concerned appears to be a section intermediate between the proximal and distal convolution. He found in the instance of a kidney of a living frog poisoned with a suitable dose of mercuric chloride during life, that the circulation through every visible glomerulus is extraordinarily active.. Fluid is being separated from the blood within the Malpighian bodies, not at a slower rate but on the contrary at a much faster rate than normal. If phenolsulphonphthalein were injected at the beginning of the experiment, its concentration, the molecular concentration, and the electrolytic content in the secreted urine were all normal. No urine passed from the glomeruli into the ureter however. The kidney was found to be nearly or quite anuric, yet the glomeruli formed urine in excessive amounts. The bichloride destroyed the power of the tubules to extrude water and retain certain diffusible substances, consequently the question was raised as to why water of glomerular filtrate failed to reach the ureter. The answer offered was that under these abnormal conditions the osmotic pressure of the blood protein is unobstructed by the normal quantity of tubular epithelium and is able to reabsorb all or nearly all of the glomerular filtrate back into the blood.

Diabetes.

Severe oliguria and anuria may begin from 24 to 48 hours after the onset of coma. Frequently, coma has been recovered from before the onset of anuria. The mechanism has been considered to be the result of tubular injury usually attributed to ketogenic acids. Tubular injury seems seldom sufficient to explain the uremia. "The cause of anuria and azotemia in diabetic coma is not clearly established. There is evidence that the following factors may be concerned: injury, dehydration, increased endovenous metabolism, acidosis with injury of renal tubules, and decrease in blood pressure." (Bell)

Glomerulonephritis

is always associated with albuminuria, usually in large amounts. Edema is practically never lacking and is usually found to be generalized. It is not the pitting type seen in nephrosis but on the contrary is of the "intracellular type." The mechanism of this edema is not definitely known. Some investigators explain its presence on a basis of a systemic vascular constriction involving chiefly the heart, brain and kidneys. The severity of involvement being in the order named. Bell is of the impression that cardiac involvement with failure plays a prominent part, yet there are other factors, unknown at present, which are likely of great importance. There is almost invariably a distinct oliguria and often complete anuria in glomerular nephritis.

VI. The circulatory form:

A. Nerve supply of kidney and ureter.

"The extrinsic nerve supply of the kidney is derived from the renal plexus which extends from the aortic plexus along the renal artery to the hilum of the kidney. The renal plexus is made up of branches arising from the center of the semilunar ganglion, the lesser splanchnics, and the major splanchnics, a branch arising from the first lumber ganglion, and small branches arising from the first small ganglion which lies on the posterior aspect of the superior mesenteric artery. The renal plexus is also joined by one or more

slender rami from the lumbar portion of the sympathetic trunk, according to some authors. Vagus branches run directly to the renal plexus in many, but not in all cases. Direct vagus branches seem to occur more commonly on the right than on the left side. Preganglionic fibers supplying the kidney are present in all splanchnic nerves. The afferent fibers supplying the kidney are derived from the 4th to 12th thoracic segments, but clinical evidence seems to indicate that they are derived mainly from the 10th, 11th and 12th thoracic segments." (Berglund, et al)

Innervation of the ureter, on the other hand, is wholly autonomic. "Ganglion cells are limited to the adventitia, none being found in submucosa muscularis. Stripping the ureter free from the pelvis to the bladder produces no disturbance of the function but if the adventitia is stripped off atony and dilatation result. Extrinsic preganglionic innervation comes from various sources, from the renal, inferior hypogastric and vaginal plexuses, from the celiac and upper sacral ganglia and also occasionally from the spermatic, aortic and inferior mesenteric plexuses." (Berglund, et al)

Reflex anuria.

The term "reflex anuria" serves to designate that group of cases in which anatomically there is one kidney at least, which in spite of the existing pathology in the other kidney or ureter, should be able to function normally. To date, there has been no study, either clinical or experimental, that has established this as a definite clinical entity clear of question. Very few, if any, of the cases reported in the literature as cases of reflex anuria are beyond question. Experimentally, there are many points of interest. Bieter, in his studies of the splanchnic control of the glomeruli in the frog, has shown that electrical stimulation of both upper and lower splanchnics caused a marked decrease in the number of functioning glomeruli, while section of the splanchnics resulted in a considerable increase in the number of functioning glomeruli. Fee has shown that section of the nerves of the renal pedicle was accompanied by an increase in the blood flow to the kidney, increased kidney volume with a result of greater urine output. A weak stimulus to the pedicle nerves increased the renal volume and urine flow while a strong stimulus resulted in a decrease in the renal volume and flow.

By pulling on the kidney, stretching muscles, and incising the skin of dogs, Loucks and Scott were able to demonstrate, in the dog, an associated cessation of urine flow. Bieter produced a 40% decrease in the number of active glomeruli in frogs by applying a bull dog clamp to the ureter. Blum has reported a case of complete anuria resulting from a procedure in which a ligature was caught in the wall of a ureter.

Then, finally, there are those cases reported, in which anuria has appeared following pyelography and also simple ureteral catheterization in which no pathology could be demonstrated at the time. One should be justly critical about negative data leading to a diagnosis of reflex anuria in these cases.

Hysteria

is occasionally referred to as being responsible for anuria in a certain definite group of cases. Cabot reported an instance in which anuria appeared in connection with an attack of hysteria. No attempt was made to explain the pathological physiology involved in the case report. In this same paper, Cabot discussed "reflex anuria" and had this to say, "Looking at the question broadly, the arguments of those who oppose the reflex theory do not seem to me to comprehend admitted fact, and, while they excite my sympathy, they do not command my respect."

Low blood pressure,

as seen in "shock" has frequently led to anuria. Since the intrapelvic pressure is regarded as nil, as long as the filtration pressure, i.e., the pressure in the glomeruli, exceeds the osmotic colloidal pressure of the blood plasma, secretion of urine

should take place. This osmotic colloidal pressure has been taken as 25 mm. Hg. Since the glomerular pressure is assumed as two-thirds that of the renal artery, urinary secretion would be arrested only if the blood pressure fell below 45 mm. Hg. The secretory renal threshold unquestionably varies from individual to individual.

Looking at this question from the standpoint of increased intraureteral pressure, Winton states that increased pressure in the ureter does not retard urine secreted in hear-lung-kidney preparations unless the pressure exceeds 10 mm. Hg. An increase in the venous pressure less than 10 mm. Hg. is also without effect on urine flow. In partial venous obstruction, an increase in the intraureteral pressure has no effect on urine flow unless the pressure exceeds the venous pressure. Similarly, in the unobstructed kidney, increase in the pressure within the ureter will be without effect unless its value exceeds that of intrarenal pressure. The urine flow was found to be abolished by an intraureteral pressure of 20 to 30 mm. Hg. This suggests that in those cases in which there is definite stasis in the urinary tract, severe hypotension, may induce anuria more easily than in the normal kidney.

Massive disturbance of circulation.

Obstructing tumors, thrombi, emboli, and occluding arteriosclerosis of the renal vessels may all be mechanically responsible for anuria.

VII. Endocrine form

Unquestionably the pituitary, thyroid, adrenal and the pancreas by means of their endocrine secretions all exert a control effect on the elimination of urine through intimate connection with water balance and also by means of direct action on the renal parenchyma. The precise check and balance of the one on the other in this connection has not been definitely worked out. Suffice it to say at present, however, that pituitary extract seems to exert a direct action on the loop of Henle causing an increase in resorption power. Adrenalin exerts its control over urinary flow by acting on the pre and post-glomerular arterioles. A high concentration of adrenalin causes diminution in renal output by constricting the afferent renal arterioles, and a dilute concentration increasing the urinary output by constricting the efferent arteries.

Allergic and Hepatic types.

In a hurried review of the literature, little can be found in reference to allergic anuria. Oliguria and occasionally anuria is said to result from pure liver damage per se, i.e., to the exclusion of renal pathology. In this regard, "hives" of the ureter may be a factor of obstruction. Hives of the bladder has been reported.

Renal oliguria.

Bauer, according to Hinman, has described an oliguria which is the counterpart of renal polyuria of diabetes insipidus. The factors responsible for this entity are doubtless to be solved with the problems of hormonal control.

Malingering form.

There is little reference in the literature to this type of anuria but it is of course likely that some "psychotic" individual might resort to such a procedure in order to obtain a desired end.

VIII. Combination of factors

Complete tolerance

(By tolerance is meant the period of time between the onset of anuria and the first appearance of symptoms of uremia.) In these cases, there is an absence of symptoms of intoxication of the central nervous system. The patient enjoys excellent health and if it were not for the symptom or findings of anuria attention would not be attracted to him in the least. In the majority of cases, this period is said to last longer than 24 hours.

Minor intolerance

Occasionally hiccough is seen. Nausea and vomiting, slight muscular twitching, drowsiness and a general feeling of muscular fatigue and weakness are the warning signs of the onset of complete intolerance.

Complete intolerance

The usual picture of uremia with convulsions makes its appearance. The remaining associated findings depend on the etiology of the existing anuria.

There is a striking difference in the clinical picture of the anuria of sudden acute onset with no previous urinary difficulty (mercurial poisoning), as contrasted with cases in which repeated bouts of retention and moderate obstruction have been noted (ureteral stones, prostatism). In the former, the picture is quite fulminating; while in the latter, clinical uremia appears quite late and the onset of chemical uremia is not nearly so rapid and is usually of the "dry" type. Mercurial poisoning is a striking example of the acute fulminating clinical picture, especially when the lethal dose has been taken. The uremic picture is rapid in its onset. On the contrary, the case subjected to repeated attacks of urinary obstruction and in which there is mechanical obstruction as in the case of bilateral stones or tumor masses involving the ureters, the patient may live for a week or more in apparent comfort. There will be a gradual and progressive rise in the blood metabolites. Usually, there is little or no edema during the early stage at least. Drowsiness seems to characterize the picture.

Diagnosis

History

The following points in the history should be investigated in detail, history of nephritis, history of poisoning, gynecological operation, unilateral or bilateral stones in the kidneys or ureters previously diagnosed, unilateral nephrectomy for infection, malignancy involving the ureters, the bladder, the prostate, uterus, marked dehydration resulting from oligodypsia or excessive loss of fluids, attacks of hysteria, and recent transfusions.

Urological examination

The patient should first be catheterized to determine definitely whether anuria exists. The abdomen should then be palpated in order to determine the presence of masses or points of tenderness. A flat plate of the abdomen is obtained to rule out the presence of ureteral or renal stones. It must be remembered that approximately 15% of urinary tract stones are not radio opaque (Eisendrath). The patient is then prepared for cystoscopic examination, having first been given 10 cubic centimeters of indigo carmine intravenously with the hope of demonstrating its appearance in the bladder at the time of examination. Each ureter is then catheterized. If urine is obtained from either or both pelves, the catheter in such instance is left in place. In the meantime, while these preparations are being made, blood chemistries consisting of carbon dioxide combining power, blood chlorides, blood sugar, and non-protein nitrogen are to be determined.

Treatment

Treatment then follows certain lines depending in a measure on the established diagnosis.

Medical:

Watson found in 205 cases collected from the literature that 110 were treated expectantly with 80 deaths (72.7%), 95 had some sort of operative procedure with 44 deaths (46.2%). Medical treatment consists for the most part in diaphoresis, diuresis, catharsis, alkalies to combat acidosis if present, venesection, and electrical stimulation applied to the kidney regions.

Surgical:

Fluids should be given in large quantities paraorally if nausea and vomiting does not prohibit and if this is a complicating factor administration should be by the parenteral

route. McCarthy, et al, have advised the passage of the duodenal tube through which fluids are forced. Glucose solutions, given intravenously, varying from 5 to 50% have been suggested, the more dilute solutions given from the standpoint of nutrition while forcing fluids and the more concentrated solutions in the lesser amounts for the diuretic effect.

Decapsulation:

Harrison published 3 cases in 1896 in which puncture of the kidney was carried out in order to release intrarenal tension. Edebohls, in 1898, reflected the capsule of the kidney and fixed it at a higher level in one case in attempt to temporarily decompress the kidney ultimately, at the same time hoping to reestablish a better blood supply to the diseased kidney. Physiologists immediately became interested in this problem and put forward the following opposition:

1. Renal arteries are terminal.
2. Blood supply from the capsule is insignificant.
3. Chronic nephritis is a local expression of a general disease and cure must remove the cause.
4. Renal tissue once destroyed does not regenerate.

Experimetnal studies have also been carried out and such reasons as the following have been offered in opposition to the procedure:

1. A new capsule is formed in a few weeks and this capsule is much denser than the normal capsule.
2. Fewer vessels could be demonstrated in the new capsule than in the old capsule.
3. Injections into the renal arteries and aorta with the renal arteries tied give the general impression that there is little connection established between the cortical arteries and those supplying the renal parenchyma.

Bowers and Trattner rationalize decapsulation as a method of treatment of anuria on the following basis:

1. Relieves congestion by permitting the kidney to expand.

2. The capsule being under marked tension constricts the vessels at the hilus of the kidney. This may be a factor in addition to nephritis in causing renal circulatory stasis. Incision and stripping of the capsule not only releases the pull on the vessels and intrarenal tension but the necessary operative manipulation, according to Lloyd, is attended by an alteration, more or less, of the blood supply.

3. In a complete stripping operation, the nervous mechanism of the kidney, particularly at the hilus, is partially or completely damaged, thus leading to increased flow of urine. This theory is based upon the fact that section of the splanchnic nerve in animals causes a polyuria. In reviewing the literature, one finds that decapsulation, while suggested clinically as being a good procedure, is seldom done.

Nephrostomy:

Nephrostomy should be considered strongly when there is an intrinsic or extrinsic ureteral cause for the anuria. Watson has suggested that in the event that there is obstruction on both sides that a bilateral nephrostomy should be done. At any rate, most investigators make an emergency procedure out of this. It is interesting to note that Bradford found in the experimental animal that the release of complete obstruction to a kidney after 10 to 40 days would allow proper return of function in due course of time. Crabtree in discussing this problem states, "It is my belief that acute obstruction even though complete, when encountered clinically, is not serious in its after effects and we may expect

the kidney to return entirely to its normal condition when obstruction is removed." After the kidneys have returned to the peak of function following nephrostomy, provided that the cause of obstruction was not relieved at the first procedure, one should turn his attention to the etiological agent responsible for the obstruction.

Nerve blocks:

Spinal anesthesia has been used with reported good results in some cases. On the basis that blocking the splanchnics has been experimentally proven to increase the renal output, this has been done with irregular results. The latter two procedures are aimed at the so-called "reflex anuria."

Impressions

1. Diagnosis	Mechanism of Obstruction	Treatment
Bilateral stones	Mechanical obstruction. Mechanical and reflex obstruction.	Removal of obstruction. Nephrostomy.
Transfusion	Mechanical blockage. Local reaction due to anaphylactic shock. Metabolic disturbance. Toxic action.	Alkalinize urine. Venesection. Repeated venesection with transfusion. General measures.
Bichloride poisoning	Tubular degeneration with reabsorption.	General measures. Decapsulation. Splanchnic or spinal anesthesia.
Dehydration	Temporary retention of fluids.	Replace fluids.
Vascular collapse "shock".	Blood pressure below secretory level.	Restore normal blood pressure.
Diabetic coma	Questionable. Tubular degeneration.	Combat acidosis.
Reflex anuria	Questionable if exists.	General measures. Spinal or splanchnic anesthesia. Ureteral catheters.
Hysteria	Questionable. Reflex anuria basis.	General measures.
Nephritis	Failing heart. General vascular constriction.	Decapsulation. General measures.

2. Hinman's classification of anuria is given.

3. A brief discussion of each type of anuria, as listed in the classification. It is given together with some of the theories in regard to the mechanism in each instance.

4. The diagnosis of reflex anuria seems justified only after exhaustive attempt has been made to rule out all other factors.

5. The diagnosis of the etiological agent depends on a thorough history supplemented by complete urological and laboratory studies, i.e. as far as possible under the circumstances.

6. The indications for and against decapsulation are offered.

7. Attention is called to the difference between the clinical picture of the fulminating and the prolonged "chronic" type of anuria. The patient is not infrequently asymptomatic for a considerable length of time and usually presents a dry uremia in the latter type.

8. Types of anuria associated with edema may be aggravated by edema of the ureter itself? -- cardiac, glomerulo nephritis, etc.

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V. CASE REPORTS

1. - 18 years of age.
Hospital No.
Admitted: 1-28-31,
Discharged: 3-8-31

The symptoms were of short duration and of such to suggest the differential diagnosis of ectopic pregnancy, acute pelvic inflammatory disease, and appendicitis. The latter could be fairly definitely ruled out as the patient stated that she had had an appendectomy previously; the right lower abdominal scar was present. A definite diagnosis as to the true nature of the condition was not established until February 5, 1931. At this time, a cystoscopy was carried out, with the diagnosis of infected hydronephrosis on the right side. Its origin was difficult to establish. At any rate, subsequent studies seemed to warrant a right nephrectomy. This was done February 13, 1931. Studies previous to the operation showed that the left kidney appeared quite normal. The postoperative course was entirely satisfactory until February 20th. The following is a summary of the progress note by the intern: on Feb. 20; "I was called at about 5 A.M. and told that the patient could not be catheterized by the nurse and had not passed more than 5 ccm. of urine during the day and yet had taken 3,000 cc. by mouth. The nurse was told to give the patient one-sixth of a grain of morphine sulphate, and then attempt catheterization one-half hour later. This was unsuccessful. At 7:30 A.M., I passed a catheter but obtained no urine. I washed out the bladder, and apparently all of the boric acid was returned. Last night, a little blood and fluid started to drain from the lower end of the nephrectomy wound. This was increased during the day and has been very abundant, causing all of the dressings to be moist and red. The patient had vomited on 3 occasions. She complained of pain over the lumbar region and spine during the night of February 19, 1931; however, there is no pain at present. Considerable tenderness on palpation over the abdominal wound. Dressings were changed during the day and became moist almost immediately. 1500 c.c. of 10% glucose were given intravenously. The stomach was lavaged with tap water and 40 grains of soda bicarbonate were left in the stomach. This, however, was promptly vomited. At 3 P.M., the patient was again catheterized, obtaining no urine. The patient was in a semicomatose state. In the afternoon, the patient reacted quite well." The blood urea nitrogen at 9:30 A.M. showed 13 mgs. %, and at 3 P.M., 19.5 mgs. %. At 10:40 P.M. the patient was taken to the cystoscopic room; a catheter was passed up the left ureter into the pelvis of the kidney. A sudden gush of urine flowed from the catheter as though there had been some obstruction to the outflow of urine from the pelvis. The catheter was left in place for a period of several days in order that drainage might be carried out. The postoperative course from this date on was entirely uneventful. The patient was discharged from the hospital on 3-8-31.

The case was discussed with Dr. Creevy. His explanation for the attack of anuria was that edema in the retroperitoneal space about the left ureter had caused obstruction of the ureter and the urinary obstruction was relieved by insertion of the catheter into the pelvis of the kidney.

2. - 52 years of age.
Hospital No.

A diagnosis of bilateral renal stone was made. Cystoscopy was done on 7-21-36. Attempt was made to catheterize both ureters. The catheter on the left was blocked in the middle third of the ureter by two large stones. The catheter on the right side passed easily into the renal pelvis. No contrast media was injected at this time. Catheterized urine from the right renal

pelvis showed grade II pus and no red blood cells. The patient was sent from the cystoscopic operating room with an appointment to return for admission to the hospital 7 days later. She returned to the hospital on 7-29-36, stating that she had voided no urine and had no desire to micturate. The patient appeared a bit sleepy, presenting some retardation of reaction but otherwise presented nothing of particular note that had previously been noted. Blood showed 36% hemoglobin, 1,709,000 red blood cells, 18,900 white blood cells, 73% neutrophils, 27% lymphocytes; N. P.N., 169.2 mgs. The patient was clearly in the stage of complete tolerance clinically. She was taken to the cystoscopic room where an attempt was made to catheterize both ureters; however, this failed. She was then taken to the operating room where an emergency nephrostomy was established in the right kidney. The chemical uremia subsided rather remarkably. On 8-1, the non-protein nitrogen was 73.2 mgs.; 8-4, 55.5 mgs.; 8-5, 42.1 mgs.; 8-6, 43.2 mgs.; 8-8, 43.8 mgs.; 8-10, 31.4 mgs.; 8-11, 28.5 mgs. There was a gradual clinical improvement. The patient was discharged from the hospital 8-19-36. She was observed closely in the Out-patient Department and readmitted to the hospital on several occasions, and is now in the hospital having had stones removed from the right renal pelvis. The nephrostomy is still intact; however, if the nephrostomy tube is clamped off, the urine flows into the bladder freely causing the patient to suffer few if any untoward symptoms. It is the impression that the left kidney is quite dead. However, if the clinical condition warrants, it is entirely possible that the left kidney might be explored at a later date.
