

cate a deep-seated process, this in itself does not justify mastoid operation in every case, for recovery has occurred in a number of cases without mastoid operation and even without myringotomy.

#### ABSTRACT OF DISCUSSION

DR. WALTER H. SNYDER, Toledo, Ohio: I have seen only one case of the condition described by the essayist. This was in a young woman who had had an acute otitis media, complicated by mastoiditis. She had been unconscious for about a

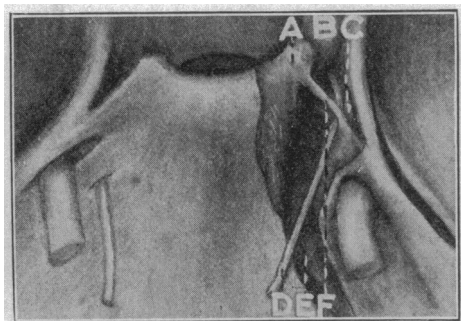


Fig. 4.—Further dissection to show sixth nerve entering cavernous sinus in relation to external wall of inferior petrosal sinus and in contact with apex of petrous pyramid. A, posterior clinoid process; B, carotid artery; C, apex of petrous pyramid; D, sixth nerve; E, inferior petrosal sinus; F, ligament.

week, or, at least, was in a deep stupor, when I first saw her. The day after her admission to the hospital she was operated on and died three days later without regaining full consciousness. The last day it was noted that she had a paralysis of the abducens on the side of the diseased mastoid. In this case the trauma of the operation did not cause the paralysis, as the tissues were very soft, no excision of the diseased bone had taken place, and it was easily removed with the hand curet, chiseling not being necessary. It is even possible that the operation did not go far enough, but the condition of the patient precluded any further investigation. We explored the tip especially and felt we had good drainage and that there was no necessity to enter the brain cavity, as apparently every part was draining and clean. Considering the anatomic relations, it is not surprising that this condition occurs; in fact, we would expect to find it much more common than it is, especially where the mastoid involvement has penetrated the inner table of the skull. It is, however, a comparatively rare condition. It is probable that many of the severe cases of mastoiditis exhibit this complication late in the disease, when the patient is moribund or lying with the eyes closed, hence is not noticed. The frequency with which this nerve is affected in basilar inflammations, especially in the syphilitic infections, would lead one to think that this complication would be more common in this rapidly spreading and especially destructive type of mastoid inflammation caused by *Streptococcus capsulatus*, of which the case I mention was an example.

DR. H. H. STARK, El Paso, Texas: I recently had a similar case in a child 4 years old. The case was in the care of a general practitioner for at least three weeks after the primary attack of sore throat, which was followed in three weeks by a discharge from the right ear. In describing the case to me the physician said that it might have been scarlet fever; that he was unable to make a diagnosis. On coming to me the child had the syndrome described, pain with paralysis of the externus on the right side, the side on which the ear was involved. The ear was discharging freely, and a roentgenogram showed involvement of the cells. The temperature at the time was 102 F. The patient was placed in the hospital, and the fever subsided in two days. The discharge continued for two weeks and then stopped. No operative procedure was done. Complete recovery occurred within two weeks after the discharge had ceased. At the time the bacteriologic examination showed a streptococcus infection. I felt that the paralysis was one of toxemia, as we had had during this period of

about six weeks several cases of infection of the same character, in which paralysis of the facial nerve occurred. One patient, a young man of 35, had a temporary hemiplegia lasting two weeks. I do not doubt Dr. Wheeler's deduction in these cases. I believe that they are due to a direct involvement of the sixth nerve from the misplaced cells.

DR. JOHN M. WHEELER, New York: If we will be on the lookout for this syndrome, we will find that this is not a rare condition. The prognosis is, on the whole, good. In some of these cases the paralysis is entirely cleared up a few days after operation; in some cases, within a few weeks or months; in some cases without operation, and in only a very few cases has the paralysis been permanent. The treatment lies entirely within the field of the ear surgeon, and the prognosis depends a great deal on his success in draining the mastoid cells.

#### ACQUIRED STRICTURE OF THE LOWER END OF THE URETER\*

ROBERT H. HERBST, M.D.

CHICAGO

Strictures of the ureter may be due to causes that are either congenital or acquired.

Acquired strictures of the ureter may be classified into those due to (1) trauma resulting from gunshot and stab wounds, postoperative conditions, labor, and passage of calculi; (2) inflammatory strictures, the result of infection. These inflammatory strictures may be classified into, (a) those that are the result of direct extension from periurethral infections, (b) those in which the infection has either ascended along the mucosa from the bladder, or has descended from the kidney, and (c) those resulting from focal infections. Strictures due to tuberculosis will not be considered in this discussion.

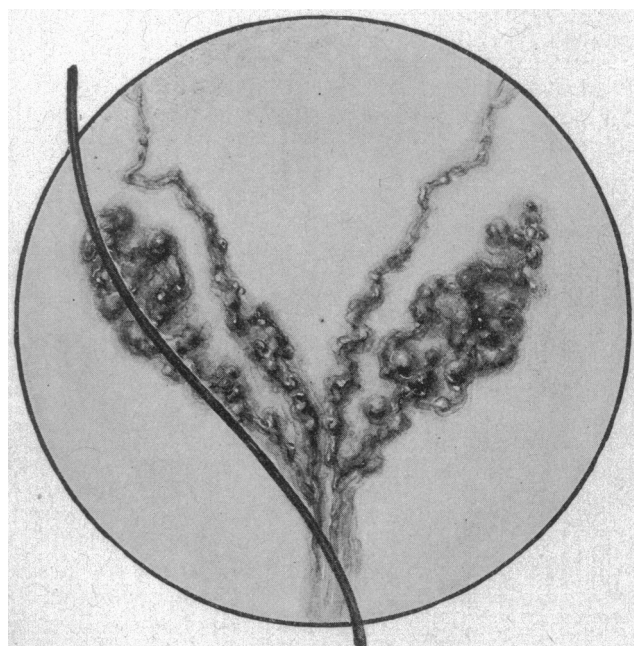


Fig. 1.—From plate made by Belfield showing relation of ureter to seminal vesicle. Vesicle filled with collargol solution. Roentgen-ray catheter in ureter.

Hunner,<sup>1</sup> in his excellent report of "One Hundred Cases of Ureteral Stricture," believes that focal infections play an important rôle in the etiology of ureteral

\* Read before the Section on Genito-Urinary Diseases at the Sixty-Ninth Annual Session of the American Medical Association, Chicago, June, 1918.

1. Hunner, G. L.: Bull. Johns Hopkins Hosp., 1918, 29, 1.

strictures. He states that he is firmly convinced that "The majority of ureteral strictures, excluding those of tuberculous origin, should be classified as simple chronic strictures and that they have their origin in an infection carried to the walls of the ureter from some distant focus, such as diseased tonsils, sinuses, teeth

acquired origin of many of them found in middle and later life. Walther<sup>5</sup> believes that stricture of the ureter is not as rare as we have been led to believe, and states that it should be remembered that this condition may be a possible cause for some of the intractable cases of renal infection failing to respond to the usual treatment.

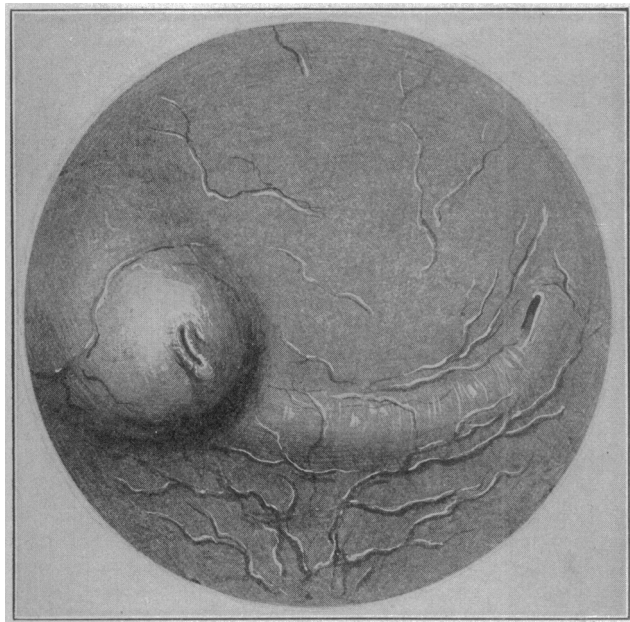


Fig. 2.—Cystoscopic view showing ureterocele of right ureter caused by stricture of lower end of ureter.

or the gastro-intestinal tract." His cases have to do entirely with women, and I can therefore understand why he omits the infection of the seminal vesicles among these foci.

Strictures resulting from infection of the seminal vesicles, either by direct extension from these organs, or possibly by organisms of focal origin, are the type to which I shall give chief consideration.

#### CONGENITAL ORIGIN OF STRICTURE

In the review of some of the earlier literature on the subject of stricture of the ureter, one is struck by the frequency of congenital origin of reported cases. In Bottomley's<sup>2</sup> extensive review we find that most of his reports are those of children, and are unquestionably due to congenital conditions. However, it is difficult to believe that some strictures, especially those showing no symptoms until middle or later life, are of congenital origin. As quoted by Moloney,<sup>3</sup> "How do you explain the delay in development in symptoms until adult life of obstruction of the ureter, when the cause is evidently congenital?" Barringer<sup>4</sup> in 1913, reporting a case of unilateral kidney calculus complicated by ureterocele of the opposite side, stated: "A ureterocele is a dilatation of the intravesical portion of the ureter. This dilatation is dependent on a narrowing or stricture of the ureteral opening. It is probable that this strictured condition is always congenital, notwithstanding that such a condition has been attributed to the passage of calculi or blood clots through a ureter." Notwithstanding these earlier opinions of congenital origin of these strictures, one finds a gradual tendency toward the belief in the

#### INFECTING ORGANISMS AS A FACTOR IN STRICTURE

The principal organisms that play the rôle of the exciting etiologic factors in the primary inflammatory stage of stricture of the lower end of the ureter are the colon bacillus, the pus coccus and the gonococcus. In the past it has been commonly believed that the gonococcus played a very small rôle in the production of strictures in this region, owing to the infrequency of infection of the bladder mucosa by this organism. However, I am satisfied that the gonococcus is responsible for no small number of strictures that occur in this location in the male, and that the preexisting infection of the ureteral wall occurs by direct extension from its closely approximated neighbor, the seminal vesicle. I shall later give briefly the history of a case which illustrates this point.

In the literature there are reported numerous cases of strictures of an inflammatory nature spreading to the ureter from adjacent organs. In 1902, Morgan<sup>6</sup> reported a case in which, after numerous operations on the urinary tract, he found the cause of the trouble to be due to an obstruction of the lower end of the ureter, caused by the binding of the ureter to the seminal vesicle, the result of an inflammatory process which had long existed in the vesicle. Belfield,<sup>7</sup> in a discussion on the subject of pus-tubes in the male,

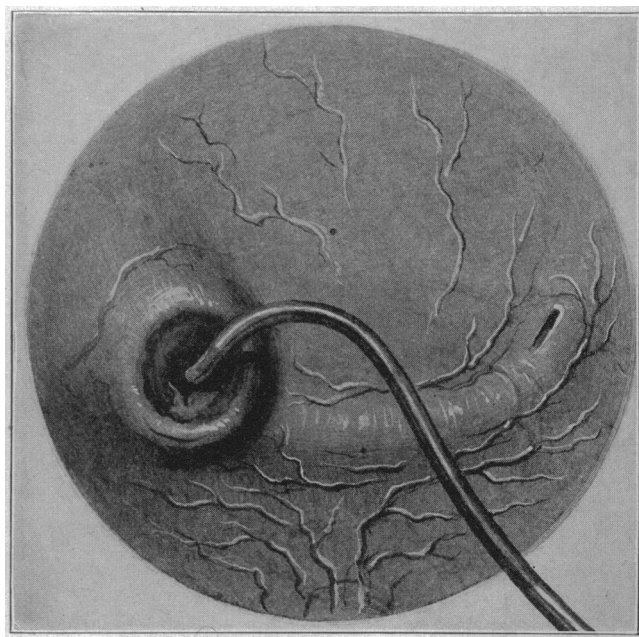


Fig. 3.—Indentation of ureterocele made by attempt to enter strictured extremity of ureter.

stated "The intimate relation between the juxtavesical ureter and the seminal duct seems, therefore, responsible for considerable kidney and ureter disease hitherto unexplored."

2. Bottomley: *Ann. Surg.*, 1910, **52**, 997.

3. Moloney: *Surg., Gynec. and Obst.*, 1917, **24**, III.

4. Barringer, B. S.: *Interstate Med. Jour.*, 1913, **20**, 343.

5. Walther: *New Orleans Med. and Surg. Jour.*, 1916, **69**, 115.

6. Morgan: *Ann. of Surg.*, 1902, p. 528.

7. Belfield, W. T.: *Tr. Am. Urol. Assn.*, 1909, **3**, 13.

McNeill<sup>8</sup> reported two cases of inflammatory strictures of the right ureter due to a pelvic abscess following ureterotomy of the left ureter. Frank,<sup>9</sup> in discussing the causes of ureteral obstruction, gave as a cause an extension of inflammation from infection of the uterus and its appendages. Again, we are all familiar with the condition found in the bladder, known as edema bullosum, commonly due to the gluing of a pus-tube to the bladder wall. If the above conditions are possible, it seems more than likely that an infection going on in the seminal vesicles may spread to the wall of the ureter.

The pathogenesis of these strictures is quite similar to that of strictures of the urethra. Let us here briefly consider the course of development of a stricture in the urethra.

First, we find the infecting organism (usually the gonococcus) destroying areas of the superficial mucosa, or what I choose to call the rubber coat of the urethra. The destruction of this mucosa is followed by infiltration of urine with its chlorid constituents. It is the chlorids which have largely to do with connective tissue proliferation and subsequent contraction of the involved area. In the development of strictures in the lower end of the ureter, we find an analogous condition, but reversed in its course. The infection spreads from the vesicle through the outer coats of the ureter, finally involving the lining mucosa with subsequent infiltration and connective tissue proliferation. For this reason some of the strictures become very dense, hard and fibrous, and take on the form of the well known impassible stricture of the urethra. The two cases reported are of this type.

#### REPORT OF CASES

CASE 1.—M. F., aged 30, gave a history of gonorrhea contracted in 1910. The urethra discharged profusely for a number of months, during which time he developed an infection of his right epididymis, for which both the right epididymis and the testicle were removed. The urethral discharge, accompanied by a severe perineal pain, continued intermittently for five years, and in 1913 an "osteopathic surgeon" made a perineal incision of some kind which left the patient with a fistula opening into the bulbous urethra.

He was admitted to my clinic in August, 1915, and, on examination, the perineal fistula mentioned in the foregoing was found, also both seminal vesicles were greatly enlarged and hard. He complained of pubic pain, and for this reason a cystoscopic examination was made which revealed a normal bladder and both ureters perfectly patent. At this time I made a perineal section and closed the opening in the urethra. He was given massage and instillations, with a hope of clearing up the vesiculitis, but after a few weeks he failed to return for treatment. Two years later, in December, 1917, he returned to the clinic, still complaining of his old condition, also of severe pain in the lower right abdomen which radiated upward toward the region of the right kidney. His vesicles were found to be in about the same condition as when he left, for the relief of which I performed a bilateral vesiculotomy. At this time a cystoscopic examination was again made, and, to my surprise, I found a well marked ureterocele on the right side of the bladder, with the orifice of the right ureter located at its summit. Numerous attempts to enter this orifice with small ureteral catheters and bougies were not successful. The roentgenogram of the entire urinary tract was negative. From these findings, the diagnosis

of acquired stricture and ureterocele of the right ureter was made. After several attempts, I succeeded in cutting the strictured orifice with a hook-shaped knife used through a Senn operating cystoscope. The small opening was further enlarged with scissors used through a Buerger operating cystoscope. The proximal end of the stricture has not been cut, but I am able to enter this with a small bougie, and expect to succeed in dilating this part of the stricture. I report this case because it illustrates that this stricture evidently developed between the two cystoscopic examinations, between which a period of two years elapsed, also that the stricture had its origin in the violent and persistent infection of the seminal vesicles.

The most common and pronounced symptom is pain, which very frequently is that of a typical renal colic, as illustrated by the following case:

CASE 2.—W. G., aged 55, single, stated that some years before he had developed a gonococcus infection which persisted for more than a year and which was accompanied by bladder symptoms; that is, frequent and imperative urination. He consulted me in September, 1913, complaining of a periodic colic-like pain in the back, which was referred to the region of the right kidney. These attacks of pain had come on about every week or two during the previous six months. They were very severe in character, requiring morphin to relieve them. I had the opportunity of seeing him in two of these attacks, and can state that they were typical of renal colic. Examination showed the prostate and seminal vesicles enlarged, the expressed specimen from which contained many pus cells. Cystoscopic examination revealed a normal bladder mucosa. In attempting to catheterize the ureters, I found a complete obstruction of the left ureter, just within the orifice. The right side was found perfectly patent. A roentgenogram of the entire urinary tract showed a small shadow which might be located near the lower end of the left ureter. After numerous unsuccessful attempts to enter the left ureter with very small ureteral bougies, a diagnosis was made of stricture of the lower end of the left ureter. Several attempts to cut the stricture through an operating cystoscope failed. A suprapubic cystotomy was then made and a small, hook-shaped ureteral knife was forced through the stricture and, by its withdrawal, the entire narrowed portion of the ureter was cut through. A small ureteral curet brought out some fine pieces of gravel which had collected at the proximal end of the stricture, and which had probably been responsible for the shadow seen in the roentgenogram. A No. 8 ureteral catheter was now readily admitted. This patient has been seen and cystoscoped a number of times during the last four years, and the stricture does not show any tendency to recur, nor has he been troubled again by the attacks of colic-like pain.

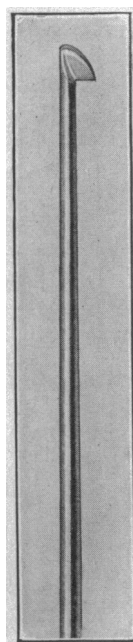


Fig. 4.—Hook-shaped knife used in cutting stricture.

#### ETIOLOGY AND TREATMENT

The spasmodic character of these attacks is likely due to complete obstruction of an already very narrow outlet of the ureter. This obstruction may be due to the admission of a pus plug, or a small piece of gravel, into the stricture area, or may be due to the same cause as is found in acute retention of the urine caused by stricture of the urethra. Under these circumstances, the pressure of the urine from behind produces an edema of a canal, already very much narrowed with resulting temporary complete obstruction. The attack promptly subsides on the passage of the obstructing foreign body, or the disappearance of the edema. The pain may be of a constant, dully aching character, referred to either side of the suprapubic region, or not infrequently radiating along the course of the ureter toward the kidney. I have seen

8. McNeill: New York Med. Jour., 1917, **106**, 786.  
9. Frank: Interstate Med. Jour., 1914, **21**, 1209.

cases of seminal vesiculitis in which the most prominent symptom was this radiating pain along the ureter, and, on catheterizing the ureter, have found what I thought at the time to be a small meatus. However, the pain disappeared after a number of dilatations of the orifice of the ureter. I think it is possible that these were cases in which there was an incipient stricture of the lower end of the ureter. I am also of the belief that in some of my cases of pyelitis which I thought I cured by pelvic lavage, the patient derived more benefit from the passage of the ureteral catheter and the dilatation of a stricture of large caliber, than from the lavage. In such cases, the urine may be perfectly negative during the attack of colic, and may show pus after its subsidence, the pus coming from the infected kidney pelvis after the stricture allows passage of urine from the affected side.

The patients may also have chills, fever, and gastrointestinal symptoms, as one observes in an acute obstruction of the ureter from other causes.

The differential diagnosis of these strictures from the symptoms is necessarily difficult. However, the cystoscope, the ureteral catheter, the olive-tipped bougie, and the roentgen ray with pyelography are absolutely essential, and will usually help one in clearing up these cases. The olive-tipped bougie is extremely valuable in the diagnosis of stricture of large caliber. When the stricture is very small, or even impassible, the ordinary ureteral catheter will clearly define the condition. In the latter type, we frequently find a decided bulging of the end of the ureter into the bladder—the condition known as ureterocele.

#### IMPORTANCE OF EARLY DIAGNOSIS

Too much importance cannot be placed on the early diagnosis of this condition, since we know that as the stricture becomes more organized, and contracts, the changes in the kidney become more serious.

Strictures of large caliber can usually be readily dilated by means of catheters, bougies and ureteral dilators. In those cases in which, on repeated attempts, we are not able to enter the strictured area, a cutting operation, either through an operating cystoscope or through a suprapubic opening, is indicated. Where this is impossible reimplantation of the ureter, or even nephrectomy, may be necessary. A hook-shaped knife, used in the two cases reported in this paper, has proved very useful in the treatment of those cases requiring a cutting operation, either through the cystoscope or through the opened bladder. To prevent subsequent contraction and recurrence, it is well to practice dilatation for some time following the cutting operation, as is commonly done in operations on the urethra. In cases in which the seminal vesicles are still the seat of infection, treatment must also be

directed to these organs, either by stripping, or by one of the operative measures, such as vasotomy or vesiculotomy.

The prognosis necessarily depends on the amount of destruction produced in the kidney. In the cases in which serious changes have occurred in this organ, especially those of long standing, little benefit may be expected from either dilatation or cutting of the strictured area. Therefore, I again call attention to the importance of an early diagnosis.

#### SUMMARY

1. Strictures of the lower end of the ureter occur more frequently than is commonly believed, and not a few of them are of the inflammatory acquired type.
2. Strictures of this part of the ureter may result from infection spreading from an adjacent seminal vesicle.
3. Strictures in this locality play an important rôle as the etiologic factor in some of the obscure infections of the kidney.
4. The importance of early diagnosis and treatment, before serious changes occur in the kidney, cannot be too strongly emphasized.

#### ABSTRACT OF DISCUSSION

DR. V. D. LESPINASSE, Chicago: This paper brings up the question of stricture of the ureter. There is no question that slight disturbances of the ureteral lumen will produce marked symptoms and marked trouble. My observation is that there is a tendency to diagnose stricture of the ureter when there is no stricture there. If the catheter goes in a centimeter or two and cannot go any farther, the inference drawn by many is that a stricture is present. This is not so. When the catheter does

not go clear to the kidney the diagnosis of ureteral stricture from this fact is not permissible. The diagnosis of stricture from the use of the catheter is quite difficult. According to Hunter's teachings, stricture of the ureter is an extremely common condition. Strictures of the ureter, particularly in the lower end of the ureter, are, at least in many cases, due to infections of the vesicles. The perivesiculitis causes a periureteritis with infiltration and loss of elasticity in the ureteral walls. Whether such a lesion should be called a stricture is doubtful, although it really is a stricture in the strict anatomic sense; but it may be simply an infiltration of the wall of the ureter, the elasticity of which is interfered with, with no real narrowing, except that in the strict sense of the term it has lost its elasticity. Of course, the treatment there is to remove the infection from the vesicle, if possible, and dilate the ureter by the use of a sound or bougie.

DR. WILLIAM F. BRAASCH, Rochester, Minn.: I question very much the possibility of making a diagnosis of stricture of the ureter by means of the ureteral catheter alone. Physiologic and anatomic changes in the ureter may give a sense of obstruction to a catheter which may lead us to believe we are dealing with a stricture. I have frequently met with such obstruction where no dilatation of the ureter above the apparent obstruction could be demonstrated in the ureterogram. We also must recognize the fact that some ureters are of much

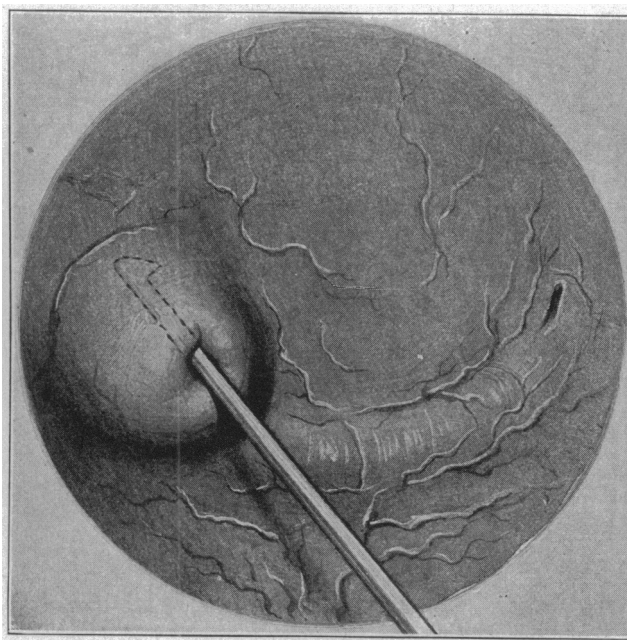


Fig. 5.—Introduction of hook-shaped knife into the strictured area.



larger caliber than others. I have frequently observed definite ureteral obstruction in normal individuals who had no abdominal pain nor any other evidence of ureteral involvement where it was impossible to introduce a No. 6 catheter up to the renal pelvis. There are so many other causes for obstruction to the ureteral catheter that it is quite impossible to make a diagnosis of stricture of the ureter because of such obstruction alone.

Although strictures of the ureter unquestionably do exist, I am certain that they do not occur as frequently as some observers would lead us to believe. I am under the impression that postmortem pathology does not bear out the assumption of the frequency of the stricture of the ureter. When found in conjunction with renal infection, it is consequent to such infection and not an etiologic factor. Actual stricture of the ureter cannot be cured by simply passing a catheter or by a single dilatation. On the contrary, it usually requires repeated dilatation at regular intervals as long as the patient lives.

DR. JOHN R. CAULK, St. Louis: There are two types of ureteral stricture, one in which there is a stricture and the other in which there is not, the latter group predominating. I am in hearty accord with what Dr. Braasch said. I have been impressed with the infrequency of true strictures of the ureter from intra-ureteral causes aside from tuberculosis and an occasional stone impaction. Strictures are not infrequently due to implications in a neighboring inflammation, such as seminal vesicle and pelvic inflammatory diseases. It has been my experience that the most important type of stricture of the ureter and the one which has caused so much confusion and, I believe, false reports, is the spasmodic stricture, or rather the spasmodic contraction of the ureter in the vicinity of an outside inflammation, particularly the seminal vesicles, and it is this condition that I wish to bring to your attention as being responsible for many of the so-called strictures. With the ureter catheter or with bougies it is impossible for any one to differentiate between them in all cases. I want to impress on you the value of large doses of atropin as a means of diagnosis between stricture and spasmodic stricture. Atropin will help the patient out of trouble very often. More attention must be paid to the tissues around the ureter and to ureteral spasm.

DR. GRANVILLE MACGOWAN, Los Angeles: Strictures occur in the ureter just as in any other tube lined with a mucous membrane, arising from influences internal or external to the ureter itself, and some are prenatal. In my experience, strictures of the ureter are usually due to pelvic troubles that produce adhesions followed by contractions in the ureter itself. Acute appendicitis sometimes produces obstructions of the ureter and causes renal colic, but that passes away along with the removal of the appendix or the subsidence of the attack. Chronic appendicitis, in which the appendix becomes bound down in the pelvis in a matted mass of inflammatory tissue, involving the ureter, sometimes will obstruct the ureter, causing Dietl's crises. I have seen two cases of this kind. There is no valid reason why stricture of the ureter should not follow the passage of a stone, although we know it does not do so usually, yet occasionally the injury must be sufficient to cause contracture especially in persons who have the tendency to keloidal degeneration.

Atropin had been used to relax these attacks. Spinal anesthesia alone will relax a patient and allow the differential diagnosis between stricture of the ureter and muscular spasm of its walls. I have three specimens of ureters which show very distinctly strictures within the bladder wall. In one of them, during life, I could pass a catheter very readily; another I never did get a catheter through. The third is merely a postmortem specimen. There can be no just contention that this disease does not exist, but it is not nearly as frequent an occurrence as urologists think it is.

DR. WILLIAM M. SPITZER, Denver: I do not know whether these strictures are true strictures or not. I have met them very frequently in cases of seminal vesiculitis. I know that I cannot pass a catheter more than 1½ c.c. That may be due to outside pressure, or it may be due to stricture, but it is very common, indeed.

DR. GUSTAV KOLISCHER, Chicago: Stricture is caused by the narrowing of the ureteral lumen, due to permanent pathologic changes in its walls. Compression is a temporary condition, due to extraneous causes. There is no doubt that such a condition will form an obstruction to the catheter. You will very rarely find true stricture of the ureter unless after infection. A simple obstruction to passing the catheter does not

prove the presence of a true stricture. It very often happens after a gynecologic operation that either on one side or the other you cannot pass a catheter, but that may be due to distortion of the ureter and not to stricture. There is only one way of proving that a stricture exists, and that is by passing an olive-tipped bougie or raying with a contrast fluid. If repeated examinations show the same result, then we have to deal with a permanent condition and can make a diagnosis of stricture.

I have always been very doubtful, just as Dr. Braasch is, as to whether it is possible to cure a ureteral stricture by inserting bougies. I cannot imagine any circumstances in which a ureteral bougie could overcome the resistance of a real ureteral stricture. If this bougie was sufficiently hard and resistant to stretch the cicatricial wall I would be

afraid to use it, because I would be afraid of perforating the wall. I believe, however, that it is possible occasionally to clear up pyelitis by regular catheterization. I agree with Dr. Braasch that we find a number of normal individuals in whom you are hardly able to enter the ureteral mouth at all, but that is not due to stricture, but to a congenital narrowing of the opening.

The rather energetic treatment recommended by Dr. Herbst in some of his cases, that is, an incision high up into the vesical wall, is rather risky. Real strictures of the ureter may occur after extensive gynecologic operations leading to extensive denudation of the ureter and followed by ureteritis, as proved by postmortems when those patients died from subsequent pyonephrosis.

DR. J. L. BOOGHER, St. Louis: I believe that these strictures indicate a pathologic change in the structure. That can take place from mere pressure; there must be a formation of fibrous tissue in the structures which is brought about by the production of cells in the tissues which cause the lessening of the blood pressure from the normal mucous membrane. The necessity on the part of nature for the production of this tissue is either because of infections or from the pressure of the mucous membranes. Without the develop-

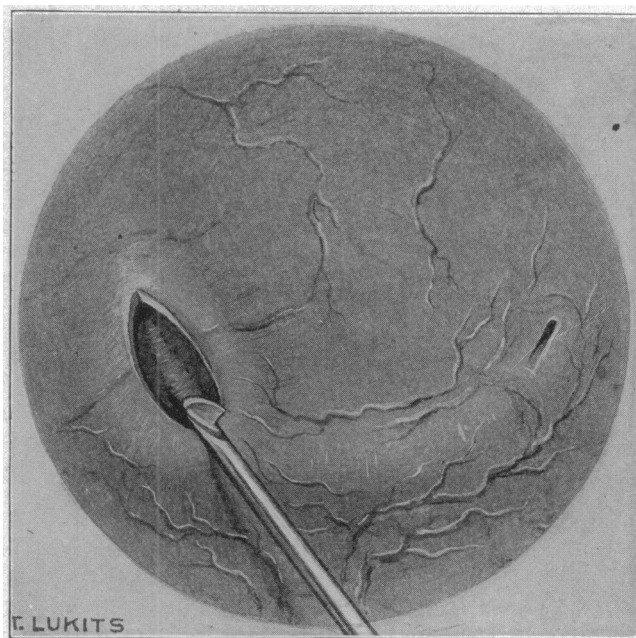


Fig. 6.—Incision of strictured area produced by withdrawal of hook-shaped knife.

ment of the spindle-shaped cell or cells of lessened blood requirements strictures cannot form. Spasmodic strictures may occur, but they cannot be called strictures; they are simply an element which produces a temporary tightening of the lumen of the vesicle; but when we have the pressure of foreign tissue brought into any sort of a channel, I do not see how the passage of any sound can change the character of that tissue. The cause of the production of the peculiar tissue which is there must be discovered and removed. I do not see why a catheter would not cause some irritation and add to the stricture more than it would detract from it.

DR. BERNARD ERDMAN, Indianapolis: With reference to Dr. Braasch's remark about the normal narrowing of the ureter, it seems to me that the meatus of the ureter bears the same relation to the ureter that the meatus of the urethra does to the urethra. It is probably the narrowest portion of the entire canal. Another point that ought to be brought out is this: All ureters do not enter the bladder in the same manner. It is sometimes a very difficult problem to pass a ureter catheter in an individual in whom I absolutely suspect no stricture at all. I would like to take issue with Dr. Boogher about the spindle cells, and I think we have a good deal of trouble with round-cell infiltration, too.

DR. J. DELLINGER BARNEY, Boston: I would like to get an expression of opinion as to the use of papaverin, mentioned by Dr. MacGowan, and others. I have tried it in several cases, and have had about as good results by simply attempting to pass a catheter.

DR. WILLIAM F. BRAASCH, Rochester, Minn.: We have used papaverin in the endeavor to remove stone from the ureter in twenty-five or thirty patients. I am under the impression that papaverin was not the greatest factor in the majority of instances in which we were successful. When a stone can be removed by cystoscopic methods, manipulation by the ureteral catheter so as to shift the stone's position is probably the most important factor. Since this is usually done when introducing papaverin, the latter has been given greater credit than it deserves.

DR. ROBERT H. HERBST, Chicago: I agree with the statement made by Dr. Braasch and Dr. Caulk, that the ureteral catheter is of very little value in the diagnosis of stricture of the ureter. There are many conditions which may cause the interruption of the catheter, both into and along the ureter, and one must be very cautious in making the diagnosis in this way without other findings. This is also true of the olive-tip bougie. When one is able to enter the narrowed portion with a catheter, the injection of a contrast fluid and the roentgen ray are of great value in making the diagnosis. However, this is not possible when the obstruction will not admit even the smallest size catheter. The two cases reported were both of the impassable type.

The cases of strictures reported in this paper were of the type which are found at the extreme lower end of the ureter and which have been preceded by severe inflammatory changes in the seminal vesicles. I believe that these obstructions are due to inflammatory changes in the wall of the ureter and not adhesions binding the ureter to the seminal vesicles. The wall of the ureter is too rigid to be compressed in this way. In opening the lower end of the ureter we must keep in mind the danger of making the incision too long. The ureter is inserted into the wall of the bladder for a distance of 2.5 c.c., and the incision must be kept within this limit.

**The Microscope.**—The first compound microscope was probably invented by the Middleburg lens grinders, Johann and Zacharias Jansen, about 1590.

## THE RATIONAL ETIOLOGY AND SATISFACTORY TREATMENT OF DACRYOCYSTITIS \*

W. R. THOMPSON, M.D.

FORT WORTH, TEXAS

The man who first suggested a lacrimal probe was thinking along scientific lines. By its use he hoped to remove the obstruction from the nasal duct, and restore its normal function. His disappointment came, however, when he learned, from repeated efforts on patients in whom the probe could be passed through the duct into the nose, that in most instances the tendency to an immediate closure was so great that the hope of draining the sac of its infectious contents had to be abandoned. The failure of the probe to give relief by drainage suggested the use of the style, which proved to be unscientific in principle and unsatisfactory in results.

For several years all contributors to this subject seem to have given up the idea of drainage or the restoration of the normal function of the lacrimal sac and nasal duct, and instead have directed their energies to sac destruction or removal, or direct drainage into the nose.

It is not my purpose to criticize the different methods of treatment. In my opinion, however, all methods depending for their success on the complete destruction of the function of the sac and duct are more or less faulty.

In the absence of adequate post-mortem information, it is impossible to determine the exact cause of nontraumatic stricture of the nasal duct. Our textbooks say that inflammation of the nasal mucous membrane and obstructions resulting from the enlargement of the inferior turbinates, nasal polypi, etc., are among the most common causes. There appear to be some very good anatomic reasons for the belief that

other and possibly more rational causes exist.

The nasal end of the nasal duct is by far the best protected of all openings entering the nasal cavity. Nature evidently realized the importance of having this duct perform its function properly at all times; to that end she displayed great wisdom in the construction and location of the inferior turbinates. The duct, as all know, enters the nasal cavity with rather an expanded orifice, immediately beneath the anterior end of the inferior turbinate. The upper border of this bone is thin and is connected to various bones along the outer wall of the nose; the lower border is free, thick, and cellular in structure. The outer surface, looking toward the opening of the duct, is concave and assists in the formation of a large and well ventilated cavity—the inferior meatus, into which the duct opens. When this bone becomes hypertrophied, the enlargement takes place on the inner or

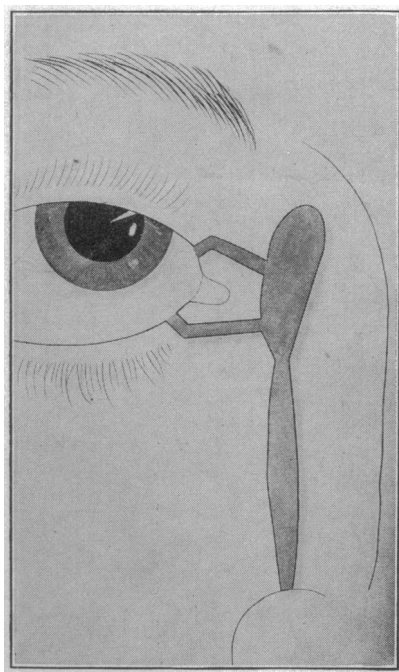


Fig. 1.—Normal sac and nasal duct.

\* Read before the Section on Ophthalmology at the Sixty-Ninth Annual Session of the American Medical Association, Chicago, June, 1918.