Pathophysiology of ureteral obstruction

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ROSE, JOHN G., AND JAY Y. GILLENNWATER. Pathophysiology of ureteral obstruction. Am. J. Physiol. 225(4): 830-837. 1973.—Forty-five experiments measuring the force of contractility within the ureteral wall, its wall tension, and the propelling force within its lumen, its intraluminal pressure, were performed on 29 dogs. The parameters of wall tension and intraluminal pressure served as an excellent measure of ureteral function in both normal and pathologically altered ureters. Our studies illustrate that ureteral peristalsis is partially impaired in obstructed but uninfected ureters; whereas, it is totally impaired when obstruction coexists with infection.

THE FOLLOWING PAPER is concerned with answering the question, how is ureteral function altered in the obstructed ureter? In order to answer this question, and thereby provide a clearer understanding of the pathophysiology of ureteral obstruction, it is necessary first to define and next to measure those parameters of ureteral function that are responsible for transporting urine.

The first of these parameters is a circumferentially directed tensile stress within the ureteral wall that has been termed ureteral wall tension. This force per unit area acts to coapt the ureteral wall and constrict the lumen, resulting in the generation of contractions. The second parameter, intraluminal ureteral pressure, is the pressure produced within the ureteral lumen that acts to propel urine distally toward the bladder.

The measurement of intraluminal ureteral pressure was originally accomplished by Trattner (16) in 1924. Since then, others (2, 4, 5, 7-9, 15) have refined his technique, which essentially consists of placing a catheter into the ureteral lumen and connecting it to a pressure transducer. The measurement of ureteral wall tension has recently been described by Rose et al. (14). It is accomplished by calculating wall tension from intraluminal pressure and ureteral wall dimensions.

By comparing these parameters of wall tension and intraluminal pressure in normal and occluded ureters, one can determine how obstruction impairs ureteral function.

MATERIALS AND METHODS

Procedure

Forty-five experiments, in which intraluminal ureteral pressure, ureteral diameter, aortic blood pressure, and respirations were monitored on a Sanborn eight-channel recorder, and in which ureteral wall tension was calculated, were performed on 29 female beagle dogs weighing between 10 and 15 kg.

Four groups of animals were studied. Group I (controls) consisted of 21 experiments on 11 animals with normal, unobstructed ureters; Group II (acute ureteral obstruction) of five experiments on five animals whose ureters were ligated with 2-0 silk while they were being monitored for the above-mentioned parameters; Group III (chronic ureteral obstruction) of 13 experiments on 7 animals that underwent unilateral ureteral occlusion 2 weeks to 4 months prior to evaluation; and Group IV (chronic ureteral obstruction and infection) of six experiments on six animals that underwent chronic unilateral ureteral occlusion plus inoculation with greater than $10^6$ organisms of E. coli type 04 above the point of occlusion prior to evaluation.

Group I (controls). Each animal was anesthetized with 6% sodium pentobarbital, 30 mg/kg, iv. A no. 16 Foley catheter was inserted into the bladder and left indwelling in order to permit continuous urine drainage throughout each experiment. Respirations were measured with a pneumograph, while aortic blood pressure was measured with a Statham transducer connected to a polyethylene catheter which was inserted into the femoral artery through a cutdown. The kidney and ureter were exposed retroperitoneally using a flank incision. Extensive care was taken not to disturb the blood supply to the ureter. A strainometer (Fig. 1) was placed around the ureter in order to measure changes in ureteral diameter. The instrument we used is a modification of one used by Peterson (10, 12, 13) to measure wall tension in blood vessels. It consists of an arclike shoe into which the ureter is placed and a delicate lever connected to a transducer, which rests on the anterior surface of the ureter and detects changes in ureteral diameter. The strainometer lever is so carefully balanced that it does not compress the ureter or alter its peristaltic activity. Intraluminal ureteral pressure was measured with a Statham transducer connected to a no. 23-gauge polyethylene catheter introduced into the ureteral lumen distal to the strainometer (Fig. 2). The tip of the ureteral catheter was placed exactly beneath the proximal margin of the strainometer lever.

Preceding and following each recording with the strainometer, it was calibrated with steel cylinders of a known diameter. By using simple extrapolations, one could determine the outside ureteral diameter at any given moment during the experiment.

At the conclusion of each experiment, ureteral urine cultures were obtained, and the segment of ureter being
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FIG. 1. Illustration of strainometer used to monitor continuously changes in outside ureteral diameter that occur during ureteral peristalsis. Arrows point to transducer that is connected to strainometer lever. Lever is shown resting on arclike shoe into which ureter is placed. As ureter alters its diameter, lever is deflected. Type and amplitude of deflection are detected by transducer and recorded.

FIG. 2. Diagram illustrating placement of strainometer around ureter and position of ureteral pressure catheter.

studied was clamped between two hemostats welded together at a fixed distance (Fig. 3) in order to minimize distortion and shrinkage of the specimen. The specimen was then placed in Formalin for 3 days prior to the preparation of slides of ureteral cross sections. The slides were examined under a calibrated dissecting microscope, and ureteral wall thickness and luminal diameter were measured in different sagittal and coronal planes.

Ureteral wall tension was calculated using a standard formula, which will be discussed below.

Group II (acute ureteral obstruction). Each animal was anesthetized with sodium pentobarbital and a Foley catheter was inserted into its bladder. Respirations and aortic blood pressure were monitored. Using a flank approach, the kidney and ureter were exposed retroperitoneally. A strainometer was placed around the ureter to record changes in outside ureteral diameter, and a catheter was inserted into the ureteral lumen to detect intraluminal pressure. A 2-0 silk ligature was placed loosely around the lower ureter, distal to the strainometer and the ureteral pressure catheter. Following a control recording of all parameters, the ligature around the ureter was gently tied, occluding the ureteral lumen. The parameters of intraluminal pressure, ureteral diameter, aortic blood pressure, and respirations were then monitored continuously for 2-3 hr.

At the conclusion of each experiment, urine cultures and tissue specimens were obtained, and ureteral wall tension was calculated.

Group III (chronic ureteral obstruction). Each animal underwent total, unilateral ureteral occlusion via a midline suprapubic approach. The ureters were ligated 3–4 cm proximal to the vesico-ureteral junction. The duration of obstruction ranged from 2 weeks to 4 months.

Following a predetermined period of obstruction, each animal was anesthetized and a Foley catheter was inserted into its bladder. Respirations and aortic blood pressure were monitored. The obstructed ureter was exposed retroperitoneally through a flank incision. Recordings of intraluminal ureteral pressure and the changes in ureteral diameter were made. Urine cultures as well as tissue specimens of the obstructed ureters were obtained, and ureteral tensions were calculated.

Group IV (chronic ureteral obstruction and infection). Each animal underwent unilateral ureteral occlusion via a midline suprapubic approach. Following 10–14 days of ureteral obstruction, the obstructed ureter was surgically exposed via a midline incision. Using a no. 27-gauge needle connected to a 2.5-ml syringe, greater than 10^6 organisms of E. coli 04 were inoculated into the obstructed ureteral lumen proximal to the point of obstruction. Leakage of E. coli from the ureteral lumen was prevented by holding the site of inoculation with a gauze pad for 10 min. The midline incision was then closed.

At intervals of 3 days to 1 week following inoculation with E. coli, both the obstructed and infected ureter and its normal contralateral mate were studied by recording...
FIG. 4. A: diagram illustrating inside ureteral radius, a, and outside ureteral radius, b. B: diagram illustrating circumferentially directed ureteral wall tension and radially directed intraluminal ureteral pressure.

I: ureteral pressure, aortic blood pressure, respirations, and ureteral diameter, and by calculating ureteral wall tension. At the conclusion of each experiment, bilateral ureteral urine cultures and histologic specimens were obtained.

Calculations

A more detailed discussion of the calculation of ureteral wall tension has been previously reported (14). Presently, a brief resume is offered.

Ureteral wall tension was calculated using the following formula as derived by Peterson and Attinger (1, 12, 13)

\[ T = \frac{P_i(a^2 + b^2)}{(b^2 - a^2)} \]

where \( T \) equals wall tension at the mucosal surface, \( P_i \) equals intraluminal pressure, b equals outside radius, and a equals luminal radius (Fig. 4).

In our experiments, intraluminal pressure, \( P_i \), is measured directly from a transducer connected to a catheter that is inserted into the ureteral lumen. The outside ureteral radius, b, is one-half the outside ureteral diameter that is measured by the strainometer. The luminal radius, a, is equal to the outside radius minus the ureteral wall thickness.¹

RESULTS

Group I—Normal Ureteral Peristalsis

The strainometer continually monitored the changes in ureteral diameter that occurred with normal peristalsis. Figure 5 illustrates the downward deflections in the strainometer tracing that were recorded during ureteral contractions. These corresponded to the decreases in outside ureteral diameter that resulted during contractions as the ureteral walls apposed. The small upward deflections that immediately preceded the contractile waves in the strainometer tracing were due to the increases in outside ureteral diameter that resulted from distention of the ureteral wall by urine boluses as they were propelled down the ureter.

Associated with each contraction, there was a corresponding ureteral pressure wave. When the ureter was not contracting, the ureteral pressure remained at base-line levels. However, when the ureter contracted, the ureteral pressure rose to peak levels. Figure 5 illustrates the relationship in time between the ureteral pressure wave, the urine bolus, and the ureteral contraction. It can be seen that the onset of the ureteral pressure wave was detected just as the bolus passed down the ureter, while the peak of the ureteral pressure wave was detected as the ureter was contracting.

The strainometer recordings and the ureteral pressure recordings demonstrated that ureteral contractions are smooth, regular waves that occur at a slightly irregular frequency. This frequency ranged between 3 and 10 contractions/min and averaged 6.7 contractions/min.

With each contraction, there was an increase in ureteral wall tension, which returned to base-line levels during periods of ureteral rest (Fig. 5). The mean increase in ureteral wall tension associated with contractions was \( 2.09 \times 10^4 \text{ dynes/cm}^2 \). This increase above base-line tension is statistically significant (\( T_{1, 40} = 6.28, P < 0.0001 \)), using the Student t test.

The mean base-line ureteral wall tension was \( 1.27 \times 10^4 \text{ dynes/cm}^2 \), the range being \( 0.44 \times 10^4 \text{ dynes/cm}^2 \) to \( 2.76 \times 10^4 \text{ dynes/cm}^2 \). The mean peak ureteral tension was \( 3.35 \times 10^4 \text{ dynes/cm}^2 \), whereas the range of peak ureteral tensions was \( 1.36 \times 10^4 \text{ dynes/cm}^2 \) to \( 6.29 \times 10^4 \text{ dynes/cm}^2 \) (Fig. 6).

The increase in ureteral pressure associated with contractions, i.e., the amplitude of the pressure wave, occurred when wall tension reached peak levels and averaged 14.8

¹ This determination of luminal radius is an oversimplification. A more accurate and detailed determination is to be found in ref. 14.
mm Hg. This increase is statistically significant ($T_1, 40 = 6.75, P < 0.0001$), using the Student $t$ test. The amplitude of the pressure wave ranged from 6 to 35 mm Hg. Base-line ureteral pressure averaged 8.2 mm Hg and ranged between 3 and 18 mm Hg. Peak ureteral pressure averaged 23.0 mm Hg and ranged between 10 and 45 mm Hg (Fig. 6). Urine cultures in the unobstructed ureters revealed no growth.

**Group II—Acute Ureteral Obstruction**

In the initial minutes following acute ureteral occlusion, there was an increase in the frequency of ureteral contractions, an increase in the amplitude of pressure waves associated with contractions, and an increase in the amount of deflection (mm ureteral diameter) representative of contractions in the strainometer recording (Fig. 7). The rate of ureteral contractions usually increased significantly within 3 min of ureteral obstruction. Associated with this were marked increases in peak ureteral wall tension. Base-line ureteral wall tension also increased; however, these increases were proportionately less than increases in peak tension. Therefore, the change in ureteral tension associated with contractions, i.e., the peak minus the base-line tension, was significantly increased. There were analogous increases in peak intraluminal ureteral pressure which were significantly greater than the increases in base-line intraluminal ureteral pressure. Hence, within the initial minutes of ureteral occlusion, the amplitude of the ureteral pressure waves accompanying ureteral contractions was increased (Fig. 8).

After 5-20 min of ureteral obstruction, base-line ureteral wall tension became elevated and approached peak values, whereas peak tensions changed less dramatically (Fig. 8). Therefore, the changes in ureteral wall tension associated with contractions diminished. Similarly, base-line ureteral pressures increased disproportionately greater than peak ureteral pressures, thereby decreasing the amplitude of ureteral pressure waves. The frequency of ureteral contractions continued to remain elevated. However, contractions became more difficult to detect, since the amount of deflec-

![Fig. 6. Upper graph: mean base line and peak ureteral wall tensions ± S.E. found in 21 experiments on normal ureters, 13 experiments on chronically obstructed ureters, and 6 experiments on chronically obstructed and infected ureters. Lower graph: mean base line and peak intraluminal ureteral pressures ± S.E. found in 21 experiments on normal ureters, 13 experiments on chronically obstructed ureters, and 6 experiments on chronically obstructed and infected ureters.](http://ajplegacy.physiology.org/)

![Fig. 7. Recording of aortic blood pressure, intraluminal ureteral pressure, outside ureteral diameter, and respirations in an acutely obstructed ureter.](http://ajplegacy.physiology.org/)
tion representing contractions in the strainometer recording and the amplitude of the ureteral pressure waves in the ureteral pressure recording both diminished (Fig. 7).

As ureteral obstruction continued (it usually was monitored for 2 hr), the ureteral pressure recording became a flat line in all five experiments, whereas the strainometer recording became a flat line in three of five experiments (Fig. 7). In the remaining two experiments (subjects 5001 and 5003), only very slight deflections could be detected in the strainometer recording.

Within 30 min to 3 hr following ureteral occlusion, depending on the individual subject, base-line and peak ureteral wall tension continued to increase proportionately, there being essentially no significant difference between the two. Likewise, ureteral pressure became a flat line, so that base-line and peak values became equal, while the amplitude of pressure associated with contractions became zero (Fig. 8). Urine cultures in acutely obstructed ureters revealed no growth.

**Group III—Chronic Ureteral Obstruction**

Ureteral occlusion was carried out for periods of 2 weeks to 4 months. Following these periods of chronic obstruction, ureteral diameter increased as expected. Each of these dilated ureters exhibited contractions that were visually observed as well as detected in the strainometer recordings. However, these contractile waves were irregular in frequency and amplitude when compared to those of normal ureters. In addition, the contractile waves in chronically obstructed ureters were not preceded by the upward deflections in the strainometer recording that are characteristic of urine boluses formed in normal ureters (Figs. 5 and 9).

Base-line and peak ureteral wall tensions were increased in chronic ureteral obstruction. Base-line tension ranged from $3.24 \times 10^4$ dynes/cm² to $22.6 \times 10^4$ dynes/cm², the mean being $10.1 \times 10^4$ dynes/cm². Peak ureteral tension ranged from $3.73 \times 10^4$ dynes/cm² to $27.0 \times 10^4$ dynes/cm², the mean being $12.4 \times 10^4$ dynes/cm² (Fig. 6). The changes in ureteral wall tension associated with contractions, i.e., the elevations above the base-line tension, were not statistically significant in chronically obstructed ureters ($T_1, 24 = 0.86, P = 0.40$), whereas they were in control ureters ($T_1, 40 = 6.28, P = 0.0001$), as computed by the Student $t$ test.

After chronic ureteral obstruction, base-line ureteral pressure was within the range of control values obtained for unobstructed ureters. It was 7–15 mm Hg. The mean base-line pressure was 10.1 mm Hg for chronically obstructed ureters. Peak ureteral pressures were within the normal range following chronic ureteral obstruction; however, their mean values were less than those of unobstructed ureters. The range was 11–44 mm Hg. The mean peak pressure was 17.9 mm Hg (Fig. 6).

Ureteral pressure waves, i.e., the elevations above base-line intraluminal pressure that are associated with contractions in normal unobstructed ureters, were not always associated with contractions in chronically obstructed ureters (Fig. 9). When ureteral pressure waves were associated with contractions in chronically obstructed ureters, their amplitude was reduced below control values. The amplitude of
pressure waves ranged from 3 to 11 mm Hg in chronically obstructed ureters. The mean amplitude was 5.6 mm Hg in obstructed ureters (Fig. 6).

The frequency of ureteral contractions was not altered following chronic ureteral obstruction. It ranged from 2 to 10 contractions/min and averaged 7.2 contractions/min. Urine cultures in chronically obstructed ureters revealed no growth.

**Group IV—Chronic Ureteral Obstruction and Infection**

In ureters that were chronically obstructed and infected (urine cultures showed *E. coli* greater than $10 \times 10^8$ organisms/ml), there was an absence of ureteral contractions in each strainometer recording (Fig. 10). These ureters were unable to increase their wall tensions and their intraluminal pressures above baseline levels (Fig. 6).

Ureteral wall tension ranged from $1.91 \times 10^4$ dynes/cm$^2$ to $18.1 \times 10^4$ dynes/cm$^2$ with a mean of $6.89 \times 10^4$ dynes/cm$^2$. Intraluminal ureteral pressure ranged from 8 to 25 mm Hg with a mean of 16 mm Hg.

The contralateral ureters of these animals exhibited normal ureteral peristalsis. Ureteral contractions were readily detectable, and ureteral tension and pressure were within normal limits. Urine cultures of the contralateral ureters revealed no growth.

**DISCUSSION**

An understanding of the urodynamics in the normal ureter is an essential prerequisite to any attempt at explaining the pathophysiology of ureteral obstruction. Therefore, it seems appropriate to begin this discussion with an analysis of ureteral function in the unobstructed ureter.

It has been demonstrated that the normal ureter functions by forming urine into elongated boluses and establishing increases in intraluminal pressure behind these boluses to aid in their distal transport (2–5, 7–9, 11, 15, 16). It was assumed that the inherent contractile properties of the ureter were responsible for initiating this process; however, definitive experimental evidence to support this assumption was lacking. Our experiments, by measuring the contractile force within the ureteral wall and its relationship to the generation of contractions, the formation of ureteral urine into boluses, and the establishment of increases in intraluminal pressure, provide proof for this assumption and add a new dimension to the understanding of ureteral urine transport.

The basis for this understanding resides in the fact that the contractile force within the ureteral wall, its wall tension, is responsible for maintaining the characteristic states of relaxation and contraction that are essential to the transport of ureteral urine. Our data bear this out by showing that when ureteral wall tension is maintained at baseline levels, the ureter remains in a state of rest. During these periods of ureteral rest, intraluminal radius is maintained at a length that causes intraluminal pressure to be maintained at baseline levels. When wall tension increases to peak levels, this increase in circumferentially directed force causes the ureteral walls to coapt and the lumen to constrict, resulting in the generation of contractions. The luminal constriction associated with these contractions forms ureteral urine into boluses and decreases intraluminal radius, causing a corresponding increase in intraluminal pressure. The sequence of these events can be viewed on one of our standard recordings (Fig. 5). It can be seen that urine boluses are formed just ahead of the contracting segments of ureter in which intraluminal pressure is built up to peak levels. This temporal arrangement of urine boluses and pressure waves results in the distal propulsion of the boluses down the ureter.

It may therefore be concluded that the normal mechanics of ureteral urine transport are primarily dependent on the ability of the ureter to increase its contractile force above baseline levels in order to bring about the generation of contractions, the formation of ureteral urine into boluses, and the establishment of increases in pressure behind these boluses.

By analyzing the parameters of ureteral wall tension, intraluminal ureteral radius, and intraluminal ureteral pressure simultaneously during acute ureteral occlusion,
it is possible to gain new insights into the pathophysiology of acute ureteral obstruction in terms of the forces generated within the ureteral wall and lumen. The relationship between these parameters is expressed by equation 1. It illustrates that wall tension is completely related to intraluminal radius and pressure. The interrelationship of these three parameters upon one another, however, may change, depending on varying physiologic conditions. For example, during normal ureteral peristalsis, the parameter of wall tension will influence directly the intraluminal radius and pressure. For, as wall tension is increased and the ureter contracts, intraluminal radius is decreased and intraluminal pressure is increased. Thus, in this instance, radius and pressure are primarily determined by tension. In other physiologic situations, the reverse may occur, i.e., ureteral wall tension may be determined by intraluminal pressure and radius. For example, when obstruction to ureteral urine flow exists in the presence of continued renal output, as it does in acute ureteral occlusion, the pressure within the ureteral lumen builds up. Correspondingly, this increase in pressure distends the ureteral lumen and increases intraluminal radius. As equation 1 illustrates, simultaneous increases in intraluminal pressure and radius must be accompanied by increases in wall tension. Thus, in this instance, namely, acute ureteral obstruction, ureteral wall tension can be primarily determined by intraluminal pressure and radius.

Our data demonstrate that the increases in wall tension that result from increases in intraluminal pressure, secondary to obstruction, differ from the increases in wall tension that are primarily responsible for generating contractions. The former increases are increases in base-line wall tension, whereas the latter are increases above base-line wall tension. By distinguishing between these two types of elevations in ureteral wall tension and by applying this distinction to the study of acute obstruction, it is possible to determine the types of tensile force that the ureter is generating during the various stages of acute obstruction and the relationship of these forces to overall ureteral function.

In the first few minutes following acute ureteral occlusion, both types of increases in ureteral wall tension occur, i.e., base line and peak. However, the peak elevations, i.e., the increases above base-line levels, are markedly increased in comparison to base-line elevations. These large increases above base-line levels represent the amount of ureteral force that is being applied to the generation of contractions. As expected, these forceful contractions are capable of producing marked elevations in the amplitude of intraluminal ureteral pressure waves. The slight increases in base-line wall tension that occur within the initial minutes following ureteral obstruction represent the force that the ureteral wall must generate, even during periods of rest, to counteract the increase in base-line intraluminal pressure that is built up behind an obstruction. During the first 1–3 min of obstruction, the contractile force clearly exceeds the counteracting force.

Anywhere from 5–20 min following ureteral occlusion, the base-line pressure that is built up within the ureteral lumen secondary to obstruction becomes significantly elevated. Correspondingly, the base line tension that the ureteral wall generates to counter this distending pressure is also proportionately increased. On the other hand, the contractile force that the ureteral wall generates, i.e., its increase above base-line tension, is markedly diminished when compared to that generated in the initial minutes following ureteral obstruction. These meager increases in contractile force are only capable of producing slight increases in intraluminal pressure above base-line levels.

As ureteral occlusion continues beyond 2 hr, the ureter utilizes even greater amounts of its force to counteract the building pressure within its lumen, until all measurable force within the ureteral wall is exerted to maintain ureteral tone against the radially directed pressure within the lumen. At this point, the ureter is unable to increase its wall tension above base-line levels and is therefore unable to generate contractions.

Thus, it seems that during the initial minutes of acute ureteral obstruction, the ureter generates forceful contractions by increasing the tensile force within its walls above base-line levels. However, as acute obstruction progresses, the ureter must utilize the greater portion of its tensile force to maintain its wall tone against the increased pressure building within its lumen, leaving little, and eventually no force, for the generation of contractions.

As ureteral occlusion is continued for weeks, the pressure that is built up within the ureteral lumen secondary to acute obstruction continues to exert its radially directed force on the ureteral walls, until the walls no longer maintain their tone. At this point, the ureteral walls begin to decompensate and are distended. Decompensation continues, and ureteral luminal radius and length are increased, until the markedly dilated and tortuous ureters that are the hallmark of chronic ureteral obstruction result.

Since ureteral luminal radius and length are increased in chronically obstructed ureters, the luminal volume over which urine is distributed is also increased. This increase in volume brings about a decrease in base-line intraluminal pressure from the high levels that were built up after acute ureteral occlusion to the essentially normal levels that exist following chronic occlusion. Simultaneously accounting for this reduction in base-line intraluminal pressure is the reduction in ipsilateral renal blood flow that occurs following chronic ureteral occlusion (17). The decrease in renal blood flow to the obstructed kidney brings about decreases in glomerular filtration, renal output, and intratubular hydrostatic pressure (6), all of which serve to lower base-line intraluminal pressure.

It is to be expected that the decreases in intraluminal pressure that result from chronic obstruction are decreases in base-line pressures; for the conditions no longer exist as they did in acute obstruction, where urine was being formed into ureters whose walls resisted extensive dilatation by maintaining their tone. Once the ureteral walls decompensate, as they do in chronic obstruction, the ureter reaches a new equilibrium whereby it maintains normal base-line intraluminal pressures at the expense of increasing its radius and length. During this new equilibrium, base-line wall tension is elevated, for it follows that if base-line pressure is normal and ureteral radius is increased, base-line tension must increase (equation 1). Indeed, this is the case as confirmed by our data.

The function of a ureter whose base-line wall tension is
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elevated secondary to dilatation but whose base-line intraluminal pressure is normal, i.e., a chronically obstructed ureter, is entirely different from a ureter that has been acutely obstructed for periods of a few hours. With chronic obstruction, the ureter is able to increase its wall tension above base-line levels and is therefore able to generate contractions. However, the force of these contractions is markedly reduced when compared to contractions in normal, unobstructed ureters. Furthermore, in contrast to normal ureters, not all contractions in chronically obstructed ureters are capable of producing simultaneous increases in intraluminal pressure above base-line levels. When they do produce increases in intraluminal pressure above base-line levels, the magnitude of these increases is greatly reduced when compared to normal ureters.

Thus, it seems that chronically obstructed ureters are unable to generate contractions that are forceful enough to coapt their walls effectively. Therefore, they are unable to produce significant driving pressures within their lumens, so that their ability to transport urine is somewhat reduced.

When chronic ureteral obstruction is complicated by severe infection, the ureter is totally unable to increase its wall tension above base-line levels and is therefore unable to generate contractions. Chronically obstructed and infected ureters are also totally unable to increase their intraluminal pressures above base-line levels, probably because they are unable to coapt their walls. Therefore, they are unable to develop any driving pressure within their lumens to propel urine distally. Needless to say, ureteral urine transport is severely impaired in these ureters.

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