Chapter 12. Urinary Obstruction & Stasis

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Urinary Obstruction & Stasis: Introduction

Because of their potential damage on renal function, urinary obstruction and stasis are important urologic disorders. Ureteral obstruction leads to hydronephrosis, kidney atrophy that may terminate in renal failure. Furthermore, obstruction often is complicated by infection, which causes additional damage to the organs involved.

Classification

Obstruction may be classified according to cause (congenital or acquired), duration (acute or chronic), degree (partial or complete), and level (upper or lower urinary tract).

Etiology

Congenital anomalies, more common in the urinary tract than in any other organ system, are generally obstructive. In adult life, many types of acquired obstructions can occur.

Congenital

The common sites of congenital narrowing are the external meatus in boys (meatal stenosis) or just inside the external urinary meatus in girls, the distal urethra (stenosis), posterior urethral valves, ectopic ureters, ureteroceles, and the ureterovesical and ureteropelvic junctions (Beganovic´ et al, 2007; Tan and Smith, 2004). Another congenital cause of urinary stasis is damage to sacral roots 2–4 as seen in spina bifida and myelomeningocele. Vesicoureteral reflux causes both vesical and renal stasis (see Chapter 13).

Acquired

Acquired obstructions are numerous and may be primary in the urinary tract or secondary to retroperitoneal lesions that invade or compress the urinary passages. Among the common causes are (1) urethral stricture secondary to infection or injury; (2) benign prostatic hyperplasia or cancer of the prostate; (3) vesical tumor involving the bladder neck or one or both ureteral orifices; (4) local extension of cancer of the prostate or cervix into the base of the bladder, occluding the ureters; (5) compression of the ureters at the pelvic brim by metastatic nodes from cancer of the prostate or cervix; (6) ureteral stone; (7) retroperitoneal fibrosis or malignant tumor; and (8) pregnancy.

Neurogenic dysfunction affects principally the bladder. The upper tracts are damaged secondarily by ureterovesical obstruction or reflux and, often, by complicating infection. Severe constipation, especially in children, can cause bilateral hydroureteronephrosis from compression of the lower ureters.

Elongation and kinking of the ureter secondary to vesicoureteral reflux commonly lead to ureteropelvic obstruction and hydronephrosis. Unless a voiding cystourethrogram is obtained in children with this lesion, the primary cause may be missed and improper treatment given.

Pathogenesis and Pathology

Obstruction and neuropathic vesical dysfunction have the same effects on the urinary tract. These changes can best be understood by considering the effects of (1) a severe meatal stricture on the lower tract (distal to the bladder neck), (2) a large obstructing prostate on the midtract (bladder), and (3)

an impacted stone in the ureter on the upper tract (ureter and kidney).

Lower Tract (eg, Urethral Stricture)

Hydrostatic pressure proximal to the obstruction causes dilation of the urethra. The wall of the urethra may become thin, and a diverticulum may form. If the urine becomes infected, urinary extravasation may occur, and periurethral abscess can result. The prostatic ducts may become widely dilated.

Midtract (eg, Prostatic Hyperplasia)

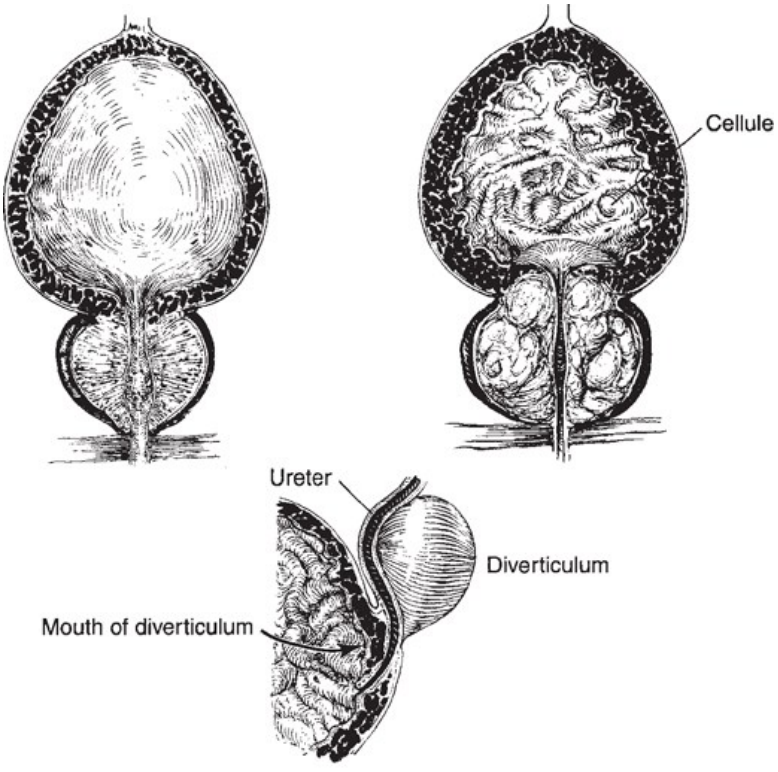
In the earlier stages (compensatory phase), the muscle wall of the bladder becomes hypertrophied and thickened. With decompensation, it becomes less contractile and, therefore, weakened (Lieber et al, 2010).

Stage of Compensation

To balance the increasing outlet resistance, the bladder musculature hypertrophies. Its thickness may double or triple. Complete emptying of the bladder is thus made possible.

Hypertrophied muscle may be seen endoscopically. With secondary infection, the effects of infection are often superimposed. There may be edema of the submucosa, which may be infiltrated with plasma cells, lymphocytes, and polymorphonuclear cells. At cystoscopy, surgery, or autopsy, the following evidence of this compensation may be visible (Figure 12–1):

Figure 12–1.



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Changes in the bladder developing from obstruction. ***Upper left:*** Normal bladder and prostate. ***Upper right:*** Obstructing prostate causing trabeculation, cellule formation, and hypertrophy of the interureteric ridge. ***Bottom:*** Marked trabeculation (hypertrophy) of the vesical musculature; diverticulum displacing left ureter. (Redrawn and modified, with permission, from Tanagho EA, Pugh RCB: The anatomy and function of the ureterovesical junction. Br J Urol 1963;35:151.)

Trabeculation of the Bladder Wall

The wall of the distended bladder is normally quite smooth. With hypertrophy, muscle bundles with deposit of interstitial collage fibers become taut and give a coarsely interwoven appearance to the mucosal surface commonly described as trabeculation. The trigonal muscle and the interureteric ridge, which normally are only slightly raised above the surrounding tissues, respond to obstruction by hypertrophy of their smooth musculature. The ridge then becomes prominent. This trigonal hypertrophy causes increased resistance to urine flow in the intravesical ureteral segments. It is this mechanism that causes relative functional obstruction of the ureterovesical junctions, leading to back pressure on the kidney and hydroureteronephrosis. The obstruction increases in the presence of significant residual urine, which further stretches the ureterotrigonal complex (Tanagho and Meyers, 1965). (A urethral catheter relieves the obstruction somewhat by eliminating the trigonal stretch. Definitive prostatectomy leads to permanent release of stretch and gradual softening of trigonal hypertrophy with relief of the obstruction.)

Cellules

Normal intravesical pressure is about 30 cm of water at the beginning of micturition. Pressures two to four times as great may be reached by the trabeculated (hypertrophied) bladder in its attempt to force urine past the obstruction. This pressure tends to push mucosa between the superficial muscle bundles, causing the formation of small pockets, or cellules (Figure 12–1).

Diverticula

If cellules force their way entirely through the musculature of the bladder wall, they become saccules, then actual diverticula, which may be embedded in perivesical fat or covered by peritoneum, depending on their location. Diverticula have no muscle wall and are therefore unable to expel their contents into the bladder efficiently even after the primary obstruction has been removed. When secondary infection occurs, it is difficult to eradicate; surgical removal of the diverticula may be required. If a diverticulum pushes through the bladder wall on the anterior surface of the ureter, the ureterovesical junction will become incompetent (see Chapter 13).

Mucosa

In the presence of acute infection, the mucosa may be reddened and edematous. This may lead to temporary vesicoureteral reflux in the presence of a “borderline” junction. The chronically inflamed membrane may be thinned and pale. In the absence of infection, the mucosa appears normal.

Stage of Decompensation

The compensatory power of the bladder musculature varies greatly. One patient with prostatic enlargement may have only mild symptoms of prostatism but a large obstructing gland that can be palpated rectally and observed cystoscopically; another may suffer acute retention and yet have a gland of normal size on rectal palpation and what appears to be only a mild obstruction cystoscopically.

In the face of progressive outlet obstruction, possibly aggravated by prostatic infection with edema or by congestion, decompensation of the detrusor may occur, resulting in the presence of large amount of residual urine after voiding. The amount may range up to 500 mL or more.

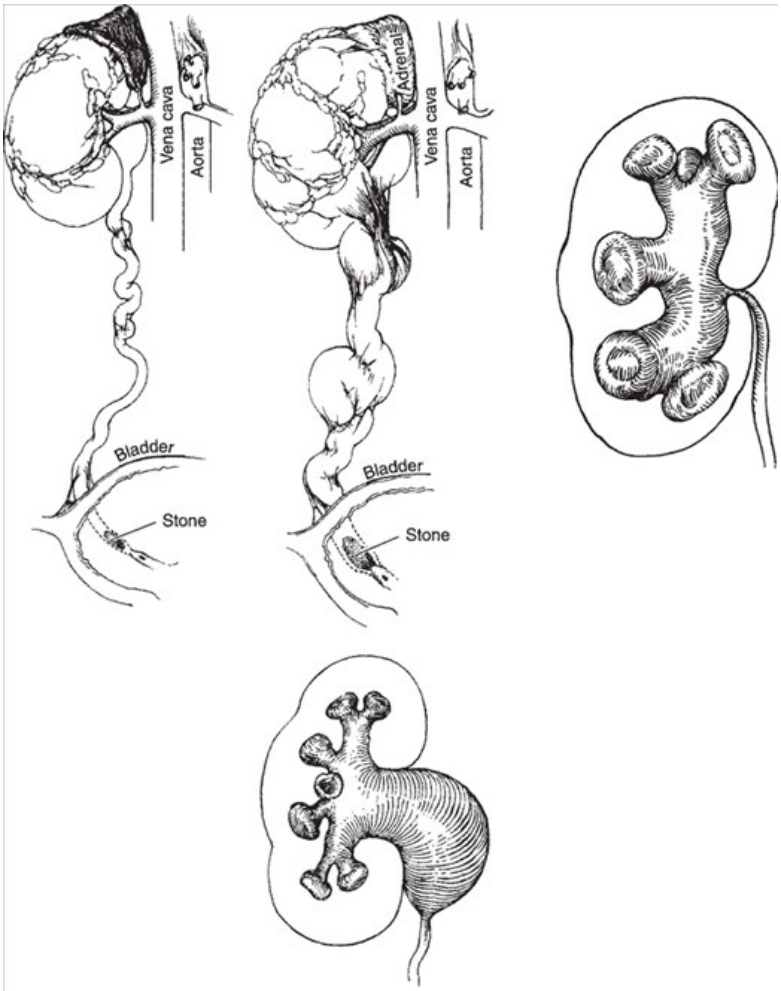
Upper Tract

Ureter

In the early stages of obstruction, intravesical pressure is normal while the bladder fills and is increased only during voiding. The pressure is not transmitted to the ureters and renal pelves because of the competence of the ureterovesical “valves.” (A true valve is not present; the ureterotrigonal unit, by virtue of its intrinsic structure, resists the retrograde flow of urine.) However, owing to trigonal hypertrophy (see Section “Trabeculation of the bladder wall”) and to the resultant increase in resistance to urine flow across the terminal ureter, there is progressive back pressure on the ureter and kidney, resulting in ureteral dilatation and hydronephrosis. Later, with the phase of decompensation accompanied by residual urine, there is an added stretch effect on the already hypertrophied trigone that increases appreciably the resistance to flow at the lower end of the ureter and induces further hydroureteronephrosis. With decompensation of the ureterotrigonal complex, the valve-like action may be lost, vesicoureteral reflux occurs, and the increased intravesical pressure is transmitted directly to the renal pelvis, aggravating the degree of hydroureteronephrosis (Riccabona, 2010; Routh et al, 2010).

Secondary to the back pressure resulting from reflux or from obstruction by the hypertrophied and stretched trigone or by a ureteral stone, the ureteral musculature thickens in its attempt to push the urine downward by increased peristaltic activity (stage of compensation). This causes elongation and some tortuosity of the ureter (Figure 12–2). At times, this change becomes marked, and bands of fibrous tissue develop. On contraction, the bands further angulate the ureter, causing secondary ureteral obstruction. Under these circumstances, removal of the obstruction below may not prevent the kidney from undergoing progressive obstruction due to the secondary ureteral obstruction.

Figure 12–2.



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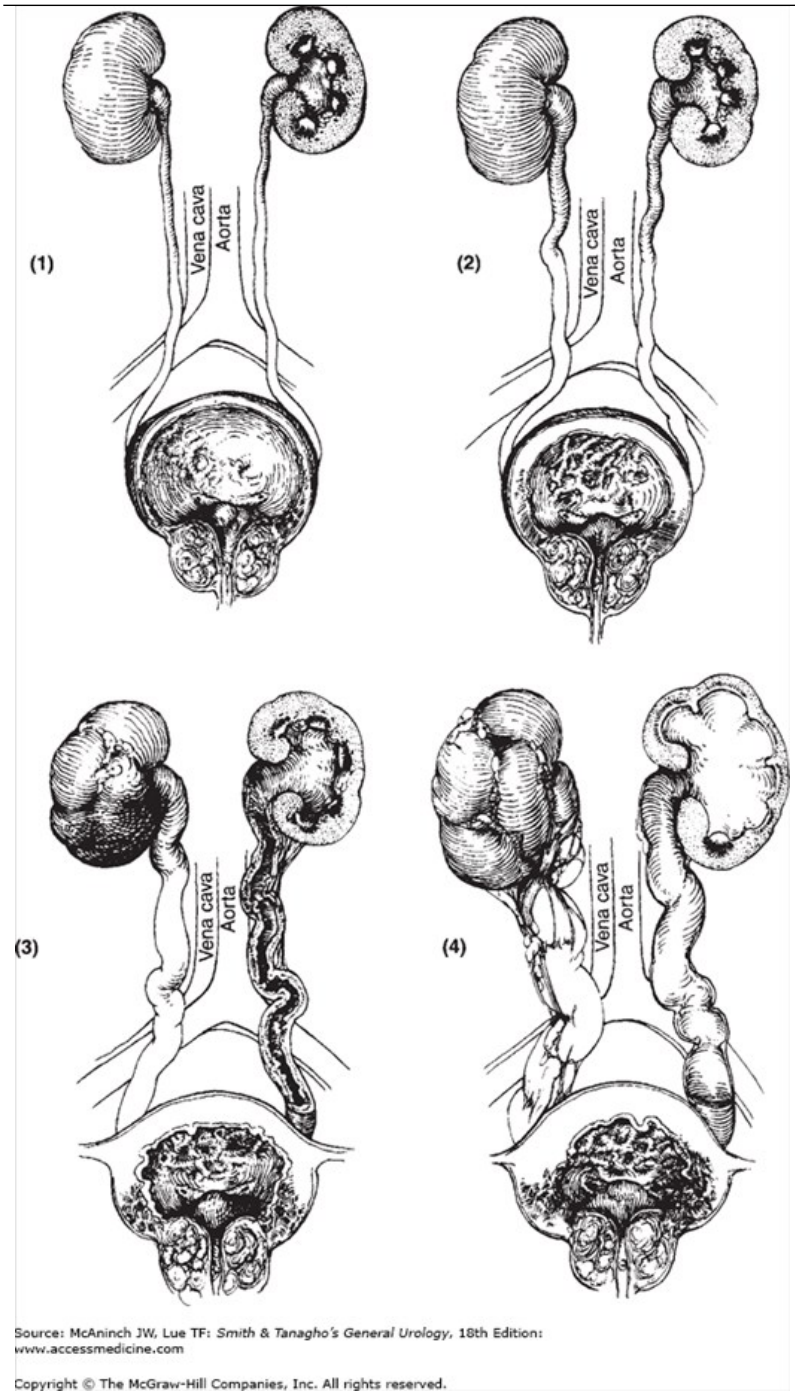
Mechanisms and results of obstruction. ***Upper left:*** Early stage. Elongation and dilatation of ureter due to mild obstruction. ***Upper center:*** Later stage. Further dilatation and elongation with kinking of the ureter; fibrous bands cause further kinking. ***Upper right:*** Intrarenal pelvis. Obstruction transmits all back pressure to parenchyma. ***Lower:*** Extrarenal pelvis, when obstructed, allows some of the increased pressure to be dissipated by the pelvis. (Reproduced, with permission, from Tanagho EA et al: Primary vesicoureteral reflux: Experimental studies of its etiology. J Urol 1965;93:165.)

Finally, because of increasing pressure, the ureteral wall becomes attenuated and therefore loses its contractile power (stage of decompensation). Dilatation may be so extreme that the ureter resembles a loop of bowel (Gimpel et al, 2010) (Figures 12–3 and 13–8, upper right).

Figure 12–3.



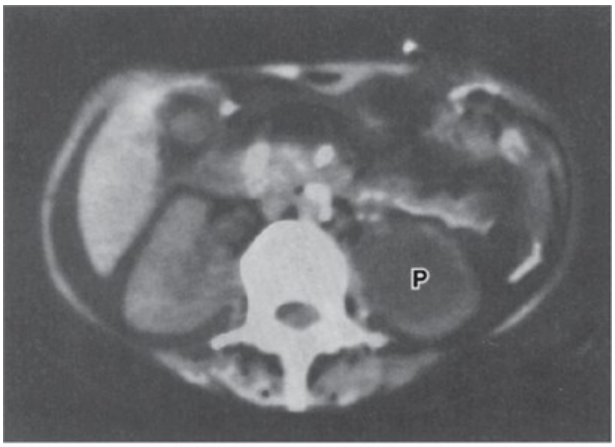
Pathogenesis of bilateral hydronephrosis. Progressive changes in bladder, ureters, and kidneys from obstruction of an enlarged prostate: thickening of bladder wall, dilatation and elongation of ureters, and hydronephrosis. (Reproduced, with permission, from Tanagho EA: Ureteroceles: Embryogenesis, pathogenesis and management. J Cont Educ Urol [Feb] 1979;18:13.)

Kidney

The pressure within the renal pelvis is normally close to zero. When this pressure increases because of obstruction or reflux, the pelvis and calyces dilate. The degree of hydronephrosis that develops depends on the duration, degree, and site of the obstruction (Figure 12–4). The higher the obstruction, the greater the effect on the kidney. If the renal pelvis is entirely intrarenal and the obstruction is at the ureteropelvic junction, all the pressure will be exerted on the parenchyma (Klein et al, 2010). If the renal pelvis is extrarenal, only part of the pressure produced by a ureteropelvic stenosis is exerted on the parenchyma; this is because the extrarenal pelvis is embedded in fat and dilates more readily, thus "decompressing" the

calyces (Figure 12–2).

Figure 12–4.



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Hydronephrotic left renal pelvis. Low-density mass (P) in left renal sinus had attenuation value similar to that of water, suggesting the correct diagnosis. Unless intravenous contrast material is used, differentiation from peripelvic cyst may be difficult.

In the earlier stages, the renal pelvic musculature undergoes compensatory hypertrophy in its effort to force urine past the obstruction. Later, however, the muscle becomes stretched and atonic (and decompensated).

The progression of hydronephrotic atrophy is as follows (Chevalier, 2010; Rodriguez, 2004):

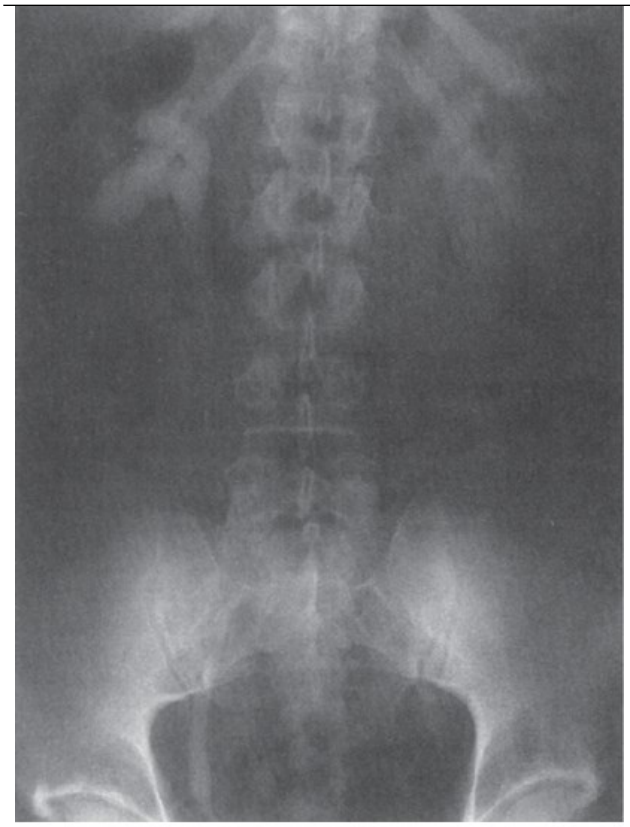
1. The earliest changes in the development of hydronephrosis are seen in the calyces. The end of a normal calyx is concave because of the papilla that projects into it; with increase in intrapelvic pressure, the fornices become blunt and rounded. With persistence of increased intrapelvic pressure, the papilla becomes flattened, then convex (clubbed) as a result of compression enhanced by ischemic atrophy (Figure 12–5). The parenchyma between the calyces is affected to a lesser extent. The changes in the renal parenchyma are due to (a) compression atrophy from increase in intrapelvic pressure (more accentuated with intrarenal pelves) and (b) ischemic atrophy from hemodynamic changes, mainly manifested in arcuate vessels that run at the base of the pyramids parallel to the kidney outline and are more vulnerable to compression between the renal capsule and the centrally increasing intrapelvic pressure.

2. This spotty atrophy is caused by the nature of the blood supply of the kidney. The arterioles are "end arteries"; therefore, ischemia is most marked in the areas farthest from the interlobular arteries. As the back pressure increases, hydronephrosis progresses, with the cells nearest the main arteries exhibiting the greatest resistance.

3. This increased pressure is transmitted up the tubules. The tubules become dilated, and their cells atrophy from ischemia. It should be pointed out that a few instances of dilated renal pelves and calyces are not due to the presence of obstruction. Rarely, the renal cavities are congenitally capacious and thus simulate hydronephrosis. More commonly, hydronephrosis may occur in childhood owing to the back pressure associated with vesicoureteral reflux. If the valvular incompetence resolves (and this is common), some degree of the hydronephrotic changes may persist. These persisting changes may cause the physician to suspect the presence of obstruction, which may lead to unnecessary surgery. A radioisotope renogram to assess ureteral drainage function can be performed to determine whether organic obstruction is present.

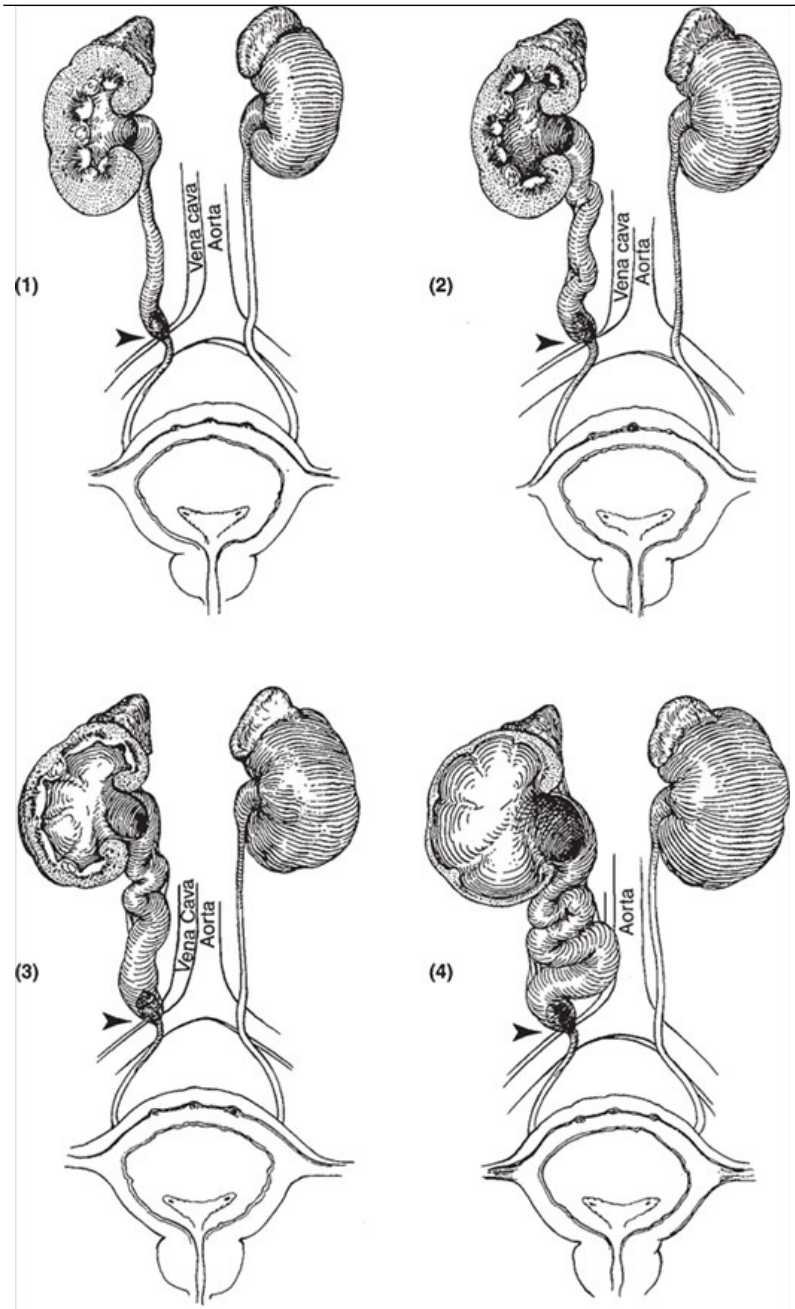
4. Only in unilateral hydronephrosis are the advanced stages of hydronephrotic atrophy seen. Eventually, the kidney is completely destroyed and appears as a thin-walled sac filled with clear fluid (water and electrolytes) or pus (Figure 12–6).

Figure 12–5.



Lower right ureteral obstruction. Mild-to-moderate dilatation of the collecting system with rounded blunting of the calyces.

Figure 12–6.



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Pathogenesis of unilateral hydronephrosis. Progressive changes in ureter and kidney secondary to obstructing calculus (*arrowheads*). As the right kidney undergoes gradual destruction, the left kidney gradually enlarges (compensatory hypertrophy).

If obstruction is unilateral, the increased intrarenal pressure causes some suppression of renal function on that side. The closer the intrapelvic pressure approaches the glomerular filtration pressure (6–12 mm Hg), the less urine can be secreted. Glomerular filtration rate and renal plasma flow are reduced, concentrating power is gradually lost, and the urea–creatinine concentration ratio of urine from the hydronephrotic kidney is lower than that of urine from the normal kidney.

Hydronephrotic atrophy is an unusual type of pathologic change. Other secretory organs (eg, the submaxillary gland) cease secreting when their ducts are obstructed. This causes primary (disuse) atrophy. The completely obstructed kidney, however, continues to secrete urine. (If this were not so, hydronephrosis could not occur, since it depends on increased intrarenal pressure.) As urine is excreted into the renal pelvis, fluid and, particularly,

soluble substances are reabsorbed, through either the tubules or the lymphatics. This has been demonstrated by injecting phenolsulfonphthalein (PSP) into the obstructed renal pelvis. It disappears (is reabsorbed) in a few hours and is excreted by the other kidney. If the intrapelvic pressure in the hydronephrotic kidney rapidly increases to a level approaching filtration pressure (resulting in cessation of filtration), a safety mechanism is activated that produces a break in the surface lining of the collecting structure at the weakest point—the fornices. This leads to escape and extravasation of urine from the renal pelvis into the parenchymal interstitium (pyelointerstitial backflow). The extravasated fluid is absorbed by the renal lymphatics, and the pressure in the renal pelvis drops, allowing further filtration of urine. This explains the process by which the markedly hydronephrotic kidney continues to function. Further evidence of the occurrence of extravasation and reabsorption is that the markedly hydronephrotic kidney does not contain urine in the true sense; only water and a few salts are present.

Functional impairment in unilateral hydronephrosis, as measured by excretory urograms or renal scans, is greater and increases faster than that seen in bilateral hydronephrotic kidneys showing comparable damage on urography. As unilateral hydronephrosis progresses, the normal kidney undergoes compensatory hypertrophy (particularly in children) of its nephrons (renal counterbalance), thereby assuming the function of the diseased kidney in order to maintain normal total renal function. For this reason, successful anatomic repair of the ureteral obstruction of such a kidney may fail to improve its powers of waste elimination.

If both kidneys are equally hydronephrotic, a strong stimulus is continually being exerted on both to maintain maximum function. This is also true of a hydronephrotic solitary kidney. Consequently, the return of function in these kidneys after repair of their obstructions is at times remarkable.

The extent of recovery after partial obstruction is difficult to determine preoperatively. Renal scanning with dimercaptosuccinic acid (DMSA) is most helpful. Temporary drainage, especially by nephrostomy, followed by tests to assess renal function is the best measure.

Physiologic Explanation of Symptoms of Bladder Neck Obstruction

The following hypothesis has been proposed to explain the syndrome known as “prostatism,” which occurs with progressive vesical obstruction:

> The bladder, like the heart, is a hollow muscular organ that receives fluid and forcefully expels it. And, like the heart, it reacts to an increasing workload by going through the successive phases of compensation and finally decompensation.
> Normally, contraction of the detrusor muscle in the presence of urine pulls the bladder neck open and the urine is expelled. The intravesical pressure generated in this instance varies between 20 and 40 cm of water.
> With bladder neck obstruction, hypertrophy of the vesical musculature develops, allowing the intravesical voiding pressure to rise to 50–100 cm or more of water in order to overcome the increased outlet resistance. Despite this, the encroaching prostate appears to interfere with the mechanisms that ordinarily open the internal orifice. Also, the contraction phase may not last long enough for all of the urine to be expelled; “exhaustion” of the muscle occurs prematurely. The refractory phase then sets in, and the detrusor is temporarily unable to respond to further stimuli. A few minutes later, voiding may be initiated again and completed.

Compensation Phase

Stage of Irritability

In the earliest stages of obstruction of the bladder neck, the vesical musculature begins to hypertrophy. The force and size of the urinary stream remain normal because the balance is maintained between the expelling power of the bladder and urethral resistance. During this phase, however, the bladder appears to be hypersensitive. As the bladder is distended, the need to void is felt. In patients with a normal bladder, these early urges can be inhibited, and the bladder relaxes and distends to receive more urine. However, in patients with a hypertrophied detrusor, the contraction of the detrusor is so strong that it virtually goes into spasm, producing the symptoms of an irritable bladder. The earliest symptoms of bladder neck obstruction, therefore, are urgency (even to the point of incontinence) and frequency, both day and night. Needs to mention C fibers here.

Stage of Compensation

As the obstruction increases, further hypertrophy of the muscle fibers of the bladder occurs, and the power to empty the bladder completely is thereby maintained. During this period, in addition to urgency and frequency, the patient notices hesitancy in initiating urination while the bladder develops contractions strong enough to overcome resistance at the bladder neck. The obstruction causes some loss in the force and size of the urinary stream and the stream becomes slower as vesical emptying nears completion (exhaustion of the detrusor as it nears the end of the contraction phase).

Decompensation Phase

If vesical tone becomes impaired or if urethral resistance exceeds detrusor power, some degree of decompensation occurs. The contraction phase of the vesical muscle becomes too short to completely expel the contents of the bladder, and some urine remains in the bladder (residual urine).

Acute Decompensation

The tone of the compensated vesical muscle can be temporarily impaired by rapid filling of the bladder (high fluid intake) or by overstretching of the detrusor (postponement of urination though the urge is felt). This may cause increased difficulty of urination, with marked hesitancy and the need for straining to initiate urination; a very weak and small stream; and termination of the stream before the bladder completely empties (residual urine). Acute and sudden complete urinary retention may also occur.

Chronic Decompensation

As the degree of obstruction increases, a progressive imbalance between the power of the bladder musculature and urethral resistance develops. Therefore, it becomes increasingly difficult to expel all the urine during the contraction phase of the detrusor. The symptoms of obstruction become more marked. The amount of residual urine gradually increases, and this diminishes the functional capacity of the bladder. Progressive frequency of urination is noted. On occasion, as the bladder decompensates, it becomes overstretched and attenuated. It may contain 1000–3000 mL of urine. It loses its power of contraction, and overflow (paradoxic) incontinence results.

Clinical Findings

Symptoms

Lower and Midtract (Urethra and Bladder)

Symptoms of obstruction of the lower and midtract are typified by the symptoms of urethral stricture, benign prostatic hyperplasia, neurogenic bladder, and tumor of the bladder involving the vesical neck. The principal symptoms are hesitancy in starting urination, lessened force and size of the stream, and terminal dribbling. Hematuria, which may be partial (eg with stricture); initial or terminal (eg with prostatic congestion); or total (eg with vesical tumor). Other symptoms are burning on urination, cloudy urine (due to complicating infection), and occasionally acute urinary retention (Elbadawi, 1998a and 1998b).

Upper Tract (Ureter and Kidney)

Symptoms of obstruction of the upper tract are typified by the symptoms of ureteral stricture or ureteral or renal stone. The principal complaints are pain in the flank radiating along the course of the ureter, gross total hematuria (from stone), gastrointestinal symptoms, chills, fever, burning on urination, and cloudy urine with onset of infection, which is the common sequel to obstruction or vesicoureteral reflux. Nausea, vomiting, loss of weight and strength, and pallor are due to uremia secondary to bilateral hydronephrosis. A history of vesicoureteral reflux in childhood may be significant (Aslan and Kogan, 2003). Obstruction of the upper tract may be silent even when uremia supervenes.

Signs

Lower and Midtract

Palpation of the urethra may reveal induration about a stricture. Rectal examination may show atony of the anal sphincter (damage to the sacral nerve roots) or benign or malignant enlargement of the prostate. Vesical distention may be found.

Although observation of the force and caliber of the urinary stream affords a rough estimate of maximum flow rate, the rate can be measured accurately with a urine flowmeter or, even more simply, by the following technique: Have the patient begin to void. When observed maximum flow has been reached, interpose a container to collect the urine and simultaneously start a stopwatch. After exactly 5 seconds, remove the container. The flow rate in milliliters per second can easily be calculated. The normal urine flow rate is 20–25 mL/s in males and 25–30 mL/s in females. Any flow rate <15

mL/s should be regarded with suspicion. A flow rate <10 mL/s is indicative of obstruction or weak detrusor function. Flow rates associated with an atonic neurogenic bladder (diminished detrusor power), or with urethral stricture or prostatic obstruction (increased urethral resistance), may be as low as 3–5 mL/s. A cystometrogram can differentiate between these two causes of impaired flow rate. After definitive treatment of the cause, the flow rate should return to normal.

In the presence of a vesical diverticulum or vesicoureteral reflux, although detrusor power is normal, the urinary stream may be impaired because of the diffusion of intravesical pressure into the diverticulum and vesicoureteral junction as well as the urethra. Excision of the diverticulum or repair of the vesicoureteral junctions leads to efficient expulsion of urine via the urethra.

Upper Tract

An enlarged kidney may be discovered by palpation or percussion. Renal tenderness may be elicited if infection is present. Cancer of the cervix may be noted; it may invade the base of the bladder and occlude one or both ureteral orifices, or its metastases to the iliac lymph nodes may compress the ureters. A large pelvic mass (tumor, pregnancy) can displace and compress the ureters. Children with advanced urinary tract obstruction (usually due to posterior urethral valves) may develop ascites. Rupture of the renal fornices allows leakage of urine retroperitoneally; with rupture of the bladder, urine may pass into the peritoneal cavity through a tear in the peritoneum (Kibar et al, 2010).

Laboratory Findings

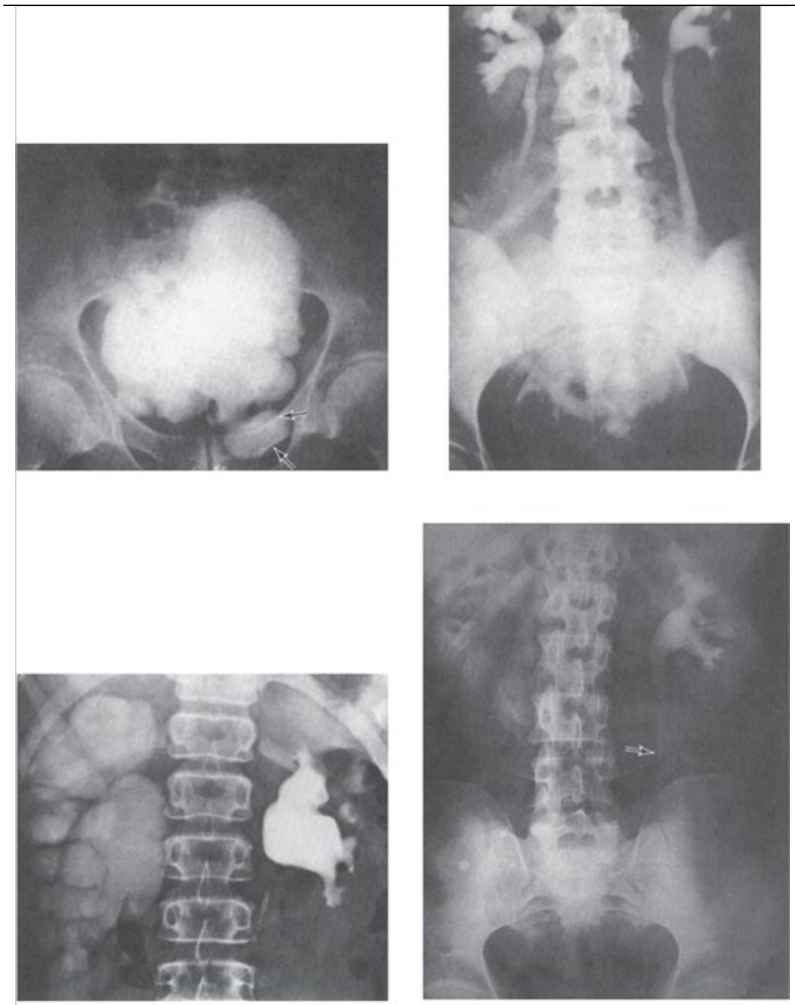
Anemia may be found secondary to chronic infection or in advanced bilateral hydronephrosis (stage of uremia). Leukocytosis is to be expected in the acute stage of infection. Little, if any, elevation of the white blood count accompanies the chronic stage.

Large amounts of protein are usually not found in the obstructive uropathies. Casts are not common from hydronephrotic kidneys. Microscopic hematuria may indicate renal or vesical infection, tumor, or stone. Pus cells and bacteria may or may not be present. In the presence of significant bilateral hydronephrosis, both urea and creatinine are elevated.

Urinary Tract Imaging (Figure 12–7)

A plain film of the abdomen may show enlargement of renal shadows, calcific bodies suggesting ureteral or renal stone, or tumor metastases to the bones of the spine or pelvis. Metastases in the spine may be the cause of spinal cord damage (neuropathic bladder); if they are osteoblastic, they are almost certainly from cancer of the prostate.

Figure 12–7.



Changes in bladder, ureters, and kidneys caused by obstruction. ***Upper left:*** Cystogram showing benign prostatic enlargement and multiple diverticula. Arrows point to femoral hernia that probably developed as a result of straining to urinate. ***Upper right:*** Pregnancy. Significant dilatation and elongation of upper right ureter due to compression at the pelvic line. Left side normal. ***Lower left:*** Excretory urogram, 70 minutes after injection. Advanced right hydronephrosis secondary to ureteropelvic obstruction. Mild ureteropelvic obstruction on left. ***Lower right:*** Stone in left ureter (at *arrow*) with mild hydronephrosis.

Excretory urograms reveal almost the entire story unless renal function is severely impaired. They are more informative when partial obstruction is present because the radiopaque material is retained. These urograms demonstrate the degree of dilatation of the pelves, calyces, and ureters. The point of ureteral stenosis is revealed. Segmental dilatation of the lower end of a ureter implies the possibility of vesicoureteral reflux (Figure 12–7), which can be revealed by voiding cystography. The cystogram may show trabeculation as an irregularity of the vesical outline and may show diverticula. Vesical tumors, nonopaque stones, and large intravesical prostatic lobes may cause radiolucent shadows. A film taken immediately after voiding will show residual urine.

Retrograde cystography shows changes of the bladder wall caused by distal obstruction (trabeculation, diverticula) or demonstrates the obstructive lesion itself (enlarged prostate, posterior urethral valves, cancer of the bladder). If the ureterovesical valves are incompetent, ureteropyelograms may be obtained by reflux.

Retrograde urograms may show better detail than the excretory type, but care must be taken not to overdistend the passages with too much opaque fluid; small hydronephroses can be made to look quite large. The degree of ureteral or ureterovesical obstruction can be judged by the degree of delay of drainage of the radiopaque fluid instilled.

Computed tomography (CT), magnetic resonance imaging (MRI), and sonography can also help determine the extent of dilatation and parenchymal atrophy (Silverman et al, 2009). CT scan with and without contrast is the best modality when stone obstruction is suspected (Kennish et al, 2010). To avoid irradiation in pregnant women, MRI and sonography are the preferred tests. However, both tests have limited value in delineating ureteral anatomy if it is not dilated.

Isotope Scanning (Nuclear Renography)

The glomerular agent Tc 99m **diethylenetriaminepentaacetic acid** (DTPA) and the tubular agent Tc 99m MAG3 are most commonly used in the evaluation of obstruction. However, to predict functional recovery, **DMSA**, a cortical agent, has been shown to be superior to tubular selective agents DTPA or MAG3. In the presence of obstruction, the radioisotope renogram may show depression of both the vascular and secretory phases and a rising rather than a falling excretory phase due to retention of the isotope-containing urine in the renal pelvis. Furosemide is often given 20 minutes after the tracer is given to induce diuresis, which helps the interpretation of the clearance curve.

Instrumental Examination

Exploration of the urethra with a catheter or other instrument is a valuable diagnostic measure. Passage may be blocked by a stricture or tumor. Spasm of the external sphincter may make passage difficult. Passage of the catheter immediately after voiding allows estimation of the amount of residual urine in the bladder. Bladder ultrasound can also accurately measure the amount of postvoid residual urine and determine outlet obstruction (Housami et al, 2009). Residual urine is common in bladder neck obstruction (enlarged prostate), cystocele, and neurogenic (neuropathic) bladder.

Measurement of vesical tone by means of cystometry is helpful in diagnosing neurogenic bladder and in differentiating between bladder neck obstruction and vesical atony. Inspection of the urethra, bladder, ureter, and renal pelvis by means of panendoscopy, cystoscopy, or ureteroscopy may reveal the primary obstructive cause (Van Cangh et al, 2001). Catheters may be passed to the renal pelves and urine specimens obtained. The function of each kidney may be measured, and retrograde ureteropyelograms can be obtained (Whitaker and Buxton-Thomas, 1984).

Differential Diagnosis

A thorough examination usually leaves no doubt about the diagnosis. The differential diagnosis under these circumstances is rarely difficult. If seemingly simple infection does not respond to medical therapy or if infection recurs, obstruction, a foreign body, or vesicoureteral reflux is the probable cause, and complete study of the urinary tract is indicated.

Complications

Stagnation of urine leads to infection, which then may spread throughout the entire urinary system. Once established, infection is difficult and at times impossible to eradicate even after the obstruction has been relieved.

Often, the invading organisms are urea splitting (*Proteus,* staphylococci), which causes the urine to become alkaline. Calcium salts precipitate and form bladder or kidney stones more easily in alkaline urine. If both kidneys are affected, the result may be renal insufficiency. Secondary infection increases renal damage.

Pyonephrosis is the end stage of a severely infected and obstructed kidney. The kidney is functionless and filled with thick pus. At times, a plain film of the abdomen may show an air urogram caused by gas liberated by infecting organisms.

Treatment

Relief of Obstruction

Treatment of the main causes of obstruction and stasis (benign prostatic hyperplasia, cancer of the prostate, neurogenic bladder, ureteral stone, posterior urethral valves, and ureteral stenosis) is described in detail elsewhere in this book (Glassberg, 2001; Hashim and Abrams, 2010; Myers and McAninch, 2009).

Lower Tract Obstruction (Distal to the Bladder)

With patients in whom secondary renal or ureterovesical damage (reflux in the latter) is minimal or nonexistent, correction of the obstruction is sufficient. If significant reflux is demonstrated and does not subside spontaneously after relief of obstruction, surgical repair may be needed. Repair becomes imperative if there is considerable hydronephrosis in addition to reflux. Preliminary drainage of the bladder by an indwelling catheter or other means of diversion (eg, loop ureterostomy) is indicated in order to preserve and improve renal function. If, after a few months of drainage, reflux persists, the incompetent ureterovesical junction should be surgically repaired. Persistent obstruction from prostatic enlargement of urethral stricture may also require surgical intervention (Andrich and Mundy, 2008; Robert et al, 2011; Roehrborn, 2011).

Upper Tract Obstruction (above the Bladder)

If tortuous, kinked, dilated, or atonic ureters have developed secondary to lower tract obstruction (so that they are themselves obstructive), vesical drainage will not protect the kidneys from further damage; the urine proximal to the obstruction must be diverted by nephrostomy or ureterostomy. The kidneys then may regain some function. Over a period of many months, the ureter may become less tortuous and less dilated; its obstructive areas may open up. If radiopaque material instilled into the nephrostomy tube passes readily to the bladder, it may be possible to remove the nephrostomy tube. If obstruction or reflux persists, surgical repair is indicated. Permanent urinary diversion (eg, ureteroileal conduit) may be necessary.

If one kidney has been irreversibly damaged, as measured by kidney function tests, urography, sonography, CT scan, or scintigraphy, nephrectomy may be necessary.

Eradication of Infection

Once the obstruction is removed, every effort should be made to eradicate infection. If the infection has been severe and prolonged, antibiotics may fail to sterilize the urinary tract. On the other hand, the incidence of urinary tract infection is low in children whose upper tract obstruction is diagnosed prenatally (Roth et al, 2009).

Prognosis

No simple statement can be made about the prognosis in this group of patients. The outcome depends on the cause, site, degree, and duration of the obstruction. The prognosis is also definitely influenced by complicating infection, particularly if the infection has been present for a long time.

If renal function is fair to good, if the obstruction or other causes of stasis can be corrected, and if complicating infection can therefore be eradicated, the prognosis is generally excellent.

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