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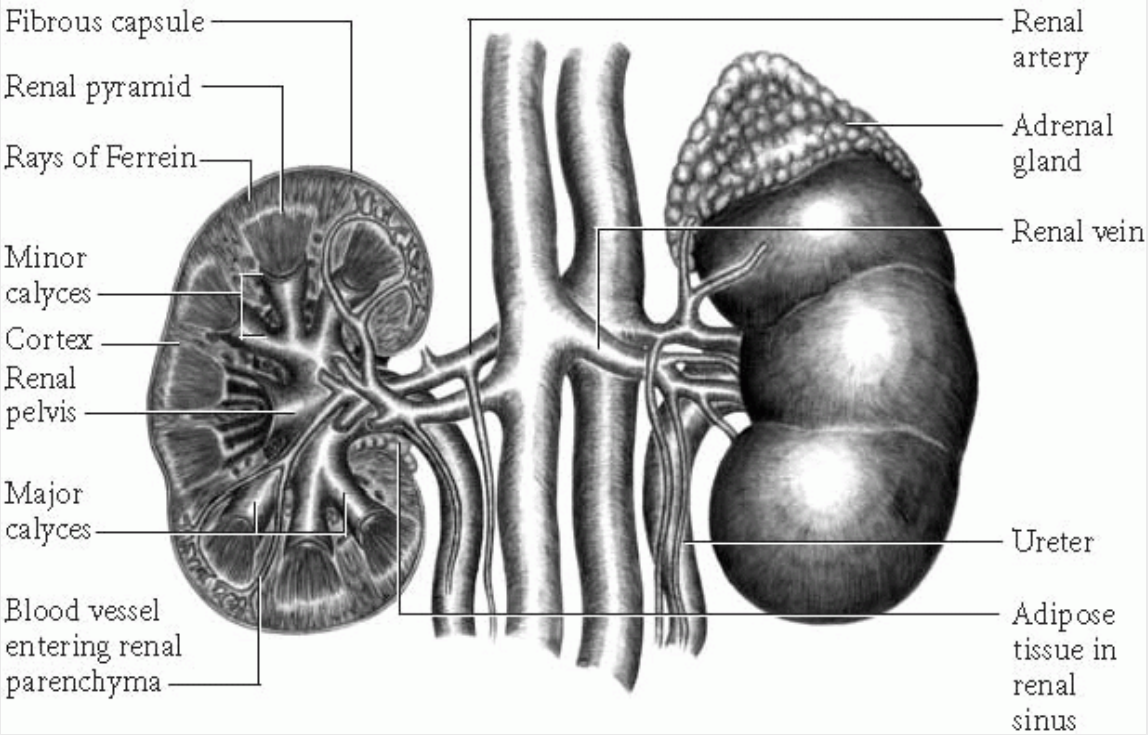
Title: ***Professional Guide to Diseases, 9th Edition***

7

Renal and urologic disorders

STRUCTURE OF THE KIDNEYS

The major components of the kidneys are depicted below.



Introduction

The kidneys are located retroperitoneally in the lumbar area, with the right kidney a little lower than the left because of the liver mass above

it. The left kidney is slightly longer than the right and closer to the midline. The kidneys move as body position changes. They're covered by the fibrous capsule, perirenal fat, renal fascia, and pararenal fat. (See *Structure of the kidneys*.)

Renal arteries branch into five segmental arteries that supply different areas of the kidneys. The segmental arteries then branch into several divisions from which the afferent arterioles and vasa recta arise. Renal veins follow a similar branching pattern, characterized by stellate vessels and segmental branches, and empty into the inferior vena cava. The tubular system receives its blood supply from a peritubular capillary network of vessels.

The gross structure of each kidney includes the lateral and medial margins, the hilus, the renal sinus, and renal parenchyma. The hilus, located at the medial margin, is the indentation where the blood and lymph vessels enter the kidney and the ureter emerges. The hilus leads to the renal sinus, which is a spacious cavity filled with adipose tissue, branches of the renal vessels, calyces, the renal pelvis, and the ureter. The renal sinus is surrounded by parenchyma, which consists of a cortex and a medulla.

The cortex (outermost layer of the kidney) contains glomeruli (parts of the nephron), cortical arches (areas that separate the medullary pyramids from the renal surface), columns of Bertin (areas that separate the pyramids from one another), and medullary rays of Ferrein (long, delicate processes from the bases of the pyramids that mix with the cortex). The medulla contains pyramids (cone-shaped structures of parenchymal tissue), papillae (apical ends of the pyramids through which urine oozes into the minor calyces), and papillary ducts of Bellini (collecting ducts in the pyramids that empty into the papillae).

Ureters

The ureters are a pair of retroperitoneally located, mucosa-lined, fibromuscular tubes that transport urine from the renal pelvis to the urinary bladder. Although the ureters have no sphincters, their oblique entrance into the bladder creates a mucosal fold that may produce a sphincterlike action.

The adult urinary bladder is a spherical, hollow muscular sac, with a normal capacity of 300 to 600 ml. It's located anterior and inferior to the peritoneal cavity, and posterior to the pubic bones. The gross structure of the bladder includes the fundus (large central, posterosuperior portion of the bladder), the apex (anterosuperior region), the body (posteroinferior region containing the ureteral orifices), and the urethral orifice, or neck (most inferior portion of the bladder). The three orifices comprise a triangular area called the *trigone*.

Nephrons

The functional units of each kidney are its 1 to 3 million nephrons. Each nephron is composed of the renal corpuscle and the tubular system. The renal corpuscle includes the glomerulus (a network of minute blood vessels) and Bowman's capsule (an epithelial sac surrounding the glomerulus that's part of the tubular system). The renal corpuscle has a vascular pole, where the afferent arteriole enters and the efferent arteriole emerges, and a urinary pole that narrows to form the beginning of the tubular system. The tubular system includes the proximal convoluted tubule, the loop of Henle, and the distal convoluted tubule. The last portion of the nephron consists of the collecting duct.

Innervation and vasculature

The kidneys are innervated by sympathetic branches from the celiac plexus, upper lumbar splanchnic and thoracic nerves, and the intermesenteric and superior hypogastric plexuses, which form a plexus around the kidneys. Similar numbers of sympathetic and parasympathetic nerves from the renal plexus, superior hypogastric plexus, and intermesenteric plexus innervate the ureters. Nerves that arise from the inferior hypogastric plexuses innervate the bladder. The parasympathetic nerve supply to the bladder controls micturition.

The ureters receive their blood supply from the renal, vesical, gonadal, and iliac arteries, and the abdominal aorta. The ureteral veins follow the arteries and drain into the renal vein. The bladder receives blood through vesical arteries. Vesical veins unite to form the pudendal plexus, which empties into the iliac veins. A rich lymphatic system drains the renal cortex, the kidneys, the ureters, and the bladder.

Homeostasis

Through the production and elimination of urine, the kidneys maintain homeostasis. These vital organs regulate the volume, electrolyte concentration, and acid-base balance of body fluids; detoxify the blood and eliminate wastes; regulate blood pressure; and aid in erythropoiesis. The kidneys eliminate wastes from the body through urine formation (by glomerular filtration, tubular reabsorption, and tubular secretion) and excretion. Glomerular filtration, the process of filtering the blood flowing through the kidneys, depends on the permeability of the capillary walls, vascular pressure, and filtration pressure. The normal glomerular filtration rate (GFR) is about 120 ml/minute.

Clearance measures function

Clearance is the volume of plasma that can be cleared of a given substance per unit of time, and depends on how renal tubular cells handle the substance that has been filtered by the glomerulus:

- If the tubules don't reabsorb or secrete the substance, clearance equals the GFR.
- If the tubules reabsorb it, clearance is less than the GFR.
- If the tubules secrete it, clearance is greater than the GFR.
- If the tubules reabsorb and secrete it, clearance is less than, equal to, or greater than the GFR.

The most accurate measure of glomerular function is creatinine clearance, because this substance is filtered only by the glomerulus and isn't reabsorbed by the tubules.

The transport of filtered substances in tubular reabsorption or secretion may be active (requiring the expenditure of energy) or passive (requiring none). For example, energy is required to move sodium across tubular cells (active transport), but none is required to move urea (passive transport). The amount of reabsorption or secretion of a substance depends on the maximum tubular transport capacity for that substance; that is, the greatest amount of a substance that can be reabsorbed or secreted per minute without saturating the system.

ELDER TIP

After age 40, a person's renal function begins to diminish; if he lives to age 90, it may have decreased by as much as 50%. This change is reflected in a decreased GFR; it's caused by age-related changes in the renal vasculature that disturb glomerular hemodynamics as well as by reduced cardiac output and atherosclerotic changes that reduce renal blood flow by more than 50%.

Water regulation

Hormones partially control water regulation by the kidneys. Hormonal control depends on the response of osmoreceptors to changes in osmolality. The two hormones involved are antidiuretic hormone (ADH), produced by the pituitary gland, and aldosterone, produced by the adrenal cortex. ADH alters the collecting tubules' permeability to water. When plasma concentration of ADH is high, the tubules are very permeable to water, so a greater amount of water is reabsorbed, creating a high concentration but small volume of urine. The reverse is true if ADH concentration is low.

Aldosterone regulates sodium and water reabsorption from the distal tubules. High plasma aldosterone concentration promotes sodium and water reabsorption from the tubules and decreases sodium and water excretion in the urine; low plasma aldosterone concentration promotes sodium and water excretion. Aldosterone also helps control the distal tubular secretion of potassium. Other factors that determine potassium secretion include the amount of potassium ingested, number of hydrogen ions secreted, level of intracellular potassium, amount of sodium in the distal tubule, and the GFR.

The countercurrent mechanism — composed of a multiplication system and an exchange system that occur in the renal medulla via the limbs of the loop of Henle and the vasa recta—is the method by which the kidneys concentrate urine. It achieves active transport of sodium and chloride between the loop of Henle and the medullary interstitial fluid. Failure of the countercurrent mechanism produces polyuria and nocturia.

To regulate acid-base balance, the kidneys secrete hydrogen ions, reabsorb sodium and bicarbonate ions, acidify phosphate salts, and synthesize ammonia— which keep the blood at its normal pH of 7.37 to 7.43.

The kidneys assist in regulating blood pressure by synthesizing and secreting renin in response to an actual, or perceived, decrease in the volume of extracellular fluid. Renin, in turn, acts on a substrate to form angiotensin I, which is converted to angiotensin II. Angiotensin II increases arterial blood pressure by peripheral vasoconstriction and stimulation of aldosterone secretion. The resulting increase in the aldosterone level promotes the reabsorption of sodium and water to correct the fluid deficit and renal ischemia.

The kidneys secrete erythropoietin in response to decreased oxygen tension in the renal blood supply. Erythropoietin then acts on the bone marrow to increase the production of red blood cells (RBCs).

Renal tubular cells synthesize active vitamin D and help regulate calcium balance and bone metabolism.

Clinical assessment

Assessment of the renal and urologic systems begins with an accurate patient history and requires a thorough physical examination and certain laboratory data and test results from invasive and noninvasive procedures. When obtaining a patient history, ask about symptoms that pertain specifically to the pathology of the renal and urologic systems, such as frequency or urgency, and about the presence of any systemic diseases that can produce renal or urologic dysfunction, such as hypertension, diabetes mellitus, or bladder infections. Family history may also suggest a genetic predisposition to certain renal diseases, such as glomerulonephritis or polycystic kidney disease. Also, ask what medications the patient

has been taking; abuse of analgesics or antibiotics may cause nephrotoxicity.

Physical examination for renal disease

The first step in physical examination is careful observation of the patient's overall appearance, because renal disease affects all body systems. Examine the patient's skin for color, turgor, intactness, and texture; mucous membranes for color, secretions, odor, and intactness; eyes for periorbital edema and vision; general activity for motion, gait, and posture; muscle movement for motor function and general strength; and mental status for level of consciousness, orientation, and response to stimuli. (See *Common renal symptoms*.)

Renal disease causes distinctive changes in vital signs: hypertension due to fluid and electrolyte imbalances and hyperactivity of the renin-angiotensin system; a strong, fast, irregular pulse due to fluid and electrolyte imbalances; hyperventilation to compensate for metabolic acidosis; and an increased susceptibility to infection due to overall decreased resistance. Palpation and percussion may reveal little because the kidneys and bladder are difficult to palpate unless they are enlarged or distended.

Noninvasive tests and monitoring

Laboratory tests analyze serum levels of chemical substances such as uric acid, creatinine, and blood urea nitrogen; tests also determine urine characteristics, including the presence of RBCs, white blood cells, casts, or bacteria; specific gravity and pH; and physical properties, such as clarity, color, and odor. (See *Serum and urine values in renal disease*, pages 376 and 377.)

- Intake and output assessment: Fluid intake and output measurement helps determine the patient's hydration status but isn't a reliable method of evaluating renal function because urine output varies with different types of renal disorders. To provide the most useful and accurate information, use calibrated containers, establish baseline values for each patient, compare measurement patterns, and validate intake and output measurements by checking the patient's weight daily. Monitor all fluid losses—including blood, vomitus, and diarrhea. Also assess wound and stoma drainage daily.

COMMON RENAL SYMPTOMS

Symptom	Possible cause

Dribbling	Prostatic enlargement, strictures
Dysuria	Infection, inflammation of bladder or urethra
Edema	Nephrotic syndrome, failure
Frequency	Infection, diabetes, bladder tumors, medications
Hematuria	Glomerular diseases, trauma, neoplasms, renal calculi
Hesitancy	Neurogenic bladder, infection
Incontinence	Infection, neoplasms, prolapsed uterus
Nocturia	Infection, nephrotic syndrome, diabetes, medications
Oliguria	Failure, insufficiency, neoplasms
Proteinuria	Glomerular diseases, infection
Pyuria	Infection
Renal colic	Calculi
Urgency	Infection, prostatic disease, medications

- Specimen collection: Meticulous specimen collection is vital for valid laboratory data. If the patient is collecting the specimen, explain the importance of cleaning the meatal area thoroughly. The culture specimen should be caught midstream, in a sterile container; a specimen for urinalysis, in a clean container, preferably at the first voiding of the day. Begin a 24-hour specimen collection after discarding the first voiding; such specimens often necessitate special handling or preservatives. When obtaining a urine specimen from a catheterized patient, remember to avoid taking the specimen from the collection bag; instead, aspirate a sample through the collection port in the catheter, with a sterile needle and a syringe.

- Kidney-ureter-bladder radiography: This test assesses size, shape, position, and areas of calcification of these organs.
- Ultrasonography: This safe, painless procedure allows for visualization of the renal parenchyma, calyces, pelvis, ureters, and bladder. Because the test doesn't depend on renal function, it's useful in patients with renal failure and in detecting complications after kidney transplantation.
- Invasive tests: See *Invasive diagnostic tests for assessing the renal and urologic systems*, pages 378 and 379.

SERUM AND URINE VALUES IN RENAL DISEASE

	Normal serum value	Deviation	Normal urine value	Deviation
Sodium	135 to 145 mEq/L	↑or N	30 to 280 mEq/L	V
Potassium	3.8 to 5.5 mEq/L	↑	25 to 100 mEq/L	↓
Chloride	100 to 108 mEq/L	↑	110 to 250 mEq/24 hr	↓
Calcium	8.9 to 10.1 mg/dl	↓	Female: < 250 mg/24 hr Male: < 275 mg/24 hr	↓
Phosphorus	2.5 to 4.5 mg/dl	↑ or N	1 g/24 hr	V
Magnesium	1.7 to 2.1 mEq/L	↑	< 150 mg/24 hr	↓
Carbon dioxide combining power	22 to 34 mEq/L	↓		
Specific gravity			1.005 to 1.035	↓
pH			4.5 to 8	↑

Blood urea nitrogen	8 to 20 mg/dl	↑	10 to 20 g/L	↓
Creatinine	*Female:* 0.6 to 0.9 mg/dl *Male:* 0.8 to 1.2 mg/dl	↑	*Female:* 0 to 80 mg/24 hr *Male: 0* to 40 mg/24 hr	↓
Osmolality	280 to 295 mOsm/kg	V	500 to 1,400 mOsm/kg	↓
Uric acid	*Female:* 2.3 to 6 mg/dl *Male:* 4.3 to 8 mg/dl	↑		
Glucose	70 to 100 mg/dl	N	0	N
Protein	6.9 to 7.9 g/dl	↓ or N	0	V
Hematocrit	*Female:* 38% to 46% *Male:* 42% to 54%	↓		
Hemoglobin	*Female:* 12 to 16 g/dl *Male:* 14 to 18 g/dl	↓		
White blood cells	4,000 to 10,000/mcl		none	V
Red blood cells			none	V
Casts			none	V
Bacteria			none	V
Alkaline phosphatase	*Female age 24 to 65:* 82 to 282 U/L *Male age 19 or older:* 98 to 251 U/L	↑		
KEY: ↑ = increased; ↓ = decreased; N = normal; V = varies				

Treatment methods

Treatment of intractable renal or urinary system dysfunction may require urinary diversion, dialysis, or kidney transplantation. Urinary diversion is the surgical creation of an outlet for excreting urine. The types of urinary diversion include ileal conduit, cutaneous ureterostomy, ureterosigmoidostomy, and creation of a rectal bladder.

In dialysis, a semipermeable membrane, osmosis, and diffusion imitate normal renal function by eliminating excess body fluids, maintaining or restoring plasma electrolyte and acid-base balance, and removing waste products and dialyzable poisons from the blood. Dialysis is most often used for patients with acute or chronic renal failure. The two most common types of dialysis are peritoneal dialysis and hemodialysis.

INVASIVE DIAGNOSTIC TESTS FOR ASSESSING THE RENAL AND UROLOGIC SYSTEMS

Several invasive tests are available to assist in diagnosis of renal and urologic problems. The most serious complication which occurs with many of these procedures is hypersensitivity to the contrast media. Symptoms may include increased pulse rate, itching, hives, chills, fever, dyspnea, and shock.

Procedure	Purpose	Special considerations
Computed tomography scan (CT) or magnetic resonance imaging (MRI)	Although CT scan and MRI can be noninvasive, I.V. contrast material is often given to enhance the views obtained. These tests are especially helpful in evaluating renal or bladder mass lesions.	*After:* After using I.V. contrast medium, observe for hypersensitivity reaction and hematoma at injection site.
Cystoscopy	A fiber-optic scope is used to visualize the inside of the bladder in cystoscopy.	*Before:* Give sedatives as ordered. *After:* Offer increased fluids; administer analgesics; watch for

		hematuria and signs of perforation, hemorrhage, and infection (chills, fever, increased pulse rate, shock).
Cystourethrography	In cystourethrography, X-rays and I.V. contrast material are used to determine size and shape of the bladder and urethra.	*During:* Catheterize the patient. *After:* Offer increased fluids; observe for hypersensitivity reaction.
Cystometry	Cystometry is used to evaluate bladder pressure, sensation, and capacity. A catheter is introduced into the bladder, and saline solution is instilled. Results are shown on a computer at the time of testing.	*Before:* Observe voiding; catheterize for residual urine. *After:* Remove catheter; watch for stress incontinence when patient coughs; watch voiding; catheterize for residual urine.
Excretory urography	Excretory urography uses X-rays and I.V. contrast material to allow visualization of renal parenchyma, calyces, pelves, ureters, and bladder.	*After:* Observe for hypersensitivity reaction; watch for hematomas at injection site.
Nephrotomography	In nephrotomography, I.V. contrast material and tomography are used to visualize parenchymia, calyces, and pelves in layers.	*After:* Observe for hypersensitivity reaction.
Renal angiography	Renal angiography uses contrast material injected into a catheter in the femoral artery or vein to allow visualization of the arterial tree and capillaries as well as venous drainage of the kidney.	*After:* Observe for hypersensitivity reaction, hematomas and hemorrhage at injection site, and nephrotoxicity. Offer increased fluids.
Renal scan	In renal scan, radioisotopes	*After:* Observe for hypersensitivity

	are administered I.V., and images of the kidney are taken at intervals to determine renal function.	reaction.
Renal biopsy	The specimen obtained in renal biopsy is used to develop histologic diagnosis and determine therapy and prognosis.	*Before:* Make sure the patient's clotting times, prothrombin times, and platelet count are recorded on his chart. *After:* Apply gentle pressure to the bandage site. Watch for hemorrhage and hematoma at the biopsy site and look for hematuria.

In peritoneal dialysis, a dialysate solution is infused into the peritoneal cavity. Substances then diffuse through the peritoneal membrane. Waste products remain in the dialysate solution and are removed.

Hemodialysis separates solutes by differential diffusion through a cellophane membrane placed between the blood and the dialysate solution, in an external receptacle. Because the blood must actually pass out of the body into a dialysis machine, hemodialysis requires an access route to the blood supply by an arteriovenous fistula or cannula or by a bovine or synthetic graft.

When caring for a patient with such an access route, monitor the patency of the access route, prevent infection, and promote safety and adequate function. After dialysis, watch for such complications as headache, vomiting, agitation, and twitching.

Patients with end-stage renal disease may benefit from kidney transplantation, despite its limitations: a shortage of donor kidneys, the chance of transplant rejection, and the need for lifelong medications and follow-up care. After kidney transplantation, maintain fluid and electrolyte balance, prevent infection, monitor for rejection, and promote psychological well-being.

CONGENITAL ANOMALIES

Medullary sponge kidney

In medullary sponge kidney, the collecting ducts in the renal pyramids dilate, and cavities, clefts, and cysts form in the medulla. This disease may affect only a single pyramid in one kidney or all pyramids in both kidneys. The kidneys are usually somewhat enlarged but may be of normal size; they appear spongy.

Because this disorder is usually asymptomatic and benign, it's often overlooked until the patient reaches adulthood. The prognosis is generally very good. Medullary sponge kidney is unrelated to medullary cystic disease; these conditions are similar only in the presence and location of the cysts.

Causes and incidence

Medullary sponge kidney may be transmitted as an autosomal dominant trait, but this remains unproven. Most nephrologists consider it a congenital abnormality.

Although medullary sponge kidney may be found in both sexes and in all agegroups, it primarily affects males ages 40 to 70. It occurs in about 1 in every 5,000 to 20,000 persons.

Complications

- Calcium oxalate calculi
- Infection

Signs and symptoms

Symptoms usually appear only as a result of complications and are seldom present before adulthood. Complications include formation of calcium oxylate stones, which lodge in the dilated cystic collecting ducts or pass through a ureter, and infection secondary to dilation of the ducts. These complications, which occur in about 30% of patients, are likely to produce severe colic, hematuria, lower urinary tract infection ([UTI]; burning on urination, urgency, frequency), and pyelonephritis.

Secondary impairment of renal function from obstruction and infection occurs in only about 10% of patients.

Diagnosis

Excretory urography is usually the key to diagnosis, often showing a characteristic flowerlike appearance of the pyramidal cavities when they fill with contrast material. It may also show renal calculi. Urinalysis is generally normal unless complications develop; however, it may show a slight reduction in concentrating ability or hypercalciuria. Diagnosis must distinguish medullary sponge kidney from renal tuberculosis, renal tubular acidosis, and papillary necrosis.

Treatment

Treatment focuses on preventing or treating complications caused by stones and infection. Specific measures include increasing fluid intake and monitoring renal function and urine. New symptoms necessitate immediate evaluation.

Because medullary sponge kidney is a benign condition, surgery is seldom necessary, except to remove stones during acute obstruction. Only serious, uncontrollable infection or hemorrhage requires nephrectomy.

Special considerations

Patient care includes explaining the disease to the patient and his family and reassuring them that the condition is benign and the prognosis good.

- To prevent infection, instruct the patient to bathe often and use proper toilet hygiene; this is especially important for a female patient because the proximity of the urinary meatus and the anus increases the risk of infection.
- If infection occurs, stress the importance of completing the prescribed course of antibiotic therapy.
- Emphasize the need for adequate fluid intake.
- Explain all diagnostic procedures, and provide emotional support. Teach the patient how to collect a clean-catch urine specimen for

culture. Check for allergy to excretory urography dye.

- When the patient is hospitalized for a stone, strain all urine, administer analgesics

  as ordered, and force fluids. Before discharge, tell the patient to watch for and report any signs of stone passage and UTI.

Polycystic kidney disease

Polycystic kidney disease is an inherited disorder characterized by multiple, bilateral, grapelike clusters of fluid-filled cysts that grossly enlarge the kidneys, compressing and eventually replacing functioning renal tissue. (See *Polycystic kidney.*) The disease appears in two distinct forms: The infantile form typically causes stillbirth or early neonatal death, although some infants may survive for 2 years, then develop fatal renal, cardiac, or respiratory failure. The adult form begins insidiously but usually becomes obvious between ages 30 and 50; rarely, it causes no symptoms until the patient is in his 70s. In the adult form, renal deterioration is more gradual but, as in the infantile form, progresses relentlessly to fatal uremia.

The prognosis in adults is extremely variable. Progression may be slow, even after symptoms of renal insufficiency appear. However, after uremic symptoms develop, polycystic kidney disease is usually fatal within 4 years, unless the patient receives treatment with dialysis, kidney transplantation, or both.

Causes and incidence

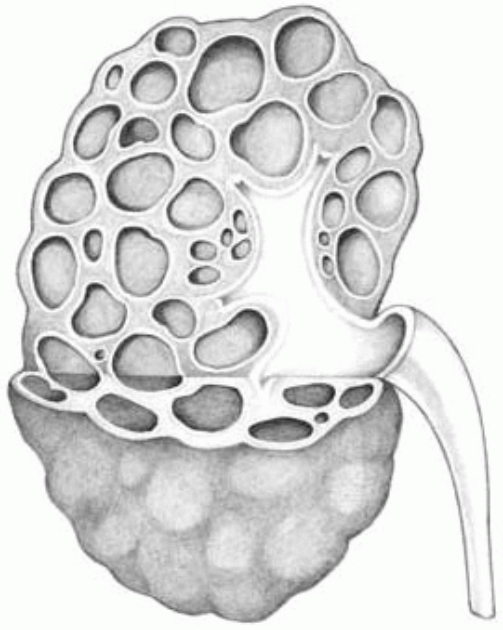
While both types of polycystic kidney disease are genetically transmitted, the incidence in two distinct age groups and different inheritance patterns suggest two unrelated disorders. The infantile type appears to be inherited as an autosomal recessive trait, whereas the adult type seems to be an autosomal dominant trait. The gene has been located on chromosome 6, supporting the premise that this is a single genetic disease with variable phenotype presentation.

Polycystic kidney disease reportedly affects 1 in every 1,000 Americans; yet that number may be even higher because some cases from patients

who aren't symptomatic go unreported. Both types of polycystic kidney disease affect males and females equally.

POLYCYSTIC KIDNEY

In polycystic kidney disease, cysts are seen in grapelike clusters, as shown below.



Complications

- Hypertension
- Urinary tract infection (UTI)
- Kidney infection
- End-stage kidney disease
- Kidney stones
- Ruptured cysts

Signs and symptoms

PEDIATRIC TIP

The neonate with infantile polycystic disease often has pronounced epicanthal folds, a pointed nose, a small chin, and floppy, lowset ears (Potter facies). At birth, he has huge bilateral masses on the flanks that are symmetrical, tense, and can't be transilluminated. He characteristically shows signs of respiratory distress and heart failure. Eventually, he develops uremia and renal failure. Accompanying hepatic fibrosis may cause portal hypertension and bleeding varices to develop, requiring sclerotherapy or portacaval shunting.

Adult polycystic kidney disease is commonly asymptomatic through the patient's 40s, but may induce nonspecific symptoms, such as hypertension, polyuria, and

recurrent UTIs. Later, the patient develops overt symptoms related to the enlarging kidney mass, such as lumbar pain, widening girth, and swollen or tender abdomen. Abdominal pain is usually worsened by exertion and relieved by lying down. In advanced stages, this disease may cause recurrent hematuria, life-threatening retroperitoneal bleeding resulting from cyst rupture, proteinuria, and colicky abdominal pain from the ureteral passage of clots or calculi. Generally, about 10 years after symptoms appear, progressive compression of kidney structures by the enlarging mass produces renal failure and uremia. Hypertension is found in about 20% to 30% of children and up to 75% of adults due to intrarenal ischemia, which activates the renin-angiotensin system.

Diagnosis

A family history and a physical examination revealing large bilateral, irregular masses in the flanks strongly suggest polycystic kidney disease. In advanced stages, grossly enlarged and palpable kidneys make the diagnosis obvious. In patients with these findings, the following laboratory results are typical:

- Excretory urography reveals enlarged kidneys, with elongation of pelvis, flattening of the calyces, and indentations caused by cysts.

Excretory urography of the neonate shows poor excretion of contrast medium.

- Ultrasound and computed tomography scan show kidney enlargement and the presence of cysts; tomography demonstrates multiple areas of cystic damage. Ultrasonography is the preferred imaging technique because it's less expensive, doesn't require contrast or radiation exposure, and is easily and safely performed on children and pregnant females.
- Urinalysis and creatinine clearance tests are nonspecific tests that evaluate renal function and reveal urine protein or blood in the urine.

Diagnosis must rule out the presence of renal tumors.

Treatment

Polycystic kidney disease can't be cured. The primary goal of treatment is preserving renal parenchyma and preventing infectious complications. Management of secondary hypertension will also help prevent rapid deterioration in function. Progressive renal failure requires treatment similar to that for other types of renal disease, including dialysis or, rarely, kidney transplantation.

When adult polycystic kidney disease is discovered in the asymptomatic stage, careful monitoring is required, including urine cultures and creatinine clearance tests every 6 months. Prompt and vigorous antibiotic treatment is needed when a urine culture reveals infection—even when the patient is asymptomatic. As renal impairment progresses, selected patients may undergo dialysis, transplantation, or both. Cystic abscess or retroperitoneal bleeding may require surgical drainage; intractable pain (a rare symptom) may also require surgery. However, because this disease affects both kidneys, nephrectomy usually isn't recommended because it increases the risk of infection in the remaining kidney.

Special considerations

Because polycystic kidney disease is usually relentlessly progressive, comprehensive patient teaching and emotional support are essential.

- Refer the young adult patient or the parents of infants with polycystic kidney disease for genetic counseling. Parents will probably have many questions about the risk to other offspring.
- Provide supportive care to minimize any associated symptoms. Carefully assess the patient's lifestyle and his physical and mental status; determine how rapidly the disease is progressing. Use this information to plan individualized patient care.
- Acquaint yourself with all aspects of end-stage renal disease, including dialysis and transplantation, so you can provide appropriate care and patient teaching as the disease progresses.
- Explain all diagnostic procedures to the patient or to his family if the patient is an infant. Before beginning excretory urography or other procedures that use an iodinebased contrast medium, determine whether the patient has ever had an allergic reaction to iodine or shellfish. Even if the

  patient has no history of allergy, watch for an allergic reaction after performing the procedures.
- Administer antibiotics as ordered for UTI. Stress to the patient the need to take the medication exactly as prescribed, even if symptoms are minimal or absent.

ACUTE RENAL DISORDERS

Acute renal failure

Acute renal failure is the sudden interruption of kidney function due to obstruction, reduced circulation, or renal parenchymal disease. It's usually reversible with medical treatment; otherwise, it may progress to end-stage renal disease, uremic syndrome, and death.

Causes and incidence

The causes of acute renal failure are classified as prerenal, intrinsic (or parenchymal), and postrenal. Prerenal failure is associated with diminished blood flow to the kidneys, possibly resulting from hypovolemia, shock, severe anaphylaxis, embolism, blood loss, sepsis,

pooling of fluid in ascites or burns, or from cardiovascular disorders, such as heart failure, arrhythmias, and tamponade.

Intrinsic renal failure results from damage to the kidneys themselves, usually due to acute tubular necrosis, but possibly due to acute poststreptococcal glomerulonephritis, systemic lupus erythematosus, periarteritis nodosa, vasculitis, sickle-cell disease, bilateral renal vein thrombosis, nephrotoxins, chronic misuse of nonsteroidal anti-inflammatory drugs, radiopaque contrast agents, ischemia, renal myeloma, acute pyelonephritis, and exposure to heavy metals, such as lead or mercury.

Postrenal failure results from bilateral obstruction of urinary outflow. Its multiple causes include kidney stones, blood clots, papillae from papillary necrosis, tumors, benign prostatic hyperplasia, strictures, and urethral edema from catheterization.

In the United States, the annual incidence of acute renal failure is 100 cases for every million people. It's diagnosed in 1% of hospital admissions. Hospital-acquired acute renal failure occurs in 4% of all admitted patients and 20% of patients who are admitted to critical care units.

Complications

- Volume overload
- Pulmonary edema
- Electrolyte imbalance
- Metabolic acidosis

Signs and symptoms

Acute renal failure is a critical illness. Its early signs are oliguria, azotemia and, rarely, anuria. Electrolyte imbalance, metabolic acidosis, and other severe effects follow, as the patient becomes increasingly uremic and renal dysfunction disrupts other body systems:

- *GI:* anorexia, nausea, vomiting, diarrhea or constipation, stomatitis, bleeding, hematemesis, dry mucous membranes, uremic breath

- *Central nervous system (CNS):* headache, drowsiness, irritability, confusion, peripheral neuropathy, seizures, coma
- *Cutaneous:* dryness, pruritus, pallor, purpura and, rarely, uremic frost
- *Cardiovascular:* early in the disease, hypotension; later, hypertension, arrhythmias, fluid overload, heart failure, systemic edema, anemia, altered clotting mechanisms
- *Respiratory:* pulmonary edema, Kussmaul's respirations.

Fever and chills indicate infection, a common complication.

Diagnosis

The patient's history may include a disorder that can cause renal failure. Blood test results indicating intrinsic acute renal failure include elevated blood urea nitrogen, serum creatinine, and potassium levels and low blood pH, bicarbonate, hematocrit (HCT), and hemoglobin (Hb) level. Urine specimens show casts, cellular debris, decreased specific gravity and, in glomerular diseases, proteinuria and urine osmolality close to serum osmolality. Urine sodium level is less than 20 mEq/L if oliguria is due to decreased perfusion; more than 40 mEq/L if due to an intrinsic problem. Other studies include renal ultrasonography, kidney-ureter-bladder X-rays, cautious use of excretory urography, renal scan, and nephrotomography.

Treatment

The goal of treatment is to correct or eliminate any reversible causes of kidney failure, such as obstructive uropathy, volume depletion, or the use of kidney-toxic medications. Supportive measures include a diet high in calories and low in protein, sodium, and potassium, with supplemental vitamins and restricted fluids. Meticulous electrolyte monitoring is essential to detect hyperkalemia. If hyperkalemia occurs, acute therapy may include dialysis, I.V. administration of hypertonic glucose, insulin infusion, and sodium bicarbonate, and administration of a potassium exchange resin (orally by enema) to remove potassium from the body.

If measures fail to control uremic symptoms, hemodialysis or peritoneal dialysis may be necessary. Continuous arteriovenous hemodiafiltration and continuous venovenous hemodiafiltration are alternative hemodialysis techniques for the treatment of acute renal failure. They're generally reserved for when intermittent dialysis fails to control hypervolemia or uremia, or for patients for whom peritoneal dialysis isn't possible.

Special considerations

Patient care includes careful monitoring and dietary education.

- Measure and record intake and output, including all body fluids, such as wound drainage, nasogastric output, and diarrhea. Weigh the patient daily.
- Assess Hb levels and HCT and replace blood components, as indicated. *Don't* use whole blood if the patient is prone to heart failure and can't tolerate extra fluid volume. Packed red blood cells deliver the necessary blood components without added volume.
- Monitor vital signs. Watch for and report any signs of pericarditis (pleuritic chest pain, tachycardia, pericardial friction rub), inadequate renal perfusion (hypotension), and acidosis.
- Maintain proper electrolyte balance. Strictly monitor potassium levels. Watch for symptoms of hyperkalemia (malaise, anorexia, paresthesia, or muscle weakness) and electrocardiogram changes (tall, peaked T waves, widening QRS segment, and disappearing P waves), and report them immediately. Avoid administering medications containing potassium.

ALERT

Assess the patient frequently, especially during emergency treatment to lower potassium levels. If the patient receives hypertonic glucose and insulin infusions, monitor potassium levels. If you give sodium polystyrene sulfonate rectally, make sure the patient doesn't retain it and become constipated, to prevent bowel perforation.

- Maintain nutritional status. Provide a high-calorie, low-protein, low-sodium, and low-potassium diet, with vitamin supplements. Give the anorectic patient small, frequent meals.
- Use sterile technique, because the patient with acute renal failure is highly susceptible to infection. Personnel with upper respiratory tract infections shouldn't provide care for the patient.
- Prevent complications of immobility by encouraging frequent coughing and deep breathing and by performing passive range-of-motion exercises. Help the patient walk as soon as possible.
- Provide good mouth care frequently because mucous membranes are dry. If stomatitis occurs, an antibiotic solution may be ordered. Have the patient swish the solution around in his mouth before swallowing.
- Monitor for GI bleeding by guaiac testing all stools for blood. Administer medications carefully, especially antacids and stool softeners. Use aluminum-hydroxide-based antacids; magnesium-based antacids can cause serum magnesium levels to rise to critical levels.
- Use appropriate safety measures, such as side rails and restraints, because the patient with CNS involvement may be dizzy or confused.

- Provide emotional support to the patient and his family. Reassure them by clearly explaining all procedures.
- If the patient requires hemodialysis, check the blood access site (arteriovenous fistula, subclavian or femoral catheter) as per facility protocol or every 2 hours for patency and signs of clotting. Don't use the arm with the shunt or fistula for taking blood pressures or drawing blood. Weigh the patient before beginning dialysis. During dialysis, monitor vital signs, clotting times, blood flow, the function of the vascular access site, and arterial and venous pressures. Watch for complications, such as septicemia, embolism, hepatitis, and rapid fluid and electrolyte loss. After dialysis, monitor vital signs and the vascular access site; weigh the patient; watch for signs of fluid and electrolyte imbalances.
- During peritoneal dialysis, position the patient carefully. Elevate the head of the bed to reduce pressure on the diaphragm and aid respiration. Be alert for signs of infection (cloudy drainage, elevated

temperature) and, rarely, bleeding. If pain occurs, reduce the amount of dialysate. Monitor the diabetic patient's blood glucose periodically, and administer insulin as ordered. Watch for complications, such as peritonitis, atelectasis, hypokalemia, pneumonia, and shock.

- Use standard precautions when handling all blood and body fluids.

PREVENTION

- *Evaluate all drugs the patient is taking and determine which drugs may be affected by or have an effect on renal function.*
- *Aggresively restore fluid volume after major surgery or trauma.*

Acute pyelonephritis

Acute pyelonephritis (also known as *acute infective tubulointerstitial nephritis*) is a sudden inflammation caused by bacteria that primarily affects the interstitial area and the renal pelvis or, less often, the renal tubules. It's one of the most common renal diseases. With treatment and continued follow-up care, the prognosis is good, and extensive permanent damage is rare. (See *Chronic pylenonephritis,* page 386.)

Causes and incidence

Acute pyelonephritis results from bacterial infection of the kidneys. Infecting bacteria usually are normal intestinal and fecal flora that grow readily in urine. The most common causative organism is *Escherichia coli,* but *Proteus, Pseudomonas, Staphylococcus aureus,* and *Enterococcus faecalis* (formerly *Streptococcus faecalis*) may also cause this infection.

Typically, the infection spreads from the bladder to the ureters, then to the kidneys, as in vesicoureteral reflux due to congenital weakness at the junction of the ureter and the bladder. Bacteria refluxed to intrarenal tissues may create colonies of infection within 24 to 48 hours. Infection may also result from instrumentation (such as catheterization, cystoscopy, or urologic surgery), from a hematogenic infection (as in septicemia or endocarditis), or possibly from lymphatic infection.

Pyelonephritis may also result from an inability to empty the bladder (for example, in patients with neurogenic bladder), urinary stasis, or urinary obstruction due to tumors, strictures, or benign prostatic hyperplasia.

Pyelonephritis occurs more commonly in women, probably because of a shorter urethra and the proximity of the urinary meatus to the vagina and the rectum—both of which allow bacteria to reach the bladder more easily—and a lack of the antibacterial prostatic secretions produced by men. Incidence increases with age and is higher in the following groups:

- *Sexually active women:* Intercourse increases the risk of bacterial contamination.
- *Pregnant women:* About 5% develop asymptomatic bacteriuria; if untreated, about 40% develop pyelonephritis.
- *Diabetics:* Neurogenic bladder causes incomplete emptying and urinary stasis; glycosuria may support bacterial growth in the urine.
- *Persons with other renal diseases:* Compromised renal function aggravates susceptibility.

CHRONIC PYELONEPHRITIS

Chronic pyelonephritis is a persistent kidney inflammation that can scar the kidneys and may lead to chronic renal failure. Its etiology may be bacterial, metastatic, or urogenous. This disease is most common in patients who are predisposed to recurrent acute pyelonephritis, such as those with urinary obstructions or vesicoureteral reflux.

Patients with chronic pyelonephritis may have a childhood history of unexplained fevers or bedwetting. Clinical effects may include flank pain, anemia, low urine specific gravity, proteinuria, leukocytes in urine and, especially in late stages, hypertension. Uremia rarely develops from chronic pyelonephritis unless structural abnormalities exist in the excretory system. Bacteriuria

may be intermittent. When no bacteria are found in the urine, diagnosis depends on excretory urography (renal pelvis may appear small and flattened) and renal biopsy.

Effective treatment of chronic pyelonephritis requires control of hypertension, elimination of the existing obstruction (when possible), and long-term antimicrobial therapy.

Complications

- Recurrence of pyelonephritis
- Sepsis
- Perinephric abscess
- Renal failure

Signs and symptoms

Typical clinical features include urgency, frequency, burning during urination, dysuria, nocturia, and hematuria (usually microscopic but may be gross). Urine may appear cloudy and have an ammonia-like or fishy odor. Other common symptoms include a temperature of 102° F (38.9° C) or higher, shaking chills, flank pain, anorexia, and general fatigue.

These symptoms characteristically develop rapidly over a few hours or a few days. Although these symptoms may disappear within days, even without treatment, residual bacterial infection is likely and may cause symptoms to recur later.

ELDER TIP

Elderly patients may exhibit altered mental status or GI or pulmonary symptoms rather than the usual febrile responses to pyelonephritis.

PEDIATRIC TIP

In children younger than age 2, fever, vomiting, nonspecific abdominal complaints, or failure to thrive

may be the only signs of acute pyelonephritis.

Diagnosis

Diagnosis requires urinalysis and culture. Typical findings include:

- Pyuria (pus in urine): Urine sediment reveals the presence of leukocytes singly, in clumps, and in casts; and, possibly, a few red blood cells.
- Significant bacteriuria: Urine culture reveals more than 100,000 organisms/µl of urine.
- Low specific gravity and osmolality: These findings result from a temporarily decreased ability to concentrate urine.
- Slightly alkaline urine pH.
- Proteinuria, glycosuria, and ketonuria: These conditions are less common.
- Costovertebral angle tenderness

Excretory urography or computed tomography (CT) scan of the kidneys, ureters, and bladder also help in the evaluation of acute pyelonephritis by revealing calculi, tumors, or cysts in the kidneys and the urinary tract. In addition, excretory urography may show asymmetrical kidneys.

Treatment

Treatment centers on antibiotic therapy appropriate to the specific infecting organism after identification by urine culture and sensitivity studies. When the infecting organism can't be identified, therapy usually consists of a broad-spectrum antibiotic. Urinary analgesics are also appropriate.

ALERT

If a woman is pregnant, antibiotics must be prescribed cautiously.

Symptoms may disappear after several days of antibiotic therapy. Although urine usually becomes sterile within 48 to 72 hours, the course

of such therapy is 10 to 14 days. Follow-up treatment may include reculturing urine 1 week after drug therapy stops, then periodically for the next year to detect residual or recurring infection. Most patients with uncomplicated infections respond well to therapy and don't suffer reinfection.

In infection from obstruction or vesicoureteral reflux, antibiotics may be less effective; treatment may then necessitate surgery to relieve the obstruction or correct the anomaly. Patients at high risk of recurring urinary tract and kidney infections, such as those with prolonged use of an indwelling catheter or maintenance antibiotic therapy, require long-term follow-up. Recurrent episodes of acute pyelonephritis can eventually result in chronic pyelonephritis.

Special considerations

Patient care is supportive during antibiotic treatment of the underlying infection.

- Administer antipyretics for fever.
- Encourage fluids to achieve urine output of more than 2,000 ml/day. This helps to empty the bladder of contaminated urine. Don't encourage intake of more than 2 to 3 qt (2 to 3 L) because this may decrease the effectiveness of the antibiotics.
- Provide an acid-ash diet to prevent stone formation.
- Teach proper technique for collecting a clean-catch urine specimen. Be sure to refrigerate or culture a urine specimen within 30 minutes of collection to prevent overgrowth of bacteria.
- Stress the need to complete prescribed antibiotic therapy, even after symptoms subside. Encourage long-term follow-up care for high-risk patients.

To prevent acute pyelonephritis:

PREVENTION

- *Observe strict sterile technique during catheter insertion and care.*

- *Instruct women to prevent bacterial contamination by wiping the perineum from front to back after defecation.*
- *Advise routine checkups for patients with a history of urinary tract infections. Teach them to recognize signs of infection, such as cloudy urine, burning on urination, urgency, and frequency, especially when accompanied by a low-grade fever.*

Acute poststreptococcal glomerulonephritis

Acute poststreptococcal glomerulonephritis (APSGN), also known as *acute glomerulonephritis,* is a relatively common bilateral inflammation of the glomeruli. It usually follows a streptococcal infection of the respiratory tract or, less often, a skin infection such as impetigo.

Causes and incidence

APSGN results from the entrapment and collection of antigen-antibody (produced as an immunologic mechanism in response to streptococcus) in the glomerular capillary membranes, inducing inflammatory damage and impeding glomerular function. Sometimes, the immune complement further damages the glomerular membrane. The damaged and inflamed glomerulus loses the ability to be selectively permeable, and allows red blood cells (RBCs) and proteins to filter through as the glomerular filtration rate (GFR) falls. Uremic poisoning may result.

APSGN is most common in boys ages 3 to 7, but it can occur at any age. Incidence is rising in the United States and Europe, with epidemics occurring in developing countries in Africa, the West Indies, and the Middle East.

Up to 95% of children and up to 70% of adults with APSGN recover fully; the rest may progress to chronic renal failure within months.

Complications

- Acute or chronic kidney failure

- End-stage renal disease
- Hypertension
- Heart failure
- Pulmonary edema

- Chronic glomerulonephritis
- Nephrotic syndrome

Signs and symptoms

APSGN begins within 1 to 3 weeks after untreated pharyngitis. Symptoms include mild to moderate edema, oliguria (less than 400 ml/24 hours), proteinuria, azotemia, hematuria, and fatigue. Mild to severe hypertension may result from either sodium or water retention (due to decreased GFR) or inappropriate renin release. Heart failure from hypervolemia leads to pulmonary edema.

PEDIATRIC TIP

The presenting features of APSGN in children may be encephalopathy with seizures and focal neurological deficits.

Diagnosis

Diagnosis requires a detailed patient history and assessment of clinical symptoms and laboratory tests.

Urinalysis typically reveals proteinuria and hematuria. RBCs, white blood cells, and mixed cell casts are common in urinary sediment. Elevated serum creatinine levels and low creatinine clearance accompany impaired glomerular filtration. Elevated antistreptolysin-O titers (in 80% of patients), elevated streptozyme and anti-DNase B titers, and low serum complement levels verify recent streptococcal infection. A throat culture may also show group A beta-hemolytic streptococcus. Renal ultrasound may show a normal or slightly enlarged kidney. A renal biopsy may confirm the diagnosis or assess renal tissue status.

Treatment

The goals of treatment are relief of symptoms and prevention of complications. Vigorous supportive care includes bed rest, fluid and dietary sodium restrictions, and correction of electrolyte imbalances (possibly with dialysis, although this is rarely necessary). Therapy may include diuretics to reduce extracellular fluid overload and an antihypertensive. The use of antibiotics is recommended for 7 to 10 days if staphylococcal infection is documented. Otherwise, antibiotic use is controversial.

Special considerations

APSGN usually resolves within 2 weeks, so patient care is primarily supportive.

- Check vital signs and electrolyte values. Monitor intake and output and daily weight. Assess renal function daily through serum creatinine, blood urea nitrogen, and urine creatinine clearance levels. Watch for and immediately report signs of acute renal failure (oliguria, azotemia, and acidosis).
- Consult the dietitian to provide a diet high in calories and low in protein, sodium, potassium, and fluids.
- Protect the debilitated patient against secondary infection by providing good nutrition, using good hygienic technique, and preventing contact with infected persons.
- Bed rest is necessary during the acute phase. Allow the patient to *gradually* resume normal activities as symptoms subside.
- Provide emotional support for the patient and his family. If the patient is on dialysis, explain the procedure fully.
- Advise the patient with a history of chronic upper respiratory tract infections to immediately report signs of infection (fever, sore throat).
- Tell the patient that follow-up examinations are necessary to detect chronic renal failure. Stress the need for regular blood pressure, urinary protein, and renal function assessments during the convalescent months to detect recurrence. After APSGN, gross

hematuria may recur during nonspecific viral infections; abnormal urinary findings may persist for years.

- Encourage pregnant women with a history of APSGN to have frequent medical evaluations because pregnancy further stresses the kidneys and increases the risk of chronic renal failure.

Acute tubular necrosis

Acute tubular necrosis (ATN), also known as *acute tubulointerstitial nephritis,* accounts for about 75% of all cases of acute renal failure and is the most common cause of acute renal failure in critically ill patients. ATN injures the tubular segment of the nephron, causing renal failure and uremic syndrome. Mortality ranges from

40% to 70%, depending on complications from underlying diseases. Nonoliguric forms of ATN have a better prognosis.

Causes and incidence

ATN results from ischemic or nephrotoxic injury, most commonly in debilitated patients, such as the critically ill or those who have undergone extensive surgery. In ischemic injury, disruption of blood flow to the kidneys may result from circulatory collapse, severe hypotension, trauma, hemorrhage, dehydration, cardiogenic or septic shock, surgery, anesthetics, or reactions to transfusions. Nephrotoxic injury may follow ingestion of certain chemical agents or result from a hypersensitive reaction of the kidneys. (See *Rise in nephrotoxic injury*.) Because nephrotoxic ATN doesn't damage the basement membrane of the nephron, it's potentially reversible. However, ischemic ATN can damage the epithelial and basement membranes and can cause lesions in the renal interstitium. ATN may result from:

- diseased tubular epithelium that allows leakage of glomerular filtrate across the membranes and reabsorption of filtrate into the blood
- obstruction of urine flow by the collection of damaged cells, casts, red blood cells (RBCs), and other cellular debris within the tubular walls

- ischemic injury to glomerular epithelial cells, resulting in cellular collapse and decreased glomerular capillary permeability
- ischemic injury to vascular endothelium, eventually resulting in cellular swelling and obstruction.

Complications

- Fluid and electrolyte imbalance
- Heart failure
- GI hemorrhage
- Pulmonary edema

Signs and symptoms

Nephrotoxic injury causes multiple symptoms similar to those of renal failure, particularly azotemia, anemia, acidosis, overhydration, and hypertension. Some patients may also experience fever, rash, and eosinophilia. However, ATN is usually difficult to recognize in its early stages because effects of the critically ill patient's primary disease may mask the symptoms of ATN. The first recognizable effect may be decreased urine output. Generally, hyperkalemia and the characteristic uremic syndrome soon follow, with oliguria (or, rarely, anuria) and confusion, which may progress to uremic coma. Other possible complications may include heart failure, uremic pericarditis, pulmonary edema, uremic lung, anemia, anorexia, intractable vomiting, and poor wound healing due to debilitation.

RISE IN NEPHROTOXIC INJURY

Incidence of acute tubular necrosis (ATN) due to ingestion or inhalation of toxic substances is rising. ATN may occur in hospitalized patients following exposure to toxic agents, such as antibiotics, chemotherapeutic agents, or contrast material. Other nephrotoxic agents include pesticides, fungicides, heavy metals (for example, mercury, arsenic, lead, bismuth, uranium), and organic solvents containing carbon tetrachloride or

ethylene glycol, such as cleaning fluids or industrial solvents. Ingestion of these substances may be accidental or intentional.

ALERT

Fever and chills may signal the onset of an infection, which is the leading cause of death in ATN.

Diagnosis

Diagnosis is usually delayed until the condition has progressed to an advanced stage. The most significant laboratory clues are urinary sediment containing RBCs and casts, and dilute urine of a low specific gravity (1.010), low osmolality (less than 400 mOsm/kg), and high sodium level (40 to 60 mEq/L). Blood studies reveal elevated blood urea nitrogen and serum creatinine levels, anemia, defects in platelet adherence, metabolic acidosis, and

hyperkalemia. An electrocardiogram may show arrhythmias (due to electrolyte imbalances) and, with hyperkalemia, widening QRS segment, disappearing P waves, and tall, peaked T waves.

Treatment

Treatment consists of identifying the nephrotoxic substance, eliminating its use, and removing it from the body, possibly by hemodialysis or hemoperfusion in extreme cases. Initial treatment may include administration of diuretics and infusion of a large volume of fluids to flush tubules of cellular casts and debris and to replace fluid loss. However, this treatment carries a risk of fluid overload. Long-term fluid management requires daily replacement of projected and calculated losses (including insensible loss). Diet may be high in carbohydrates and low in protein, sodium, and potassium to minimize their build up in the body. During the course of acute renal failure, treatment is supportive.

Other appropriate measures to control complications include transfusion of packed RBCs for anemia and administration of antibiotics for infection. Epogen may be given to stimulate RBC production as an alternative to blood transfusion. Hyperkalemia may require emergency

I.V. administration of 50% glucose, regular insulin, and sodium bicarbonate. Sodium polystyrene sulfonate with sorbitol may be given by mouth or by enema to reduce extracellular potassium levels. Peritoneal dialysis or hemodialysis may be needed if the patient is catabolic.

Special considerations

Patient care is largely supportive.

- Maintain fluid balance. Watch for fluid overload, a common complication of therapy. Accurately record intake and output, including wound drainage, nasogastric output, and hemodialysis and peritoneal dialysis balances. Weigh the patient daily.
- Monitor hemoglobin (Hb) levels and hematocrit, and administer blood products as needed. Use fresh packed cells instead of whole blood to prevent fluid overload and heart failure.
- Maintain electrolyte balance. Monitor laboratory results, and report imbalances. Enforce dietary restriction of foods containing sodium and potassium, such as bananas, orange juice, and baked potatoes. Check for potassium content in prescribed medications (for example, potassium penicillin). Provide adequate calories and essential amino acids, while restricting protein intake to maintain an anabolic state. Total parenteral nutrition may be indicated in the severely debilitated or catabolic patient.
- Use sterile technique, particularly when handling catheters because the debilitated patient is vulnerable to infection. Immediately report fever, chills, delayed wound healing, or flank pain if the patient has an indwelling catheter in place.
- Watch for complications. If anemia worsens (pallor, weakness, lethargy with decreased Hb level), administer RBCs as ordered. For acidosis, give sodium bicarbonate or assist with dialysis in severe cases, as ordered. Watch for signs of diminishing renal perfusion (hypotension and decreased urine output). Encourage coughing and deep breathing to prevent pulmonary complications.
- Perform passive range-of-motion exercises. Provide good skin care; apply lotion or bath oil for dry skin. Help the patient to walk as soon as possible, but guard against exhaustion.

- Provide reassurance and emotional support. Encourage the patient and his family to express their fears. Fully explain each procedure; repeat the explanation each time the procedure is done. Help the patient and his family set realistic goals according to individual prognosis.
- To prevent ATN, make sure patients are well hydrated before surgery or after X-rays that use a contrast medium. Administer mannitol, as ordered, to high-risk patients before and during these procedures. Carefully monitor patients receiving blood transfusions to detect early signs of transfusion reaction (fever, rash, chills), and discontinue such transfusion immediately.

PREVENTION

- *Immediately treat disorders that cause a decrease in blood flow and oxygenation to the kidneys, such as severe hypotension and cardiogenic or septic shock.*
- *Proper hydration before surgery is very important. Adequate hydration after radio contrast dyes may allow for their excretion and reduce the risk of kidney damage.*
- *Proper typing and crossmatching before blood transfusions reduce the risk of incompatibility reactions.*
- *When a patient is exposed to drugs toxic to the renal system, monitor him carefully by checking levels regularly.*
- *Patients with diseases, such as diabetes, liver disease, and cardiac disorders, are at higher risk for ATN. These patients need to be well-controlled in order to prevent the risk of ATN.*

Renal infarction

Renal infarction is the formation of a coagulated, necrotic area in one or both kidneys that results from renal blood vessel occlusion. The location

and size of the infarction depend on the site of vascular occlusion; most often, infarction affects the renal cortex, but it can extend into the medulla. (See *Sites of renal infarction*.) Residual renal function after infarction depends on the extent of the damage from the infarction.

Causes and incidence

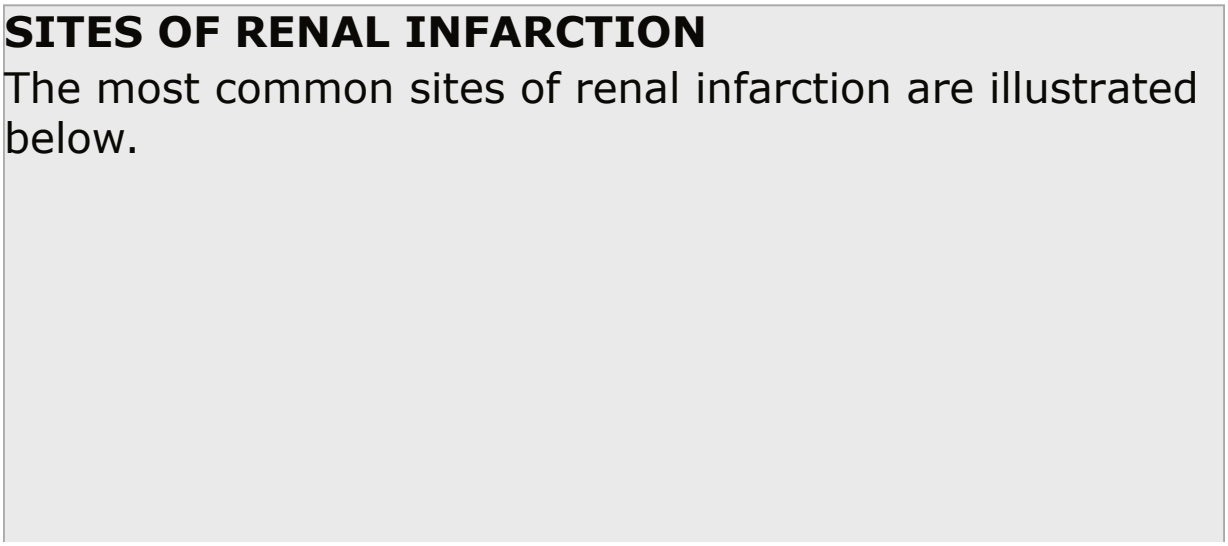
In 75% of patients, renal infarction results from renal artery embolism secondary to mitral stenosis, infective endocarditis, atrial fibrillation, microthrombi in the left ventricle, rheumatic valvular disease, or recent myocardial infarction. The embolism reduces the rate of blood flow to renal tissue and leads to ischemia. The rate and degree of blood flow reduction determine whether or not the insult will be acute or chronic as arterial narrowing progresses. Less common causes of renal infarction are atherosclerosis, with or without thrombus formation, and thrombus from flank trauma, sickle cell anemia, scleroderma, polyarteritis nodosa, and arterionephrosclerosis.

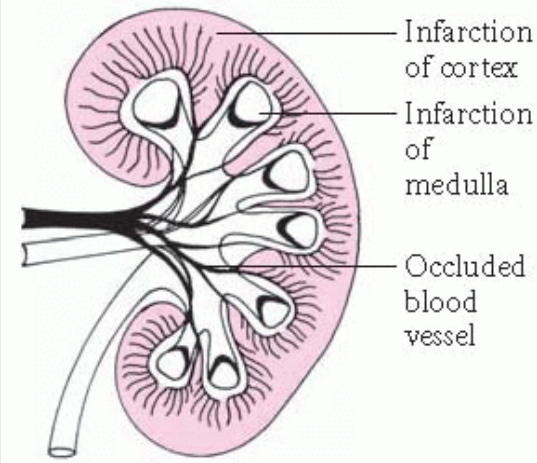
Complications

- End-stage renal disease
- Granular kidneys
- Severe hypertension
- Cardiac hypertrophy
- Heart failure

SITES OF RENAL INFARCTION

The most common sites of renal infarction are illustrated below.





Signs and symptoms

Although renal infarction may be asymptomatic, typical symptoms include severe upper abdominal pain or gnawing flank pain and tenderness, costovertebral tenderness, fever, anorexia, nausea, and vomiting. Gross hematuria may be present. When arterial occlusion causes infarction, the affected kidney is small and not palpable. Renovascular hypertension, a frequent complication that may occur several days after infarction, results from reduced blood flow, which stimulates the renin-angiotensin mechanism.

Diagnosis

A history of predisposing cardiovascular disease or other factors in a patient with typical clinical features strongly suggests renal infarction. Firm diagnosis requires appropriate laboratory tests:

- Urinalysis reveals proteinuria and microscopic hematuria.
- Urine enzyme levels, especially lactate dehydrogenase (LD) and alkaline phosphatase,

  are often elevated as a result of tissue destruction.
- Serum enzyme levels, especially aspartate aminotransferase, alkaline phosphatase, and LD, are elevated. Blood studies may also reveal leukocytosis and increased erythrocyte sedimentation rate.

- Excretory urography shows diminished or absent excretion of contrast dye, indicating vascular occlusion or urethral obstruction.
- Isotopic renal scan, a benign, noninvasive technique, demonstrates absent or reduced blood flow to the kidneys.

CONFIRMING DIAGNOSIS

Renal arteriography provides absolute proof of infarction but is used as a last resort because it's a high-risk procedure.

Treatment

Infection in the infarcted area or significant hypertension may require surgical repair of the occlusion or nephrectomy. Surgery to establish collateral circulation to the area can relieve renovascular hypertension. Persistent hypertension may respond to antihypertensives and a low-sodium diet. Additional treatments may include administration of intra-arterial streptokinase, lysis of blood clots, and heparin therapy. For bilateral emboli, intra-arterial streptokinase or transluminal angioplasty is recommended. If effective renal blood flow is obtained, long-term anticoagulation is recommended.

Special considerations

Assess the degree of renal function and offer supportive care to maintain homeostasis.

- Monitor intake and output, vital signs (particularly blood pressure), electrolytes, and daily weight. Watch for signs of fluid overload, such as dyspnea, tachycardia, pulmonary edema, and electrolyte imbalances.
- Carefully explain all diagnostic procedures.
- Provide reassurance and emotional support for the patient and his family.
- Encourage the patient to return for follow-up examination, which usually includes excretory urography or a renal scan to assess regained renal function.

Renal calculi

Renal calculi or nephrolithiasis (commonly called *kidney stones*) may form anywhere in the urinary tract but usually develop in the renal pelvis or the calyces of the kidneys. Calculi formation follows precipitation of substances normally dissolved in the urine, such as calcium oxalate, calcium phosphate, magnesium ammonium phosphate or, occasionally, urate or cystine. (See *How urine pH affects calculi formation*.) Renal calculi vary in size and may be solitary or multiple. They may remain in the renal pelvis or enter the ureter and may damage renal parenchyma; large calculi cause pressure necrosis. In certain locations, calculi cause obstruction, with resultant hydronephrosis, and tend to recur.

Causes and incidence

Although the exact cause of renal calculi is unknown, predisposing factors include:

- *Dehydration:* Decreased urine production concentrates calculus-forming substances.
- *Infection:* Infected, damaged tissue serves as a site for calculus development; pH changes provide a favorable medium for calculus formation (especially for magnesium ammonium phosphate or calcium phosphate calculi); or infected calculi (usually magnesium ammonium phosphate or staghorn calculi) may develop if bacteria serve as the nucleus in calculus formation. Infections may promote destruction of renal parenchyma.
- *Obstruction:* Urinary stasis (as in immobility from spinal cord injury) allows calculus constituents to collect and adhere, forming calculi. Obstruction also promotes infection, which, in turn, compounds the obstruction.
- *Metabolic factors:* These factors may predispose to renal calculi: hyperparathyroidism, renal tubular acidosis, elevated uric acid (usually with gout), defective metabolism of oxalate, genetic defect in metabolism of cystine, and excessive intake of vitamin D or dietary calcium.

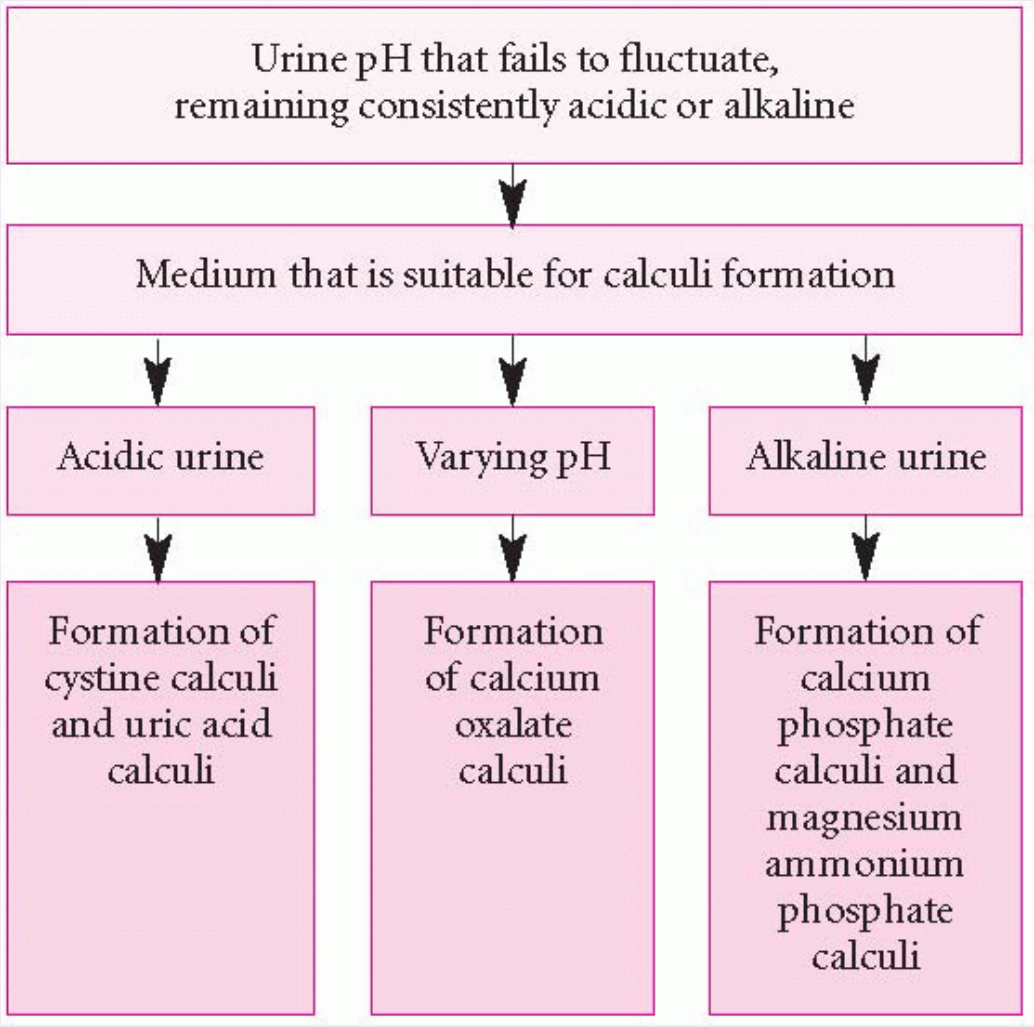
Among Americans, renal calculi develop in 2% to 10% of the population, with people living in southeastern states having an increased risk. They're more common in

men (especially those ages 30 to 40) than in women by a 3:1 ratio. They're rare in children.

PATHOPHYSIOLOGY

HOW URINE pH AFFECTS CALCULI FORMATION

The pH of the urine influences the types of calculi that may form.



Some types of calculi tend to be familial; some are associated with other conditions, such as bowel disease, ileal bypass for obesity, or renal

tubule defects. Calcium calculi are most common, accounting for over 75% of all calculi, and are two to three times more common in males, usually appearing between ages 20 and 30. The calcium may combine with other substances, such as oxalate (the most common substance), phosphate, or carbonate, to form the stone. Oxalate is present in certain foods. Diseases of the small intestine increase the tendency to form calcium oxalate calculi. Recurrence is likely.

Uric acid calculi are also more common in men and make up about 6% of all calculi. These calculi are associated with gout and chemotherapy. Cystine calculi, which make up about 2% of all calculi, may form in people with cystinuria, a hereditary disorder affecting both men and women. Struvite calculi, accounting for about 15% of all calculi, are mainly found in women as a result of a urinary tract infection (UTI). They can grow very large and may obstruct the kidney, ureter, or bladder.

Indavir stones appear in patients with human immunodeficiency virus who are treated with the protease inhibitor indinavir.

Complications

- Damage or destruction of renal parenchyma
- Pressure necrosis
- Hydronephrosis
- Bleeding

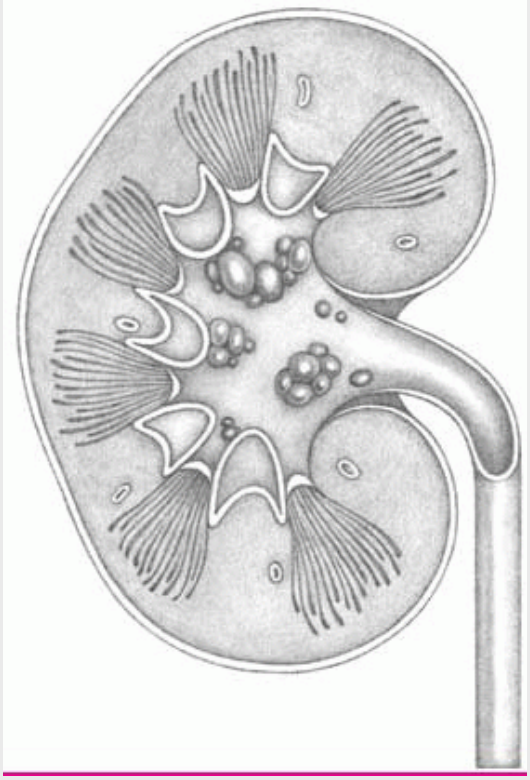
Signs and symptoms

Clinical effects vary with size, location, and etiology of the calculi. Pain, the key symptom, usually results from obstruction; large, rough calculi occlude the opening to the ureter and increase the frequency and force of peristaltic contractions. The pain of classic renal colic travels from the costovertebral angle to the flank, to the suprapubic region and external genitalia. The intensity

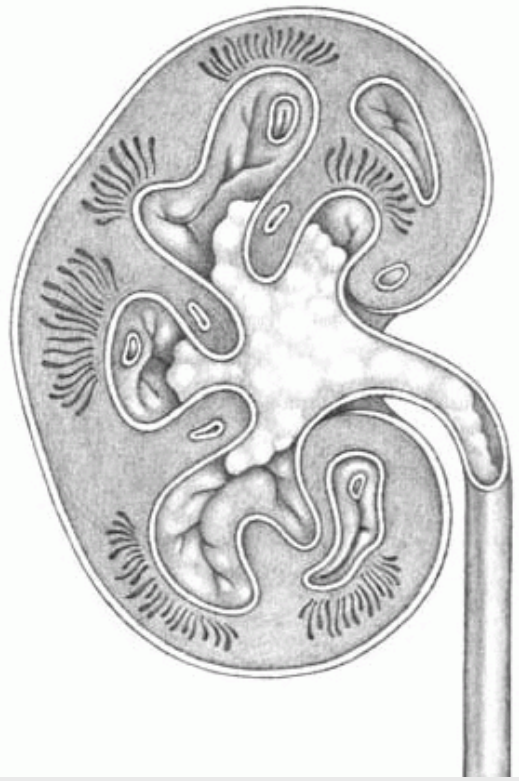
of this pain fluctuates and may be excruciating at its peak. If calculi are in the renal pelvis and calyces, pain may be more constant and dull. Back pain (from calculi that produce an obstruction within a kidney) and severe abdominal pain (from calculi traveling down a ureter) may also occur. (See *Types of renal calculi*.) Nausea and vomiting usually accompany severe pain.

TYPES OF RENAL CALCULI

Multiple small calculi may vary in size; they may remain in the renal pelvis or pass down the ureter.



A staghorn calculus (a cast of the calyceal and pelvic collecting system) may form from a stone that stays in the kidney.



Other associated signs include fever, chills, hematuria (when calculi abrade a ureter), abdominal distention, pyuria and, rarely, anuria (from bilateral obstruction, or unilateral obstruction in the patient with one kidney).

Diagnosis

Diagnosis is based on the clinical picture and the following tests:

- Computed tomography scan or magnetic resonance imaging are highly sensitive for identifying hydronephrosis and detecting small renal and urethral stones.
- Excretory urography may be used for diagnosis of obstruction by urinary calculus.
- Kidney-ureter-bladder X-rays reveal most renal calculi.
- Calculus analysis shows mineral content.

CONFIRMING DIAGNOSIS

Excretory urography confirms the diagnosis and determines size and location of calculi.

- Kidney ultrasonography is an easily performed, noninvasive, nontoxic test to detect obstructive changes such as hydronephrosis.
- Urine culture of midstream sample may indicate UTI.
- Urinalysis may be normal, or may show increased specific gravity and acid or alkaline pH suitable for different types of stone formation. Other urinalysis findings include hematuria (gross or microscopic), crystals (urate, calcium, or cystine), casts, and pyuria with or without bacteria and white blood cells.

- A 24-hour urine collection is evaluated for calcium oxalate, phosphorus, and uric acid excretion levels.
- Serial blood calcium and phosphorus levels detect hyperparathyroidism and show increased calcium level in proportion to normal serum protein.

PREVENTION

PREVENTING RENAL CALCULI

The formation of kidney stones may be prevented by making certain lifestyle changes. If these lifestyles changes don't help in the prevention of kidney stone formation, drugs are needed. Advise your patient to make these changes.

Maintain hydration

The most important lifestyle change is drinking plenty of fluids, especially water. An adult needs to drink at least 3½ quarts per day. Drinking plenty of fluids helps to flush out the kidneys.

Adjust diet

For a patient who tends to form calcium and oxalate stones, suggest an oxalaterestricted diet. Foods that are high in oxalate should be avoided. These foods include

rhubarb, star fruit, beets, beet greens, collards, okra, refried beans, spinach, swiss chard, sweet potatoes, sesame seeds, almonds, and soy products. Restricting calcium in the diet doesn't seem to reduce the risk of kidney stones. Diets high in calcium actually help reduce the risk of oxalate stone formation. Calcium binds with oxalate so it can't be absorbed in the gastrointestinal tract and excreted by the kidneys.

All patients who form kidney stones should be on low sodium diets. Sodium intake should be restricted to 3 to 4 grams sodium per day. Also, diets very low in animal proteins may help reduce the risk of stones.

Take prescribed drugs

Drugs can be helpful in preventing stone formation. The drugs prescribed depend on the type of stone. Some stones, such as calcium stones, develop more easily in more alkaline environments, and some stones, such as uric acid stones, develop in an acid environment. Patients with struvite stones are treated with antibiotics to prevent infection. Cystine stones are the hardest to treat, and they're treated with a urine alkalinizer and chelator (D-Penicillamine and Thiola).

Increased blood uric acid levels may indicate gout as the cause. Diagnosis must rule out appendicitis, cholecystitis, peptic ulcer, and pancreatitis as potential sources of pain.

Treatment

Because 90% of renal calculi are smaller than 5 mm in diameter, treatment usually consists of measures to promote their natural passage. Along with vigorous hydration, such treatment includes antimicrobial therapy (varying with the cultured organism) for infection, analgesics such as meperidine for pain, and diuretics to prevent urinary stasis and further calculus formation (thiazides decrease calcium excretion into the urine). Prophylaxis to prevent calculus formation includes a low-

calcium diet for absorptive hypercalciuria, parathyroidectomy for hyperparathyroidism, allopurinol for uric acid calculi, and daily administration of ascorbic acid by mouth to acidify the urine. (See *Preventing renal calculi*.)

Calculi too large for natural passage may require surgical removal. When a calculus is in the ureter, a cystoscope may be inserted through the urethra and the calculus manipulated with catheters or retrieval instruments. Extraction of calculi from other areas (kidney calyx, renal pelvis) may necessitate a flank or lower abdominal approach. Percutaneous ultrasonic lithotripsy and extracorporeal shock wave lithotripsy shatter the calculus into fragments for removal by suction or natural passage.

Special considerations

Patient care includes confirming the diagnosis, facilitating passage of the stone, and prevention of future occurrences.

- To aid diagnosis, maintain a 24- to 48-hour record of urine pH, with nitrazine pH paper; strain all urine through gauze or a tea strainer, and save all solid material recovered for analysis.
- To facilitate spontaneous passage, encourage the patient to walk if possible. Also promote sufficient intake of fluids to maintain a urine output of 3 to 4 L/day (urine should be very dilute and colorless). To help acidify urine, offer fruit juices, particularly cranberry juice. If the patient can't drink the required amount of fluid, supplemental I.V. fluids may be given. Record intake and output and daily weight to assess fluid status and renal function.
- Stress the importance of proper diet and compliance with drug therapy. For example, if the patient's stone is caused by a hyperuricemic condition, advise him (or whoever prepares his meals) to avoid foods high in purine. Restrict protein to 60 g/day to decrease calcium and uric acid, and limit sodium to 3 to 4 g/day. Oxalate foods are restricted.
- If surgery is necessary, give reassurance by supplementing and reinforcing what the surgeon has told the patient about the

procedure. The patient is apt to be fearful, especially if surgery includes removal of a kidney, so emphasize the fact that the body can adapt well to one kidney. If he's to have an abdominal or flank incision, teach deep-breathing and coughing exercises.

- After surgery, the patient will probably have an indwelling catheter or a nephrostomy tube. Unless one of his kidneys was removed, expect bloody drainage from the catheter. Never irrigate the catheter without a physician's order. Check dressings regularly for bloody drainage, and know how much drainage to expect. Immediately report suspected hemorrhage (excessive drainage, rising pulse rate). Use sterile technique when changing dressings or providing catheter care.
- Watch for signs of infection (rising fever, chills), and give antibiotics as ordered. To prevent pneumonia, encourage frequent position changes, and ambulate the patient as soon as possible. Have him hold a small pillow over the operative site to splint the incision and thereby facilitate deep-breathing and coughing exercises.
- Before discharge, teach the patient and his family the importance of following the prescribed dietary and medication regimens to prevent recurrence of calculi. Encourage increased fluid intake. If appropriate, show the patient how to check his urine pH, and instruct him to keep a daily record. Tell him to immediately report symptoms of acute obstruction (pain, inability to void).

Renal vein thrombosis

Renal vein thrombosis—clotting in the renal vein—results in renal congestion, engorgement and, possibly, infarction. Thrombosis may affect both kidneys and may occur in an acute or a chronic form. Chronic thrombosis usually impairs renal function, causing nephrotic syndrome. Abrupt onset of thrombosis that causes extensive damage may precipitate rapidly fatal renal infarction. If thrombosis affects both kidneys, the prognosis is poor. However, less severe thrombosis that affects only one kidney, or gradual progression that allows development of collateral circulation, may preserve partial renal function.

Causes and incidence

Renal vein thrombosis often results from a tumor that obstructs the renal vein (usually hypernephroma). Other causes include thrombophlebitis of the inferior vena cava (may result from abdominal trauma) or blood vessels of the legs, heart failure, and periarteritis.

PEDIATRIC TIP

In infants, renal vein thrombosis usually follows diarrhea that causes severe dehydration.

Chronic renal vein thrombosis is often a complication of other glomerulopathic diseases, such as amyloidosis, systemic lupus erythematosus, diabetic nephropathy, and membranoproliferative glomerulonephritis.

Complications

- Fatal renal infarction
- Disseminated intravascular coagulation

Signs and symptoms

Clinical features of renal vein thrombosis vary with speed of onset. Rapid onset of venous obstruction produces severe lumbar pain and tenderness in the epigastric region and the costovertebral angle. Other characteristic features include fever, leukocytosis, pallor, hematuria, proteinuria, peripheral edema and, when the obstruction is bilateral, oliguria and other uremic signs. The kidneys enlarge and become easily palpable. Hypertension is unusual but may develop.

Gradual onset causes symptoms of nephrotic syndrome. Peripheral edema is possible but pain is generally absent. Other clinical signs include proteinuria, hypoalbuminemia, and hyperlipidemia.

Infants with this disease have enlarged kidneys, oliguria, and renal insufficiency that may progress to acute or chronic renal failure.

Diagnosis

A variety of tests are used to confirm diagnosis of renal vein thrombosis.

- Computed tomography scan with contrast is the procedure of choice because it reveals enlargement and distention of the affected renal vein with visualization of the clots within the vein.
- Excretory urography provides reliable diagnostic evidence. In acute renal vein thrombosis, the kidneys appear enlarged and excretory function diminishes. Contrast medium seems to “smudge” necrotic renal tissue. In chronic thrombosis, it may show ureteral indentations that result from collateral venous channels.

CONFIRMING DIAGNOSIS

Renal arteriography and biopsy may also confirm the diagnosis. Venography confirms thrombosis.

- Urinalysis reveals gross or microscopic hematuria, proteinuria (more than 2 g/day in chronic disease), casts, and oliguria.
- Blood studies show leukocytosis, hypoalbuminemia, and hyperlipidemia.
- Magnetic resonance imaging, abdominal X-ray, or abdominal ultrasound may show occlusion of the renal vein.

Treatment

Treatment is most effective for gradual thrombosis that affects only one kidney. Anticoagulation therapy reduces the incidence of new thrombus formation and often reverses the deterioration of renal function. Heparin is the initial therapy of choice. After 5 to 7 days, warfarin (Coumadin) therapy can be instituted for long-term care. Streptokinase or urokinase infusion may be successful in early resolution of acute renal vein thrombosis. Surgery is rarely used; it must be performed within 24 hours of thrombosis but even then has limited success because thrombi often extend into the small veins. Extensive intrarenal bleeding may necessitate nephrectomy.

Patients who survive abrupt thrombosis with extensive renal damage develop nephrotic syndrome and require treatment for renal failure, such as dialysis and, possibly, transplantation.

PEDIATRIC TIP

Some infants with renal vein thrombosis recover completely following heparin therapy or surgery; others suffer irreversible kidney damage.

Special considerations

Patient care includes careful monitoring of symptoms for signs of improvement or worsening.

- Assess renal function regularly. Monitor vital signs, intake and output, daily weight, and electrolytes.
- Administer diuretics for edema as ordered, and enforce dietary restrictions of sodium and potassium intake.
- Monitor closely for signs of pulmonary emboli (chest pain, dyspnea).
- If heparin is given by constant I.V. infusion, frequently monitor partial thromboplastin time to determine the patient's response.

  Dilute the drug; administer it by infusion pump or controller, so the patient receives the least amount necessary.

ALERT

During anticoagulant therapy, watch for and report signs of bleeding, such as tachycardia, hypotension, hematuria, bleeding from nose or gums, ecchymoses, petechiae, and tarry stools. Instruct the patient on maintenance warfarin therapy to use an electric razor and a soft toothbrush, and to avoid trauma. Suggest that he wear a medical identification bracelet, and tell him to avoid aspirin, which aggravates bleeding tendencies. Stress the need for close follow-up.

CHRONIC RENAL DISORDERS

Nephrotic syndrome

Nephrotic syndrome is a condition characterized by marked proteinuria, hypoalbuminemia, hyperlipidemia, and edema. (See *What happens in nephrotic syndrome*.) Although nephrotic syndrome isn't a disease itself, it results from a specific glomerular defect and indicates renal damage. The prognosis is highly variable, depending on the underlying cause. Some forms may progress to end-stage renal failure.

Causes and incidence

About 75% of nephrotic syndrome cases result from primary (idiopathic) glomerulonephritis. Classifications include:

- In *lipid nephrosis (nil lesions),* the main cause of nephrotic syndrome in children, the glomerulus looks normal by light microscopy. Some tubules may contain increased lipid deposits.
- *Membranous glomerulonephritis,* the most common lesion in adult idiopathic nephrotic syndrome, is characterized by uniform thickening of the glomerular basement membrane containing dense deposits and eventually progresses to renal failure.
- *Focal glomerulosclerosis* can develop spontaneously at any age, follow renal transplantation, or result from heroin abuse. Reported incidence of this condition is 10% in children with nephrotic syndrome and up to 20% in adults. Lesions initially affect the deeper glomeruli, causing hyaline sclerosis, with later involvement of the superficial glomeruli. These lesions generally cause slowly progressive deterioration in renal function. Remissions occur occasionally.
- In *membranoproliferative glomerulonephritis,* slowly progressive lesions develop in the subendothelial region of the basement membrane. Lesions may follow infection, particularly streptococcal infection. This disease occurs primarily in children and young adults.

Other causes of nephrotic syndrome include metabolic diseases such as diabetes mellitus; collagen-vascular disorders, such as systemic lupus erythematosus and periarteritis nodosa; circulatory diseases, such as heart failure, sickle cell anemia, and renal vein thrombosis; nephrotoxins, such as mercury, gold, and bismuth; allergic reactions; and infections, such as tuberculosis or enteritis. Other possible causes are pregnancy, hereditary nephritis, multiple myeloma, and other

neoplastic diseases. These diseases increase glomerular protein permeability, leading to increased urinary excretion of protein, especially albumin, and subsequent hypoalbuminemia.

Nephrotic patients have an increased risk of infection, particularly of peritonitis.

PEDIATRIC TIP

Black children appear to be at greater risk for peritonitis.

Complications

- Atherosclerosis
- Renal vein thrombosis
- Acute renal failure
- Chronic renal failure
- Infections
- Malnutrition
- Fluid overload

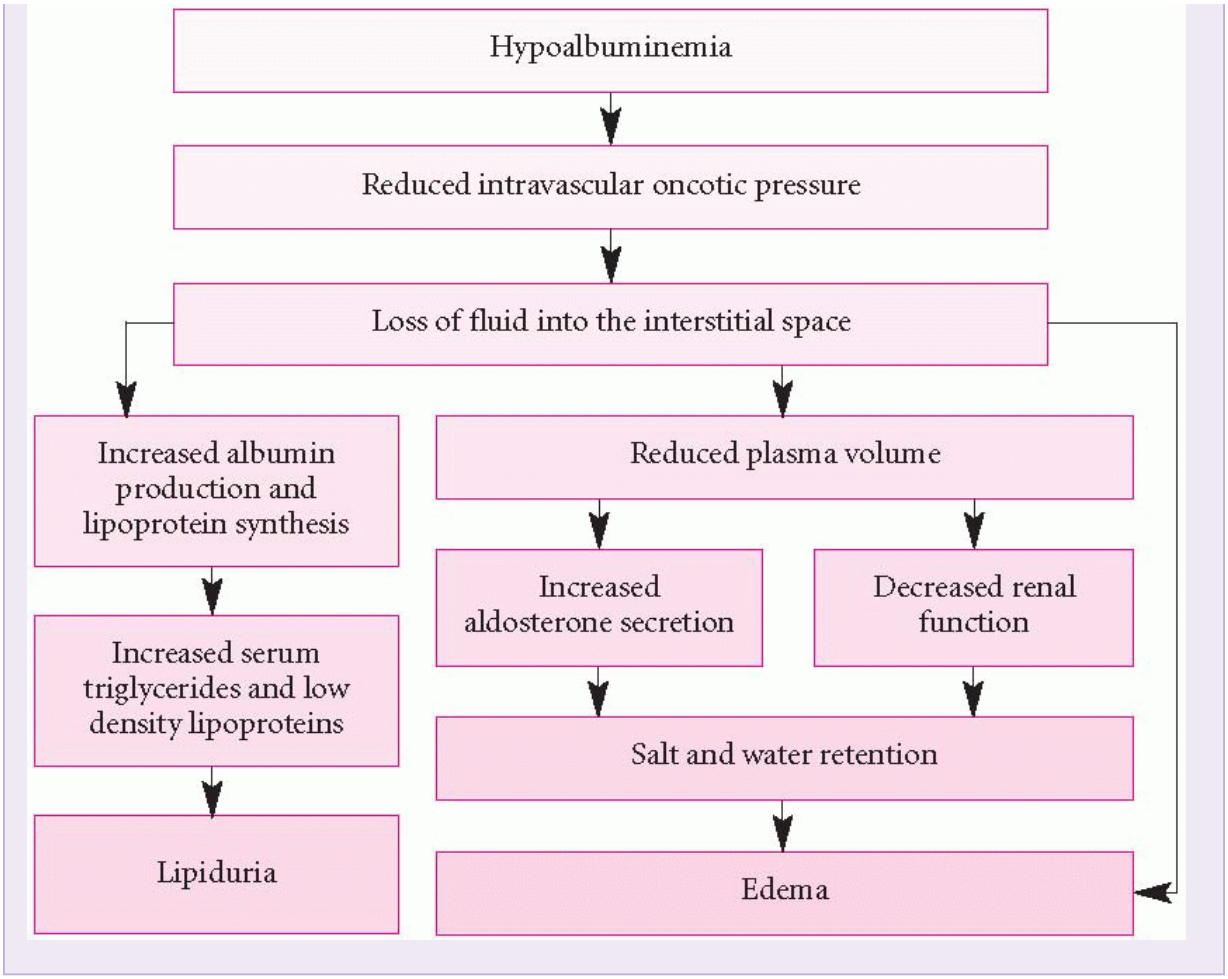
Signs and symptoms

The dominant clinical feature of nephrotic syndrome is mild to severe dependent edema of the ankles or sacrum, or periorbital edema, especially in children. Edema may

lead to ascites, pleural effusion, and swollen external genitalia. Accompanying symptoms may include orthostatic hypotension, lethargy, anorexia, depression, and pallor. Major complications are malnutrition, infection, coagulation disorders, thromboembolic vascular occlusion, and accelerated atherosclerosis.

PATHOPHYSIOLOGY

WHAT HAPPENS IN NEPHROTIC SYNDROME



Diagnosis

Consistent proteinuria in excess of 3.5 g for 24 hours strongly suggests nephrotic syndrome; examination of urine also reveals increased number of hyaline, granular, and waxy, fatty casts, and oval fat bodies. Serum values that support the diagnosis are increased cholesterol, phospholipids, and triglycerides and decreased albumin levels. Histologic identification of the lesion requires kidney biopsy. Other tests may be done to rule out metabolic causes.

Treatment

The goals of treatment of nephrotic syndrome are to relieve symptoms, prevent complications, and delay progressive kidney damage. Treatment of the causative disorder—possibly lifelong—is necessary to control nephrotic syndrome. Corticosteroid, immunosuppressive,

antihypertensive, and diuretic medications may help control symptoms. Antibiotics may be needed to control infections. Angiotensinconverting enzyme inhibitors may significantly reduce the degree of protein loss in urine and are therefore typically prescribed for the treatment of nephrotic syndrome.

Treatment of hypertension and of high cholesterol and triglyceride levels are also recommended to reduce the risk of atherosclerosis and complications. Dietary limitation of cholesterol and saturated fats may be of little benefit because the high levels

that accompany this condition seem to result from overproduction by the liver rather than from excessive fat intake. High-protein diets are of debatable value. In many patients, reducing the amount of protein in the diet produces a decrease in urine protein. In most cases, a moderateprotein diet (1 g/kg of body weight per day) is usually recommended. Sodium may be restricted to help control edema. Vitamin D may need to be replaced if nephrotic syndrome is chronic and unresponsive to therapy. Blood thinners may be required to treat or prevent clot formation.

Supportive treatment consists of protein replacement with infusion of salt-poor albumin or with a nutritional diet of 1.5 g protein/kg of body weight, with restricted sodium intake of 0.5 to 1 g/day; diuretics for edema; and antibiotics for infection.

Some patients respond to an 8-week course of corticosteroid therapy (such as prednisone), followed by a maintenance dose. Others respond better to a combination course of prednisone and azathioprine or cyclophosphamide.

Special considerations

Patient care includes identification and treatment of the underlying cause accompanied by supportive care during treatment.

- Frequently check urine protein. (Urine containing protein appears frothy.)
- Measure blood pressure while the patient is supine and also while he's standing; immediately report a drop in blood pressure that exceeds 20

mm Hg.

ALERT

After kidney biopsy, watch for bleeding and shock.

- Monitor intake and output and check weight at the same time each morning—after the patient voids and before he eats—and while he's wearing the same kind of clothing. Ask the dietitian to plan a high-protein, low-sodium diet.
- Provide good skin care because the patient with nephrotic syndrome usually has edema.
- To avoid thrombophlebitis, encourage activity and exercise, and provide antiembolism stockings as ordered.
- Watch for and teach the patient and his family how to recognize adverse drug effects, such as bone marrow toxicity from cytotoxic immunosuppressants and cushingoid symptoms (muscle weakness, mental changes, acne, moon face, hirsutism, girdle obesity, purple striae, bone fractures, amenorrhea) from long-term steroid therapy. Other steroid complications include masked infections, increased susceptibility to infections, ulcers, GI bleeding, and steroid-induced diabetes; a steroid crisis may occur if the drug is discontinued abruptly. To prevent GI complications, administer steroids with an antacid or with cimetidine or ranitidine. Explain that steroid adverse effects will subside when therapy stops.
- Offer the patient and his family reassurance and support, especially during the acute phase, when edema is severe and the patient's body image changes.

Chronic glomerulonephritis

A slowly progressive disease, chronic glomerulonephritis is characterized by inflammation of the glomeruli, which results in sclerosis, scarring, and eventual renal failure. This condition usually remains subclinical until the progressive phase begins, marked by proteinuria, cylindruria (presence of granular tube casts), and hematuria. By the time it produces symptoms, chronic glomerulonephritis is usually irreversible.

Causes and incidence

Common causes of chronic glomerulonephritis include primary renal disorders, such as membranoproliferative glomerulonephritis, membranous glomerulopathy, focal glomerulosclerosis, rapidly progressive glomerulonephritis and, less often, poststreptococcal glomerulonephritis. Systemic disorders that may cause chronic glomerulonephritis include lupus erythematosus, Goodpasture's syndrome, or hemolytic-uremic syndrome.

Chronic glomerulonephritis is twice as common in men as it is in women.

Complications

- End-stage renal disease
- Severe hypertension
- Heart failure
- Increased susceptibility to infections
- Nephrotic syndrome

Signs and symptoms

Chronic glomerulonephritis typically develops insidiously and asymptomatically, usually over many years. At any time, however, it may suddenly become progressive, producing nephrotic syndrome, hypertension, proteinuria, and hematuria. In late stages of progressive chronic glomerulonephritis, it may accelerate to uremic symptoms, such as azotemia, nausea, vomiting, pruritus, dyspnea, malaise, and fatigability. Mild to severe edema and anemia may accompany these symptoms. Severe hypertension may cause cardiac hypertrophy, leading to heart failure, and may accelerate the development of advanced renal failure, eventually necessitating dialysis or transplantation.

Diagnosis

Patient history and physical assessment seldom suggest glomerulonephritis. Suspicion develops from urinalysis revealing

proteinuria, hematuria, cylindruria, and red blood cell casts. Rising blood urea nitrogen and serum creatinine levels indicate advanced renal insufficiency. X-ray or ultrasound shows smaller kidneys. Kidney biopsy identifies the underlying disease and provides data needed to guide therapy.

Treatment

Treatment is essentially nonspecific and symptomatic, with its goals to control hypertension with antihypertensives and a sodium-restricted diet, to correct fluid and electrolyte imbalances through restrictions and replacement, to reduce edema with diuretics such as furosemide, and to prevent heart failure. Treatment may also include antibiotics (for symptomatic urinary tract infections [UTIs]), dialysis, or transplantation.

Special considerations

Patient care is primarily supportive, focusing on continual observation and sound patient teaching.

- Accurately monitor vital signs, intake and output, and daily weight to evaluate fluid retention. Observe for signs of fluid, electrolyte, and acid-base imbalances.
- Ask the dietitian to plan low-sodium, high-calorie meals with adequate protein.
- Administer medications as ordered, and provide good skin care (because of pruritus and edema) and oral hygiene. Instruct the patient to continue taking prescribed antihypertensives as scheduled, even if he's feeling better, and to report any adverse effects. Advise him to take diuretics in the morning, so he won't have to disrupt his sleep to void. Teach him how to assess ankle edema.
- Warn the patient to report signs of infection, particularly UTI, and to avoid contact with persons who have infections. Urge follow-up examinations to assess renal function.
- Help the patient adjust to this illness by encouraging him to express his feelings. Explain all necessary procedures beforehand, and answer the patient's questions about them.

Renovascular hypertension

Renovascular hypertension is a rise in systemic blood pressure resulting from stenosis of the major renal arteries or their branches or from intrarenal atherosclerosis. This narrowing or sclerosis may be partial or complete, and the resulting blood pressure elevation, benign or malignant.

Causes and incidence

Stenosis or occlusion of the renal artery stimulates the affected kidney to release the enzyme renin, which converts angiotensinogen —a plasma protein—to angiotensin I. As angiotensin I circulates through the lungs and liver, it converts to angiotensin II, which causes peripheral vasoconstriction, increased arterial pressure

and aldosterone secretion and, eventually, hypertension.

Atherosclerosis (especially in older men) and fibromuscular diseases of the renal artery wall layers—such as medial fibroplasia and, less commonly, intimal and subadventitial fibroplasia—are the primary causes in 95% of all patients with renovascular hypertension. Other causes include arteritis, anomalies of the renal arteries, embolism, trauma, tumor, and dissecting aneurysm. Less than 5% of patients with high blood pressure display renovascular hypertension; it's most common in persons younger than age 30 or older than age 50.

PEDIATRIC TIP

Fibromuscular dysplasia is the most common cause of renovascular hypertension in children. The surgical cure rate is very high.

Complications

- Heart failure
- Myocardial infarction
- Stroke

- Renal failure (occasionally)

Signs and symptoms

In addition to elevated systemic blood pressure, renovascular hypertension usually produces symptoms common to hypertensive states, such as headache, palpitations, tachycardia, anxiety, lightheadedness, decreased tolerance of temperature extremes, retinopathy, and mental sluggishness. Significant complications include heart failure, myocardial infarction, stroke and, occasionally, renal failure.

Diagnosis

Diagnosis is confirmed by the following tests:

CONFIRMING DIAGNOSIS

Arterial digital subtraction angiography with assays of venous renin is the definitive diagnostic procedure. When stenosis is significant, transluminal angioplasty can be done during the same procedure.

- Gadolinium enhanced magnetic resonance angiography can identify turbulent blood flow indicative of renal stenosis.
- Duplex Doppler ultrasonography scans the renal artery and will reveal stenosis but results vary.
- Oral captopril renography is the simplest, noninvasive test for detection of renovascular hypertension but has a relatively high false-positive rate.

Treatment

Surgery, the treatment of choice, is performed to restore adequate circulation and to control severe hypertension or severely impaired renal function by renal artery bypass, endarterectomy, arterioplasty or, as a last resort, nephrectomy. Balloon catheter renal artery dilation is used in selected cases to correct renal artery stenosis without the risks and

morbidity of surgery. Symptomatic measures include antihypertensives, diuretics, and a sodium-restricted diet.

Medications that may be used in an attempt to control blood pressure include diuretics, beta-adrenergic blockers, calcium channel blockers, angiotensin-converting enzyme inhibitors, angiotensin-receptor blockers, and alpha-adrenergic blockers. Diazoxide or nitroprusside may be given in the hospital if symptoms are acute. Response to medications is highly individual and the dosage or specific drug used may need frequent adjustment.

Lifestyle changes may be recommended, including weight, exercise, dietary adjustments, smoking cessation, and avoidance of alcohol. These habits add to the effects of hypertension in causing complications.

Special considerations

The care plan must emphasize helping the patient and his family understand renovascular hypertension and the importance of following the prescribed treatment.

- Accurately monitor intake and output and daily weight. Check blood pressure in both arms regularly, with the patient lying down and standing. A drop of 20 mm Hg or more on arising may necessitate an adjustment in antihypertensive medications. Assess renal function daily.
- Maintain fluid and sodium restrictions. Explain the purpose of a low-sodium diet.
- Explain the diagnostic tests, and prepare the patient appropriately; for example, adequately

  hydrate the patient before tests that use contrast media. Make sure the patient isn't allergic to the dye used in diagnostic tests. After excretory urography or arteriography, watch for complications.
- If a nephrectomy is necessary, reassure the patient that the remaining kidney is adequate for renal function.

ALERT

Postoperatively, watch for bleeding and hypotension. If the sutures around the renal vessels slip, the patient can quickly go into shock because kidneys receive 25% of cardiac output.

- Provide a quiet, stress-free environment if possible. Urge the patient and his family members to have regular blood pressure screenings.

PREVENTION

Preventing atherosclerosis can help prevent renovascular hypertension. To reduce the risk of atherosclerosis, give patients this advice.

- *Stop smoking because smoking damages the arteries.*
- *Get regular exercise. Regular exercise improves circulation and helps develop collateral blood vessels.*
- *Eat a healthy diet. Eating a diet low in saturated fats, cholesterol, and sodium can help control weight, blood pressure, and cholesterol.*
- *Manage stress. Some stress-reduction techniques include deep breathing and stretching exercises.*

Hydronephrosis

Hydronephrosis is an abnormal dilation of the renal pelvis and the calyces of one or both kidneys, caused by an obstruction of urine flow in the genitourinary tract. Although partial obstruction and hydronephrosis may not produce symptoms initially, the pressure built up behind the area of obstruction eventually results in symptomatic renal dysfunction.

Causes and incidence

Almost any type of obstructive uropathy can result in hydronephrosis. The most common causes are benign prostatic hyperplasia, urethral strictures, and calculi; less common causes include strictures or stenosis of the ureter or bladder outlet, congenital abnormalities, abdominal tumors, blood clots, and neurogenic bladder. If obstruction is in the

urethra or bladder, hydronephrosis is usually bilateral; if obstruction is in a ureter, it's usually unilateral. Obstructions distal to the bladder cause the bladder to dilate and act as a buffer zone, delaying hydronephrosis. Total obstruction of urine flow with dilation of the collecting system ultimately causes complete cortical atrophy and cessation of glomerular filtration.

Hydronephrosis occurs in 1 out of every 100 people.

Complications

- Pyelonephritis
- Paralytic ileus
- Renal failure

Signs and symptoms

Clinical features of hydronephrosis vary with the cause of the obstruction. In some patients, hydronephrosis produces no symptoms or only mild pain and slightly decreased urinary flow; in others, it may produce severe, colicky renal pain or dull flank pain that may radiate to the groin, and gross urinary abnormalities, such as hematuria, pyuria, dysuria, alternating oliguria and polyuria, or complete anuria. Other symptoms of hydronephrosis include nausea, vomiting, abdominal fullness, pain on urination, dribbling, or hesitancy. Unilateral obstruction may cause pain on only one side, usually in the flank area.

The most common complication of an obstructed kidney is infection (pyelonephritis) due to stasis that exacerbates renal damage and may create a life-threatening crisis. Paralytic ileus frequently accompanies acute obstructive uropathy.

Diagnosis

CONFIRMING DIAGNOSIS

While the patient's clinical features may suggest hydronephrosis, excretory urography, isotope renography (radioisotope scan of the kidneys), computed

tomography scan of the kidneys or abdomen, abdominal magnetic resonance imaging, renal ultrasound,

and renal function studies are necessary to confirm it.

Treatment

The goals of treatment are to preserve renal function and prevent infection through surgical removal of the obstruction, such as dilation for stricture of the urethra or prostatectomy for benign prostatic hyperplasia.

If renal function has already been affected, therapy may include a diet low in protein, sodium, and potassium. This diet is designed to stop the progression of renal failure before surgery. Inoperable obstructions may necessitate decompression and drainage of the kidney using a nephrostomy tube placed temporarily or permanently in the renal pelvis or placement of a ureteral stent to allow the ureter to drain. Concurrent infection requires appropriate antibiotic therapy.

Special considerations

Explain hydronephrosis as well as the purpose of excretory urography and other diagnostic procedures. Check the patient for allergy to excretory urography dye.

- Administer medication for pain, as needed and prescribed.
- Postoperatively, closely monitor intake and output, vital signs, and fluid and electrolyte status. Watch for a rising pulse rate and cold, clammy skin, which indicate possible impending hemorrhage and shock. Monitor renal function studies daily.
- If a nephrostomy tube has been inserted, check it frequently for bleeding and patency. Irrigate the tube only as ordered, and don't clamp it.
- If the patient is to be discharged with a nephrostomy tube in place, teach him how to care for it properly.

PREVENTION

To prevent progression of hydronephrosis to irreversible renal disease, urge older men (especially those with family histories of benign prostatic hyperplasia or prostatitis) to have routine medical checkups. Teach them to recognize and report symptoms of hydronephrosis (colicky pain, hematuria) or urinary tract infection.

Renal tubular acidosis

Renal tubular acidosis (RTA)—a syndrome of persistent dehydration, hyperchloremia, hypokalemia, metabolic acidosis, and nephrocalcinosis—results from the kidneys' inability to conserve bicarbonate. This disorder occurs as distal RTA (Type I, or classic RTA) or proximal RTA (Type II). The prognosis is usually good but depends on the severity of renal damage that precedes treatment.

Causes and incidence

Metabolic acidosis usually results from renal excretion of bicarbonate. However, metabolic acidosis associated with RTA results from a defect in the kidneys' normal tubular acidification of urine.

Distal RTA results from an inability of the distal tubule to secrete hydrogen ions against established gradients across the tubular membrane. This results in decreased excretion of titratable acids and ammonium, increased loss of potassium and bicarbonate in the urine, and systemic acidosis. Prolonged acidosis causes mobilization of calcium from bone and, eventually, hypercalciuria, predisposing the kidney to the formation of renal calculi. Distal RTA may be classified as primary or secondary.

- *Primary distal RTA* may occur sporadically or through a hereditary defect and is most prevalent in women, older children, adolescents, and young adults.

It can also be caused by certain drugs, such as amphotericin B, lithium, and analgesics.

- *Secondary distal RTA* has been linked to many renal or systemic conditions, such as starvation, malnutrition, hepatic cirrhosis, and several genetically transmitted disorders.

Proximal RTA results from defective reabsorption of bicarbonate in the proximal tubule. This causes bicarbonate to flood the distal tubule, which normally secretes hydrogen ions, and leads to impaired formation of titratable acids and ammonium for excretion. Ultimately, metabolic acidosis results. Proximal RTA occurs in two forms:

- In *primary proximal RTA*, the reabsorptive defect is idiopathic and is the only disorder present.
- In *secondary proximal RTA*, the reabsorptive defect may be one of several defects and is due to proximal tubular cell damage from a disease such as Fanconi's syndrome.

Complications

- Pyelonephritis
- Hypercalciuria
- Renal calculi

Signs and symptoms

PEDIATRIC TIP

In infants, RTA produces anorexia, vomiting, occasional fever, polyuria, dehydration, growth retardation, apathy, weakness, tissue wasting, constipation, nephrocalcinosis, and rickets.

In children and adults, RTA may lead to urinary tract infection, rickets, and growth problems. Possible complications of RTA include nephrocalcinosis and pyelonephritis.

Diagnosis

CONFIRMING DIAGNOSIS

Demonstration of impaired acidification of urine with systemic metabolic acidosis confirms distal RTA. Demonstration of bicarbonate wasting caused by impaired reabsorption confirms proximal RTA.

Other relevant laboratory results show:

- decreased serum bicarbonate, pH, potassium, and phosphorus
- increased serum chloride and alkaline phosphatase
- alkaline pH, with low titratable acids and ammonium content in urine; increased urinary bicarbonate and potassium; low specific gravity.

In later stages, X-rays may show nephrocalcinosis.

Treatment

Supportive treatment for patients with RTA requires replacement of those substances being abnormally excreted, especially bicarbonate, and may include sodium bicarbonate tablets or solution to control acidosis. Potassium may be given by mouth for dangerously low potassium levels. Vitamins D and calcium supplements are usually avoided because the tendency toward nephrocalcinosis persists even after bicarbonate therapy. If pyelonephritis occurs, treatment may include antibiotics as well.

Treatment for renal calculi secondary to nephrocalcinosis varies and may include supportive therapy until the calculi pass or until surgery for severe obstruction is performed.

Special considerations

Urge compliance with all medication instructions. Inform the patient and his family that the prognosis for RTA and bone lesion healing is directly related to the adequacy of treatment.

- Monitor laboratory values, especially potassium, for hypokalemia.
- Test urine for pH, and strain it for calculi.
- If rickets develops, explain the condition and its treatment to the patient and his family.

- Teach the patient how to recognize signs and symptoms of calculi (hematuria, low abdominal or flank pain). Tell him to immediately report signs and symptoms.
- Instruct the patient with low potassium levels to eat foods with a high potassium content, such as bananas, leafy green vegetables, and baked potatoes. Orange juice is also high in potassium.
- Because RTA may be caused by a genetic defect, encourage family members to seek genetic counseling or screening for this disorder.

Chronic renal failure

Chronic renal failure is usually the end result of a gradually progressive loss of renal function; occasionally, it's the result of a rapidly progressive disease of sudden onset. Few symptoms develop until after more than 75% of glomerular filtration is lost; then the remaining normal parenchyma deteriorates progressively, and symptoms worsen as renal function decreases.

If this condition continues unchecked, uremic toxins accumulate and produce potentially fatal physiologic changes in all major organ systems. If the patient can tolerate it, maintenance dialysis or kidney transplantation can sustain life. (See *Comparing peritoneal dialysis and hemodialysis*. Also see *Continuous ambulatory peritoneal dialysis,* page 408.)

Causes and incidence

Diabetes and hypertension are the primary causes of chronic renal failure, accounting for two-thirds of cases. Other causes of chronic renal failure include:

- chronic glomerular disease such as glomerulonephritis
- chronic infections, such as chronic pyelonephritis or tuberculosis
- congenital anomalies such as polycystic kidneys
- vascular diseases such as renal nephrosclerosis
- obstructive processes such as calculi

- collagen diseases such as systemic lupus erythematosus
- nephrotoxic agents such as long-term aminoglycoside therapy.

These conditions gradually destroy the nephrons and eventually cause irreversible renal failure. Similarly, acute renal failure that fails to respond to treatment becomes chronic renal failure.

This syndrome may progress through the following stages:

- reduced renal reserve (creatinine clearance glomerular filtration rate [GFR] is 40 to 70 ml/minute)
- renal insufficiency (GFR 20 to 40 ml/ minute)
- renal failure (GFR 10 to 20 ml/minute)
- end-stage renal disease (GFR less than 10 ml/minute).

Chronic renal failure and end-stage renal disease affect about 2 out of 1,000 people in the United States.

Complications

- Fatal physiologic changes in all major organs
- Anemia
- Peripheral neuropathy
- Cardiopulmonary complication
- GI complication
- Sexual dysfunction
- Dry skin
- Fractures

Signs and symptoms

Chronic renal failure produces major changes in all body systems:

- *Renal and urologic:* Initially, salt-wasting and consequent hyponatremia produce hypotension, dry mouth, loss of skin turgor, listlessness, fatigue, and nausea; later, somnolence and confusion

develop. As the number of functioning nephrons decreases, so does the kidneys' capacity to excrete sodium, resulting in salt retention and overload. Accumulation of potassium causes muscle irritability, then muscle weakness as the potassium level continues to rise. Fluid overload and metabolic acidosis also occur. Urinary output decreases; urine is very dilute and contains casts and crystals.

- *Cardiovascular:* Renal failure leads to hypertension, arrhythmias (including life-threatening ventricular tachycardia or fibrillation), cardiomyopathy, uremic pericarditis, pericardial effusion with possible cardiac tamponade, heart failure, and periorbital and peripheral edema.
- *Respiratory:* Pulmonary changes include reduced pulmonary macrophage activity with increased susceptibility to infection, pulmonary edema, pleuritic pain, pleural friction rub and effusions, crackles, thick sputum, uremic pleuritis and uremic lung (or uremic pneumonitis), dyspnea due to heart failure, and Kussmaul's respirations as a result of acidosis.
- *GI:* Inflammation and ulceration of GI mucosa cause stomatitis, gum ulceration and bleeding and, possibly, parotitis, esophagitis, gastritis, duodenal ulcers, lesions on the small and large bowel, uremic colitis, pancreatitis, and proctitis. Other GI symptoms include a metallic taste in the mouth, uremic fetor (ammonia smell to breath), anorexia, nausea, and vomiting.
- *Cutaneous:* Typically, the skin is pallid, yellowish bronze, dry, and scaly. Other cutaneous symptoms include severe itching; purpura; ecchymoses; petechiae; uremic frost (most often in critically ill or terminal patients); thin, brittle fingernails with characteristic lines; and dry, brittle hair that may change color and fall out easily.
- *Neurologic:* Restless leg syndrome, one of the first signs of peripheral neuropathy, causes pain, burning, and itching in the legs and feet, which may be relieved by voluntarily

shaking, moving, or rocking them. Eventually, this condition progresses to paresthesia and motor nerve dysfunction (usually bilateral footdrop) unless dialysis is initiated. Other signs and symptoms include muscle cramping and twitching, shortened memory and attention

span, apathy, drowsiness, irritability, confusion, coma, and seizures. EEG changes indicate metabolic encephalopathy.

- *Endocrine:* Common endocrine abnormalities include stunted growth patterns in children (even with elevated growth hormone levels), infertility and decreased libido in both sexes, amenorrhea and cessation of menses in females, and impotence, decreased sperm production, and testicular atrophy in males. Increased aldosterone secretion (related to increased renin production) and impaired carbohydrate metabolism (increased blood glucose levels similar to diabetes mellitus) may also occur.
- *Hematopoietic:* Anemia, decreased red blood cell (RBC) survival time, blood loss from dialysis and GI bleeding, mild thrombocytopenia, and platelet defects occur. Other problems include increased bleeding and clotting disorders, demonstrated by purpura, hemorrhage from body orifices, easy bruising, ecchymoses, and petechiae.
- *Skeletal:* Calcium-phosphorus imbalance and consequent parathyroid hormone imbalances cause muscle and bone pain, skeletal demineralization, pathologic fractures, and calcifications in the brain, eyes, gums, joints, myocardium, and blood vessels. Arterial calcification may produce coronary artery disease. In children, renal osteodystrophy (renal rickets) may develop.

COMPARING PERITONEAL DIALYSIS AND HEMODIALYSIS

Advantages, disadvantages, and complications of peritoneal dialysis and hemodialysis are described below.

Type	Advantages	Disadvantages	Possible complications
Peritoneal dialysis	▪ Can be performed immediately ▪ Requires less complex equipment and less specialized personnel than hemodialysis ▪ Requires small amounts of heparin or none at all	▪ Contraindicated within 72 hours of abdominal surgery ▪ Requires 48 to 72 hours for significant response to treatment ▪ Severe protein loss necessitates high-	▪ Bacterial or chemical peritonitis ▪ Pain (abdominal, low back, shoulder) ▪ Shortness of breath, or dyspnea ▪ Atelectasis and pneumonia

	▪ No blood loss; minimal cardiovascular stress ▪ Can be performed by patient anywhere (continuous ambulatory peritoneal dialysis), without assistance and with minimal patient teaching ▪ Allows patient independence without long interruptions in daily activities because exchange may be done at night while he sleeps ▪ Lower infection rate ▪ Lower cost	protein diet (up to 100 g/day) ▪ High risk of peritonitis; repeated bouts may cause scarring, preventing further treatments with peritoneal dialysis ▪ Urea clearance less than with hemodialysis (60%)	▪ Severe loss of protein into the dialysis solution in the abdominal cavity (10 to 20 g/day) ▪ Fluid overload ▪ Excessive fluid loss ▪ Constipation ▪ Catheter site inflammation, infection, or leakage ▪ Anorexia ▪ Hypertriglyceridemia ▪ Abdominal hernias
Hemodialysis	▪ Takes only 3 to 5 hours per treatment ▪ Faster results in an acute situation ▪ Total number of hours of maintenance treatment that's only half that of peritoneal dialysis ▪ In an acute situation, can use an I.V. route without a surgical access route	▪ Requires surgical creation of a vascular access between circulation and dialysis machine ▪ Requires complex water treatment, dialysis equipment, and highly trained personnel ▪ Requires administration of larger amounts of heparin ▪ Confines patient to special treatment unit	▪ Septicemia ▪ Air emboli ▪ Rapid fluid and electrolyte imbalance (disequilibrium syndrome) ▪ Hemolytic anemia ▪ Metastatic calcification ▪ Increased risk of hepatitis ▪ Hypotension or hypertension ▪ Itching ▪ Pain (generalized or in chest) ▪ Heparin overdose, possibly causing hemorrhage ▪ Leg cramps ▪ Nausea and vomiting

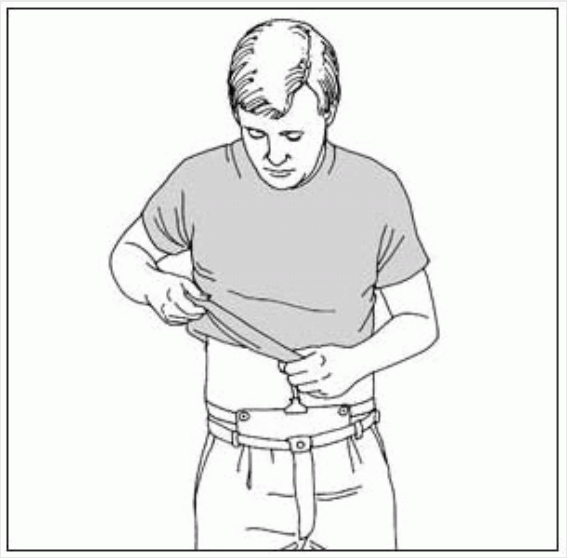
			▪ Headache

CONTINUOUS AMBULATORY PERITONEAL DIALYSIS

Continuous ambulatory peritoneal dialysis is a useful alternative to hemodialysis in patients with renal failure. Using the peritoneum as a dialysis membrane, it allows almost uninterrupted exchange of dialysis solution. With this method, four to six exchanges of fresh dialysis solution are infused each day. The approximate dwell-time for daytime exchanges is 5 hours; for overnight exchanges, the dwell-time is 8 to 10 hours. After each dwell-time, the patient removes the dialyzing solution by gravity drainage. This form of dialysis offers the unique advantages of a simple, easily taught procedure and patient independence from a special treatment center.

After applying a mask, the patient attaches a bag of dialysate to the tube entering his abdominal area so that the fluid flows into the peritoneal cavity. The patient can remove the mask when this step is completed.

While the dialysate remains in the peritoneal cavity, the patient rolls up the bag, places it under his shirt, and goes about his normal activities.



After applying a mask, the patient unrolls the bag and suspends it below his pelvis to allow the dialysate to drain from the peritoneal cavity back into the bag.

Diagnosis

Diagnosis of chronic renal failure is based on clinical assessment, a history of chronic progressive debilitation, and gradual deterioration of renal function as determined by creatinine clearance tests. The following laboratory findings also aid in diagnosis:

- Blood studies show elevated blood urea nitrogen, serum creatinine, and potassium levels; decreased arterial pH and bicarbonate; and low hemoglobin (Hb) level and hematocrit (HCT).
- Urine specific gravity becomes fixed at 1.010; urinalysis may show proteinuria, glycosuria, erythrocytes, leukocytes, and casts, depending on the etiology.
- X-ray studies include kidney-ureter-bladder films, excretory urography, nephrotomography, renal scan, and renal arteriography.

- Renal or abdominal computed tomography scan, magnetic resonance imaging, or ultrasound indicate changes associated with chronic renal failure, including abnormally small size in both kidneys.
- Kidney biopsy allows histologic identification of the underlying pathology.

Treatment

Treatment focuses on controlling the symptoms, minimizing complications, and slowing the progression of the disease. Associated diseases that cause or result from chronic renal failure must be controlled such as hypertension. Conservative treatment aims to correct specific symptoms. A low-protein diet reduces the production of end products of protein metabolism that the kidneys can't excrete. (A patient receiving continuous peritoneal dialysis should have a high-protein diet.) A high-calorie diet prevents ketoacidosis and the negative nitrogen balance that results in catabolism and tissue atrophy, and restricts sodium and potassium.

Maintaining fluid balance requires careful monitoring of vital signs, weight changes, and urine volume (if present). If some renal function remains, administration of loop diuretics such as furosemide, and fluid restriction can reduce fluid retention. Cardiac glycosides may be used to mobilize edema fluids; antihypertensives, to control blood pressure and associated edema. Antiemetics taken before meals may relieve nausea and vomiting; cimetidine or ranitidine may decrease gastric irritation. Methylcellulose or docusate can help prevent constipation.

Treatment may also include regular stool analysis (guaiac test) to detect occult blood and, as needed, cleaning enemas to remove blood from the GI tract. Anemia necessitates iron and folate supplements; severe anemia requires infusion of fresh frozen packed cells or washed packed cells. However, transfusions relieve anemia only temporarily.

Epoetin alpha (erythropoietin) increases RBC production.

Drug therapy often relieves associated symptoms: an antipruritic, such as trimeprazine or diphenhydramine, for itching and aluminum hydroxide gel to lower serum phosphate levels. The patient may also

benefit from supplementary vitamins (particularly B vitamins and vitamin D) and essential amino acids.

ALERT

Careful monitoring of serum potassium levels is necessary to detect hyperkalemia. Emergency treatment for severe hyperkalemia includes dialysis therapy and administration of 50% hypertonic glucose I.V., regular insulin, calcium gluconate I.V., sodium bicarbonate I.V., and cation exchange resins such as sodium polystyrene sulfonate.

ALERT

Cardiac tamponade resulting from pericardial effusion may require emergency pericardial tap or surgery.

Blood gas measurements may indicate acidosis; intensive dialysis and thoracentesis can relieve pulmonary edema and pleural effusions.

Hemodialysis or peritoneal dialysis (particularly continuous ambulatory peritoneal dialysis and continuous cyclic peritoneal dialysis) can help control most manifestations of end-stage renal disease; altering dialyzing bath fluids can correct fluid and electrolyte disturbances. But anemia, peripheral neuropathy, cardiopulmonary and GI complications, sexual dysfunction, and skeletal defects may persist. Maintenance dialysis itself may produce complications, such as protein wasting, refractory ascites, and dialysis dementia. Kidney transplantation may eventually be the treatment of choice for some patients with end-stage renal disease.

PEDIATRIC TIP

Children require more dialysis in relation to their body weight than adults because their metabolic rates and, therefore, food intake, are higher.

Special considerations

Because chronic renal failure has such widespread clinical effects, it requires meticulous and carefully coordinated supportive care.

- Good skin care is important. Bathe the patient daily, using superfatted soaps, oatmeal baths, and skin lotion without alcohol to ease pruritus. Don't use glycerincontaining soaps because they'll cause skin drying. Give good perineal care, using mild soap and water. Pad the side rails to guard against ecchymoses. Turn the patient often, and use a convoluted foam mattress to prevent skin breakdown.
- Provide good oral hygiene. Brush the patient's teeth often with a soft brush or sponge tip to reduce breath odor. Sugarless hard candy and mouthwash minimize bad taste in the mouth and alleviate thirst.
- Offer small, palatable meals that are also nutritious; try to provide favorite foods within dietary restrictions. Encourage intake of high-calorie foods. Instruct the outpatient to avoid high-sodium foods and high-potassium foods. Encourage adherence to fluid and protein restrictions. To prevent constipation, stress the need for exercise and sufficient dietary bulk.
- Watch for hyperkalemia. Observe for cramping of the legs and abdomen, and diarrhea. As potassium levels rise, watch for muscle irritability and a weak pulse rate. Monitor the electrocardiogram for tall, peaked T waves, widening QRS segment, prolonged PR interval, and disappearance of P waves, indicating hyperkalemia.
- Assess hydration status carefully. Check for jugular vein distention, and auscultate the lungs for crackles. Measure daily intake and output carefully, including all drainage, emesis, diarrhea, and blood loss. Record daily weight, presence or absence of thirst, axillary sweat, dryness of tongue, hypertension, and peripheral edema.
- Monitor for bone or joint complications. Prevent pathologic fractures by turning the patient carefully and ensuring his safety. Provide passive range-of-motion exercises for the bedridden patient.
- Encourage deep breathing and coughing to prevent pulmonary congestion. Listen often for crackles, rhonchi, and decreased breath sounds. Be alert for clinical effects of pulmonary edema (dyspnea, restlessness, crackles). Administer diuretics and other medications, as ordered.

- Maintain strict sterile technique. Use a micropore filter during I.V. therapy. Watch

  for signs of infection (listlessness, high fever, and leukocytosis). Urge the outpatient to avoid contact with infected persons during the cold and flu season.
- Carefully observe and document seizure activity. Infuse sodium bicarbonate for acidosis, and sedatives or anticonvulsants for seizures, as ordered. Pad the side rails and keep an oral airway and suction setup at bedside. Assess neurologic status periodically, and check for Chvostek's and Trousseau's signs, indicators of low serum calcium levels.
- Observe for signs of bleeding. Watch for prolonged bleeding at puncture sites and at the vascular access site used for hemodialysis. Monitor Hb levels and HCT, and check stool, urine, and vomitus for blood.
- Report signs of pericarditis, such as a pericardial friction rub and chest pain.

ALERT
Watch for the disappearance of friction rub, with a drop of 15 to 20 mm Hg in blood pressure during inspiration (paradoxical pulse)—an early sign of pericardial tamponade.

- Schedule medications carefully. Give iron before meals, aluminum hydroxide gels after meals, and antiemetics, as necessary, a half hour before meals. Administer antihypertensives at appropriate intervals. If the patient requires a rectal infusion of sodium polystyrene sulfonate for dangerously high potassium levels, apply an emollient to soothe the perianal area. Be sure the sodium polystyrene sulfonate enema is expelled; otherwise, it will cause constipation and won't lower potassium levels. Recommend antacid cookies as an alternative to aluminum hydroxide gels needed to bind GI phosphate.

If the patient requires dialysis:

- Prepare the patient by fully explaining the procedure. Be sure that he understands how to protect and care for the arteriovenous shunt, fistula, or other vascular access. Check the vascular access site per facility protocol or every 2 hours for patency and check the limb for adequate blood supply and intact nervous function (temperature, pulse rate, capillary refill, and sensation). If a fistula is present, feel for a thrill and listen for a bruit. Use a gentle touch to avoid occluding the fistula. Report signs of possible clotting. Don't use the arm with the vascular access site to take blood pressure readings, draw blood, or give injections as these procedures may rupture the fistula or occlude blood flow.
- Withhold the 6 a.m. (or morning) dose of antihypertensive on the morning of dialysis, and instruct the outpatient to do the same.
- Use standard precautions when handling body fluids and needles.
- Monitor Hb levels and HCT. Assess the patient's tolerance of his levels. Some individuals are more sensitive to lower levels than others. Instruct the anemic patient to conserve energy and to rest frequently.
- After dialysis, check for disequilibrium syndrome, a result of sudden correction of blood chemistry abnormalities. Symptoms range from a headache to seizures. Also, check for excessive bleeding from the dialysis site. Apply pressure dressing or absorbable gelatin sponge, as indicated. Monitor blood pressure carefully after dialysis.
- A patient undergoing dialysis is under a great deal of stress, as is his family. Refer them to appropriate counseling agencies for assistance in coping with chronic renal failure.

LOWER URINARY TRACT DISORDERS

Lower urinary tract infection

Cystitis and urethritis, the two forms of lower urinary tract infection (UTI), are nearly 10 times more common in women than in men and affect about 10% to 20% of all women at least once. Lower UTI is also a prevalent bacterial disease in children, with girls again most commonly affected. In men and children, lower UTIs are frequently related to anatomic or physiologic abnormalities and therefore require extremely

close evaluation. UTIs often respond readily to treatment but recurrence and resistant bacterial flare-up during therapy are possible.

PEDIATRIC TIP

Have a work-up done on all children with proven UTI to exclude an abnormality of the urinary tract that would predispose them to renal damage.

Causes and incidence

Most lower UTIs result from ascending infection by a single, gram-negative, enteric bacterium, such as *Escherichia coli, Klebsiella, Proteus, Enterobacter, Pseudomonas,* or *Serratia*. However, in a patient with neurogenic bladder, an indwelling catheter, or a fistula between the intestine and bladder, lower UTI may result from simultaneous infection with multiple pathogens. Infection can result from a breakdown in local defense mechanisms in the bladder that allow bacteria to invade the bladder mucosa and multiply. These bacteria can't be readily eliminated by normal micturition.

Bacterial flare-up during treatment is generally caused by the pathogenic organism's resistance to the prescribed antimicrobial therapy. The presence of even a small number (less than 10,000/µl) of bacteria in a midstream urine sample obtained during treatment casts doubt on the effectiveness of treatment.

In 99% of patients, recurrent lower UTI results from reinfection by the same organism or from some new pathogen; in the remaining 1%, recurrence reflects persistent infection, usually from renal calculi, chronic bacterial prostatitis, or a structural anomaly that may become a source of infection.

The high incidence of lower UTI among women may result from the shortness of the female urethra (1¼" to 2" [3 to 5 cm]), which predisposes women to infection caused by bacteria from the vagina, perineum, rectum, or a sexual partner. (See *Preventing UTIs*.) Men are less vulnerable because their urethras are longer (7¼" [18.4 cm]) and because prostatic fluid serves as an antibacterial shield. However, in men older than age 60, incidence rates match those of women. In both

men and women, infection usually ascends from the urethra to the bladder.

ELDER TIP

As a person ages, the bladder muscles weaken, which may result in incomplete bladder emptying and chronic urine retention—factors that predispose the older person to bladder infections.

Complications

- Chronic or recurrent UTI
- Complicated UTI
- Kidney infection

Signs and symptoms

Lower UTI usually produces urgency, frequency, dysuria, cramps or spasms of the bladder, itching, a feeling of warmth during urination, nocturia, and possibly urethral discharge in males. Inflammation of the bladder wall also causes hematuria and fever. Other common features include low back pain, malaise, nausea, vomiting, abdominal pain or tenderness over the bladder area, chills, and flank pain.

ELDER TIP

The most common initial symptoms of lower UTI in elderly patients are lethargy and a change in mental status.

Diagnosis

Characteristic clinical features and a microscopic urinalysis showing red blood cells and white blood cells greater than 10/high-power field suggest lower UTI.

CONFIRMING DIAGNOSIS

A clean-catch midstream urine specimen revealing a bacterial count above 100,000/µl confirms the diagnosis.

Lower counts don't necessarily rule out infection, especially if the patient is voiding frequently because bacteria require 30 to 45 minutes to reproduce in urine. Careful midstream, clean-catch collection is preferred to catheterization, which can reinfect the bladder with urethral bacteria.

Sensitivity testing determines the appropriate therapeutic antimicrobial. If patient history and physical examination warrant, a blood test or a stained smear of the discharge rules out venereal disease. Voiding cystoureterography or excretory urography may detect congenital anomalies that predispose the patient to recurrent UTIs.

Treatment

Appropriate antimicrobials are the treatment of choice for most initial lower UTIs. A course of antibiotic therapy lasting from

7 to 10 days is standard, but recent studies suggest that a single dose of an antibiotic or an antibiotic regimen of 3 to 5 days length may be sufficient to render the urine sterile. After 3 days of antibiotic therapy, urine culture should show no organisms. If the urine isn't sterile, bacterial resistance has probably occurred, making the use of a different antimicrobial necessary. Single-dose antibiotic therapy with amoxicillin or co-trimoxazole may be effective in females with acute noncomplicated UTI. A urine culture taken 1 to 2 weeks later indicates whether or not the infection has been eradicated.

PREVENTION

PREVENTING UTIs

Following a few simple steps can help reduce the risk of developing a urinary tract infection (UTI). Women in particular may benefit from these suggestions.

Maintain hydration

Suggest drinking plenty of liquids, especially water. Cranberry juice may have infection-fighting properties.

Urinate promptly

Tell the patient to urinate promptly when the urge arises; urination shouldn't be restricted for a long time after the urge to void is felt.

Maintain hygiene

Wiping from front to back after urinating and after a bowel movement helps prevent bacteria in the anal region from spreading to the vagina and urethra. Also, after intercourse, the bladder should be emptied as soon as possible. Drinking a glass of water also helps flush bacteria.

Avoid irritation

Use of deodorant sprays or other feminine products, such as douches and powders, should be avoided because they can irritate the urethra.

Recurrent infections due to infected renal calculi, chronic prostatitis, or structural abnormality may necessitate surgery; prostatitis also requires long-term antibiotic therapy. In patients without these predisposing conditions, long-term, low-dosage antibiotic therapy is the treatment of choice.

PEDIATRIC TIP

Fluoroquinolones aren't used for children because of possible adverse effects on developing cartilage.

Special considerations

The care plan should include careful patient teaching, supportive measures, and proper specimen collection.

- Explain the nature and purpose of antimicrobial therapy. Emphasize the importance of completing the prescribed course of therapy or, with long-term prophylaxis, of adhering strictly to the ordered dosage. Urge the patient to drink plenty of water (at least eight glasses a day). Stress the need to maintain a consistent fluid intake of about 2

qt (2 L)/day. More or less than this amount may alter the effect of the prescribed antimicrobial. Fruit juices, especially cranberry juice, and oral doses of vitamin C may help acidify the urine and enhance the action of the medication.

- Watch for GI disturbances from antimicrobial therapy. Nitrofurantoin macrocrystals, taken with milk or a meal, prevent such distress. If therapy includes phenazopyridine, warn the patient that this drug may turn urine red-orange.
- Suggest warm sitz baths for relief of perineal discomfort. If baths aren't effective, apply heat sparingly to the perineum but be careful not to burn the patient. Apply topical antiseptics, such as povidone-iodine ointment, on the urethral meatus as necessary.

- Collect all urine samples for culture and sensitivity testing carefully and promptly. Teach a woman how to clean the perineum properly and keep the labia separated during voiding. A noncontaminated midstream specimen is essential for accurate diagnosis.
- To prevent recurrent lower UTIs, teach a woman to carefully wipe the perineum from front to back and to clean it thoroughly with soap and water after defecation. Advise an infection-prone woman to void immediately after sexual intercourse. Stress the need to drink plenty of fluids routinely and to avoid postponing urination. Recommend frequent comfort stops during long car trips. Also stress the need to completely empty the bladder. To prevent recurrent infections in men, urge prompt treatment of predisposing conditions such as chronic prostatitis. Have the patient use a commode rather than a bedpan to promote sitting up, which assists in emptying the bladder.

Vesicoureteral reflux

In vesicoureteral reflux, urine flows from the bladder back into the ureters and eventually into the renal pelvis or the parenchyma. Because the bladder empties poorly, urinary tract infection (UTI) may result, possibly leading to acute or chronic pyelonephritis with renal damage.

Vesicoureteral reflux is most common during infancy in boys and during early childhood (ages 3 to 7) in girls. Primary vesicoureteral reflux that

results from congenital anomalies is most prevalent in females and is rare in blacks. Up to 25% of asymptomatic siblings of children with diagnosed primary vesicoureteral reflux also show reflux.

Causes and incidence

In patients with vesicoureteral reflux, incompetence of the ureterovesical junction and shortening of intravesical ureteral musculature allow backflow of urine into the ureter when the bladder contracts during voiding. Incompetence may result from congenital anomalies of the ureters or bladder, including short or absent intravesical ureter, ureteral ectopia lateralis (greater-than-normal lateral placement of ureters), and gaping ureteral orifice; inadequate detrusor muscle buttress in the bladder, stemming from congenital paraureteral bladder diverticulum; acquired diverticulum (from outlet obstruction); flaccid neurogenic bladder; and high intravesical pressure from outlet obstruction or an unknown cause. Vesicoureteral reflux may also result from cystitis, with inflammation of the intravesical ureter, which causes edema and fixation of the intramural ureter and usually leads to reflux in persons with congenital ureteral or bladder anomalies or other predisposing conditions.

Reflux nephropathy occurs in about 4 out of 1,000 asymptomatic people. However, in infants and children who experience UTIs, its prevalence approaches 40% to 50%. Reflux nephropathy may lead to chronic renal failure and end-stage renal disease.

Complications

- Recurrent UTI
- Chronic pyelonephritis
- Renal scarring
- Hypertension

Signs and symptoms

Vesicoureteral reflux typically manifests itself as the signs and symptoms of UTI: frequency, urgency, burning on urination, hematuria, foul-

smelling urine and, in infants, dark, concentrated urine. With upper urinary tract involvement, signs and symptoms usually include high fever, chills, flank pain, vomiting, and malaise.

PEDIATRIC TIP

In children, fever, nonspecific abdominal pain, and diarrhea may be the only clinical effects. Rarely, children with minimal symptoms remain undiagnosed until puberty or later, when they begin to exhibit clear signs of renal impairment (anemia, hypertension, and lethargy).

Diagnosis

Symptoms of UTI provide the first clues to diagnosis of vesicoureteral reflux. In infants, hematuria or strong-smelling urine may be the first indication; palpation may

reveal a hard, thickened bladder (hard mass deep in the pelvis) if posterior urethral valves are causing an obstruction in male infants.

Cystoscopy, with instillation of a solution containing methylene blue or indigo carmine dye, may confirm the diagnosis. After the bladder is emptied and refilled with clear sterile water, color-tinged efflux from either ureter positively confirms reflux.

Other pertinent laboratory studies include the following:

- Clean-catch urinalysis shows a bacterial count greater than 100,000/µl. Microscopic examination may reveal white blood cells, red blood cells, and an increased urine pH in the presence of infection. Specific gravity less than 1.010 demonstrates inability to concentrate urine.
- Laboratory studies reveal elevated creatinine levels (more than 1.2 mg/dl) and elevated blood urea nitrogen levels (more than 18 mg/dl), indicating advanced renal dysfunction.
- Excretory urography may show dilated lower ureter, ureter visible for its entire length, hydronephrosis, calyceal distortion, and renal scarring.

- Voiding cystourethrography (either fluoroscopic or radionuclide) identifies and determines the degree of reflux and shows when reflux occurs. It may also pinpoint the causative anomaly. In this procedure, contrast material is instilled into the bladder, and X-rays are taken before, during, and after voiding. Nuclear cystography and renal ultrasound may also be used to detect reflux.
- Abdominal computed tomography scan or ultrasound of the kidneys or abdomen shows hydronephrosis, reflux, a small kidney, or scarring.
- Catheterization of the bladder after the patient voids determines the amount of residual urine.

Treatment

The goal of treatment in a patient with vesicoureteral reflux is to prevent pyelonephritis and renal dysfunction with antibiotic therapy and, when necessary, vesicoureteral reimplantation. Appropriate surgical procedures create a normal valve effect at the junction by reimplanting the ureter into the bladder wall at a more oblique angle.

Antimicrobial therapy is usually effective for reflux that's secondary to infection, reflux related to neurogenic bladder and, in children, reflux related to a short intravesical ureter (which abates spontaneously with growth). Reflux related to infection generally subsides after the infection is cured. However, 80% of females with vesicoureteral reflux will have recurrent UTIs within a year. Recurrent infection requires long-term prophylactic antibiotic therapy and careful patient follow-up (cystoscopy and excretory urography every 4 to 6 months) to track the degree of reflux.

UTI that recurs despite adequate prophylactic antibiotic therapy necessitates vesicoureteral reimplantation or reconstructive repair. Bladder outlet obstruction in neurogenic bladder requires surgery only if renal dysfunction is present. After surgery, as after antibiotic therapy, close medical follow-up is necessary (excretory urography every 2 to 3 years and urinalysis once per month for 1 year), even if symptoms haven't recurred.

Special considerations

Patient care includes education and postoperative support.

- To ensure complete emptying of the bladder, teach the patient with vesicoureteral reflux to double void (void once and then try to void again in a few minutes). Because his natural urge to urinate may be impaired, advise him to void every 2 to 3 hours whether or not he feels the urge.

PEDIATRIC TIP

Because the diagnostic tests may frighten the child, encourage one of his parents to stay with him during all procedures. Explain the procedures to the parents and to the child, if he's old enough to understand.

- If surgery is necessary, explain postoperative care: suprapubic catheter in the male, indwelling catheter in the female; and, in both, one or two ureteral catheters or splints brought out of the bladder through a small abdominal incision. The suprapubic or indwelling catheter keeps the bladder empty and prevents pressure from stressing the surgical wound; ureteral

  catheters drain urine directly from the renal pelvis. After complicated reimplantations, all catheters remain in place for 7 to 10 days. Explain that the child will be able to move and walk with the catheters but must be very careful not to dislodge them.
- Postoperatively, closely monitor fluid intake and output. Give analgesics and antibiotics, as ordered. Make sure the catheters are patent and draining well. Maintain sterile technique during catheter care. Watch for fever, chills, and flank pain, which suggest a blocked catheter.
- Before discharging the patient, stress the importance of close follow-up care and adequate fluid intake throughout childhood.
- Instruct parents to watch for and report recurring signs of UTI (painful, frequent, burning urination; foul-smelling urine).
- If the child is taking antimicrobial drugs, make sure his parents understand the importance of completing the prescribed therapy or maintaining low-dose prophylaxis.

Neurogenic bladder

Neurogenic bladder (also known as *neuromuscular dysfunction of the lower urinary tract, neurologic bladder dysfunction,* and *neuropathic bladder*) refers to all types of bladder dysfunction caused by an interruption of normal bladder innervation. Subsequent complications include incontinence, residual urine retention, urinary infection, stone formation, and renal failure. A neurogenic bladder can be spastic (hypertonic, reflex, or automatic) or flaccid (hypotonic, atonic, nonreflex, or autonomous).

Causes and incidence

At one time, neurogenic bladder was thought to result primarily from spinal cord injury; now, it appears to stem from a host of underlying conditions:

- cerebral disorders, such as stroke, brain tumor (meningioma and glioma), Parkinson's disease, multiple sclerosis, dementia, and incontinence caused by aging
- spinal cord disease or trauma, such as herniated vertebral disks, spina bifida, myelomeningocele, spinal stenosis (causing cord compression) or arachnoiditis (causing adhesions between the membranes covering the cord), cervical spondylosis, myelopathies from hereditary or nutritional deficiencies and, rarely, tabes dorsalis
- disorders of peripheral innervation, including autonomic neuropathies resulting from endocrine disturbances such as diabetes mellitus (most common)
- metabolic disturbances, such as hypothyroidism, porphyria, or uremia (infrequent)
- acute infectious diseases such as transverse myelitis
- heavy metal toxicity
- chronic alcoholism
- collagen diseases such as systemic lupus erythematosus
- vascular diseases such as atherosclerosis

- distant effects of cancer such as primary oat cell carcinoma of the lung
- herpes zoster
- syphilis
- sacral agenesis.

An upper motor neuron lesion (above S2 to S4) causes spastic neurogenic bladder, with spontaneous contractions of detrusor muscles, elevated intravesical voiding pressure, bladder wall hypertrophy with trabeculation, and urinary sphincter spasms. A lower motor neuron lesion (below S2 to S4) causes flaccid neurogenic bladder, with decreased intravesical pressure, increased bladder capacity and large residual urine retention, and poor detrusor contraction.

Complications

- Calculus formation
- Incontinence
- Renal failure
- Residual urine retention
- UTI

Signs and symptoms

Neurogenic bladder produces a wide range of clinical effects, depending on the underlying cause and its effect on the structural integrity of the bladder. Usually, this disorder causes some degree of incontinence, changes in initiation or interruption of micturition, and the inability to empty the bladder completely. Other effects of neurogenic bladder include vesicoureteral reflux,

deterioration or infection in the upper urinary tract, and hydroureteral nephrosis.

Depending on the site and extent of the spinal cord lesion, spastic neurogenic bladder may produce involuntary or frequent scanty urination, without a feeling of bladder fullness, and possibly

spontaneous spasms of the arms and legs. Anal sphincter tone may be increased. Tactile stimulation of the abdomen, thighs, or genitalia may precipitate voiding and spontaneous contractions of the arms and legs. With cord lesions in the upper thoracic (cervical) level, bladder distention can trigger hyperactive autonomic reflexes, resulting in severe hypertension, bradycardia, and headaches.

Flaccid neurogenic bladder may be associated with overflow incontinence, diminished anal sphincter tone, and a greatly distended bladder (evident on percussion or palpation), but without the accompanying feeling of bladder fullness due to sensory impairment.

Diagnosis

The patient's history may include a condition or disorder that can cause neurogenic bladder, incontinence, and disruptions of micturition patterns. Voiding cystourethrography evaluates bladder neck function, vesicoureteral reflux, and continence.

Urodynamic studies help evaluate how urine is stored in the bladder, how well the bladder empties, and the rate of movement of urine out of the bladder during voiding. These studies consist of four components:

- Urine flow study (uroflow) shows diminished or impaired urine flow.
- Cystometry evaluates bladder nerve supply, detrusor muscle tone, and intravesical pressures during bladder filling and contraction.
- Urethral pressure profile determines urethral function with respect to the length of the urethra and the outlet pressure resistance.
- Sphincter electromyelography correlates the neuromuscular function of the external sphincter with bladder muscle function during bladder filling and contraction. This evaluates how well the bladder and urinary sphincter muscles work together.
- Retrograde urethrography reveals the presence of strictures and diverticula. This test may not be performed on a routine basis.

Treatment

The goals of treatment are to maintain the integrity of the upper urinary tract, control infection, and prevent urinary incontinence through

evacuation of the bladder, drug therapy, surgery or, less commonly, neural blocks and electrical stimulation.

Techniques of bladder evacuation include Credé's method, Valsalva's maneuver, and intermittent self-catheterization. Credé's method—application of manual pressure over the lower abdomen— promotes complete emptying of the bladder. After appropriate instruction, most patients can perform this maneuver themselves. Even when patients perform this maneuver properly, however, Credé's method isn't always successful and doesn't always eliminate the need for catheterization.

Intermittent self-catheterization—more effective than either Credé's method or Valsalva's maneuver—has proved to be a major advance in the treatment of neurogenic bladder because it allows complete emptying of the bladder without the risks that an indwelling catheter poses. Generally, men can perform this procedure more easily than women, but women can learn self-catheterization with the help of a mirror. Intermittent self-catheterization, in conjunction with a bladder-retraining program, is especially useful for patients with flaccid neurogenic bladder.

Drug therapy for neurogenic bladder may include bethanechol and phenoxybenzamine to facilitate bladder emptying and propantheline, methantheline, flavoxate, dicyclomine, and imipramine to facilitate urine storage.

When conservative treatment fails, surgery may correct the structural impairment through transurethral resection of the bladder neck, urethral dilatation, external sphincterotomy, or urinary diversion procedures. Implantation of an artificial urinary sphincter may be necessary if permanent incontinence follows surgery for neurogenic bladder.

Special considerations

Care for patients with neurogenic bladder varies according to the underlying cause and method of treatment.

- Explain all diagnostic tests clearly so the patient understands the procedure, time involved, and possible results. Assure the patient that

the lengthy diagnostic process is necessary to identify the most effective treatment plan. After the treatment plan is chosen, explain it to the patient in detail.

- Use strict sterile technique during insertion of an indwelling catheter (a temporary measure to drain the incontinent patient's bladder). Don't interrupt the closed drainage system for any reason. Obtain urine specimens with a syringe and smallbore needle inserted through the aspirating port of the catheter itself (below the junction of the balloon instillation site). Irrigate in the same manner if ordered.
- Clean the catheter insertion site with soap and water at least twice a day. Don't allow the catheter to become encrusted. Use a sterile applicator to apply antibiotic ointment around the meatus after catheter care. Keep the drainage bag below the tubing, and don't raise the bag above the level of the bladder. Clamp the tubing, or empty the bag before transferring the patient to a wheelchair or stretcher to prevent accidental urine reflux. If urine output is considerable, empty the bag more frequently than once every 8 hours because bacteria can multiply in standing urine and migrate up the catheter and into the bladder.
- Watch for signs of infection (fever, cloudy or foul-smelling urine). Encourage the patient to drink plenty of fluids to prevent calculus formation and infection from urinary stasis. Try to keep the patient as mobile as possible. Perform passive range-of-motion exercises if necessary.
- If urinary diversion procedure is to be performed, arrange for consultation with an enterostomal therapist, and coordinate the care plans.
- Before discharge, teach the patient and his family evacuation techniques as necessary (Credé's method, intermittent catheterization). Counsel him regarding sexual activities. Remember, the incontinent patient feels embarrassed and distressed. Provide emotional support.

Congenital anomalies of the ureter, bladder, and urethra

The most common congenital malformations of the ureter, bladder, and urethra include duplicated ureter, retrocaval ureter, ectopic orifice of the ureter, stricture or stenosis of the ureter, ureterocele, exstrophy of the bladder, congenital bladder diverticulum, hypospadias, and epispadias. Some of these abnormalities are obvious at birth; others aren't apparent and are recognized only after they produce symptoms. (See *Congenital urologic anomalies*.)

Causes and incidence

Congenital anomalies of the ureter, bladder, and urethra are among the most common birth defects, occurring in about 5% of all births. Their causes are unknown; diagnosis and treatment vary.

Complications

- Calculi
- Hematuria
- Infections

Special considerations

PEDIATRIC TIP

Because these anomalies aren't always obvious at birth, carefully evaluate the newborn's urogenital function. Document the amount and color of urine, voiding pattern, strength of stream, and any indications of infection, such as fever and urine odor. Tell parents to watch for these signs at home. In all children, watch for signs of obstruction, such as dribbling, oliguria or anuria, abdominal mass, hypertension, fever, bacteriuria, or pyuria.

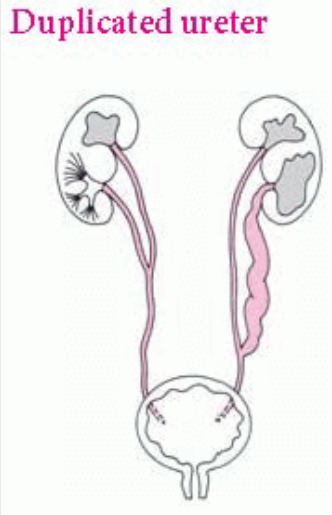
- Monitor renal function daily; record intake and output accurately.
- Follow strict sterile technique in handling cystostomy tubes or indwelling urinary catheters.

- Make sure that ureteral, suprapubic, or urethral catheters remain in place and

  don't become contaminated. Document the type, color, and amount of drainage.

CONGENITAL UROLOGIC ANOMALIES

Duplicated ureter



Pathophysiology

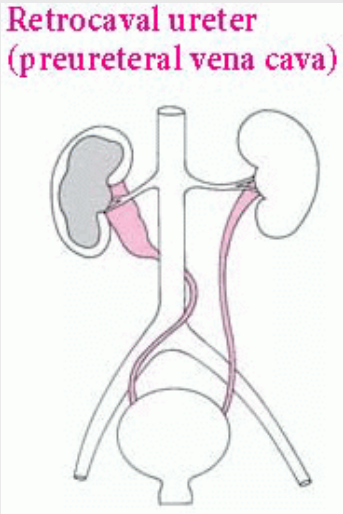
▪ Most common ureteral anomaly

▪ *Complete,* a double collecting system with two separate pelves, each with its own ureter and orifice

▪ *Incomplete* (y type), two separate ureters join before entering bladder

Clinical features

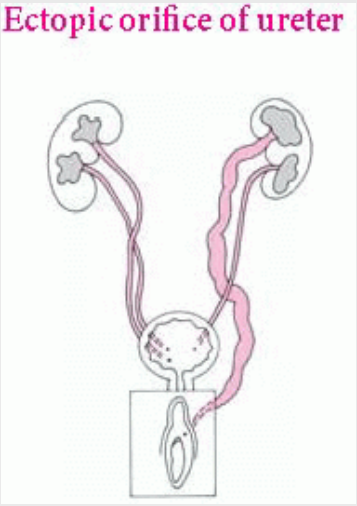
Retrocaval ureter (preureteral vena cava)



Pathophysiology

▪ The right ureter passes behind the inferior vena cava before entering the bladder. Compression of the ureter between the vena cava and the spine causes dilation and elongation of the pelvis; hydroureter and hydronephrosis; and fibrosis and stenosis of the ureter in the compressed area.

Ectopic orifice of ureter



Pathophysiology

▪ Ureters single or duplicated in females, ureteral orifice usually inserts in urethra or vaginal vestibule, beyond external urethral sphincter; in males, in prostatic urethra, or in seminal vesicles or vas deferens

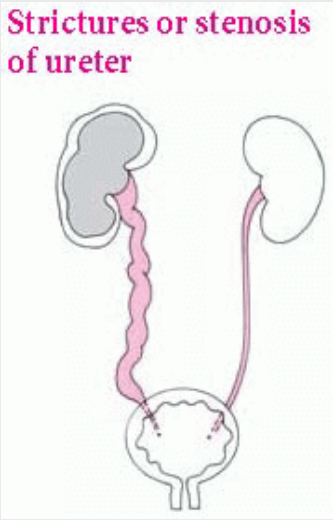
Clinical features

▪ Persistent or recurrent infection
▪ Frequency, urgency, or burning on urination
▪ Diminished urine output
▪ Flank pain, fever, and chills

Diagnosis and treatment

▪ Excretory urography
▪ Voiding cystoscopy
▪ Cystoureterography
▪ Retrograde pyelography
▪ Surgery for obstruction, reflux, or severe renal damage

Strictures or stenosis of ureter



Pathophysiology

▪ Most common site, the distal ureter above ureterovesical junction; less common, ureteropelvic

▪ Relatively uncommon; higher incidence in males

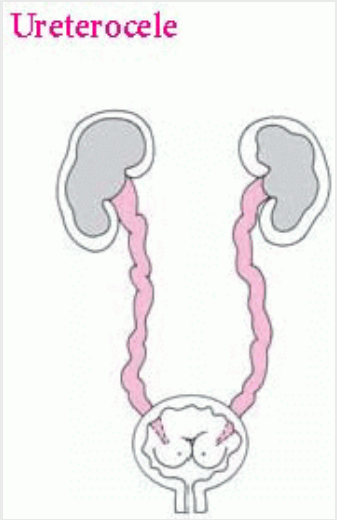
Clinical features

▪ Right flank pain
▪ Recurrent urinary tract infection
▪ Renal calculi
▪ Hematuria

Diagnosis and treatment

▪ Excretory urography demonstrates superior ureteral enlargement with spiral appearance
▪ Surgical resection and anastomosis of ureter with renal pelvis, or reimplantation into bladder

Ureterocele



Pathophysiology

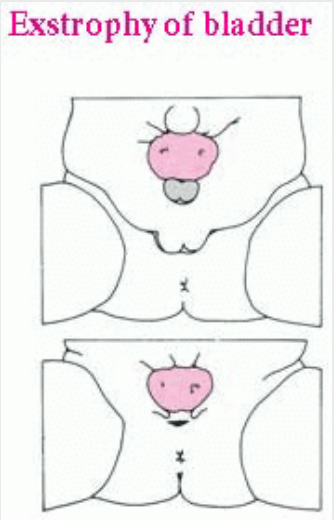
▪ Bulging of submucosal ureter into bladder can be 1 or 2 cm, or can almost fill entire bladder

▪ Symptoms rare when ureteral orifice opens between trigone and bladder neck
▪ Obstruction, reflux, and incontinence (dribbling) in 50% of females
▪ In males, flank pain, frequency, urgency

Diagnosis and treatment

▪ Excretory urography
▪ Urethroscopy, vaginoscopy
▪ Voiding cystourethrography
▪ Resection and ureteral reimplantation into bladder for incontinence

Exstrophy of bladder

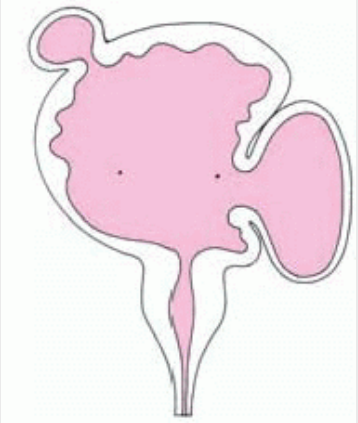


Pathophysiology

▪ Absence of anterior abdominal and bladder wall allows the bladder to protrude onto abdomen

junction; rare, the midureter ▪ Discovered during infancy in 25% of patients; before puberty in most ▪ More common in males ***Clinical features*** ▪ Megaloureter or hydroureter (enlarged ureter), with hydronephrosis when stenosis occurs in distal ureter ▪ Hydronephrosis alone when stenosis occurs at ureteropelvic junction ***Diagnosis and treatment*** ▪ Ultrasound ▪ Excretory urography ▪ Voiding cystography ▪ Surgical repair of stricture; nephrectomy for severe renal damage	▪ Unilateral, bilateral, ectopic with resulting hydroureter and hydronephrosis ***Clinical features*** ▪ Obstruction ▪ Persistent or recurrent infection ***Diagnosis and treatment*** ▪ Voiding cystourethrography ▪ Excretory urography and cystoscopy show thin, translucent mass ▪ Surgical excision or resection of ureterocele, with reimplantation of ureter	▪ In males, undescended testes and epispadias; in females, cleft clitoris, separated labia, or absent vagina ▪ Skeletal or intestinal anomalies possible ***Clinical features*** ▪ Obvious at birth, with urine seeping onto abdominal wall from abnormal ureteral orifices ▪ Surrounding skin excoriated; exposed bladder mucosa ulcerated; infection; related abnormalities ***Diagnosis and treatment*** ▪ Excretory urography ▪ Surgical closure of defect, and bladder and urethra reconstruction during infancy to allow pubic bone fusion; alternative treatment: protective dressing and diapering; urinary diversion eventually necessary for most patients
Congenital bladder diverticulum	**Hypospadias**	**Epispadias**

Congenital bladder diverticulum



Pathophysiology

▪ Circumscribed pouch or sac (diverticulum) of bladder wall

▪ Can occur anywhere in bladder, usually lateral to ureteral orifice. Large diverticulum at orifice can cause reflux.

Clinical features

▪ Fever, frequency, and painful urination

▪ Urinary tract infection

▪ Cystitis, particularly in males

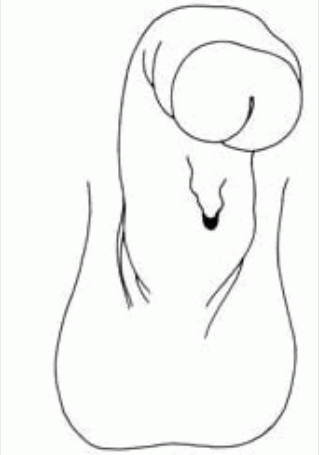
Diagnosis and treatment

▪ Excretory urography shows diverticulum

▪ Retrograde cystography shows vesicoureteral reflux in ureter

▪ Surgical correction for reflux

Hypospadias



Pathophysiology

▪ Urethral opening on ventral surface of penis or, in females (rare), within vagina

▪ Occurs in 1 in 300 live male births; genetic factor suspected in less severe cases

Clinical features

▪ Usually associated with chordee, making normal urination with penis elevated impossible

▪ Absence of ventral prepuce

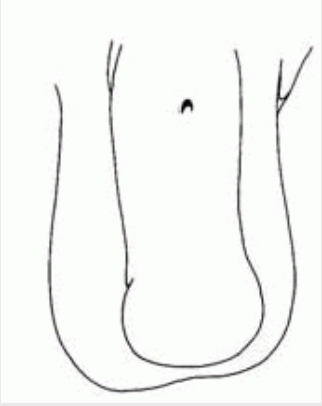
▪ Vaginal discharge in females

Diagnosis and treatment

▪ Mild disorder requires no treatment

▪ Surgical repair of severe anomaly usually necessary before child reaches school age

Epispadias



Pathophysiology

▪ Urethral opening on dorsal surface of penis; in females, a fissure of the upper wall of urethra

▪ A rare anomaly; usually more common in males; often accompanies bladder exstrophy

Clinical features

▪ In mild cases, orifice appears along dorsum of glans; in severe cases, along dorsum of penis

▪ In females, bifid clitoris and short, wide urethra

Diagnosis and treatment

▪ Surgical repair, in several stages, almost always necessary

- Apply sterile saline pads to protect the exposed mucosa of the newborn with bladder exstrophy. Don't use heavy clamps on the

umbilical cord, and avoid dressing or diapering the infant. Place the infant in an incubator, and direct a stream of saline mist onto the bladder to keep it moist. Use warm water and mild soap to keep the surrounding skin clean. Rinse well, and keep the area as dry as possible to prevent excoriation.

- Infant boys diagnosed with hypospadias and episiadias should not be circumcised because the foreskin can be used in the surgical repair.
- Provide reassurance and emotional support to the parents. When possible, allow them to participate in their child's care to promote normal bonding. As appropriate, suggest or arrange for genetic counseling.

PROSTATE AND EPIDIDYMIS DISORDERS

Prostatitis

Prostatitis, inflammation of the prostate gland, may be acute or chronic. Acute prostatitis most often results from gram-negative bacteria and is easy to recognize and treat. However, chronic prostatitis, the most common cause of recurrent urinary tract infections (UTIs) in males, is less easy to recognize.

Causes and incidence

About 80% of bacterial prostatitis cases result from infection by *Escherichia coli;* the rest are due to infection by *Klebsiella, Enterobacter, Proteus, Pseudomonas, Streptococcus,* or *Staphylococcus.* These organisms probably spread to the prostate by the bloodstream or from ascending urethral infection, invasion of rectal bacteria via lymphatics, reflux of infected bladder urine into the prostate ducts or, less commonly, infrequent or excessive sexual intercourse or such procedures as cystoscopy or catheterization. Chronic prostatitis usually results from bacterial invasion from the urethra.

It's estimated that 2 of every 10,000 people who seek outpatient care do so because of prostatitis. As many as 35% of males older than age 50 have chronic prostatitis; about 50% of males will be diagnosed with prostatitis at some point in their lives.

Complications

- UTI
- Prostatic abscess
- Pyelonephritis
- Epididymitis

Signs and symptoms

Acute prostatitis begins with fever, chills, low back pain, myalgia, perineal fullness, and arthralgia. Urination is frequent and urgent. Dysuria, nocturia, and urinary obstruction may also occur. The urine may appear cloudy. When palpated rectally, the prostate is tender, indurated, swollen, firm, and warm.

Chronic bacterial prostatitis sometimes produces no symptoms but usually elicits the same urinary symptoms as the acute form but to a lesser degree. UTI is a common complication. Other possible signs include painful ejaculation, hemospermia, persistent urethral discharge, and sexual dysfunction.

Diagnosis

Characteristic rectal examination findings suggest prostatitis. In many cases, a urine culture can identify the causative infectious organism.

CONFIRMING DIAGNOSIS

A firm diagnosis depends on a comparison of urine cultures of specimens obtained by the Meares and Stamey technique. This test requires four specimens: one collected when the patient starts voiding (voided bladder one); another midstream; another after the patient stops voiding and the physician massages the prostate to produce secretions (expressed prostate secretions; and a final voided specimen. A significant increase

in colony count in the prostatic specimens confirms prostatitis.

Treatment

Systemic antibiotic therapy chosen according to the infecting organism is the treatment of choice for acute prostatitis. If sepsis is likely, I.V. antibiotics may be given until sensitivity test results are known. If test results and clinical response are favorable, parenteral therapy continues for 48 hours to 1 week, after which an oral agent is substituted for 30 days. For infections caused by a sexually transmitted disease, injection of ceftriaxone followed by a 10-day course of doxycycline or floxacin is effective.

Supportive therapy includes bed rest, adequate hydration, and administration of analgesics, antipyretics, sitz baths, and stool softeners as necessary. Diet therapy includes avoiding substances that irritate the bladder, such as alcohol, caffeinated food and beverages, citrus juices, and hot or spicy foods. Increasing the intake of fluids (1,893 to 3,785 ml/day) encourages frequent urination that will help flush the bacteria from the bladder. In symptomatic chronic prostatitis, regular massage of the prostate is most effective. Regular ejaculation may help promote drainage of prostatic secretions. Anticholinergics and analgesics may help relieve nonbacterial prostatis symptoms.

If drug therapy is unsuccessful, treatment may include transurethral resection of the prostate, which requires removal of all infected tissue. However, this procedure usually isn't performed on young adults because it may cause retrograde ejaculation and sterility. Total prostatectomy is curative but may cause impotence and incontinence.

Special considerations

Patient care is primarily supportive.

- Ensure bed rest and adequate hydration. Provide stool softeners and administer sitz baths, as ordered.

- As needed, prepare to assist with suprapubic needle aspiration of the bladder or a suprapubic cystostomy.
- Emphasize the need for strict adherence to the prescribed drug regimen. Instruct the patient to drink at least 8 glasses of water a day. Have him report adverse drug reactions (rash, nausea, vomiting, fever, chills, and GI irritation).

Epididymitis

This infection of the epididymis, the testicle's cordlike excretory duct, is one of the most common infections of the male reproductive tract. It usually affects adults and is rare before puberty. Epididymitis may spread to the testicle itself, causing orchitis; bilateral epididymitis may cause sterility. (See *Orchitis*, page 424.)

Causes and incidence

Epididymitis is usually a complication of pyogenic bacterial infection of the urinary tract (urethritis or prostatitis). The pyogenic organisms, such as staphylococci, *Escherichia coli,* streptococci, *Chlamydia trachomatis*, and *Neisseria gonorrhoeae*, reach the epididymis through the lumen of the vas deferens. Rarely, epididymitis is secondary to a distant infection, such as pharyngitis or tuberculosis, that spreads through the lymphatics or, less commonly, the bloodstream. Other causes include trauma, gonorrhea, syphilis, or a chlamydial infection. Trauma may reactivate a dormant infection or initiate a new one. Epididymitis may be a complication of prostatectomy and may also result from chemical irritation by extravasation of urine through the vas deferens. The incidence is about 600,000 cases per year, with the highest prevalence in young males ages 19 to 35.

Complications

- Bilateral epididymitis can cause sterility
- Orchitis

Signs and symptoms

The key symptoms are pain, extreme tenderness, and swelling in the groin and scrotum with erythema, high fever, malaise, and a characteristic waddle—an attempt to protect the groin and scrotum during walking. An acute hydrocele may also result from inflammation.

ORCHITIS

Orchitis, an infection of the testicles, is a serious complication of epididymitis. It may also result from mumps, which may lead to sterility. Orchitis may, rarely, result from other systemic infections, testicular torsion, or severe trauma. Its typical effects include unilateral or bilateral tenderness, sudden onset of pain, and swelling of the scrotum and testicles. The affected testicle may be red. Nausea and vomiting also occur. Sudden cessation of pain indicates testicular ischemia, which may result in permanent damage to one or both testicles.

Treatment consists of immediate antibiotic therapy; in orchitis due to mumps, diethylstilbestrol may be given to relieve pain, swelling, and fever. Severe orchitis may require surgery to incise and drain the hydrocele and improve testicular circulation. Other treatment is similar to that for epididymitis. To prevent orchitis due to mumps, stress the need for prepubertal males to receive mumps vaccine (or gamma globulin injection after contracting mumps).

Diagnosis

Clinical features suggest epididymitis but diagnosis is actually made with the aid of laboratory tests:

- urinalysis: increased white blood cell (WBC) count indicates infection
- urine culture and sensitivity tests: may identify causative organism
- serum WBC count: more than 10,000/µl in infection.

ALERT

Scrotal ultrasonography may help differentiate acute epididymitis from other conditions, such as testicular torsion, which is a surgical emergency.

Testicular scan (nuclear medicine scan) may be done to rule out torsion. In epididymitis, increased blood flow is also demonstrated.

Treatment

The goal of treatment is to reduce pain and swelling and combat infection. Therapy must begin immediately, particularly in the patient with bilateral epididymitis because sterility is always a threat. During the acute phase, treatment consists of bed rest, scrotal elevation with towel rolls or adhesive strapping, broad-spectrum antibiotics, and analgesics. An ice bag applied to the area may reduce swelling and relieve pain (heat is contraindicated because it may damage germinal cells, which are viable only at or below normal body temperature). When pain and swelling subside and allow walking, an athletic supporter may prevent pain. Occasionally, corticosteroids may be prescribed to help counteract inflammation but their use is controversial.

ELDER TIP

In the older patient undergoing open prostatectomy, bilateral vasectomy may be necessary to prevent epididymitis as a postoperative complication; however, antibiotic therapy alone may prevent it. When epididymitis is refractory to antibiotic therapy, epididymectomy under local anesthetic is necessary.

Special considerations

Patient care includes support and monitoring for worsening of symptoms.

- Watch closely for abscess formation (localized, hot, red, tender area) or extension of infection into the testes. Closely monitor temperature, and ensure adequate fluid intake.

- Because the patient is usually very uncomfortable, administer analgesics as necessary. During bed rest, check often for proper scrotum elevation.
- Before discharge, emphasize the importance of completing the prescribed antibiotic therapy, even after symptoms subside. Educate the patient regarding preventing the transmission of sexually transmitted diseases, as appropriate.
- If the patient faces the possibility of sterility, suggest supportive counseling as necessary.

Benign prostatic hyperplasia

Although most men older than age 50 have some prostatic enlargement, in benign prostatic hyperplasia (BPH), also known as *benign prostatic hypertrophy,* the prostate gland enlarges sufficiently to compress the urethra and cause some overt urinary obstruction. Depending on the size of the enlarged prostate, the age and health of the patient, and the extent of obstruction, BPH is treated symptomatically or surgically.

Causes and incidence

Evidence suggests a link between BPH and hormonal activity. As men age, production of androgenic hormones decreases, causing an imbalance in androgen and estrogen levels, and high levels of dihydrotestosterone, the main prostatic intracellular androgen. Other causes include neoplasm, arteriosclerosis, diabetes, inflammation, and metabolic or nutritional disturbances.

Whatever the cause, BPH begins with changes in periurethral glandular tissue. As the prostate enlarges, it may extend into the bladder and obstruct urinary outflow by compressing or distorting the prostatic urethra. BPH may also cause a pouch to form in the bladder that retains urine when the rest of the bladder empties. This retained urine may lead to calculus formation or cystitis.

The likelihood of developing an enlarged prostate increases with age. A small amount of prostate enlargement is present in many men older than age 40 and more than 90% of men older than age 80. It's estimated that

by 2006, 115 million men age 50 and older will develop BPH. Blacks, with an incidence of 224.3 cases per 100,000 people, are at the greatest risk, present with more advanced disease, and have a poorer diagnosis. Whites, by comparison, have an incidence of 150.3 cases per 100,000 people while Asians have an incidence of 82.2 cases per 100,000 people.

Complications

- Urinary stasis
- Urinary tract infection (UTI)
- Calculi
- Bladder wall trabeculation
- Detrusor muscle hypertrophy
- Bladder diverticula and saccules
- Urethral stenosis
- Hydronephrosis
- Paradoxical incontinence
- Acute or chronic renal failure
- Acute postobstructive diuresis

Signs and symptoms

Clinical features of BPH depend on the extent of prostatic enlargement and the lobes affected. Characteristically, the condition starts with a group of symptoms known as *prostatism*: reduced urine stream caliber and force, urinary hesitancy, and difficulty starting micturition (resulting in straining, feeling of incomplete voiding, and an interrupted stream). As the obstruction increases, it causes frequent urination with nocturia, dribbling, urine retention, incontinence, and possibly hematuria. Physical examination indicates a visible midline mass above the symphysis pubis that represents an incompletely emptied bladder; rectal palpation discloses an enlarged prostate. Examination may detect secondary anemia and, possibly, renal insufficiency secondary to obstruction.

As BPH worsens, complete urinary obstruction may follow infection or use of decongestants, tranquilizers, alcohol, anti-depressants, or anticholinergics. Complications include infection, renal insufficiency, hemorrhage, and shock.

Diagnosis

Clinical features and a rectal examination are usually sufficient for diagnosis. Other findings help to confirm it:

- Excretory urography may indicate urinary tract obstruction, hydronephrosis, calculi or tumors, and filling and emptying defects in the bladder.
- Elevated blood urea nitrogen and serum creatinine levels suggest renal dysfunction.
- Urinalysis and urine culture show hematuria, pyuria and, when the bacterial count exceeds 100,000/µl, UTI.

When symptoms are severe, a cystourethroscopy is definitive, but this test is performed only immediately before surgery to help determine the best procedure.

It can show prostate enlargement, bladder wall changes, and a raised bladder.

Treatment

Conservative therapy includes prostate massages, sitz baths, fluid restriction for bladder distention, and antimicrobials for infection. If symptoms are mild, methods for relief may include avoiding alcohol and caffeine, especially after dinner; urinating when the urge is first felt; avoiding over-the-counter cold and sinus medications that contain decongestants or antihistamines because they can increase BPH symptoms; keeping warm and exercising regularly because cold weather and lack of physical activity may worsen symptoms; performing pelvic strengthening exercises (Kegel exercises); reducing stress because nervousness and tension can lead to more frequent urination. Some men have had success taking extracts of saw palmetto berries, an herb that has been used to ease prostate symptoms. Fat-soluble saw palmetto

extract that has been standardized to contain 85% to 95% fatty acids and sterols is more effective. Regular ejaculation may help relieve prostatic congestion.

Urine flow rates can be improved with alpha1-adrenergic blockers, which relieve bladder outlet obstruction by preventing contractions of the prostatic capsule and bladder neck. Finasteride lowers levels of hormones produced by the prostate, reduces the size of the prostate gland, increases urine flow rate, and decreases symptoms of BPH. It may take 3 to 6 months before a significant improvement in symptoms occurs. Potential adverse effects related to finasteride include decreased sex drive and impotence.

Surgery is the only effective therapy to relieve acute urine retention, hydronephrosis, severe hematuria, recurrent UTIs, and other intolerable symptoms. A transurethral resection may be performed if the prostate weighs less than 2 oz (56.7 g). In this procedure, a resectoscope removes tissue with a wire loop and electric current. In high-risk patients, continuous drainage with an indwelling urinary catheter alleviates urine retention. Transurethral needle ablation may be used to heat and destroy prostate tissue by radiofrequency; this helps spare surrounding tissue.

The following procedures involve open surgical removal:

- *suprapubic (transvesical) resection:* most common and useful when prostatic enlargement remains within the bladder
- *retropubic (extravesical) resection:* allows direct visualization; potency and continence are usually maintained.

Balloon dilatation of the prostate is still being investigated. Balloon dilatation or balloon urethroplasty involves passing a flexible balloon catheter through the urethra at the level of the prostate while being guided by fluoroscope. The balloon is inflated for a short time to distend the prostatic urethra.

Special considerations

Prepare the patient for diagnostic tests and surgery, as appropriate.

- Monitor and record the patient's vital signs, intake and output, and daily weight. Watch closely for signs of postobstructive diuresis (such as increased urine output and hypotension), which may lead to serious dehydration, lowered blood volume, shock, electrolyte loss, and anuria.
- Administer antibiotics, as indicated, for UTI, urethral instrumentation, and cystoscopy.
- If urine retention is present, insert an indwelling urinary catheter (although this is usually difficult in a patient with BPH). If the catheter can't be passed transurethrally, assist with suprapubic cystostomy (under local anesthetic). Watch for rapid bladder decompression.

After prostatic surgery:

- Maintain patient comfort, and watch for and prevent postoperative complications. Observe for immediate dangers of prostatic bleeding (shock and hemorrhage). Check the catheter often (every 15 minutes for the first 2 to 3 hours) for patency and urine color; check dressings for bleeding.
- Postoperatively, many urologists insert a three-way catheter and establish continuous bladder irrigation. Keep the catheter open at a rate sufficient to maintain returns that are clear and light pink. Watch for fluid overload from absorption of the irrigating fluid into systemic circulation. If

  a regular catheter is used, observe it closely. If drainage stops because of clots, irrigate the catheter, as indicated, usually with 80 to 100 ml of normal saline solution, while maintaining strict sterile technique.

ALERT

Watch for septic shock, the most serious complication of prostatic surgery, which may cause severe chills, sudden fever, tachycardia, hypotension, or other signs of shock. Start rapid infusion of antibiotics I.V. as indicated. Watch for pulmonary embolus, heart failure, and renal shutdown. Monitor vital signs, central venous pressure,

and arterial pressure continuously. The patient may need intensive supportive care in the intensive care unit.

- Administer anticholinergics, as indicated, to relieve painful bladder spasms that often occur after transurethral resection.
- Provide patient comfort measures after an open procedure: suppositories (except after perineal prostatectomy), analgesic medication to control incisional pain, and frequent dressing changes.
- Continue infusing I.V. fluids until the patient can drink sufficient fluids (2 to 3 L/day) to maintain adequate hydration.
- Administer stool softeners and laxatives, as needed, to prevent straining. Don't check for fecal impaction because a rectal examination may precipitate bleeding.
- After the catheter is removed, the patient may experience frequency, dribbling, and occasional hematuria. Reassure him that he'll gradually regain urinary control.
- Reinforce prescribed limits on activity. Warn the patient against lifting, strenuous exercise, and long automobile rides because these increase the risk of bleeding. Also caution the patient to restrict sexual activity for at least several weeks after discharge from the hospital.
- Instruct the patient to follow the prescribed oral antibiotic drug regimen, and tell him the indications for using gentle laxatives. Urge him to seek medical care immediately if he can't void, he passes bloody urine, or he develops a fever.

Selected references

Chisholm-Burns, M.A., et al. *Pharmacology Principles and Practice*. New York: McGraw-Hill Book Co., 2007.

Dogan, H.S., and Tekal, S. “Management of Pediatric Stone Disease,” *Current Urology Reports* 8(2):163-73, March 2007.