23 The Urinary System

**Objectives**

In this chapter we will study

- the common signs of urinary system disorders;
- diagnostic procedures applicable to urinary system disorders;
- urinary tract infections, particularly cystitis and pyelonephritis;
- glomerulopathies such as glomerulonephritis and nephrotic syndrome;
- neurogenic bladder; and
- how diuretics can be used to treat certain urinary disorders.

**Assessment of the Urinary System**

The urinary system carries out a wide variety of homeostatic functions. It regulates acid-base balance, body water (and blood pressure), and mineral balance. It also plays a vital role in removing metabolic waste products and detoxifying and removing chemicals from the body (see *A&P*, p. 882). Disorders of the urinary system therefore have a far-reaching impact on the other body systems.

**Common Signs and Symptoms of Urinary Disorders**

As with other body systems discussed previously, some of the signs and symptoms of urinary system disorders—such as fever, malaise, and weight loss—are nonspecific. Therefore, the patient history, physical examination, and clinical tests are essential to achieving an accurate diagnosis.

Specific symptoms of urinary problems include changes in urinary frequency, abnormal urination patterns, changes in the volume of urine produced, abnormal appearance of the urine, and pain upon urination. In addition, individuals with urinary system diseases often have generalized edema and hypertension because their kidneys are unable to properly regulate the body’s water and sodium ion balance.

**Changes in Urinary Frequency**

The average adult voids 700 to 2,000 mL of urine over the course of a day, usually distributed in four to six voids. If the frequency of urination increases without a corresponding increase in volume, a number of pathologies are suggested. These include urinary bladder trauma or infection (*cystitis*), urethral infection (*urethritis*), prostate enlargement, production of acidic urine, tumors in the urinary tract, and damage to either the urinary system or the regions of the nervous system that regulate it.

**Abnormal Urination Patterns**

**Urinary incontinence** (*enuresis*), the inability to retain urine, may be a “normal” effect of aging in both men and women, or it may be a sign of urinary system pathology. In women, stretching of the pelvic floor during childbirth may result in incontinence associated with mild physical stress such as sneezing or coughing (*stress incontinence*). Other causes of incontinence include damage to the urinary tract due to childbirth or prostate removal, neurogenic bladder dysfunction, or a congenital defect such as *extrophy* of the bladder, in which the bladder is everted due to the absence of portions of the lower abdominal wall and anterior wall of the bladder. Incontinence may be prevented in part by doing Kegel exercises—rhythmically tightening the pelvic muscles as if trying to stop the urine stream. This strengthens the muscles of the pelvic floor and reduces the incidence of incontinence.

During the first 2 to 3 years of life, bedwetting (*nocturnal enuresis*) is relatively common. But after 3 years of age, continued enuresis may indicate neurogenic bladder disease, impaired or delayed neuromuscular development of the urinary tract, or a behavioral problem. **Nocturia** (having to void during the night) may simply be due to excessive fluid intake in the late evening—or it may indicate the presence of renal or prostate disease.

**Changes in the Volume of Urine Produced**

Changes in daily urine volume may signal disease. **Polyuria**, or *diuresis* (daily urine volume exceeding 2 L/day), suggests that the ability to concentrate urine has been impaired or that the kidney has been damaged. In **oliguria** (urine volume...
production of less than 500 mL per day) or anuria (urine production of less than 100 mL per day), the patient is at risk of azotemia (the accumulation of nitrogenous wastes in the blood) or uremia (toxic effects of the accumulated wastes). Oliguria and anuria can be caused by an obstruction of the urinary tract, renal ischemia, congestive heart failure, shock, or pyelonephritis.

**Abnormal Appearance of the Urine** Changes in the appearance (and odor) of the urine may simply be due to something the patient has eaten, or they may indicate a pathology. The color of urine can vary considerably, ranging from a deep amber to a light, almost colorless yellow. These color variations reflect the body’s state of hydration. In normal urine samples, color intensity is an indicator of urine concentration. The darker the color, the more concentrated the urine. Food pigments excreted in the urine are usually red or yellow, while excretion of B vitamins causes the color to become a brighter yellow. Additionally, certain drugs excreted in the urine can cause the color to change from yellow to brown, black, blue, green, or red. If diet and drugs can be ruled out as causes of a color change, disease may be indicated. For example, myoglobin, hemoglobin, and erythrocytes in the urine produce a red or brown color, while bilirubin causes the urine to appear bluish-green to brownish-black. A white, cloudy appearance can occur if the urine contains pus, bacteria, lipids, or semen, or if the urine is alkaline and salts precipitate from solution. Frothy or foamy urine indicates excessive amounts of protein. The exact cause of a change in the appearance of the urine can be determined through urinalysis.

**Pain upon Urination** Pain or discomfort during urination (dysuria) is most often localized to the site of trauma or infection. It can be an extremely important diagnostic sign. Dysuria is a common symptom of cystitis or urethritis or of a urinary tract obstruction such as renal calculi (kidney stones, composed of crystallized urinary salts) or an enlarged prostate gland. When the pain is localized to the lumbar back and radiates laterally and to the upper quadrant (right or left), kidney infection or trauma is the most likely cause. Pain superior to the pubic region suggests cystitis.

**Tests Used in Diagnosing Urinary System Disorders**

If a clinician suspects a urinary system disorder based on the physical examination and patient history, a number of diagnostic procedures are available to determine whether the system is functioning normally or not. The most commonly used noninvasive test is urinalysis, in which a freshly voided urine sample is analyzed (see A&P, pp. 901–2). Urinalysis is also used to screen for drug use (legal or illegal) because of the kidney’s important role in removing metabolic wastes and toxins. This procedure is becoming more widely used by businesses and the governing bodies of various sports (for example, the Olympic Games and national sports teams) to ensure compliance with drug use policies.

Urinalysis provides a wealth of diagnostic data. Chemical techniques are used to determine the amount of protein, glucose, ketones, blood, nitrites, and hydrogen ions (pH) in the urine as well as whether metabolites of drugs or toxins are present. Urinalysis has been made easier by the development of test strips that are dipped into the urine. Each strip has chemical reagents that change color in the presence of various urine solutes. Elevated amounts of one or more chemicals may indicate various disorders of the urinary and other systems. For example, proteinuria (protein in the urine) is a common indicator of damage to the glomeruli. Ketonuria (ketones in the urine) suggests a condition causing metabolic acidosis such as diabetes mellitus or starvation. Glycosuria (glucose in the urine) is diagnostic of various forms of diabetes, usually diabetes mellitus.

The solute concentration of the urine can be measured with an osmometer, while a urinometer may be used to determine the specific gravity of a urine sample (see A&P, p. 902). Changes in osmolarity or specific gravity suggest either increased or decreased numbers of particles (ions, molecules, and cells) in the urine. Coupled with other tests, a change in urine osmolarity or specific gravity may support the diagnosis of a disease.

In addition, urine can be centrifuged to concentrate its solids, and the sediment examined microscopically for the presence of cells or casts.
(cylindrical masses of mucoproteins formed in the kidney tubule that may contain cells, protein, or fat). The significance of casts in some urinary diseases is explained later in this chapter. Some epithelial cells are expected to be present in the urine due to the natural shedding of cells by the urinary tract. But cells that shouldn’t be present in large numbers include blood cells (erythrocytes or leukocytes) and bacteria. Erythrocytes suggest inflammation, tumors, renal calculi, or trauma; leukocytes indicate a urinary tract infection (UTI), and bacteria, of course, confirm this. Urine cytology (examining the cells in urine) sometimes reveals urinary tract cancer.

In addition to urinalysis, many renal function tests have been developed to identify kidney diseases and measure their severity. These tests usually involve measuring the clearance of a specific chemical from the body; this is determined by comparing the concentration of the chemical in blood and urine samples. The clearance of a specific compound can be used to calculate the renal plasma flow or glomerular filtration rate (see A&P, pp. 903–4).

The ability of the kidneys to concentrate the urine can also be measured. Exogenous antidiuretic hormone (ADH) is given to the patient, and the urine osmolarity is measured 1 hour later. The administered ADH should cause the urine to be more concentrated by increasing renal water retention. If retention is impaired, ADH does not increase the urine osmolarity.

Imaging techniques are also used to obtain information about the status of the urinary system. The system can be examined by CT and MRI scans, ultrasound, angiography, and techniques in which the urinary tract is infused with a radiopaque substance to make it visible by X ray. These X-ray techniques include the following:

- **Intravenous urography**, an iodinated benzoic acid derivative is administered intravenously. The kidney rapidly filters the chemical, and the iodine makes the image more opaque on an X ray, so that the kidney and lower urinary tract can be viewed clearly. This technique is often used to locate sites of renal injury.

- **Cystography** uses a radiopaque agent infused by catheter into the bladder. This technique allows for controlled filling of the bladder with the agent and consequently a clearer image than is possible with intravenous urography. It is most often used to diagnose neurogenic bladder, a ruptured bladder, or cystitis.

- **Retrograde pyelography** employs a radiopaque chemical infused through a catheter until it fills the renal calices, renal pelvis, ureters, and bladder. Because this technique does not require the radiopaque agent to be filtered by the kidney, it provides a more detailed image, especially of the lower urinary tract (urinary bladder and urethra).

In addition to imaging techniques, renal biopsy can be used to confirm a diagnosis.

### Urinary Tract Infections: Cystitis and Pyelonephritis

Urinary tract infections (UTIs) may occur in any portion of the urinary tract. Bacteria (*Escherichia coli*, *Pseudomonas*, *Klebsiella*, *Staphylococcus*, and *Proteus*) are the most commonly encountered microbial agents. These bacteria usually move from the perineum to the urethral orifice and then progress in a retrograde manner into the urethra, urinary bladder, ureters, and finally the kidneys. People most at risk of UTI include prepubertal children, the elderly, sexually active women, pregnant women, people who practice poor hygiene, and individuals diagnosed with neurogenic bladder, urinary tract obstruction, or diabetes mellitus. It has been estimated that at least 10% to 20% of all women in the United States have lower urinary tract infections at any one time.

Several physiological factors limit the occurrence of urinary tract infections, including the bactericidal effects of urea, the acidic pH of the urine, the “washing out” of bacteria during micturition, and closure of the urethral openings of the bladder during micturition, thus minimizing urine reflux. In men, the length of the urethra and secretions from the prostate also reduce the potential for UTI.

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The most common UTI is **cystitis**—bladder inflammation accompanied by dysuria, increased urinary frequency and urgency, and pain in the pubic and lumbar regions (see A&P, clinical insight 23.3, p. 906). The urine may also appear cloudy due to the presence of bacteria (*bacteriuria*) or pus (*pyuria*). If infection causes bleeding in the bladder, *hematuria* may also occur. Severe or prolonged infections may induce ulceration of the bladder (*ulcerative cystitis*) or death of bladder cells (*gangrenous cystitis*). Cystitis is diagnosed through the patient history and urinalysis. The most common treatment is antibiotics, but in cases
where urinary tract obstruction is the suspected cause, the obstruction must be treated as well. **Pyelonephritis**, infection of the renal pelvis and medulla, is another form of UTI. Most often, the same bacteria that cause cystitis cause pyelonephritis; however, this disease may also be caused by a virus or fungus. Pathogens may invade the kidneys when urinary obstruction causes a backflow of urine from the bladder to the kidneys, or they may invade by way of the blood. One or both kidneys may be involved. As with cystitis, most cases of pyelonephritis occur in women. As the infection intensifies, abscesses and necrosis may occur within the kidney, evidenced by pyuria and leading to scarring of the kidney and atrophy of renal tubules.

Signs and symptoms of pyelonephritis include fever, pain in the groin and/or flank, dysuria, and increased urinary frequency. Diagnosis of pyelonephritis is through urinalysis and urine culture, since it is often difficult to distinguish between cystitis and pyelonephritis based on clinical symptoms alone. Urinalysis reveals leukocyte-containing casts and more leukocytes than would be present in cystitis. Urine culture shows more antibody-coated bacteria than would be identified in cystitis.

Pyelonephritis is treated the same as cystitis, except that antibiotics specific for the causative agent are used and the duration of antibiotic treatment is longer. In addition, the patient’s urine is rechecked through cultures at 1 and 4 weeks after treatment, especially if symptoms recur. This precaution is due to the increase in the numbers of antibiotic-resistant bacteria and the possibility of more severe damage to the kidneys.

**Glomerulopathies:**
**Glomerulonephritis and Nephrotic Syndrome**

Glomerulopathies are diseases that affect mainly the function of the glomerulus. These disorders may be the primary disease, or they may result secondarily from some other systemic disease. All cases are distinguished by damage to the glomeruli with changes in capillary permeability. The two most common glomerulopathies are **glomerulonephritis** and **nephrotic syndrome**, both of which can affect people of any age, sex, or ethnic group.

**Glomerulonephritis** is an inflammation of the glomeruli due to infection, systemic disease, drugs, toxins, or an immune disorder. Due to the wide variety of causes, glomerulonephritis is often characterized by the specific causative agent or the progression of the disease (acute, chronic, or rapidly progressive). In general, the signs and symptoms of all types are similar, differing only slightly. A general discussion of glomerulonephritis is presented here.

Patients with glomerulonephritis usually show marked changes in urine composition due to changes in glomerular permeability. They typically exhibit proteinuria, hematuria, leukocyturia, and casts. Other signs include edema, systemic hypertension, and elevated **blood urea nitrogen (BUN)**. Renal function tests reveal a decreased glomerular filtration rate. Renal biopsy is often performed to determine the type and extent of injury to the kidneys.

Treatment of glomerulonephritis centers on the causative agent and managing the accompanying dysfunction. Although each type of glomerulonephritis would have a specific treatment regimen, all are aimed at controlling edema, hypertension, hyperkalemia, and hyperlipidemia. Acute glomerulonephritis leads to death in 2% to 5% of patients, while patients with chronic glomerulonephritis may live productive lives for up to 20 years after diagnosis.

**Nephrotic syndrome** is a kidney disease defined by the excretion of more than 3.5 g of protein per day in the urine. It is often accompanied by an abnormally low level of serum albumin. Since plasma protein level is a major factor in the osmotic uptake of water from the tissue fluid, this state of hypoproteinemia typically results in edema and ascites. Thus, a triad of proteinuria, hypoproteinemia, and edema is the chief sign of nephrotic syndrome. Nephrotic syndrome has a variety of forms and causes. It can be congenital, or it can be triggered by glomerulonephritis, certain drugs (such as street heroin), certain infections (such as HIV and some tropical parasitic infections), and some systemic diseases (such as diabetes mellitus and systemic lupus erythematosus).

Patients may first notice foamy or frothy urine. Subsequently, the patient history and physical examination often reveal anorexia, edema, abdominal pain, and muscle wasting. The edema may be localized to a single body region and may lead to other signs and symptoms such as chest pain and dyspnea, stemming from pleural effusion. Edema often appears in the face in the
morning and in the knees or ankles later in the day.

Nephrotic syndrome is suspected when the aforementioned triad of signs appears, and is confirmed by additional blood and urine findings, including elevated levels of serum lipids, fatty casts and blood cells in the urine, and urinary excretion of more than 3.5 g of protein per day. A final confirmation often comes from a renal biopsy showing damage to the glomerular basement membrane and the presence of autoantibodies in the glomerulus. Nephrotic syndrome can lead to renal failure and death. Some other consequences of nephrotic syndrome include susceptibility to infection due to the loss of immunoglobulins from the blood, prolonged blood clotting due to the loss of clotting factors, iron-deficiency anemia due to the loss of iron-transport proteins from the blood, and atherosclerosis due to the elevated serum lipid levels.

Treatment of nephrotic syndrome is twofold, involving first treating the causative agent and then the associated symptoms. The most common treatments include dietary restrictions (low salt and low fat), protein supplements to restore the plasma albumin level, anti-inflammatory drugs (most often steroids), and diuretics to control edema or hypertension. If damage to the kidney is severe, lifelong hemodialysis may be required.

Neurogenic Bladder

Neurogenic bladder results when the innervation of the bladder is disrupted. The disruption can be at the level of the brain, spinal cord, or peripheral nerves supplying the bladder. Disruption of the micturition reflex leads to incontinence, increased urination frequency, or a urinary obstruction that causes retention of urine within the bladder. There are a wide variety of causes of neurogenic bladder, including congenital abnormalities, trauma such as a ruptured intervertebral disc, and diseases such as syphilis, diabetes mellitus, central nervous system tumors, demyelinating or degenerative nervous system disease, or a cerebrovascular accident. However, the most common cause is spinal cord injury that results in paraplegia.

Although nervous control of the bladder is impaired in all cases of neurogenic bladder, the effect of the disruption is not always the same. For example, in the absence of nervous regulation, the bladder may be in either a hypotonic (flaccid) or spastic (contracted) state. In general, congenital defects produce hypotonic bladder, while trauma and disease produce either hypotonic or spastic bladder.

The signs and symptoms of neurogenic bladder vary due to the wide variety of causes. Diagnosis involves imaging techniques such as intravenous urography, ultrasound, and cystography, as well as assessment of the micturition flow rate. Treatment depends on the cause. Many patients are treated with catheterization and medications to improve bladder control. Patients must be continually monitored for renal function, infection, and the formation of calculi. Limiting dietary calcium intake minimizes calculus formation. If not treated and managed properly, neurogenic bladder can cause other renal disorders and impair renal function.

Using Diuretics to Treat Urinary System Disorders

Diuretics are chemicals that increase urine volume (see A&P, p. 903). They are usually used to reduce total body water and blood pressure. Thus, they are helpful in controlling hypertension, edema, congestive heart failure, and some other diseases. Diuretics are divided into different classes based on their mechanism of action:

- **Aldosterone antagonists** These are agents that work on the distal convoluted tubule to block the sodium-retaining action of aldosterone. Thus, more sodium remains in the renal tubule and passes in the urine, and where sodium goes, water follows.

- **Carbonic anhydrase (CAH) inhibitors** CAH is employed in the proximal convoluted tubules to break carbonic acid down into H⁺ and HCO₃⁻. The H⁺ is then normally excreted in exchange for Na⁺. By blocking the action of CAH, CAH inhibitors reduce H⁺ secretion and Na⁺ retention. Thus, again, more sodium and more water pass in the urine.

- **Osmotic diuretics** These are drugs that are freely filtered by the glomerulus but not reabsorbed by the renal tubules. Thus, they remain in the tubules, increasing the osmolarity of the tubular fluid. This osmolarity opposes tubular reabsorption of water, so more water is passed in the urine.

- **Sodium and chloride reabsorption inhibitors** These diuretics work mainly in the nephron loop and are therefore also called loop diuretics. They block the reabsorption of
Na\(^+\), K\(^+\), and Cl\(^-\) and thus promote salt and water elimination.

Each of these diuretics is appropriate to the treatment of different diseases, and they vary in their side effects. Some may cause hyperkalemia and others hypokalemia; some cause alkalosis and others acidosis; and so on. Before prescribing any of them, the clinician must have a thorough understanding of the disease at issue, the patient’s overall physiological condition, and the mode of action and side effects of each diuretic.

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**Case Study 23  The International Student with Renal Disease**

Haddi is a young Nigerian woman studying in the United States. One afternoon in March, she reports to the university health center complaining that she doesn’t feel well, she has no appetite, and her stomach hurts. She is told to come back first thing the next morning to give a urine specimen and have a physical examination. That day, her urine specimen is oddly frothy, and the nurse notes that Haddi’s eyelids are puffy. A dipstick urinalysis shows a high concentration of protein in the urine. Since this indicates a possibly serious disorder, the clinic refers Haddi to a urologist.

In taking Haddi’s history, the urologist learns that Haddi often notices that her face is puffy in the morning, and by afternoon she frequently has swelling in the knees and ankles. She occasionally has abdominal pains and sometimes difficulty breathing. The urologist asks Haddi about her travel history and history of other illnesses. Haddi says that she goes home to Nigeria during Christmas and summer breaks, and that she almost always gets malaria when she is there. She last went home in December, and had a bout of malaria then, as usual, but obtained treatment and her symptoms (chills and fever) disappeared. The urologist admits Haddi to the hospital for overnight observation and a 24-hour urine collection. Some of the results of her physical examination and laboratory work are shown here.

**Vital signs:**
- Oral temperature = 98.6°F (37.0°C)
- Heart rate = 68 beats/min
- Respiratory rate = 24 breaths/min
- Blood pressure = 131/73 mmHg

**Physical examination:**
- Edema of lower limbs, mild ascites

**Blood:**
- Hematocrit (Hct) = 34%
- RBC count = 3.3 x 10\(^6\)/μL
- Total protein = 3.1 g/dL
- Albumin = 1.6 g/dL

Sodium = 136 mEq/L
Other serum electrolytes = Normal
Blood urea nitrogen (BUN) = 57 mg/dL (mild azotemia)
Lipids: Fat droplets present
Low-density lipoproteins = 220 mg/dL
Triglycerides = 165 mg/dL
Cholesterol = 238 mg/dL

**Urine:**
- pH = 5.5
- Specific gravity = 1.052
- Protein excretion = 15.5 g/day
- Glucose and ketones = Negative
- Appearance: Light yellow, frothy.
- Urine culture: No pathogenic microorganisms.
- Sediment shows fatty casts, RBCs, and WBCs.
- Dipstick tests show proteinuria and hematuria.

On the basis of these findings and with Haddi’s consent, the urologist orders a renal biopsy. The histopathologist observes disruption of the glomerular basement membranes, and a stain for immunoglobulins in the glomerulus is positive.

The urologist diagnoses Haddi with nephrotic syndrome. He explains to her that nephrotic syndrome can be triggered by certain forms of malaria, and often develops a few months after a malarial attack. He says that her blood work shows no signs of malarial parasites at present, and Haddi says she has not had any of the fever and chills of malaria since returning to school for the semester. The physician advises her that nephrotic syndrome often clears up when the underlying cause is successfully treated, as her malaria appears to be. He warns her, however, that repeated bouts of malaria can worsen the condition and cause potentially fatal renal failure, and furthermore that malaria sometimes does not yield to drug therapy in people with nephrotic syndrome. These facts make it critically important, he says, that she take extreme measures to avoid malaria-carrying mosquitoes when she goes home and that she carefully observe malaria prophylaxis—taking
drugs in advance of her trips home to prevent malaria infection even if she is bitten.

In the meantime, the physician advises that Haddi remain in the hospital for treatment. She receives furosemide, a diuretic to treat her edema; an immunosuppressant to control the immune attack on her glomeruli; and I.V. albumin. She is placed on a low-fat, low-salt diet. From March through May, Haddi’s serum albumin returns to a normal level of 3.5 g/dL, her urinary protein excretion declines to a low level, and she is gradually withdrawn from the diuretic and immunosuppressant. Before traveling home in May, she takes a regimen of chloroquine for protection against malaria.

Based on this case study and other information in this chapter, answer the following questions.

1. Nephrotic syndrome is sometimes caused by diabetes mellitus. How do we know this is not the cause in Haddi’s case?
2. In nephrotic syndrome, what accounts for the froth in a freshly collected urine specimen?
3. Which data obtained from Haddi’s blood and urine are especially consistent with the edema she experiences?
4. Explain the pathophysiological reasons that Haddi has ascites, azotemia, and hematuria.
5. Why is Haddi given intravenous albumin? Which of her symptoms would be relieved by this treatment?
6. Aside from malaria prophylaxis, what are some other protective measures Haddi could take on her trips home in order to reduce her risk of kidney failure?

Howard, a 65-year-old male, is prescribed an osmotic diuretic, mannitol, for the treatment of hypertension. Explain how mannitol would affect his blood pressure and his daily urine output.

8. Based on your knowledge of the role of the renal tubule in regulating ion balance, explain how a diuretic could induce hyperkalemia. Then explain how a different diuretic might induce hypokalemia.

9. Susan, a 12-year-old girl, is brought to her pediatrician for a routine physical. Her mother mentions that Susan seems to be drinking a lot more water than normal. Urinalysis reveals elevated specific gravity, decreased pH, and glycosuria. Which of the following clinical signs would support a diagnosis of diabetes mellitus?
   a. ketonuria
   b. pyuria
   c. oliguria
   d. hemoglobinuria
   e. bright yellow urine

10. For each of the four diseases listed below, you are given a choice of two diagnostic methods. State which of the two methods you would use and why.
   - cystitis: retrograde pyelography or urine specific gravity?
   - pyelonephritis: urine culture or renal clearance test?
   - diabetes mellitus: chemical urinalysis or renal biopsy?
   - nephrotic syndrome: urine culture or chemical urinalysis?

Selected Clinical Terms

anuria A state of severely reduced urine output, under 100 mL/day, presenting a threat of azotemia and uremia.

cast A cylindrical, solid mass found in the urine sediments, molded by the renal tubule and composed of protein and various combinations of blood cells, epithelial cells, fat, bacteria, and crystals; typically an indication of serious renal pathology.

dysuria Pain upon urination.

enuresis Incontinence; the inability to retain urine or control its elimination.

hematuria Blood in the urine.

nocturia Having to urinate during the night.

oliguria A state of inadequate urine output, under 500 mL/day.

polyuria A state of excessive urine output, exceeding 2 L/day.

pyuria Pus in the urine.