The kidneys regulate the body’s fluid, electrolyte, and acid-base balances while removing toxic substances from the blood and excreting them in urine. The kidneys also play a significant role in erythropoietin and prostaglandin synthesis, in insulin degradation, and in the renin-angiotensin-aldosterone system.

Many common disease processes and injuries can interfere with normal renal function. Some of these disorders can result in renal failure (see Chapter 36). Because of the potential seriousness of any renal problem, the client and his or her significant others have physical as well as psychological needs. You need to know about the whole person and the person’s family and maintain awareness of appropriate interventions.

EXTRARENAL CONDITIONS

Many conditions located primarily in other parts of the body affect the kidneys, such as diabetes mellitus, hypertension, and sepsis. This chapter provides a brief description of the renal implications of these extrarenal conditions. For further discussion, see Chapters 45, 52 and 81.

DIABETES MELLITUS

One of the most common extrarenal diseases affecting the kidney is diabetes mellitus. Diabetic nephropathy, a progressive process, commonly leads to renal failure. About 30% of clients with end-stage renal disease (ESRD) (also known as stage 5 chronic kidney disease [CKD]) have diabetes mellitus. Researchers estimate that 25% to 50% of clients with insulin-dependent diabetes mellitus (IDDM, or type 1 diabetes mellitus) develop ESRD within 10 to 20 years of beginning insulin therapy. Renal disease also occurs in the non–insulin-dependent or type 2 diabetic client. The incidence of proteinuria is about 25% after 20 years of diabetes mellitus.

Several pathologic changes lead to renal failure in clients who have diabetes mellitus. The most common is a characteristic intercapillary glomerulosclerosis, or scarring of the capillary loops. Progressive microangiopathy, called nephrosclerosis, affects the afferent and efferent arterioles and eventually scars the glomerulus, tubules, and interstitium. Pyelonephritis (kidney infection) can scar the renal parenchyma and lead to ischemia. It may also lead to renal papillary necrosis and sloughing of the papillae. Neurogenic bladder dysfunction may contribute to renal failure. The high incidence of urinary tract infection or the increased pressure in the kidney caused by the backup of urine may also contribute to renal dysfunction.

Initially, the sclerotic, or hardening, process of glomerulosclerosis increases renal vascular resistance, contributing to systemic hypertension. This does not cause renal insufficiency. Indeed, the glomerular filtration rate (GFR) may increase as much as 20% to 50% above the normal GFR during this early “silent” phase. It is now recognized that microalbuminemia (measurable by assay) occurs quite some time before clinical proteinuria. If it is diagnosed, it may be a much earlier indicator of eventual renal failure. As more nephrons are destroyed, available functioning renal tissue decreases and the client begins to show clinical proteinuria (a key manifestation), hypertension, edema, and evidence of renal failure.

The kidney metabolizes 30% to 40% of insulin, and as renal function declines the degradation of insulin also decreases, resulting in a lower insulin requirement.

<table>
<thead>
<tr>
<th>Table</th>
<th>Less Common Renal Infectious Processes</th>
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<tbody>
<tr>
<td>Be sure to check out the bonus material on the Evolve website and the CD-ROM, including free self-assessment exercises.</td>
<td><a href="http://evolve.elsevier.com/Black/medsurg">http://evolve.elsevier.com/Black/medsurg</a></td>
</tr>
</tbody>
</table>
Renal failure may be initially identified when the client is evaluated for recurrent insulin reactions. Researchers hope the sclerotic process can be slowed by the following:

- Carefully controlling hypertension (see the Translating Evidence into Practice feature on Managing Hypertension in Diabetic Clients to Slow Progression of Renal Disease below)
- Adjusting insulin therapy and carefully monitoring blood glucose level to maintain euglycemia
- Restricting dietary protein (see the Complementary and Alternative Therapy feature on Slowing the Progression of Diabetic Nephropathy with a Unique Diet, p. 781)

Regardless of diabetic control, however, renal failure inevitably develops within 5 to 10 years after the appearance of significant proteinuria.

**HYPERTENSION**

Because the kidneys receive a large share of cardiac output, renal function can affect or be affected by cardiovascular changes. Renal blood flow determines the GFR, which directly affects renal function. Hypertension is one condition that can either cause or be affected by renal disease. For example, renovascular hypertension results from renal artery stenosis or renal infarction. The reduction in renal blood flow activates the renin-angiotensin-aldosterone system and increases systemic blood pressure.

Renal hypertension associated with parenchymal renal disease (e.g., glomerulonephritis, polycystic disease, pyelonephritis) usually results from the kidney’s decreasing ability to excrete salt and water. Other causes include increased renin release from increased glomerular perfusion and inadequacy of renal vasodilating substances, as occurs with analgesic nephropathy. Among clients with renal failure, 80% to 85% of hypertension results from excess salt and water retention; renovascular hypertension accounts for up to 15% of all systemic hypertension.

On the other hand, sustained systemic high blood pressure adversely affects the kidneys. Researchers report that nephrosclerosis can be seen microscopically in clients who have had uncontrolled hypertension for more than 5 years, although all other renal diagnostic tests may be normal. Kidney damage is the direct result of

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**Managing Hypertension in Diabetic Clients to Slow Progression of Renal Disease**

Both diabetes mellitus and hypertension can result in renal failure. One third of hypertensive males lose renal function over 7 years. It has been estimated that 5% of hypertensive clients with elevated serum creatinine levels will require dialysis therapy.1-4 Diabetic nephropathy may reduce glomerular filtration rate by 10 to 12 ml/min/year if hypertension is untreated. It has been found that lowering blood pressure to less than 130/80 mm Hg in clients with chronic kidney disease slows renal disease progression.1,2,4 Clients with proteinuria of greater than 1 g/24 hour benefit from even tighter control of blood pressure to levels of less than 125/75 mm Hg. Lowering blood pressure reduces mortality in those at risk for cardiovascular events, including diabetic clients.1,3

Despite this evidence, management of blood pressure in diabetic clients is less than optimal. Chronic disease management focusing on care processes and intermediate outcomes such as glycemic control found positive outcomes in type 2 diabetic clients. A recent randomized trial of a specialized clinic focusing on intensified multiple risk factor intervention showed improved microvascular disease and a trend toward improved macrovascular disease in diabetic clients within 4 years.7 Similar benefits were seen in a before-and-after study of diabetic clients with more advanced chronic kidney disease.8 Multidisciplinary clinics offering care by nurses and physicians, and sometimes other professionals, have demonstrated improved outcomes.8

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**IMPLICATIONS**

Control of blood pressure in diabetic clients and good disease management can improve outcomes and can slow progression of renal disease in diabetic clients.5 Nurses, in their roles as primary health care providers and as members of specialized disease management teams, can reduce the morbidity and complications associated with diabetes mellitus and hypertension. Further research should address the specific interventions and strategies that are most effective.

**REFERENCES**

In general, clients with end-stage renal disease are encouraged to follow a low-protein diet to slow the progression of their disease. However, a recent study challenged this notion by using a carbohydrate-restricted, low-iron, polyphenol-enriched diet. Foods rich in polyphenols include olive oil, cranberries, green tea, red wine, and grapes.

A total of 191 clients with type 2 diabetes mellitus and impending renal failure were included (men and women 49 to 62 years of age). Clients had a history of diabetes mellitus for 5 to 15 years and a glycosylated hemoglobin level of 6.0% to 9.3%. Current medications for both hypertension and glucose control were continued. Clients were assigned to either the low carbohydrate diet or a conventional protein-restricted diet (0.8 g/kg). The main features of the low-carbohydrate diet were a 50% reduction of carbohydrates; replacement of iron-enriched meats with iron-poor meats and foods known to inhibit iron absorption; elimination of all liquids except tea, water, and red wine; and the use of olive oil for frying and dressings. Unfortunately, compliance was not assessed.

Clients were followed for an average of 3.9 years. No significant differences were seen in the baseline characteristics of the two groups. Serum creatinine levels doubled in 21% of the clients following the lower carbohydrate diet versus 39% of the control group (p < 0.01). Thus a polyphenol-enriched diet with 50% carbohydrate reduction and low iron availability was better than a conventional protein-restricted diet in slowing the progression of diabetic nephropathy. More studies are needed, but the results are interesting.

**REFERENCE**

**HYPOTENSION**
Cardiovascular shock, or hypotension, also affects renal function. Renal vasoconstriction reduces renal blood flow. Because of the autoregulation capabilities of the kidneys (see Chapter 11, 12, 13, 32 and 81), however, GFR remains at a functional level until the advanced stages of systemic shock, at which time acute renal failure develops. Restoring systemic blood pressure usually reverses the renal vasoconstriction, and kidney function returns, typically within 2 to 8 weeks provided prolonged ischemia has not occurred. A period of polyuria (excessive urination) may follow the correction of hypovolemia, although the mechanisms for this are unclear.

Before renal function returns to normal, another oliguric period may occur, followed by a “mobilization phase” in which sequestered fluid is shifted into the intravascular space. This shift may cause some hypertension until the kidneys can remove the extra fluid. Careful assessment of the client’s fluid status and meticulous fluid management are crucial during these recovery phases.

**RHABDOMYOLYSIS**
Rhabdomyolysis is a disorder usually associated with traumatic injury of skeletal muscle tissue, which releases myoglobin and intracellular substances into the blood. It can also occur after serious crush injuries, strenuous exercise, seizures, heat stroke, prolonged coma, drug overdose, and as a side effect of the use of statins for treatment of hyperlipidemia. The resulting acute renal failure is usually reversible with treatment.

Clinical evidence of rhabdomyolysis includes fever, malaise, nausea, vomiting, muscular weakness, muscle pain, and swelling. The release of substances from damaged muscles results in myoglobinemia, myoglobinuria (which can be seen as brown urine and confirmed through urinalysis), hyperkalemia, hyperphosphatemia, hyperuricemia, and elevated creatine kinase levels. Hypocalcemia occurs initially because of the precipitation of calcium with phosphate. Later, in the diuretic phase of acute renal failure, hypercalcemia can occur as calcium is mobilized.

Treatment is typically symptomatic, including bed rest to reduce muscle metabolism and steps to correct acidosis and electrolyte imbalances and maintain normal fluid volume. In severe cases, dialytic therapy may be necessary.

**CARDIOVASCULAR DISEASE**
Cardiac disease influences kidney function primarily through its effect on cardiac output and circulating blood volume. The hemodynamic and hormonal changes of cardiac disease may decrease the kidneys’ ability to excrete sodium and water. This, in turn, increases intravascular congestion and edema and establishes a pathologic cycle.

Hemodynamic changes also occur with normal aging. Blood flow to the kidneys decreases by up to half by age 70 years, and GFR can decrease by 40% to 50% as well. Renal function deteriorates as glomeruli become sclerotic and atrophy.
PERIPHERAL VASCULAR DISEASE

Thromboembolic disease can affect the renal circulation and cause infarction of the tissue supplied by the affected blood vessel. In clients with sickle cell disease, the interstitial hypertonicity and low oxygen pressure found in the renal medulla seem to favor sickling of red blood cells in the kidney’s juxtamedullary region. These cell masses cause gross hematuria (from rupture of venules), papillary necrosis, renal infarction, concentration disturbances (from interference with the countercurrent mechanism), nephrotic syndrome, pyelonephritis, and, finally, renal failure.

In disseminated intravascular coagulation (DIC), in which diffuse clotting consumes clotting factors and causes hemorrhage in affected areas throughout the body, the kidney is the organ most affected.

SEPSIS

Extrarenal sepsis may affect kidney function either through its effect on systemic circulation or by stimulating the immune system. Renal reactions to septic shock are similar to those in hypotension. Immunologic injury can lead to glomerulonephritis (see Glomerulonephritis later in this chapter). Occasionally, pathogens may break away from extrarenal foci of infection and travel to the kidney to establish additional sites.

PREGNANCY

Pregnancy has a definite influence on kidney function. During the first trimester, the collecting system dilates and the kidneys enlarge; this may persist 9 to 12 weeks after delivery. Renal blood flow and GFR increase by 30% to 50% during pregnancy, contributing to increased creatinine clearance and decreased uric acid excretion. These normal changes (such as lower serum creatinine level) must be taken into account in interpreting laboratory findings for pregnant women. Pregnancy also increases the likelihood of proteinuria (usually transient), polyuria, and nocturia (excessive urination at night). These disorders may be caused by external bladder compression and alterations in antidiuretic hormone metabolism.

OTHER CAUSES

Kidney function is influenced by many other extrarenal disease processes, such as cancer, connective tissue disorders, and metabolic disturbances. Many systemic diseases produce clinical manifestations like those of glomerulonephritis, although they typically have other systemic features characteristic of the disease (see Glomerulonephritis). These diseases include systemic lupus erythematosus (SLE), systemic scleroderma, polyarteritis nodosa, thrombocytopenic purpura, Wegener’s granulomatosis, hemolytic-uremic syndrome, gout, amyloidosis, and Henoch-Schönlein syndrome. Diagnosis can be confirmed by renal biopsy.

Renal disease has become an increasingly common complication for people infected with the human immunodeficiency virus (HIV). Among the several renal disorders associated with HIV and acquired immunodeficiency syndrome (AIDS) are renal tuberculosis and cytomegalovirus, such malignancies as lymphoma and Kaposi’s sarcoma, and HIV-associated nephropathy, a focal glomerulosclerosis that is manifested by nephrotic syndrome (see Nephrotic Syndrome later in this chapter).

NEPHROTOXINS

Nephrotoxins have specific, destructive effects on renal cells. They can cause the following types of renal injury: acute tubular necrosis, defects in the tubular transport system, interstitial nephritis, vasculitis, and nephrotic syndrome. Box 35-1 presents some common nephrotoxic substances. Acute tubular necrosis is the most frequent injury resulting from exposure to nephrotoxins. Some nephrotoxins also cause tubular transport defects and nephrotic syndrome.

### BOX 35-1 Nephrotoxins

<table>
<thead>
<tr>
<th>ANTIBIOTICS</th>
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<tbody>
<tr>
<td>Aminoglycosides</td>
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<tr>
<td>Tetracyclines</td>
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<tr>
<td>Amphotericin B</td>
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<tr>
<td>Cephalosporins</td>
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<tr>
<td>Sulfonamides (co-trimoxazole)</td>
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<tr>
<td>Bacitracin</td>
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<td>Polymyxin</td>
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<tr>
<th>ANALGESICS</th>
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<tbody>
<tr>
<td>Salicylates</td>
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<tr>
<td>Acetaminophen</td>
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<tr>
<td>Phenacetin</td>
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<tr>
<td>NSAIDs (nonsteroidal anti-inflammatory drugs)</td>
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<tr>
<th>HEAVY METALS</th>
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<td>Lead</td>
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<td>Mercury</td>
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<td>Bismuth</td>
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<td>Arsenic</td>
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<td>Copper</td>
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<td>Cadmium</td>
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<td>Gold</td>
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<td>Lithium</td>
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<tr>
<th>POISONS</th>
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<tr>
<td>Mushrooms</td>
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<tr>
<td>Insecticides</td>
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<tr>
<td>Herbicides</td>
<td></td>
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<tr>
<td>Snake venom (bites)</td>
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<tr>
<th>ANESTHETICS</th>
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<tr>
<td>Probenecid</td>
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<tr>
<td>Phenytoin</td>
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<td>Heroin</td>
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<td>Dextran</td>
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<td>Mannitol</td>
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<td>Interleukin-2</td>
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<td>Cisplatin</td>
<td></td>
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<tr>
<td>Amphetamines</td>
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<tr>
<td>Aristolochic acid (Chinese herb)</td>
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<tr>
<th>OTHER DRUGS</th>
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<tbody>
<tr>
<td>Ethylene glycol</td>
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<tr>
<td>Gasoline</td>
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<td>Kerosene</td>
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<th>ORGANIC SOLVENTS</th>
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<tbody>
<tr>
<td>Ethylene glycol</td>
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<td>Gasoline</td>
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<td>Kerosene</td>
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All five types of kidney damage may result from nephrotoxic reactions to medications. You must know about the possible adverse effects of any medication a client takes so that you can assess and intervene appropriately. Two types of medications well-known to cause renal damage are antibiotics and certain analgesics (see Box 35-1). Because the kidneys are the major route of excretion for many antibiotics and analgesics, renal tissue is directly exposed to these compounds. Researchers estimate that 5% to 10% of clients with ESRD have analgesic nephropathy. Diuretics may have nephrotoxic effects as well and, when used aggressively, can cause hypovolemia. The longer the exposure, the higher the risk of renal toxic effects. Pre-existing renal disease, decreased renal blood flow, electrolyte imbalances, and concurrent use of other nephrotoxic medications enhance a medication’s nephrotoxic effect.

Carefully monitor renal function tests to identify early nephrotoxic reactions so that causative medications can be discontinued or the dose decreased. Closely monitor drug levels to ensure that dosages stay in the therapeutic range. Besides using these medications as briefly as possible and at as low a dose as possible, maintaining a high fluid intake may help prevent nephrotoxic effects. A high urine output keeps the medication diluted in the kidney and helps prevent crystallization.

Anesthesia reduces the kidney’s vasoconstrictive ability, which helps protect it against systemic blood pressure drops; thus the kidney is made more vulnerable to the effects of shock. In addition, certain anesthetics, particularly methoxyflurane, have a direct nephrotoxic effect. Administration of this general anesthetic agent can cause acute tubular necrosis and has been associated with fatal acute renal failure. Halothane may also adversely affect renal function.

Radioiodinated contrast agents used in radiographic and computed tomographic (CT) studies have been associated with acute tubular necrosis. Risk factors include age older than 60 years, pre-existing renal insufficiency (especially diabetic nephropathy), dehydration, low cardiac output with pre-existing renal disease, proteinuria, hypoalbuminemia, multiple myeloma, and multiple contrast studies within a 24-hour period. See the Complementary and Alternative Therapy feature below.

**Oral Acetylcysteine Supplements for Preventing Acute Deterioration in Renal Function After Coronary Angiography in Moderate Renal Insufficiency**

Acetylcysteine tablets already play several roles in medicine. For example, they seem to reduce hepatic damage from acetaminophen overdose and have a role in clearing lung function in cystic fibrosis. Other roles for this supplement are being explored. Many procedures in medicine are life-saving, but require contrast or some type of dye to help identify abnormalities in the human body. However, if the dye is given in large amounts during a procedure, it can damage the kidneys. For example, large amounts of dye are needed for visualization of the arteries during angiography and angioplasty. Those same clients do not pump blood as well from the left ventricle, which means the dye does not get diluted easily because circulation is not good.

A randomized, blinded, placebo-controlled trial with a 7-day follow-up was conducted in a university hospital in Hong Kong. A total of 200 clients (mean age 68 years, 61.5% men, 100% Chinese) with moderate chronic renal insufficiency and upcoming elective coronary angiography were studied. Clients received either oral acetylcysteine (600 mg twice daily, n = 102) or a placebo (n = 98) both the day before and the day of angiography (total treatment duration of 2 days). All clients received a nonionic, low-osmolality contrast agent (iopamidol).

Kay et al. found that in clients with moderate chronic renal insufficiency, oral acetylcysteine was safe and effective in preventing acute deterioration in renal function after elective coronary angiography. In fact, acute contrast media–induced reduction in renal function was reduced by 68% in the supplement group compared to the placebo group (NNT = 12).

Similarly, acetylcysteine was also found to be helpful for reducing renal deterioration in at-risk clients 62 years of age with histories of renal insufficiency, diabetes mellitus, elevated cholesterol, and/or smoking in Milan, Italy, who were scheduled to undergo angioplasty and possible stent placement. In a randomized study, 354 clients having angioplasty were assigned to 3 groups. One group received the standard acetylcysteine dose of 600 mg IV before the procedure and 600 mg orally twice a day for 48 hours after the procedure for a total of 3000 mg. Another group received twice that amount in the same manner. The third or control group received a placebo. Serum creatinine levels increased by 25% or more in 33% of the control group, in 15% of the standard dose group, and in 8% of the double dose group.

Therefore acetylcysteine should be considered for high-risk clients who have diabetes mellitus or chronic renal disease and are about to undergo coronary angiography.

**REFERENCES**


Using non-dye studies whenever possible and keeping the client well hydrated throughout the test will reduce the risk of acute renal failure. Baseline renal function tests before the contrast study should be available to compare with post-test findings. Monitor the client’s urine output carefully for several hours after the study is completed.

**ACQUIRED DISORDERS**

**NEPHROLITHIASIS**

Although calculi (stones) can form anywhere in the urinary tract, the most frequent site is the kidney. These stones may travel down the urinary tract, lodge anywhere along the tract, and cause obstruction and tissue damage, or they may stay in the kidney. Urolithiasis is described in detail in Chapter 34.

Treatment and nursing care of clients with renal calculi is similar to that for clients with calculi lower in the urinary tract. Damage to the kidney caused by calculi can be permanent, however, and may require nephrectomy (described later).

**PYELONEPHRITIS**

*Pyelonephritis* is an inflammation of the renal pelvis and parenchyma caused by a bacterial infection. The cause may be an active infection in the kidney or the remnants of a previous infection. The two main types of pyelonephritis are acute and chronic. They differ primarily in their clinical picture and long-term effects.

**Etiology and Risk Factors**

Sometimes an infection may be a primary disease, as happens with reduced host resistance (e.g., calculi, malignancy, hydronephrosis, or trauma). Most kidney infections, however, are extensions of infectious processes located elsewhere, especially the bladder. In Chapter 34, the etiologic mechanism and pathogenesis of infections in the lower urinary tract are discussed.

The bacteria spread to the kidney primarily by ascending the ureter to the kidney. Blood and lymphatic circulation also provide channels for the organisms. Ureteral reflux, which allows infected urine back into the ureter, and obstruction, which causes urine to back into the ureter and allows organisms to multiply, are the most common causes of ascending urinary tract infections. *Escherichia coli* is the most common bacterial organism that causes pyelonephritis.

Health promotion is key to preventing the recurrence of infection and further renal damage. The nurse provides information to clients about health and lifestyle measures to prevent urinary tract infections, including (1) perineal hygiene measures (such as wiping from front to back), (2) acidification of the urine (by drinking cranberry juice or taking ascorbic acid), and (3) ensuring adequate fluid intake. Early detection and adequate treatment of lower urinary tract infections greatly reduce the incidence of pyelonephritis.

After infection, health maintenance includes education about the importance of completing the course of antibiotics. Follow-up cultures are important with recurrent pyelonephritis to ensure that the infection has been eradicated. Health restoration measures depend on the extent of renal damage and the cause of the disease. If obstruction precipitated the infection, the cause of the obstruction must be treated.

**Acute Pyelonephritis**

Acute pyelonephritis often occurs after bacterial contamination of the urethra or after introduction of an instrument, such as a catheter or a cystoscope.

**Chronic Pyelonephritis**

Chronic pyelonephritis is more likely to occur after chronic obstruction with reflux or chronic disorders. It is slowly progressive and usually is associated with recurrent acute attacks, although the client may not have a history of acute pyelonephritis.

**Pathophysiology**

Pyelonephritis occurs when bacteria enter the renal pelvis, causing an inflammatory response and an increase in white blood cells (WBCs). The inflammation leads to edema and swelling of the involved tissue, beginning at the papillae and sometimes spreading to the cortex. The infection can be either ascending, as occurs after cystitis or prostatitis, or descending, as from a streptococcal infection in the bloodstream.

As the infection is treated and the inflammation recedes, fibrosis and scar tissue may develop. The calices become blunted with scarring in the interstitial tissues. If the infection recurs, more scar tissue develops; fibrosis and altered tubular reabsorption and secretion lead to decreased renal function.

**Acute Pyelonephritis**

Acute pyelonephritis is associated with the development of renal abscesses, perinephric abscesses, emphysematous pyelonephritis, and chronic pyelonephritis, which can lead to renal failure. Acute pyelonephritis is usually brief. It often recurs, however, either as a relapse of a previous infection not eradicated or as a new infection; 20% of these recurrences take place within 2 weeks after completion of therapy. A client must be treated adequately to prevent the development of chronic pyelonephritis.
The infection may also progress to bacteremia and urosepsis.

**Chronic Pyelonephritis**
This disease is characterized by a combination of caliceal abnormalities and overlying cortical scarring. The kidney becomes contracted, and the number of functioning nephrons decreases as they are replaced by scar tissue. Renal failure may ensue, although uremia is less common than once thought.

**Clinical Manifestations**

**Acute Pyelonephritis**
Acute pyelonephritis is characterized by enlarged kidneys, focal parenchymal abscesses, and accumulation of polymorphonuclear lymphocytes around and in the renal tubules. Typically, the client seems to be in acute distress, although in some cases this disorder causes minimal or no manifestations.

Assessment usually reveals high fever, chills, nausea, flank pain on the affected side (costovertebral angle [CVA] tenderness), headache, muscle pain, and general prostration. The pain commonly radiates down the ureter or toward the epigastrium and may be colicky if the infection is complicated by calculi or sloughed renal papillae. Commonly, the client has experienced dysuria, frequency, urgency, and other evidence of cystitis for several days. The urine may be cloudy or bloody, is foul smelling, and shows a marked increase in WBCs and casts. See Chapter 32 and Figure 32-8 for more information related to assessment of urinary tract infections.

**Chronic Pyelonephritis**
This disease has no specific manifestations of its own. Thus it is usually discovered incidentally when the client is being evaluated for hypertension or its complications. Hypertension is the most frequent manifestation of the disease. Abnormal laboratory studies may show azotemia, pyuria, anemia, acidosis, and proteinuria. They may also demonstrate poor urine-concentrating ability.

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**OUTCOME MANAGEMENT**

**Medical Management**

**Acute Pyelonephritis**
Ideal outcomes of medical management include (1) elimination of the pathogenic organisms with appropriate antibiotics, as identified by urine culture and sensitivity studies, and (2) removal of any factor or disease contributing to decreased host resistance. If calculi or other obstructions are found to be the cause of recurrent infection, appropriate treatment must be instituted.

**Inhibit Bacterial Growth.** Antibiotic therapy is based on the results of urine culture and sensitivity tests. Typically, a broad-spectrum antibiotic is prescribed; it may be changed after the results of the culture are available. Sulfonamides or the combination of sulfamethoxazole and trimethoprim is commonly used as first-line therapy unless the client is allergic to one of these drugs. Typically, antibiotic therapy continues for 10 days to 2 weeks. In severe cases of acute pyelonephritis, intravenous antibiotics may be administered. With oral therapy, the client must understand that completing the full course of antibiotic therapy is important to prevent recurrence of the infection. Recurrent infections are commonly treated with long-term prophylactic antibiotic therapy. Additional pharmacologic therapy may be needed to correct any predisposing factors.

**Relieve Pain.** Analgesic or urinary antiseptic medications can be prescribed to reduce discomfort. Antibiotics quickly reduce discomfort as well.

**Chronic Pyelonephritis**
The desired outcome of medical management is prevention of further renal damage. If bacteria are found, appropriate antibiotics are given, as in acute pyelonephritis. Chronic pyelonephritis tends to be less painful. Above all, hypertension must be controlled. Additional intervention depends on the degree of renal failure that has already occurred. Although high fluid intake may be advisable in acute pyelonephritis, it may be contraindicated in chronic pyelonephritis if the degree of renal dysfunction is significant.

**Inhibit Bacterial Growth.** Antibiotics specific to the bacteria present are given to treat chronic pyelonephritis (see Acute Pyelonephritis and Chapter 34).

**Control Hypertension.** Renal damage can cause hypertension, which can cause further renal damage. Thus it is important to control the client’s blood pressure. Reduction of dietary sodium and pharmacologic therapy may be indicated. Management of hypertension is discussed in Chapter 52.

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**Nursing Management of the Medical Client**

**Assessment.** Assessment of the client with pyelonephritis begins with a thorough history and physical examination, giving close attention to the presence of risk factors, previous urinary tract infections, hypertension, and CVA tenderness. Look for evidence of pyelonephritis.

**Diagnosis, Outcomes, Interventions**

**Diagnosis: Risk for Deficient Fluid Volume.** A common diagnosis is Risk for Deficient Fluid Volume related to fever, nausea, vomiting, and possible diarrhea.
Outcomes. The client will maintain fluid balance as evidenced by balanced intake and output, maintenance of adequate hydration, and an absence of manifestations of dehydration.

Interventions. Prepare the client for the diagnostic tests and probable antibiotic therapy. Clients with severe nausea and vomiting may require intravenous fluids. Keep in mind that overhydration may dilute antimicrobials, diminishing their effectiveness. See Chapter 34 on the nursing care of the client with cystitis.

Diagnosis: Acute Pain. Another common nursing diagnosis is Acute pain related to an inflammatory process in the kidney and possible colic.

Outcomes. The client will report either that there is no pain or that pain is controlled.

Interventions. Medications can be given to control pain caused by calculi. CVA tenderness should decrease as the antibiotics control the infection. Medication for nausea can be given as needed with antipyretics for high fevers. Adequate treatment of the infection quickly reverses the dysuria, pyuria, and frequency. Urinary analgesics (see Chapter 34) can also help the client with these problems. Fluid intake of 3 to 4 L daily is recommended. This fluid helps to dilute the urine and to reduce irritation and burning. The continual flow of urine serves to prevent stasis and discourage multiplication of bacteria in the urinary tract.

Diagnosis: Readiness for Enhanced Self-Care. Client teaching is important to promote self-care and to prevent recurrent infections. Write the diagnosis Readiness for Enhanced Self-Care to prevent recurrent infections.

Outcomes. The client will have knowledge of the treatment regimen and understand how to prevent recurrent infections as evidenced by the client’s statements and no recurrence of infection.

Interventions. The preventive measures for acute and chronic pyelonephritis are similar to those for cystitis (see Chapter 34). It is important to prevent permanent renal damage. Ensure that the client can recognize the manifestations of a urinary tract infection and knows to seek prompt medical attention when these manifestations do occur.

When the acute infection subsides, instruct the client to continue follow-up care. This care includes completing the full course of antibiotic therapy and having repeated urine cultures. Also, teach ways to prevent further infections in the urinary tract, including ensuring a high fluid intake (see Chapter 34).

It is vital that the client return for follow-up urine cultures and possibly other diagnostic tests if the cause of the pyelonephritis is not clear. Emphasize that follow-up cultures are important because bacteriuria may be present without producing any manifestations. Advise the client to report any manifestations of recurrence immediately so that retreatment can begin.

Evaluation. The infection should subside with adequate antibiotic treatment. Successful management results in reduced pain and negative findings on follow-up urine cultures. The client must also be made aware of the cause of this infection and ways to prevent further infections (see Chapter 34).

Modifications for Older Clients

In older clients, the kidneys may be less able to recover from a severe infection. Antibiotic therapy should be monitored closely because older adults often vary in their sensitivity and response to the medication. Older adults may also have altered blood levels of antibiotics because renal perfusion decreases with age, reducing the kidney’s ability to excrete drugs.

OTHER INFECTIOUS PROCESSES

Bacteria cause most cases of pyelonephritis, but renal candidiasis, a fungal infection, is becoming more prevalent. Renal tuberculosis, renal abscesses, and perinephric abscesses are other less common infectious processes. They are briefly described in the Less Common Renal Infectious Processes table on the website.

HYDRONEPHROSIS

Hydronephrosis is distention of the renal pelvis and calices caused by an obstruction of normal urine flow. Urine production continues, and the urine is trapped proximal to the obstruction. Causes of occlusion include calculus, tumor, scar tissue, congenital structural defects, and a kink in the ureter.

Whatever the cause, the accumulating urine exerts pressure on the renal pelvis wall. At low to moderate pressures, the kidney may dilate with no obvious loss of function. Over time, sustained or intermittent high pressure causes irreversible nephron destruction. In addition to pressure-related problems, pyelonephritis is always a risk because of urinary stasis.

OUTCOME MANAGEMENT

Medical Management

Treatment aims to relieve the obstruction and prevent infection. Depending on the location of the obstruction, it may involve placement of a ureteral catheter or stent above the point of obstruction. Typically, surgery is required (see Chapter 34) to relieve the obstruction and restore adequate drainage of the urinary system.
Removal of the obstruction results in sudden release of the pressure on the renal parenchyma caused by the trapped urine, which leads to diuresis. Thus postobstructive diuresis occurs and can lead to fluid and electrolyte imbalances, including dehydration. The kidney gradually begins to concentrate urine appropriately.

### Nursing Management of the Medical Client

Assessment of a client with hydronephrosis includes monitoring for the presence, location, intensity, and character of pain. Monitor urine output, and report manifestations of renal failure (oliguria, anorexia, lethargy), hematuria, and dysuria. Reduced urine output could indicate obstruction. Scan the client’s bladder to assess for any manifestations of distention or urinary retention. The kidneys, if palpated, may be tender.

Make frequent assessments, including hourly outputs; daily weights; vital signs every 30 minutes for the first 4 hours and then every 2 hours; urine for specific gravity, albumin, and glucose; and edema. Also make periodic serum electrolyte and glucose determinations, and consider the expected presence of severe fatigue caused by urinary losses and the need for frequent observation. Fluid management during this period is crucial; hourly fluid replacement is based on the previous hour’s output.

Clients who have had hydronephrosis should watch for manifestations of infection and obstruction, such as pain and reduced urine output. Avoiding urinary tract infections is important in preventing pyelonephritis and preserving renal function (see Chapter 34).

### Surgical Management

Surgery is commonly required to relieve the obstruction causing hydronephrosis. Management of the surgical client is discussed under urolithiasis in Chapter 34.

### RENAL CANCER

Benign kidney tumors are rare. Classifications include lymphangioma, lipoma, medullary fibroma, adenoma, leiomyoma, and oncocytoma. When large benign tumors occur, it is relatively impossible to distinguish them from a malignant tumor by x-ray examination. At least 85% of all renal tumors are malignant, and about 12,890 people die of kidney cancer each year. The tumors are most common in people 50 and 70 years of age. They affect men more often than women. About 51,190 new cases of renal cancer were expected to be diagnosed in the United States in 2007.1

### Etiology and Risk Factors

The exact cause of renal tumors is unknown. Some links have been established between renal cancer and smoking, hypertension, and obesity. Exposure to lead, cadmium, and phosphates also increase the risk of developing renal cancer. An inactivation of a critical gene on the short arm of chromosome 3 is thought to be related to the development of renal cancer.

Because of the possible association between smoking and renal cancer, one means of avoiding renal cancer may be to quit or not to start smoking. Avoiding exposure to chemicals such as lead, phosphate, and cadmium may also prevent some renal cancers. A lifestyle that minimizes the development of obesity and hypertension may also be helpful (see the Complementary and Alternative Therapy feature below). The cause of many renal cancers is not established, however, and prevention may not be possible.

After surgery, most clients have difficulty in dealing with cancer and the risk of recurrence. If nephrectomy is required, clients are often concerned about living with only one kidney. Assure clients that one kidney can meet the body’s needs but that care should be taken to protect that kidney. The care includes preventing injuries and infections, controlling blood pressure if necessary, and maintaining overall health and well-being through adequate nutrition and rest, for example.

### Pathophysiology

Renal cell carcinoma (RCC), or adenocarcinoma, is the most common tumor type; it accounts for 90% of all kidney cancer. Tumor growth begins in the renal cortex and

#### Complementary and Alternative Therapy

**Fatty Fish Consumption and Renal Cell Carcinoma**

The epidemiologic evidence that fatty fish consumption may be associated with lower risk of several cancers is not consistent, and no studies of renal cell carcinoma (RCC) exist. This study examined the association between fatty fish and lean fish consumption and risk of RCC in 61,433 women in the Swedish Mammography Cohort study. During a mean follow-up between 1987 and 2004 (15.3 years for a total of 940,557 person-years), 150 incident RCC cases were diagnosed. After adjustment for potential confounders, an inverse association of fatty fish consumption with the risk of RCC was found, but no association was found with lean fish consumption. Compared with no consumption, the multivariate rate ratio (RR) was 0.56 (95% confidence interval [CI], 0.35-0.91) for women eating fatty fish once a week or more. The findings suggest that consumption of fatty fish may reduce the risk of RCC in women.

**REFERENCE**

usually continues for some time before it produces manifestations. The tumor can grow very large and tends to compress the adjacent renal parenchyma rather than infiltrate it. The tumor, usually avascular, tends to surround blood vessels and constrict them. The lungs and mediastinum are the most frequent metastatic sites of occurrence. Liver, bone, skin, spleen, renal vein, and brain are other common sites of metastases.

Other types of renal cancer include (1) nephroblastoma, (2) sarcoma, and (3) epithelial tumors in the renal pelvis. Nephroblastoma, or Wilms’ tumor, is primarily a childhood disease, although it occasionally occurs in adults. The prognosis for adults is worse than that for children, with some sources reporting only a 25% survival rate. Sarcoma is infrequent and typically arises in the renal capsule. Most tumors of the renal pelvis are primarily urothelial in origin and include three tissue types: transitional cell, squamous cell, and adenocarcinoma.

Spontaneous regression of renal adenocarcinoma reportedly occurs in less than 1% of all cases. Most of these regressions occur after nephrectomy and involve metastatic areas. Authorities consider these episodes as evidence that the disease is associated with immunologic or hormonal factors.

Clinical Manifestations

Manifestations of renal malignancies vary, and tumor growth may advance significantly before the disease is discovered. It is not uncommon for the client to have clinical manifestations apparently unrelated to renal disease. Frequently, a palpable abdominal mass found during a routine physical examination arouses the first suspicion. The average time between the onset of hematuria and the onset of pain is 9 months and that between initial pain and diagnosis is 14 months. Extrarenal manifestations are commonly found before a diagnosis of renal cancer is confirmed. Up to 35% of clients have metastasis when the final diagnosis of a renal cancer is made.

The common triad of manifestations consists of hematuria, flank pain, and a palpable abdominal or flank mass. The hematuria is usually gross and intermittent, which helps to explain the client’s delay in seeking medical advice. The clinical picture also contains a combination of the following usual findings: fever, weight loss and cachexia, fatigue, hypertension, amyloidosis, thrombophlebitis, anemia, erythrocytosis, hypercalcemia, abnormal serum liver profile, and an elevated erythrocyte sedimentation rate (ESR). Less frequent findings include peripheral neuropathy, inferior vena cava obstruction, priapism, and varicocele. Hydronephrosis may occur if the tumor obstructs the ureteropelvic junction. The incidence of pulmonary embolus as a presenting manifestation may be higher than previously thought because of the high rate of vena cava and renal vein involvement. Plasma erythropoietin, renin, and chorionic gonadotropin levels are elevated, and prostaglandin production increases in renal cell carcinoma.

Several diagnostic tests help confirm a diagnosis of renal cancer. Intravenous pyelogram (IVP) is probably the most helpful in identifying a space-occupying lesion. Ultrasonography helps differentiate a cyst from a solid mass. Other noninvasive procedures include CT scan, nephrotomography, and radioisotope studies. Arteriography is used to evaluate the renal vascular system. Renal biopsy, usually done percutaneously, provides definitive data about the lesion.

Outcome Management

Staging of the tumor helps delineate the appropriate treatment and can suggest the client’s prognosis (Figure 35-1). Five-year survival rates for stage 1 are about 65%; for stage 2, about 40%; 10-year rates drop to 40% and 35%, respectively. Five-year survivals are rare in stages 3 and 4.1

Medical Management

Radiation Therapy

Radiation may be used as an adjunct with chemotherapy and surgery. Irradiation is most useful in preoperative preparation of the tumor. It is sometimes also used postoperatively to destroy residual or recurrent tumor cells, treat lymphatic involvement, and treat metastatic sites, such as bones, palliatively.

Chemotherapy

Clinical investigators continue to search for an effective chemotherapeutic regimen. Medroxyprogesterone and testosterone have been used as hormonal therapy, but
their effectiveness has been limited. Vinblastine seems to be the most effective single agent, with response rates of 25%. Combination regimens seem to increase toxic effects without improving response rates. Many agents are being studied, but renal cancer cells seem insensitive to chemotherapeutic or hormonal agents, possibly because of their slow growth rate.

**Immunotherapy**

Immunotherapy holds some promise in the treatment of renal cancer. Stimulants of the immune system have led to some positive results as long as the tumor is not too large and the immunosuppression is not too severe. There has also been some response to natural and recombinant interferon-alfa. Interleukin-2 has been approved by the Food and Drug Administration for treating renal carcinoma. Studies using vaccines are under way. Clients are immunized with irradiated tumor cells and evaluated for immune responses and clinical tumor regression. Nonmyeloablative allogeneic stem cell transplantation is another new approach. This may induce sustained regression of metastatic renal cell carcinoma in clients who have had no response to conventional immunotherapy, but further studies are required.

**Nursing Management of the Medical Client**

Nursing management of the client with renal cancer must include general aspects of care for any cancer (see Chapter 17).

**Surgical Management**

**Nephrectomy**

For renal cell carcinoma, the surgical procedure of choice is generally radical nephrectomy, which includes removal of the kidney, the adrenal gland, and perinephric fat with the retroperitoneal lymphatics. Several surgical approaches can be used to remove the diseased kidney. Transabdominal and thoracoabdominal approaches are preferred to secure the renal artery and vein and to prevent the spread of malignant cells.

A retroperitoneal approach is also possible. An incision of 6 to 10 inches is made, usually in the flank area; muscle layers are divided; and tissues are excised. The renal artery and vein are clamped and cut, and the ureter is dissected. When the tumor is in the renal pelvis, a nephroureterectomy is usually performed because of a tendency for transitional cell cancer to “seed” down the ureter into the bladder. With nephroureterectomy, a cuff of the adjacent bladder is removed. Lymphadenectomy remains controversial. Even in advanced cases, when the prognosis is poor, nephrectomy is sometimes done to relieve pain and hematuria.

If the neoplastic disease is bilateral or if there is a solitary functioning kidney, a partial nephrectomy can be done on at least one kidney, leaving enough renal tissue to support life without long-term dialysis. If partial nephrectomy is not possible in either instance, the entire kidney is removed and the client undergoes dialysis. These clients may be candidates for renal transplantation, but they are usually maintained with dialysis for about a year to watch for recurrence of the disease.

Although open nephrectomy remains the procedure of choice for many urologists, laparoscopic and robotic-assisted laparoscopic nephrectomy are being performed in a number of centers with considerable success. Four small incisions are made through fewer muscle layers. A special laparoscope is inserted through one of the incisions, and laparoscopy instruments are placed in the others. Carbon dioxide is passed through a tube in one incision to inflate the abdominal cavity, which enables the surgeon to see the organs and provides room for manipulation of instruments. At the end of the procedure, the kidney is removed through a small 2- to 3-inch incision below the navel.

The laparoscopic surgical procedure with or without robotic assistance tends to be longer (6.9 versus 2.2 hours), but clients who undergo the laparoscopic procedure require fewer analgesics, resume oral intake earlier, are discharged home earlier, and return to work sooner than those undergoing open surgery. Increasingly, removal of a kidney for organ donation is being performed laparoscopically.

**Indications.** Nephrectomy or heminephrectomy is indicated with tumors of the kidney.

**Contraindications.** As with any surgery, nephrectomy is contraindicated in clients with systemic or respiratory tract infections. General health must be satisfactory to withstand anesthesia, blood loss, and surgical stress. Any metabolic and systemic disorders should be stabilized before surgery.

**Complications.** Because the kidney is a very vascular organ, the risk of hemorrhage is high. Renal artery embolization of the affected kidney may be done to obstruct the tumor’s blood supply and reduce its vascularity, thereby reducing the risk of hemorrhage. Embolization is usually accomplished by occluding the renal artery using an absorbable gelatin sponge (Gelfoam), metal coil, barium, subcutaneous fat, isobutyl-2-cyanoacrylate, absolute ethanol, or a balloon. This procedure may also be performed to control hemorrhage in an inoperable kidney. In addition, some researchers believe that embolization may stimulate an immune response against the dying cancer cells. Other possible complications include those associated with any major surgery, such as atelectasis,
Nephrectomy reduces pain and hematuria caused by the tumor. The hospital stay is typically 4 to 6 days, with a return to work in 4 to 8 weeks. With laparoscopic nephrectomy (with or without robotic assistance), hospitalization is reduced and return to work after 2 to 4 weeks is common. Living with one kidney has few, if any, negative effects. Long-term outcomes, however, depend on the stage of the cancer.

**Outcomes.** Nephrectomy reduces pain and hematuria caused by the tumor. The hospital stay is typically 4 to 6 days, with a return to work in 4 to 8 weeks. With laparoscopic nephrectomy (with or without robotic assistance), hospitalization is reduced and return to work after 2 to 4 weeks is common. Living with one kidney has few, if any, negative effects. Long-term outcomes, however, depend on the stage of the cancer.

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### Nursing Management of the Surgical Client

**Preoperative Care**

Preoperative preparation of the client having renal surgery includes the general guidelines described in Chapter 14. Increase fluid intake, if indicated, to ensure adequate excretion of waste products before surgery. Give emotional support because the client may be anxious, not only about the surgery but also about postoperative renal function and possible recurrence of the disease. If the remaining kidney functions adequately, assure the client that this kidney can fully meet the body's needs.

**Postoperative Care**

**Assessment.** Postoperatively, monitor the client's vital signs frequently and watch for any manifestations of bleeding or hemorrhage. Bleeding may be through the incision or internal.

Surgically induced or spontaneous pneumothorax occurs occasionally after nephrectomy; monitor for this complication by assessing for sudden shortness of breath and loss of breath sounds on the affected side.

**Diagnosis, Outcomes, Interventions**

**Diagnosis: Risk for Injury: Postoperative Complications.** The nursing diagnoses are likely to include Risk for Injury: Postoperative complications related to surgical procedure. Although postoperative care is similar to that for a laparotomy, one of the greatest challenges is reestablishing effective breathing patterns. Deep breathing and coughing are difficult because the incision is very close to the diaphragm. Also, if the jackknife position is used during the operative procedure, pain and soreness in the thoracic region are increased, limiting respiratory excursion. Paralytic ileus is a common problem. Urine output must be maintained.

**Outcomes.** The client will maintain normal respiratory excursion and have no additional breath sounds and no signs of atelectasis or infection. There will be normal bowel sounds within 2 to 3 days. Urine output will be at least 0.25 ml/kg/hour if one kidney is removed and 0.5 ml/kg/hour if a partial nephrectomy is performed.

**Interventions.** Liberal use of opioids (including patient-controlled analgesia) to reduce pain and external mechanical support of the chest and abdomen with pillows or hands help the client to perform deep-breathing and coughing exercises more effectively. An incentive spirometer provides immediate feedback about the effectiveness of deep breathing.

Other interventions include carefully assessing the client's urine output and gastrointestinal status postoperatively and beginning oral intake only after adequate bowel function has resumed. Total urine output from all urine collection tubes should total 0.25 ml/kg/hour if one kidney is removed or 0.5 ml/kg/hour if a partial nephrectomy is performed. Notify the physician of lesser amounts.

Other wound drainage tubes also need to be monitored. Early ambulation is indicated.

**Diagnosis: Acute Pain.** A nursing diagnosis of Acute Pain related to surgery is common because the nephrectomy incision is extensive and causes significant discomfort. Muscle pain may develop from the prolonged position maintained during surgery.

**Outcomes.** Comfort will be attained and pain will be reduced, as indicated by the client's reports of reduced discomfort or tolerable pain, as well as by nonverbal indications of reduced discomfort, particularly during movement.

**Interventions.** The pain may be reduced by opioid analgesics (including the use of patient-controlled analgesia) and proper positioning. Epidural fentanyl or morphine sulfate can provide effective analgesia.

**Evaluation.** The client should be able to resume regular activities within 6 to 8 weeks after surgery. Long-term survival is dependent on the stage of cancer diagnosed.

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### Self-Care

With shorter hospitalizations, clients who have undergone nephrectomy may require home care and support. Clients are weakened by surgery and possibly by other treatments. Activity should increase gradually; typically, 6 weeks must elapse before clients are ready to return to work or lift more than 10 pounds.

Concern about recurrence of the cancer is common. The American Cancer Society and other support groups may be helpful in the client's adjustment to cancer. People with one kidney can lead normal lives. There is, however, a need to protect the remaining kidney by prevention of infection and trauma.

### TUBULOINTERSTITIAL DISEASE

Traditionally, the term **interstitial nephritis** has been applied to renal disease characterized by the presence...
of inflammatory cells in the spaces between the renal tubules. Not all disease processes included in this classification are inflammatory, however. Therefore the term tubulointerstitial disease is being advocated for this category of renal disorders.

Tubulointerstitial diseases are commonly classified as either acute or chronic. The acute form usually represents an allergic reaction and has a rapid onset. Assessment findings are typically related to tubular injury. Manifestations often include fever, skin rash, eosinophilia, oliguric renal failure, and occasionally gross hematuria. The disease may progress along any of three courses:

- Complete recovery
- Rapid progression to renal failure and death
- Movement to the chronic form

Although corticosteroids are commonly prescribed, their value is unclear. Treatment is similar to that for acute renal failure (see Chapter 36).

Chronic tubulointerstitial disease is characterized by progressive interstitial fibrosis and usually chronic inflammatory cell infiltration with tubular atrophy. In the terminal stages, the altered renal vasculature and renal structure make the disease virtually indistinguishable from chronic pyelonephritis.

Morphologic findings in tubulointerstitial disease include interstitial edema, cellular infiltration of the interstitium, tubular cellular atrophy and flattening, and interstitial fibrosis. As the disease progresses, renal involvement extends beyond the tubules to progressive fibrosis of Bowman’s capsule with secondary involvement of the glomeruli.

Potential causes of this pathologic process include acute pyelonephritis, septicemia, anagiaic abuse (especially with phenacetin, aspirin, and acetaminophen), immunologic mechanisms (for example, renal allograft, SLE, and Sjögren’s syndrome), heavy metal toxicity, drug toxicity, hypercalcemia, and hypocalcemia. In addition, several medication hypersensitivities can contribute. The medications involved include rifampin, penicillin and its analogs, sulfonylamides, cephalosporins, allopurinol, captopril, cimetidine, azathioprine, phenytoin, thiazide, lithium, NSAIDs, and possibly furosemide.

An early manifestation of tubulointerstitial disease is a sudden, unexplained decrease in renal function that may be mild to severe. Specifically, there may be inability to concentrate urine, salt wasting, and poor acidification of the urine leading to metabolic acidosis. Finding a variety of urine sediment abnormalities is also common. Because glucose, uric acid, phosphates, amino acids, and bicarbonate are not effectively reabsorbed in the tubules, they appear in the urine. Severe bicarbonaturia is an indicator of renal tubular acidosis. Proteinuria is less severe than with other renal disease. Systemic hypertension is a common finding.

**GLOMERULONEPHRITIS**

Glomerulonephritis encompasses a variety of diseases, most of which are caused by an immunologic reaction that results in proliferative and inflammatory changes in glomerular structure. Glomerulonephritis can be acute or chronic. It is usually manifested by either a nephrotic syndrome or a nephritic syndrome. Percutaneous renal biopsy is typically used to identify the type of glomerulonephritis, and the findings assist in planning interventions and determining the prognosis.

**NEPHROTIC SYNDROME**

Nephrotic syndrome is a set of clinical manifestations caused by protein wasting secondary to diffuse glomerular damage. Manifestations include proteinuria (>3.5 g/day), hypoalbuminemia, and edema. Abnormal permeability of the glomerular basement membrane (especially to albumin) results in loss of protein in the urine. The resulting hypoalbuminemia alters oncotic pressure in the vascular tree, and fluid moves into the interstitial spaces, causing edema. This movement stimulates plasma renin activity and augments aldosterone production; as a result, the kidney retains sodium and water, thus adding to the accumulation of extracellular fluid.

Hyperlipidemia usually occurs also, probably because of increased hepatic lipoprotein synthesis in response to decreased levels of serum albumin. Depending on the degree of renal failure, some level of normocytic anemia is common.

The causes of nephrotic syndrome are numerous. Besides glomerulonephritis, certain systemic disorders can cause it, such as diabetes mellitus, SLE, amyloidosis, hepatitis B, syphilis, carcinoma, leukemia, infectious disease, and preeclampsia. Other predisposing factors include allergic reactions, reactions to drugs (such as penicillamine, anticonvulsants, probenecid, captopril, gold salts, heroin, and NSAIDs), renal vein thrombosis, sickle cell disease, and heart failure.

Potential complications of nephrotic syndrome include the effects of extracellular fluid accumulation and the progressive development of renal failure. The client may also experience severe hypovolemia, thromboembolism, secondary aldosteronism, abnormal thyroid function, osteomalacia, and increased susceptibility to infections.

Usually, edema is the client’s chief problem. Although its onset may be insidious, it becomes massive. The client’s skin typically assumes a characteristic waxy pallor resulting from the edema rather than anemia. Other manifestations include anorexia, malaise, irritability, and abnormal or absent menses. Large amounts of protein appear in the client’s urine along with granular and epithelial cell casts and fat bodies; proteinuria may account for losses of 4 to 30 g/day. Some hematuria may be present. Serum albumin concentrations may drop as low as 1 to 2.5 g/dl.
The primary aim of treatment for nephrotic syndrome is to heal the leaking glomerular basement membrane, stop the loss of protein in the client’s urine, and break the cycle of edema. Interventions typically include maintaining the client’s fluid and electrolyte balance, reducing inflammation, preventing thrombosis, and minimizing protein loss.

Maintain Fluid and Electrolyte Balance
Unless the client is hyponatremic, fluids are not usually restricted. The client’s fluid balance, however, should be carefully monitored via daily weights, girth measurements, and intake and output determinations. These data are important because weight loss may represent true tissue loss involving protein rather than fluid.

Loop diuretics (i.e., those that work on the loop of Henle), such as furosemide (Lasix), are typically prescribed. Plasma volume expanders, such as albumin, plasma, and dextran, may be administered to raise the oncotic pressure in the vascular tree. The increased pressure pulls fluid from the extracellular spaces, making it available for kidney filtration. Diuresis in older clients must be handled with particular caution because of their reduced ability to tolerate sudden shifts in intravascular volume.

Because the kidneys have a reduced capacity to excrete sodium, mild sodium restriction usually is instituted. The diet should be as palatable as possible, however, because the client must consume adequate protein and calories. Potassium may also be restricted as serum potassium levels rise.

Because edema disrupts cellular nutrition, the client is at increased risk for skin breakdown. Thus skin care is vital. Interventions include good hygiene, massage, position changes, and possibly special mattresses. Use research-based tools to assess the client’s risk of breakdown (see Chapter 18).

Reduce Inflammation
Steroid therapy helps some clients, depending on the cause of disease. Cytotoxic agents such as cyclophosphamide and chlorambucil, indomethacin, anticoagulants, and antiplatelet agents may be used as well.

Prevent Thrombosis
Because clients with nephrotic syndrome are vulnerable to renal vein thrombosis, some are given long-term anticoagulation therapy. Teach such clients how to monitor for hemorrhage, and encourage them to carry identification that lists the drugs they take.

Minimize Protein Loss
For clients with nephrotic syndrome, most physicians recommend a protein intake of 1 to 1.5 g/kg/day with more than 35 kcal/kg/day to prevent further protein breakdown.

Twenty-four-hour urine collections are used to measure urinary protein losses and monitor the success of treatment. Treatment to reduce inflammation ultimately reduces protein loss.

An important nursing role is to help the client with nephrotic syndrome maintain health and cope with the illness. Teach the client to take prescribed medications regularly, follow the prescribed diet, and report changes in health status, such as increasing edema, reduced urine output, weight gain, respiratory distress, and signs of infection. Explain that the amount of exercise allowed is based, at least in part, on the severity of the edema. Bed rest is imposed only during severe edema. As the fluid level moves toward normal, the client is allowed more activity. Other important areas of teaching include nutrition, prevention of infection, and methods of careful self-assessment.

NEPHRITIC SYNDROME
Nephritic syndrome refers to a set of clinical manifestations that includes hematuria and at least one of the following: oliguria (urine output <400 ml/24 hour), hypertension, elevated blood urea nitrogen (BUN) level, or decreased GFR. Nephritic syndrome is common with many types of glomerulonephritis, including immunoglobulin A (IgA) nephropathy and Henoch-Schönlein purpura. Treatment includes management of the underlying disease (usually through immunosuppressive drugs, as noted later) and symptomatic treatment of blood pressure and uremia.

TYPES OF GLOMERULONEPHRITIS
There are many types of glomerulonephritis, most of which involve either nephrotic syndrome or nephritic syndrome. The diagnosis of the specific type can be made by assessment of clinical manifestations and through renal biopsy. Box 35-2 presents a classification system based on etiology. Table 35-1 describes the onset, diagnostic findings, and prognosis for various types of glomerulonephritis.

Pathophysiology
Glomerulonephritis is an immunologic disorder that causes inflammation and increased cells in the glomerulus. Because the primary function of the glomerulus is to filter blood, most cases result when antigen-antibody complexes produced by an infection elsewhere in the body become trapped in the glomerulus. This entrapment causes inflammatory damage and impedes glomerular function, reducing the glomerular membrane’s capacity for selective permeability. The source of the antigens
BOX 35-2 Classification of Glomerulonephritis Based on Etiology

**PRIMARY GLOMERULONEPHRITIS—IMMUNE RESPONSE TO PATHOGENS**
- Acute glomerulonephritis
- Postinfectious glomerulonephritis
- Group A beta-hemolytic streptococcus
- Other infectious conditions such as cytomegalovirus infection, measles, mumps, staphylococcus, or pneumococcal bacteremia
- Infectious glomerulonephritis
- Membranoproliferative glomerulonephritis
- Rapidly progressive glomerulonephritis
- Idiopathic membranous glomerulonephritis
- Immunoglobulin A (IgA) nephropathy
- Chronic glomerulonephritis

**SECONDARY GLOMERULONEPHRITIS—RELATED TO SYSTEMIC DISEASE**
- Goodpasture’s syndrome
- Hemolytic-uremic syndrome
- Henoch-Schönlein purpura
- Polyarteritis
- Progressive systemic sclerosis
- Systemic lupus erythematosus
- Wegener’s granulomatosis
- Thrombocytopenic purpura
- Postpartum renal failure

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**TABLE 35-1 Types of Glomerulonephritis Onset, Findings, and Prognosis**

<table>
<thead>
<tr>
<th>Type</th>
<th>Onset</th>
<th>Diagnostic Findings</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poststreptococcal glomerulonephritis</td>
<td>1-3 weeks after beta-hemolytic streptococcal infection of throat or skin</td>
<td>Underlying infection</td>
<td>Variable: Complete recovery to end-stage renal disease</td>
</tr>
<tr>
<td></td>
<td>Nephritic syndrome</td>
<td>Elevated antistreptolysin O titer</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Proteinuria</td>
<td>Microscopic urinary, urine with many casts</td>
<td></td>
</tr>
<tr>
<td>Membranoproliferative glomerulonephritis</td>
<td>Nephrotic syndrome sometimes preceded by a streptococcal infection</td>
<td>Hematuria (microscopic or gross)</td>
<td>Gradual progressive chronic renal failure</td>
</tr>
<tr>
<td>Rapidly progressive glomerulonephritis</td>
<td>Nephritic syndrome</td>
<td>Hematuria</td>
<td>Progresses to renal failure within weeks or months</td>
</tr>
<tr>
<td></td>
<td>Sudden</td>
<td>Edema</td>
<td></td>
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<tr>
<td></td>
<td>May follow antigen or infection</td>
<td>Hypertension</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Peak ages 40-60 yr</td>
<td>Proteinuria</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Oliguria</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Acidosis</td>
<td></td>
</tr>
<tr>
<td>Idiopathic membranous glomerulonephritis</td>
<td>Insidious</td>
<td>Asymptomatic proteinuria or nephrotic syndrome</td>
<td>Mixed: 25% have spontaneous remission, 25% have renal failure, 25% have persistent proteinuria, 25% have deteriorating renal function</td>
</tr>
<tr>
<td></td>
<td>Peak ages 40-70 yr</td>
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<td></td>
<td>Unknown antigen</td>
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</tr>
<tr>
<td>Immunoglobulin A (IgA) nephropathy (also called Berger’s disease)</td>
<td>Most common in young adults Nephritic syndrome</td>
<td>Hematuria</td>
<td>Usually progresses slowly over 10-20 yr; a small proportion progress to renal failure</td>
</tr>
<tr>
<td>Lipoid nephrosis</td>
<td>Nephrotic syndrome</td>
<td>Red blood cell casts on microscopic urinalysis</td>
<td>Generally good</td>
</tr>
<tr>
<td>Lipoid nephrosis</td>
<td>Nephrotic syndrome</td>
<td>Found on biopsy</td>
<td>May be relapses and spontaneous remission</td>
</tr>
<tr>
<td>Lipoid nephrosis</td>
<td>Insidious onset</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lipoid nephrosis</td>
<td>Found on biopsy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lipoid nephrosis</td>
<td>May be few symptoms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Focal glomerular sclerosis</td>
<td>Peaks between ages 30 and 50 yr Nephrotic syndrome</td>
<td>Found on biopsy</td>
<td>Poor, although rate of deterioration varies widely</td>
</tr>
<tr>
<td>Membranous glomerulonephritis</td>
<td>Commonly secondary to drug therapy toxins or systemic autoimmune disease Nephrotic syndrome Insidious onset</td>
<td>May be few symptoms</td>
<td>Recurs after transplantation</td>
</tr>
<tr>
<td>Membranous glomerulonephritis</td>
<td>Commonly secondary to drug therapy toxins or systemic autoimmune disease Nephrotic syndrome Insidious onset</td>
<td>Heavy proteinuria</td>
<td>Variable</td>
</tr>
<tr>
<td>Membranous glomerulonephritis</td>
<td>Commonly secondary to drug therapy toxins or systemic autoimmune disease Nephrotic syndrome Insidious onset</td>
<td></td>
<td>30% have spontaneous remission</td>
</tr>
<tr>
<td>Hemolytic-uremic syndrome</td>
<td>Follows infection with <em>Escherichia coli</em> (0157:H7 serotype) History of eating undercooked hamburger Children and older adults particularly vulnerable</td>
<td>Hemorrhagic manifestations, such as bleeding and bruising Purpura manifestations, such as acute renal failure, hemolytic anemia, and thrombocytopenia</td>
<td>Recovery rate 95% but may leave residual renal damage</td>
</tr>
</tbody>
</table>
May be either **exogenous** (e.g., after streptococcal infection) or **endogenous** (as in SLE). Evidence also indicates that some antigen-antibody complexes may form in the kidney itself.

Glomerulonephritis may also result from antibodies affixed to the glomerular basement membrane. For example, Goodpasture’s syndrome involves pulmonary hemorrhage and glomerulonephritis.

The primary pathologic processes in glomerulonephritis, lipoid nephrosis, and focal glomerular sclerosis are proliferation and inflammation; however, lipoid nephrosis and focal glomerular sclerosis are characterized by degeneration. Figure 35-2 depicts the pathophysiologic mechanisms of glomerulonephritis.

**Clinical Manifestations**

Acute glomerulonephritis may develop insidiously or suddenly, varying considerably with the pathophysiology involved. Classic manifestations of sudden onset include hematuria with red blood cell casts and proteinuria. Fever, chills, weakness, pallor, anorexia, nausea, and vomiting may be present. Generalized edema, particularly facial and peri-orbital swelling, is a typical finding. The client may have ascites, pleural effusion, and heart failure.

The client is likely to have a headache and moderate to severe hypertension. Visual acuity may be reduced because of retinal edema. Abdominal or flank pain may develop, probably because of kidney edema and distention of the renal capsule. Oliguria, even anuria, may be present for several days; the longer it persists, the more irreversible the kidney damage. In contrast, the disease may be so mild that the client reports only vague weakness, anorexia, and lethargy.

Acute glomerulonephritis can become a fulminant process, proceeding quickly to uremia or chronic glomerulonephritis. Most clients, however, start to recover within 14 days. Most clinical manifestations disappear within several weeks, although hematuria and proteinuria may be present for longer periods. If complete recovery does not occur within 2 years, it probably will not occur at all.

Some clinicians use the term **subacute glomerulonephritis** to describe disease persisting longer than 6 to 8 weeks. Although most of the manifestations of the acute disease have disappeared, the client is still at high risk for exacerbation of glomerulonephritis. The term **latent glomerulonephritis** refers to an asymptomatic condition characterized by significant albumin levels and casts in the urine for more than 1 year after acute onset. These findings indicate continued but slow parenchymal changes.

Most types of glomerulonephritis can progress to a chronic state. Sometimes, glomerulonephritis is first seen as a chronic process. Chronic glomerulonephritis progresses over an extended period, often as long as 30 years. When it progresses to end-stage renal failure, dialysis must begin or the client will die.

As the glomeruli and tubules are destroyed by the pathologic process, the kidneys shrink and become severely contracted. Fibrous and scar tissue replaces functioning renal tissue. Sclerosis of renal blood vessels also occurs. The destruction rates vary.

Common manifestations include malaise, weight loss, edema, increasing irritability and mental cloudiness, a metallic taste in the mouth, polyuria and nocturia resulting from the kidney’s inability to concentrate urine, headache, dizziness, and digestive disturbances. As the disease progresses, these manifestations intensify, and the client may experience respiratory difficulty and angina.

The cardinal manifestation of this disease is hypertension. It is not uncommon for the client to experience such complications as nosebleed, manifestations of arteriosclerosis, cardiomegaly, and hemorrhage into the kidneys, lungs, retina, or cerebrum. Edema increases as heart failure becomes more severe and the serum albumin level decreases. Examination of the eyegrounds shows vascular changes and edema of the discs. Urinalysis shows a fixed specific gravity, small amounts of proteinuria except during exacerbation, casts, WBCs, renal tubular cells, and consistent hematuria. Anemia tends to be severe.

![FIGURE 35-2 Pathophysiologic mechanisms of glomerulonephritis.](image-url)
Examining the urine usually provides the information necessary for a diagnosis of glomerulonephritis (see Chapter 32). Gross hematuria and proteinuria are the cardinal findings. The client’s urine, which may be scant, usually has a dark, smoky, cola-colored, or red-brown hue. The proteinuria produces a persistent and excessive foam. The urine may have a low pH and a specific gravity in the midnormal to high-normal range if there is enough renal damage to affect the kidneys’ ability to concentrate.

Other studies may assist in the diagnosis as well. Serum urea nitrogen and creatinine levels may be elevated, and creatinine clearance rates may be decreased. C-reactive proteins and antistreptolysin O titer are usually elevated in poststreptococcal glomerulonephritis, and the serum complement level is low in many forms of glomerulonephritis. Hematocrit and hemoglobin studies may indicate anemia, particularly if renal failure is imminent.

**OUTCOME MANAGEMENT**

**Medical Management**

Medical intervention aims to eliminate antigens, to alter the client’s immune balance, and to inhibit or alleviate inflammation to prevent further renal damage and improve kidney function. Although some clients may require initial hospitalization, treatment is typically on an outpatient basis.

**Reduce Inflammation**

Plasmapheresis has been used in some research protocols to reduce the number of antigens in certain types of glomerulonephritis, including rapidly progressive glomerulonephritis. This intervention is usually administered in conjunction with corticosteroids and immunosuppressive agents (azathioprine, cyclophosphamide). The technique is designed to remove the specific circulating antibody or mediators of the inflammatory response. Large volumes of the client’s plasma are cyclically removed and replaced with fresh frozen plasma through a continuous-flow blood cell separator.

Antibiotic therapy (such as penicillin for streptococcal organisms) is used to treat poststreptococcal glomerulonephritis. It is also used prophylactically after streptococcal infections to prevent further damage.

**Maintain Fluid and Electrolyte Balance**

Volume overload and hypertension are treated with diuretics, antihypertensives, and restriction of dietary sodium and water. Common complications of fluid overload include heart failure with pulmonary edema and increased intracranial pressure. Renal failure may develop (see Chapter 36). Appropriate monitoring is essential and should include vital signs, intake and output, and weight. Recognizing complications early facilitates prompt medical intervention.

**Nursing Management of the Medical Client**

**Assessment.** For any client with suspected glomerulonephritis, take a comprehensive history that includes upper respiratory tract infection (such as strep throat), skin infections, scarlet fever, or a history of glomerulonephritis. Also question the client about systemic disorders that might be present, such as SLE, scleroderma, amyloidosis, and hypertension. Any recent invasive procedures should also be noted.

Physical examination may reveal ascites, pleural effusion, and manifestations of heart failure with pulmonary edema. Examine the urine closely for color, amount, and abnormal substances. In particular, microscopic analysis of the urine can be a valuable diagnostic tool with glomerulonephritis; urinary casts are commonly seen under high magnification (see Chapter 32). Check the client’s vital signs closely, especially the blood pressure.

**Diagnosis, Outcomes, Interventions**

**Diagnosis: Imbalanced Nutrition: Less Than Body Requirements.** If the client has a reduced appetite or an aversion to food, the nursing diagnosis Imbalanced Nutrition: Less Than Body Requirements related to anorexia and increased metabolic demands is appropriate.

**Outcomes.** The client will maintain adequate nutritional intake, as evidenced by no weight loss, an absence of a negative nitrogen balance, and normal electrolytes.

**Interventions.** It is important to protect the kidneys while they are recovering their function. The prescribed diet is likely to be high in calories and low in protein. This diet is designed to avoid protein catabolism and enables the kidney to rest because it handles fewer protein molecules and metabolites.

The degree to which protein is restricted depends on the amount excreted in the urine and the client’s individual requirements. Sodium is also restricted, depending on the amount of edema present. Anorexia, nausea, and vomiting may interfere with adequate intake, requiring creative intervention on your part. A dietitian can help plan the client’s diet around these restrictions.

**Diagnosis: Excess Fluid Volume.** A common nursing diagnosis in glomerulonephritis is Excess Fluid Volume related to reduced urine output.

**Outcomes.** The client will maintain balanced intake and output, as evidenced by no manifestations of edema or fluid overload.

**Interventions.** Appropriate fluid balance is important. Careful monitoring of daily weight and intake and
output helps determine the progress of the edema and thus provides an estimate of renal function. Daily measurement of edematous parts (especially legs and abdomen) also provides useful, objective data. The client’s allowable fluid intake is based on the intake and output measurements. Fluid intake is usually restricted. Thirst may be relieved by offering hard candies, lemon slices, or ice chips rather than a glass of water. Assist the client to “plan” fluid distribution during the day to make the best use of allowed fluids.

**Diagnosis: Fatigue.** Another common nursing diagnosis is Fatigue related to increased metabolic demands and anemia.

**Outcomes.** The client will conserve energy through an adequate balance of rest and activity, as evidenced by absence of complaints of fatigue.

**Interventions.** Rest is essential—both physical and emotional. As mentioned, activity level correlates directly with the amount of hematuria and proteinuria. Exercise also increases catabolic activity. The allowable amount of activity depends on the results of serial urinalyses. Bed rest interspersed with periods of limited activity may continue for several weeks to months. Therefore the client may need help in arranging personal matters, such as family, home, job, finances, and community responsibilities.

Encourage the client to talk about any fears or concerns, and, if necessary, help the client deal with the emotional reactions expected during a long-term illness with a questionable prognosis. Only after handling these problems can the client rest emotionally. Appropriate diversionary activities may help the client cope with prolonged physical immobility.

**Diagnosis: Risk for Impaired Skin Integrity.** A typical nursing diagnosis is Risk for Impaired Skin Integrity related to edema.

**Outcomes.** The client will maintain tissue integrity; skin will remain intact.

**Interventions.** Edema interferes with cellular nutrition, which makes the client more susceptible to skin breakdown. Take precautions to prevent this complication. Interventions include good hygiene, massage, and position changes as well as other prophylactic measures, such as mattress devices. Use research-based tools to assess the client’s risk of breakdown (see Chapter 18).

**Diagnosis: Risk for Infection.** Another diagnosis after immunosuppressive therapy is Risk for Infection related to altered immune response secondary to treatment.

**Outcomes.** The client will maintain a healthy immune status, free of infection, as evidenced by normal temperature and an absence of local or systemic manifestations of infection.

**Interventions.** Glomerulonephritis markedly diminishes a client’s natural defenses to infection, especially to streptococcal organisms. Immunosuppressive medications and corticosteroids further reduce host resistance. Although isolation is not necessary, take care to protect the client from people with obvious infectious processes. General supportive measures help boost the client’s defense mechanisms. Client teaching should involve appropriate ways to avoid infections, especially respiratory and urinary tract infections.

**Evaluation.** The client must be able to understand the condition and the reasons for limitations, including dietary and fluid restrictions. Stress the importance of follow-up treatments to minimize the risk of recurrence.

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**Self-Care**

Clients with glomerulonephritis are followed as outpatients, often for many years. Self-care activities should include attending regular follow-up appointments, adhering to recommended medications and dietary restrictions, and monitoring for changes in condition. These are discussed further later in this chapter under Chronic Kidney Disease. It is vital that blood pressure be controlled to prevent further renal damage; many clients learn to monitor their own blood pressure at home. Because the disease may progress to renal failure, there are numerous quality-of-life issues, as discussed in Chapter 36.

**Modifications for Older Clients**

The older client is at greater risk for renal damage because of the pre-existing effects of age on the kidneys. The older client is also more likely to have concurrent chronic diseases—such as hypertension and diabetes mellitus—that may have affected the kidneys, although treatment is the same.

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**CHRONIC KIDNEY DISEASE**

Chronic kidney disease (CKD) is a rapidly growing health problem. Estimates are that 11% of the U.S. population or 19.2 million people have CKD. The Centers for Disease Control and Prevention in Atlanta has recently established a chronic kidney disease program to enhance surveillance and prevention programs for CKD at the federal and state levels.

The many diseases described in this chapter can lead to CKD, and the etiology will differ by disease. Recognition of the type of kidney disease and etiology may be useful to prevent or slow progression of the disease.

**Etiology and Risk Factors**

The increasing incidence of CKD partially reflects increased obesity-related hypertension and diabetes
Since cardiovascular disease and diabetes mellitus are frequently co-morbid conditions associated with CKD, aggressive treatment of the disease and risk factors can slow progression of the illness and limit morbidity and mortality (See the Translating Evidence into Practice feature Managing Hypertension in Diabetic Clients to Slow Progression of Renal Disease on p. 780).

The National Kidney Foundation (NKF) Kidney Disease Outcome Quality Initiative (K/DOQI) defined CKD as kidney damage with a glomerular filtration rate (GFR) <60 ml/minute/1.73 m² for more than 3 months. The NKF developed a classification system for the stages of CKD (Table 35-2). Traditionally, the classification of the type of kidney disease has been focused on pathology and etiology. The K/DOQI classification system focuses on the GFR, but it remains important to diagnose the cause of CKD.

Pathophysiology
There are many diseases that cause CKD; each has its own pathophysiology. However, there are common mechanisms for disease progression. Pathologic features include fibrosis, loss of renal cells, and infiltration of renal tissue by monocytes and macrophages. Proteinuria, hypoxia, and excessive angiotensin II production all contribute to the pathophysiology. In an attempt to maintain GFR, the glomerulus hyperfiltrates; this results in endothelial injury. Proteinuria results from increased glomerular permeability and increased capillary pressure. Hypoxia also contributes to disease progression. Angiotensin II increases glomerular hypertension, which further damages the kidney.

Clinical Manifestations
The clinical manifestations of CKD are highly variable. Many people with CKD have few if any complaints. In stage 1, clients usually have normal blood pressure, no laboratory test abnormalities, and no clinical manifestations. Clients in stage 2 are generally asymptomatic, but may develop hypertension, and laboratory test abnormalities exist. In stage 3, clients are still usually asymptomatic but laboratory values suggest abnormalities in several organ systems, and hypertension is frequently present. By stage 4, clients begin to experience clinical manifestations associated with CKD such as fatigue and poor appetite. At stage 5, full-blown clinical manifestations of end-stage renal disease (ESRD) are evident (see Chapter 36).

Proteinuria is one of the strongest predictors of progression of CKD. As the GFR declines, clients may show not only proteinuria but also hypertension, a wide range of lab abnormalities, and manifestations resulting from disorders in other organs. These disorders include anemia, metabolic acidosis, dyslipidemia, bone disease, protein-energy malnutrition, and neuropathy; alterations in health status are described in more detail in Chapter 36 in relation to ESRD.

OUTCOME MANAGEMENT
The focus of management of CKD is on slowing the progression of the disease and in preventing risk factors that lead to complications.

Medical Management
Ideal outcomes of medical management include the following:

- Controlling blood pressure (BP) to below 130/80 mm Hg
- Managing blood glucose level to maintain HbA₁c below 7%
- Managing hyperlipidemia with diet and cholesterol-lowering drugs (usually statins)
- Managing and treating emerging manifestations of renal failure including anemia, hyperphosphatemia and hyperparathyroidism, hyperkalemia, and metabolic acidosis
- Preparing clients for renal replacement therapy when necessary (see Chapter 36)

Reduce Blood Pressure
Hypertension in CKD increases the risk of loss of kidney function. The lower the BP, the lower the risk of cardiovascular disease. At blood pressures above 115/75 mm Hg, the risk of cardiovascular mortality doubles for each increase of 20 mm Hg systolic and 10 mm Hg diastolic BP. Clinical practice guidelines suggest
that systolic BP should be maintained below 130 mm Hg and diastolic BP below 80 mm Hg in people with less than 1 g of proteinuria/day and <125/75 mm Hg in people with >1 g of proteinuria/day.58

There are currently more than 125 antihypertensive agents available for the treatment of hypertension. Although many of them are very effective, control of BP is challenging. Further discussion of treatment for hypertension is included in Chapter 52. Numerous large, randomized control trials have shown that angiotensin-converting enzyme inhibitors (ACEIs) are superior to non-ACEI agents in reducing progression of renal disease, reducing ischemic heart disease and congestive heart failure event rates, and reducing mortality. ACEI and angiotensin receptor blockers (ARBs) either alone or in combination are the preferred agents for both diabetic and nondiabetic kidney disease.58 Current thinking is that combination therapy is more effective than stepped therapy.40 Because these agents carry the risk of hyperkalemia, potassium levels require close monitoring.

Reduce Serum Lipids
Cardiovascular mortality is elevated among people with CKD. For this reason, low-fat diets and administration of cholesterol-lowering medications, particularly statins, are indicated. The NKF recommends maintaining LDL <100 mg/dl, non-HDL <130 mg/dl, and triglycerides <500 mg/dl.57 Hyperlipidemia should be managed aggressively to reduce the risk of atherosclerotic cardiovascular disease.

Control Blood Glucose Level
Diabetic nephropathy can be ameliorated with aggressive control of blood glucose levels and management of hypertension. The goal is to maintain preprandial blood glucose values of 80 to 120 mg/dl in the morning and 100 to 140 mg/dl at bedtime and HbA1c levels less than 7%.

Control Phosphorus Intake
Elevations in the levels of serum phosphorus, calcium-phosphorus product, and parathyroid hormone substantially increase the risk of death. The NKF recommends that serum phosphorus level be maintained between 2.7 and 4.6 mg/dl for those with stage 3 or 4 CKD, dietary phosphorus intake should be limited, and phosphorus binders started if necessary.36

Nursing Management of the Medical Client
Assessment. For any client with CKD, take a comprehensive history that includes medications and diet currently prescribed. Check blood pressure regularly. Monitor urine studies including microalbumin and albumin levels as well as blood creatinine level, GFR, red blood cell count, and levels of electrolytes, glucose, and lipids for changes suggesting increasing renal failure. Physical assessment findings that suggest progressing renal failure may include fatigue, peripheral edema, shortness of breath, adventitious lung sounds, heart murmurs or gallops, bruising, memory loss, GI disturbances, impaired wound healing, and increased infections.

Diagnosis, Outcomes, Interventions
Diagnosis: Effective Therapeutic Regimen Management and Readiness for Enhanced Self-Care. The nursing goals for the client with CKD are to minimize the risk of progression of the disease and prevent or manage risk factors associated with morbidity and mortality. For this reason, two nursing diagnoses are Effective Therapeutic Regimen Management and Readiness for Enhanced Self-Care.

Outcomes. The client will control risk factors through self-management of medications, diet, and exercise, as evidenced by normal blood pressure and normal or stabilized laboratory values including blood glucose and creatinine levels and reduced or stabilized proteinuria.

Interventions. Nursing interventions include health education and mutual goal setting to address risk factors such as high blood pressure, poor glycemic control, proteinuria, and smoking. Blood pressure management can include dietary therapy (reduced sodium), exercise, and antihypertensive medications. Many clients are taught to monitor their blood pressure at home.

Control Blood Glucose Level. Glycemic control is addressed further in Chapter 45. Dietary management, exercise, antidiabetic agents, and insulin therapy are essential aspects of managing blood glucose levels.

Encourage Smoking Cessation. Smoking cessation assistance is another important area for prevention of risk factors. The mechanisms of the effect of smoking on kidney disease progression are unclear, but studies have shown an association between smoking and decreasing renal function. Smoking increases the risk of developing type 2 diabetes mellitus and microalbuminuria, and furthering progression of diabetic nephropathy. Smoking was related to the development of kidney disease in a longitudinal study of 2585 people with no previous history of kidney disease with a mean follow-up of 18.5 years. There are many nursing strategies to facilitate smoking cessation, but this is a difficult addiction to overcome and repeated and varied interventions may be necessary.

Diagnosis: Imbalanced Nutrition: More Than Body Requirements. Excess weight can contribute to the development of CKD. As a result, the nursing diagnosis is often
Imbalanced Nutrition: More Than Body Requirements related to sedentary lifestyle and calorie intake greater than energy used.

Outcomes. The client will demonstrate weight control as exhibited by a near-normal body weight and a body mass index (BMI) of <35 kg/m².

Interventions. Health education about both exercise and diet is a vital part of weight reduction programs. Exercise programs can vary considerably based on the client's interests, current level of activity, and present health status. Asking clients to keep a food diary can be very useful in assessing specific dietary problems. Teaching about healthy food choices is an important nursing role. Referral to a dietitian can be very helpful, particularly in complex cases. Dietitians can provide individualized instruction and sample diet plans and instruct the client further about many possible diet modifications necessary at various stages of CKD. These modifications might include reduced sodium, reduced phosphorus, low fat, low potassium, and reduced protein intake as well as reduced calories, depending on the stage of kidney disease and the laboratory value abnormalities.

Evaluation. Evaluation of interventions will involve assessing GFR to determine whether the disease has been stabilized and the progression of the disease slowed. Blood pressure, proteinuria, and body weight will be within normal limits.

Self-Care

Self-management of CKD is key to control of CKD and its complications. Clients typically feel well, so it is important to help them understand the treatment goals and risks for complications. Self-management often involves home monitoring of blood pressure, dietary modifications, medications, exercise, weight reduction, and smoking cessation.

RENAL TRAUMA

Serious kidney injury is relatively rare because of the protection afforded by the rib cage, the heavy muscles of the back, and the tough capsule surrounding the kidney. Traffic accidents and falls in which the client lands on the abdomen, flank, or back are the most common cause of injury, usually from blunt trauma. Kidney lacerations can result from fractures of the spine and ribs as well as penetrating injuries from bullets and knives.

Pathophysiology

Five categories of traumatic injury can affect the kidney:
- Contusion with intrarenal hemorrhage
- Minor laceration (rupture with subcapsular hemorrhage)
- Major laceration (rupture into the renal pelvis)
- “Fractured” kidney (shattered rupture)
- Vascular (pedicle) injury, which damages renal blood supply (Figure 35-3)

In a contusion, a hematoma develops and remains confined within the renal parenchyma. Rupture of the kidney may cause hemorrhage between the capsular walls; bleeding may or may not reach into the renal pelvis. A shattered or fractured kidney causes hemorrhage throughout the renal tissue. The pedicle holds the renal artery and other vital circulatory and nervous system connections to the kidney. Injury to the pedicle may jeopardize the life of the kidney and may occur with or without intrarenal hemorrhage.

Clinical Manifestations

The type of injury the client has suffered gives the first real key to identifying renal trauma. Commonly, the client has multiple serious injuries, and renal trauma may not be immediately apparent. Hematuria (gross or microscopic) is a cardinal manifestation and is found in about 80% of cases. However, serious renal injury can
occur without hemorrhage, and clear urine does not automatically rule out renal trauma. Other findings include shock, flank pain, and a palpable mass in the affected flank area or over the 11th or 12th rib. Paralytic ileus may also occur. You may see bruises over the client’s flank and lower back secondary to retroperitoneal hemorrhage, a development known as Grey Turner’s sign. A KUB film, IVP, retrograde pyelography, renal scan, ultrasonography, CT scan, and renal arteriography all help confirm the type and degree of kidney injury.

**Complications**

In addition to the immediate problems of hemorrhage and loss of functioning renal tissue, kidney trauma makes the client highly susceptible to a number of other problems. Even in closed injuries, there is a high risk of sepsis leading to kidney and perinephric abscesses. Secondary hemorrhage is not uncommon. Other complications include hypertension resulting from fibrosis and ischemic kidney, renal artery thrombosis, arteriovenous aneurysms, fistula formation from extravasation of urine, urinomas, and pseudocysts.

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**Medical Management**

Medical management, which primarily involves waiting and watching, is possible because the retroperitoneal space allows tamponade. In the absence of other injuries, a client with microscopic hematuria and normal findings on IVP may be observed on an ambulatory basis with careful instructions about activity restrictions and the need for adequate hydration. If there is gross hematuria, bed rest is required until the urine clears. Serial observations of the urine, hematocrit level, and vital signs are made to watch the progress of the hemorrhage. Sequential urine specimens may be collected to compare current and previous urine color and turbidity.

Even if replacement fluids are not needed, a prophylactic intravenous line may be established, and a type and crossmatch for blood may be done. If a hematoma is present or IVP shows urine extravasation, the client may receive antibiotics to prevent sepsis. The physician prescribes blood transfusions if the hematocrit is low. After the urine clears, the client can be more active. After discharge, the client needs follow-up blood pressure checks and IVP studies to rule out secondary hypertension and anatomic changes in the renal system.

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**Surgical Management**

The greatest diversity of opinion concerns proper handling of the renal damage discovered during exploration. When the other kidney is functioning effectively, the greatest diversity of opinion concerns proper handling of the renal damage discovered during exploration. When the other kidney is functioning effectively, a client with microscopic hematuria and normal IVP results who is observed on an ambulatory basis should be given careful instructions about activity restrictions and the need for adequate hydration.
avoid later sequelae, whereas others believe that the goal should be salvaging maximal renal function. The latter group advocates giving the conservative approach a fair trial and, if surgery is necessary, attempting to repair the kidney before deciding to remove it. With renal vascular injury, fewer than half of kidneys can be salvaged if the injury is 18 hours old; there is virtually no chance of renal recovery after 24 hours.

Renal hemorrhage may be controlled by injection of an autologous clot into the secondary arteries supplying the bleeding site. Blood is drawn from the client and allowed to clot. The clot is then injected angiographically. Because normal endothelium has a strong clot-lysing effect, the clot disappears from the normal adjacent vasculature after several hours and affects only the damaged portion.

If kidney repair is attempted rather than nephrectomy, the surgical procedure is designed to debride devitalized tissue, achieve hemostasis, establish a watertight seal of the collecting system, approximate the renal parenchymal edges, and drain the renal fossa.

Two surgical techniques improve the outcome of repair:

- Extracorporeal, or bench, surgery allows the kidney to be removed from the body for better visualization and manipulation of the organ during the repair process; the kidney is returned by autotransplantation. During its time outside the body, the kidney is maintained by hypothermia or by a perfusate mechanically pulsed through it.
- The slush technique, in which the kidney is immersed in iced saline slush, slows the metabolism and oxygen requirement of renal tissue, allowing longer intraoperative ischemic times. This technique causes some systemic hypothermia, but it is not significant. Pedicle vascular injury may also be repaired.

If either technique fails, nephrectomy is necessary.

**Nursing Management of the Surgical Client**

Nursing management of the client undergoing surgery for renal trauma is similar to that for other surgical procedures on the kidney (see Nephrectomy earlier in this chapter).

**Self-Care**

The client being observed on an ambulatory basis requires an appropriate teaching plan covering health maintenance activities and the need for a follow-up program. The client should promptly report any change in condition or recurrence of bleeding or pain to the physician. Strenuous activity should be avoided for several weeks or more, depending on the extent of the injury. The client must also maintain adequate hydration.

**RENAL VASCULAR ABNORMALITIES**

The kidneys depend on adequate blood circulation to nourish tissues and provide blood for filtration so that they can perform their intended functions. Anything that interferes with the normal circulatory flow significantly reduces renal function.

**RENAL ARTERY DISEASE**

**Etiology and Risk Factors**

Of all cases of renal artery disease, 90% are caused by atherosclerosis or fibromuscular dysplasia. Atherosclerosis affects males more often than females and usually involves the proximal third of the artery. Health promotion activities are the same as those for atherosclerosis as discussed with circulatory and cardiac disorders. Fibromuscular dysplasia is an alternating stenosis and dilation; arteriographic studies demonstrate a “string-of-beads” appearance of the artery. This condition affects females four to five times as often as males. Because the cause is unknown, there are no health promotion actions.

There are several other, less common, causes of renal artery disease. Cancer may obstruct the vessels. Embolism or thrombosis can cause acute obstruction. Trauma, as described earlier, can interrupt blood flow. The renal artery may be purposely occluded to produce a “medical nephrectomy” or total renal infarction; the occlusion may be done preoperatively in the case of renal adenocarcinoma or to control proteinuria or hypertension. Shredded Gelfoam can be used, or a liquid substance that polymerizes instantly when it comes in contact with blood may be injected into the renal artery. A dissecting aneurysm in the renal artery may also interrupt renal circulation.

**Pathophysiology**

The end result of any of these conditions, if severe enough, is reduced renal blood flow. The reduced flow causes renal parenchymal ischemia and, finally, renal atrophy. The role of renal artery disease in renovascular hypertension is also well documented, and hypertension alone may indicate treatment of the condition.

**Clinical Manifestations**

Because of the kidney’s compensatory mechanisms, the gradual development of renal artery stenosis from atherosclerosis and cancer may lead to few manifestations, at least until the resulting hypertension and decreasing...
renal function become evident. Acute obstruction makes itself known relatively quickly, however. Manifestations of this sudden episode include flank pain over the affected kidney or abdominal pain and fever. Atrial dysrhythmias are a frequent finding; however, because they commonly alternate with periods of normal sinus rhythm, this manifestation can be missed. Urinalysis may be normal, and blood chemistry profiles may show elevated levels of aspartate aminotransferase and lactate dehydrogenase. IVP shows a nonfunctioning kidney, and a renal scan shows no arterial blood flow.

In response to reduced renal circulation, collateral circulation helps preserve the kidney if sufficient development takes place before total obstruction. Collateral circulation, in addition to a marked reduction in filtration, renal work, and oxygen requirements, allows the kidney to tolerate ischemic periods for up to several weeks. In acute total occlusion, a normal kidney can remain viable for about 2 hours before infarction and tissue necrosis begin.

**OUTCOME MANAGEMENT**

**Surgical Management**

Treatment of the ischemic kidney usually involves surgical revascularization. Arterial endarterectomy may be done with follow-up anticoagulant or antiplatelet therapy. In the technique known as percutaneous transluminal renal angioplasty (PTRA), the vessel is cleared with a balloon catheter. If it cannot be recanalized, a renal artery resection with end-to-end anastomosis or an aortorenal bypass graft procedure may be performed.

**Percutaneous Transluminal Renal Angioplasty**

In clients undergoing PTRA, a balloon-tipped catheter is inserted, usually through the femoral artery, and threaded under radiologic guidance to the obstructed renal artery. The physician inflates the balloon and pulls it through the obstructed area, stretching it and increasing the size of the arterial lumen. A stent may be placed at the site of occlusion to maintain the size of the lumen, prevent restenosis, and minimize the risk of abrupt vessel reclosure. If a stent is placed, antiplatelet agents or anticoagulants may be prescribed to minimize the risk of acute thrombosis.

**Indications.** Renal artery angioplasty is usually the first intervention for renovascular hypertension. If angioplasty is unsuccessful or if the condition recurs, more invasive surgical approaches are considered.

**Contraindications.** Angioplasty may be contraindicated if there is previous damage to the femoral artery or severely impaired circulation to the limb.

**Complications.** Renal artery angioplasty is considered a relatively safe procedure. The overall complication rate is about 10%. The most common complications include renal artery dissection, renal artery thrombosis or occlusion, segmental renal infarction, and hematoma formation or puncture trauma.

**Outcomes.** About one third of clients with renovascular hypertension do not respond to this therapy. A significant number are cured, and about half improve significantly. The treatment can be repeated if necessary. Clients usually return home in 24 to 48 hours.

**Nursing Management of the Surgical Client**

**Preoperative Care**

Because the length of stay is limited for most clients undergoing PTRA, it is a challenge to coordinate their care during the diagnostic and intervention phases of treatment. Typically, diagnostic tests such as angiograms and blood work are completed during the week before the procedure. Teaching about angioplasty and discharge instructions must be provided in a short period.

**Postoperative Care**

Care after PTRA is similar to other care after any angioplasty. Just after the procedure, vital signs are monitored every 15 minutes for 1 hour, every 30 minutes for 2 hours, hourly for 3 hours, and then regularly according to the established protocol. Check the dressing at these times for manifestations of bleeding or hematoma formation, and check peripheral pulses to assess circulation in the affected limb. Bed rest for up to 24 hours may be ordered.

In the longer term, the client’s blood pressure should be monitored because hypertension can indicate recurrence of the lesion. Control of hypertension is crucial to preserving renal function.

After renal artery bypass graft surgery, nursing care is similar to other care after any kidney surgery, such as nephrectomy (discussed earlier in this chapter); however, in the postoperative period after an aortorenal bypass graft procedure, the client may experience an initial exacerbation of hypertension. Its cause is unclear, but it is thought to be related to systemic vasoconstriction secondary to general anesthesia and intraoperative hypothermia, severe pain, or transient renin secretion caused by clamping the aorta and manipulating the kidney. This episode usually lasts no more than 48 hours, but it can be significant and may require medical intervention. You must monitor blood pressure frequently.

**RENAL VEIN DISEASE**

The primary pathologic process involving the renal vein is thrombosis. Obstruction of venous drainage increases
interstitial pressure, which reduces renal function. Findings include severe lumbar pain, renal enlargement, proteinuria, and hematuria. If the obstruction is bilateral, oliguria and azotemia occur. Contributing factors include diabetic nephropathy, chronic glomerulonephritis, and renal amyloidosis.

Kidney survival depends largely on the development of collateral circulation before the vessel is fully occluded. Embolectomy or ligation of the renal veins may be done, and anticoagulants may be prescribed. Intravenous streptokinase is used to lyse the occluding clot. If enough renal damage has occurred, nephrectomy is an option.

**CONGENITAL DISORDERS**

Renal congenital anomalies usually involve abnormalities in the number, position, form, size, or structure of the kidneys. Blood supply may be abnormal, although malformations that significantly affect renal function are rare. Anomalies of the ureteropelvic junction usually obstruct at that point and result in hydronephrosis. Typically, this situation is discovered and treated during childhood.

**ANOMALIES INVOLVING KIDNEY NUMBER AND POSITION**

Renal agenesis is the absence of one or both kidneys. Having only one kidney presents no difficulty if this kidney functions adequately. A client can live normally with one properly functioning kidney, as kidney donors aptly demonstrate. Bilateral agenesis, however, is fatal. Even in unilateral agenesis, the functioning kidney is at high risk for development of additional anomalies.

*Supernumerary* kidneys (more than two kidneys) are usually asymptomatic and are discovered during IVP. The extra ureter enters either the ipsilateral ureter or the bladder.

*Ectopic,* or malpositioned, kidneys are usually found in the pelvis, although thoracic kidneys have been documented. Problems associated with this anomaly include respiratory difficulties, pain caused by pressure on nerves or surrounding structures, and difficulty in childbirth.

Occasionally, one kidney may be across the midline so that both kidneys are on the same side. This condition usually remains undiscovered until infection or obstruction indicates a need for x-ray examination.

**ANOMALIES INVOLVING KIDNEY FORM AND SIZE**

Anomalies of kidney form and size include aplasia, hypoplasia, dysplasia, and *horseshoe kidney.* Aplastic kidneys are small and contracted and contain no functioning renal tissue. Renal hypoplasia produces miniature kidneys with some functioning tissue. Although clinically this condition may be asymptomatic, it may cause hypertension and recurrent urinary tract infection.

Horseshoe kidney results when two kidneys are joined into a single organ whose shape somewhat resembles that of a horseshoe (Figure 35-4). The kidneys are connected, usually at the lower poles, by an isthmus of tissue. Because the developmental error interferes with normal ascent and medial rotation, the kidney is usually located in the lower lumbar region with its pelvis facing anteriorly. Although clients with horseshoe kidney may be asymptomatic, they are susceptible to hydronephrosis, infection secondary to ureteropelvic junction obstruction, and calculus formation.

**ANOMALIES INVOLVING CYSTIC DISEASE**

Cystic disease in renal tissue can range from a simple, solitary fluid-filled mass to almost complete replacement of renal structures by cystic tissue. A simple renal cyst commonly originates superficially in the renal parenchyma. It grows slowly and usually produces no manifestations until adulthood, when it may cause heaviness and pain in the abdomen and become a palpable mass. Arriving at a diagnosis may be complicated because renal cysts closely resemble malignant tumors; naturally, differentiation between the two is vital. As long as a simple renal cyst remains asymptomatic, intervention is usually unnecessary. If intervention is necessary, the cyst may be aspirated with a needle, or a partial nephrectomy may be performed to remove it.
Polycystic Kidneys

Polycystic disease of the kidney is a hereditary disorder in which grape-like cysts containing serous fluid, blood, or urine replace normal kidney tissue (Figure 35-5). The condition may develop at any age.

Infantile polycystic kidney disease is inherited as an autosomal recessive trait, and both parents must have carried the gene. It is a rare disorder that affects both kidneys and often the liver. In an infant, the disease usually causes death within days. Milder forms of the disease do not appear until childhood.

Adult polycystic disease accounts for about 10% of the clients receiving dialysis or transplantation. It is inherited as an autosomal dominant trait (see the Genetic Links feature at right). It usually appears after age 40, although it may begin as early as age 20 or as late as age 80. There are diverse manifestations; the most common are dull, aching lumbar or flank pain, which may be colicky, and hematuria. Other common findings are proteinuria, palpable kidney masses, pyuria, calculi, and uremia. Early in the disease, the ability to concentrate urine decreases. Hypertension develops along with cardiac enlargement and heart failure.

Polycystic liver disease occurs in about one third of cases, and cystic lesions are sometimes found in the thyroid, lung, pancreas, spleen, ovary, testis, epididymis, uterus, and bladder. Cerebral aneurysms occur in about 2% of clients with polycystic kidney disease.

The cystic kidney can become so enlarged that it causes severe pressure on other organs, with production of additional extrarenal manifestations. The ultimate result of this disease is ESRD (see Chapter 36). As the disease slowly progresses, renal nephrons are destroyed, renal function deteriorates, and uremia ultimately results. The mean duration of polycystic kidney disease from onset of manifestations to development of uremia varies a great deal and may be 15 to 30 years or longer.

Because there is no known way to arrest the progress of the destructive cysts, conservative medical treatment is designed to preserve kidney function. Urinary tract infection is the most common complication because of the distorted renal architecture; chronic infection can occur if resistant bacteria develop. Aggressive control of hypertension is essential.

Unlike clients with decreasing creatinine clearance rates caused by other kidney diseases, those with polycystic kidney disease seem to waste rather than retain sodium. Thus they may need increased sodium and water intake. However, if they are hypertensive, which is often the case, dietary sodium is restricted. Dietary restrictions should be individualized for all clients with chronic kidney disease. When ESRD develops, dialysis or renal transplantation is required. Nursing

**GENETIC LINKS**

**Autosomal Dominant Polycystic Kidney Disease Description**

Autosomal dominant polycystic kidney disease (ADPKD) is an adult-onset disorder characterized by progressive cyst development and bilaterally enlarged polycystic kidneys. Clinical features include renal function abnormalities, hypertension, pain, and renal insufficiency. About 50% of clients develop end-stage renal disease by about age 60. Cysts can also occur in other organs (e.g., liver, pancreas). The prevalence of ADPKD in live births ranges from 1:400 to 1:1000 with approximately 400,000 affected people in the United States.

**GENETICS**

ADPKD is caused by mutations in the *PKD1* gene (~85% of cases) located on chromosome 16 or the *PKD2* gene (~15% of cases) located on chromosome 4. Molecular genetic testing of the *PKD1* and *PKD2* genes is available clinically. As an autosomal dominant condition, offspring of an affected individual with ADPKD have a 50% chance of inheriting the disease-causing mutation. Most clients have a parent with ADPKD, but de novo mutations (new or spontaneous mutations) occur in about 10% of families.

**DIAGNOSIS/TESTING**

Diagnosis is established by imaging studies of the kidneys or by molecular genetic testing of the *PKD1* and *PKD2* genes.

**MANAGEMENT**

As yet there is no treatment specifically directed towards the disease. Current therapy is aimed at reducing morbidity and mortality from renal and other complications of the disease.
interventions for clients with renal failure are discussed in Chapter 36.

Genetic counseling (see Chapter 15) is advisable because of the hereditary nature of the disease, especially if the disease is diagnosed during childbearing years. Because the disease typically appears after the childbearing period, however, the likelihood of transmitting the disease to another generation is high. Therefore counseling the extended family is essential when the disease has been identified.

**Adult-Onset Medullary Cystic Disease**
Adult-onset medullary cystic disease, sometimes called 
uremic sponge kidney or medullary polycystic disease, is also an autosomal dominant disorder. It is similar to polycystic disease in all aspects except that it progresses to uremia rapidly after its onset in the teenage years or between ages 20 and 29. Hemodialysis and renal transplantation are likely to be required.

**Medullary Sponge Kidney**
Medullary sponge kidney is a cystic disorder in which spaces are produced at the apex of the renal pyramids. Onset peaks during adolescence or between ages 30 and 40 years. Infection, calculi, pain, and hematuria are potential complications. Renal function usually remains adequate unless the client has uncontrolled infection or calculi.

**OTHER HEREDITARY RENAL DISORDERS**
Other hereditary renal disorders include some types of chronic nephritis (such as Alport’s syndrome), congenital nephrotic syndrome, distal renal tubular acidosis, idiopathic hypercalciuria, and nephrotic diabetes insipidus. Many of these conditions are fatal during childhood, but some persist into adulthood and are discussed in the appropriate parts of this text.

**CONCLUSIONS**
Renal disorders are highly complex. You must have a clear understanding of the structure and function of the renal system to care for clients with these conditions. The outcomes of successful treatment include preservation of renal function.

**THINKING CRITICALLY**
1. A 35-year-old newlywed woman enters the emergency department with acute abdominal pain and a temperature of 101.8°F. Her abdomen is distended, and she has not had a bowel movement today, even though she is usually regular. The abdominal pain is diffuse and on the right side. She has no rebound tenderness and no pain at McBurney’s point. Her WBC is 15,000/mm³. What problems other than appendicitis might she be experiencing? What other assessments should be made?

**Factors to Consider.** What type of renal problem might she be experiencing? What teaching would she need once the diagnosis is made? What medication might be prescribed and why? What follow-up is needed?

2. L.S. is a 22-year-old male college student with a diagnosis of acute glomerulonephritis. Three weeks ago, he received oral antibiotic therapy for strep throat. He is scheduled for a renal biopsy to confirm a diagnosis of acute poststreptococcal glomerulonephritis. What other diagnostic assessments should be made? What is the expected course of treatment?

**Factors to Consider.** What type of renal problem might she be experiencing? What teaching would she need once the diagnosis is made? What medication might be prescribed and why? What follow-up is needed?

3. C.H. is a 68-year-old retired mail carrier who lives with his wife. They spend 6 months living in their home state in the Midwest and spend the winter months in Arizona. The client is admitted with a diagnosis of suspected renal cell carcinoma. Diagnostic tests confirm the diagnosis. What surgical procedure will be scheduled? What laboratory assessments are associated with this procedure?

**Factors to Consider.** What diagnostic tests were used to confirm the diagnosis of renal carcinoma? What tests will be used to determine whether radiation, chemotherapy, and immunotherapy are needed? Describe the postoperative assessments you should make for pain management, vital signs, surgical dressings, drainage tubes, and urine output. If Mr. and Mrs. H. plan to drive to Arizona a month after surgery, what should you discuss with them about their travel plans?

**Discussions for these questions can be found on the website.**

**BIBLIOGRAPHY**

Citations appearing in red refer to primary research.

Citations appearing in blue refer to evidence-based practice guidelines and protocols.


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