CHAPTER 29 Nursing Care of Clients with Kidney Disorders

LEARNING OUTCOMES

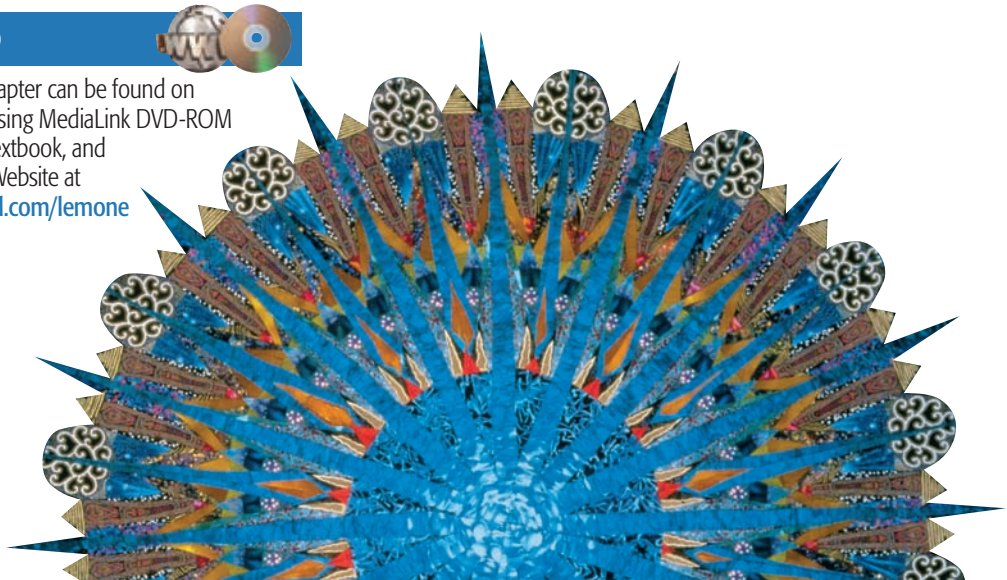
- Describe the pathophysiology of common kidney disorders, relating pathophysiology to normal physiology and manifestations of the disorder.
- Discuss risk factors for kidney disorders and nursing care to reduce these risks.
- Explain diagnostic studies used to identify disorders of the kidneys and their effects.
- Discuss the effects and nursing implications for medications and treatments used for clients with kidney disorders.
- Compare and contrast dialysis procedures used to manage acute and chronic renal failure.

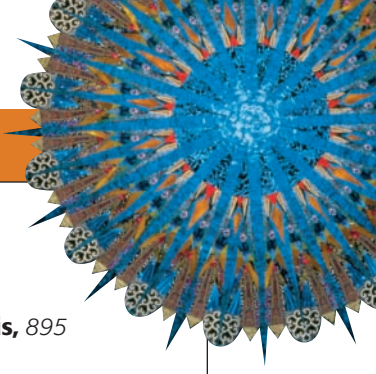
CLINICAL COMPETENCIES

- Assess the functional health status of clients with kidney disorders.
- Monitor, document, and report unexpected or abnormal manifestations in clients with kidney disorders.
- Provide appropriate and effective nursing care for clients undergoing dialysis, surgery involving the kidneys, or renal transplant.
- Based on assessment data, determine priority nursing diagnoses and interventions for clients with renal disorders.
- Plan and implement evidence-based nursing care for clients with renal disorders using research and best practices.
- Collaborate with the client and other members of the interdisciplinary team to prioritize and implement care.
- Provide teaching appropriate to the client and situation for clients with kidney disorders.
- Evaluate client responses to care, revising the plan of care as needed to promote, maintain, or restore functional health status for clients with renal disorders.

MEDIALINK

Resources for this chapter can be found on the Prentice Hall Nursing MediaLink DVD-ROM accompanying this textbook, and on the Companion Website at http://www.prenhall.com/lemone





KEY TERMS

acute renal failure, *899*
acute tubular necrosis, *901*
azotemia, *886*
chronic renal failure, *899*
continuous renal replacement therapy (CRRT), *906*
dialysate, *906*
dialysis, *906*
end-stage renal disease (ESRD), *899*
glomerular filtration rate, *883*
glomerulonephritis, *886*
hematuria, *885*
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nephrectomy, *896*
nephrotic syndrome, *888*
oliguria, *886*
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plasmapheresis, *891*
polycystic kidney disease, *884*
proteinuria, *885*
renal artery stenosis, *895*
renal failure, *899*
ultrafiltration, *906*
uremia, *906*

The internal environment of the body normally remains in a relatively constant or *homeostatic* state. The kidneys help maintain homeostasis by regulating the composition and volume of extracellular fluid. They excrete excess water and solutes and also can conserve water and solutes when deficits occur. In addition, the kidneys help regulate acid–base balance and they excrete metabolic wastes. Regulation of blood pressure is also a key function of the kidneys.

Both primary kidney disorders (such as glomerulonephritis) and systemic diseases (such as diabetes mellitus) can affect renal function. In North America, more than 20 million people are affected by kidney and urinary tract diseases (National Kidney and Urologic Diseases Information Clearinghouse [NKUDIC], 2004). Every year, approximately 1 in every 1000 people in the United States develops end-stage renal disease (ESRD), the final phase of chronic renal failure in which little or no kidney function remains. Chronic renal disease accounts for about 80,000 deaths per year and is a major cause of lost work time and wages (U.S. Renal Data System [USRDS], 2005). Ironically, the increased prevalence of chronic renal disease in recent years is partially related to the success of dialysis and transplantation.

AGE-RELATED CHANGES IN KIDNEY FUNCTION

Glomeruli in the renal cortex (see Chapter 27 ∞ for a review of normal kidney structure and function) are lost with aging, reducing kidney mass. Because of the large functional reserve of the kidneys, however, renal function remains adequate unless additional stressors affect the renal system. The **glomerular filtration rate (GFR)**, the amount of filtrate made by the kidneys per minute, declines due to age-related factors affecting the renovascular system (such as arteriosclerosis, decreased renal vascularity, and decreased cardiac output). By age 80, the GFR may be less than half of what it was at age 30.

Age-related changes in renal function have significant implications. The kidneys are less able to concentrate urine and compensate for increased or decreased salt intake. When combined with diminished effectiveness of antidiuretic hormone (ADH) and a reduced thirst response, both common in aging, this decreased ability to concentrate urine increases the risk for dehydration. Potassium excretion may be decreased because of lower aldosterone levels. As a result, fluid and electrolyte imbalances are more common and potentially critical in the older client.

Decreased GFR in the older adult also reduces the clearance of drugs excreted through the kidneys. This reduced clearance prolongs the half-life of drugs and may necessitate lower drug doses and longer dosing intervals. Common medications affected by decreased GFR include:

- Cardiac drugs: digoxin, procainamide
- Antibiotics: aminoglycosides, tetracyclines, cephalosporins
- Histamine H_2 antagonists: cimetidine
- Antidiabetic agents: chlorpropamide.

When caring for older adults, it is especially important to monitor drugs that are toxic to the renal tubules. Radiologic dyes and aminoglycoside, tetracycline, and the cephalosporin antibiotics are part of this group.

Age-related changes in renal function and related nursing implications are summarized in Table 29–1.

THE CLIENT WITH A CONGENITAL KIDNEY MALFORMATION

Congenital kidney disorders can affect the form and/or function of the kidney. Functional congenital kidney disorders are usually identified in childhood or adolescence. If function is not affected, congenital malformations may be detected only coincidentally. Malformations include agenesis, hypoplasia, alterations in kidney position, and horseshoe kidney.

Agenesis, absence of the kidney, and *hypoplasia,* underdevelopment of the kidney, typically affect only one of these paired organs. Renal function remains normal unless the unaffected kidney is compromised. Abnormal kidney position affects the ureters and urine flow, potentially leading to urinary stasis, increased risk of urinary tract infection (UTI), and lithiasis, or stone formation (see Chapter 28 ∞).

One in every 500 to 1000 people has *horseshoe kidney,* making it one of the most common renal malformations (Porth, 2005). Failure of the embryonic kidneys to ascend normally can result in a single, horseshoe-shaped organ. The two kidneys are fused at either the upper or lower pole (usually the lower). This malformation does not typically affect renal function; however, because the ureters cross the fused poles, there is an increased risk for *hydronephrosis,* or distention of the renal pelvis and

TABLE 29–1 **Nursing Implications of Age-Related Changes in Kidney Function**

FUNCTIONAL CHANGE	EFFECT	IMPLICATIONS
Decreased GFR	Decreased clearance of drugs excreted primarily through the kidneys increases drug half-life and blood levels, and risk of drug toxicity.	Monitor carefully for signs of toxicity, especially when administering digoxin, aminoglycoside antibiotics, tetracycline, vancomycin, chlorpropamide, procainamide, cimetidine, and cephalosporin antibiotics.
Decreased number of functional nephrons; lower levels of aldosterone; increased resistance to ADH	Decreased ability to conserve water and sodium; impaired potassium excretion; and decreased hydrogen ion excretion, resulting in reduced ability to compensate for acidosis.	Monitor for dehydration and hyponatremia; maintain fluid intake of 1500 to 2500 mL/day unless contraindicated; monitor for hyperkalemia, especially if taking a potassium-sparing diuretic, heparin, angiotensin-converting enzyme (ACE) inhibitor, beta-blocker, or NSAID; increased risk for acidosis.
Reduced numbers of functional nephrons	Decreased renal reserve with increased risk of failure.	Avoid giving nephrotoxic drugs if possible; monitor urine output and blood chemistries for early signs of renal failure.

calyces with urine (see Chapter 28 ∞). Recurrent UTI and renal calculi are also common in clients with horseshoe kidney.

Renal ultrasonography and intravenous pyelography are used to diagnose horseshoe kidney. Correction of the abnormality is rarely necessary, although surgical resection of the isthmus (connection between the kidneys) may be done to relieve ureteral obstruction or allow access to the abdominal aorta, which lies behind it.

Nursing care for clients with horseshoe kidney or other congenital malformations is primarily educational. Because abnormal kidney shape or position increases the risk of infection and stone formation, teach the client to maintain a fluid intake of at least 2500 mL per day. Emphasize the importance of avoiding dehydration by increasing fluids during hot weather and strenuous exercise. Teach hygiene practices such as perineal cleansing and voiding before and after intercourse to help prevent UTI. Teach the early manifestations of UTI and instruct to seek treatment promptly to prevent infection of the kidney. See Chapter 28 ∞.

THE CLIENT WITH POLYCYSTIC KIDNEY DISEASE

Polycystic kidney disease, a hereditary disease characterized by cyst formation and massive kidney enlargement, affects both children and adults. This disease has two forms: The autosomal dominant form affects adults; the autosomal recessive form is present at birth (Porth, 2005). Autosomal recessive polycystic kidney disease is rare. It usually is diagnosed prenatally or in infancy. Renal failure generally develops during childhood, necessitating kidney transplant or dialysis. Autosomal dominant polycystic kidney disease is relatively common, affecting 1 in every 300 to 1000 people and accounting for approximately 4% of clients with ESRD in the United States (Kasper et al., 2005). See the genetics box on this page. This section focuses on autosomal dominant polycystic kidney disease, the more common, adult form of the disorder.

GENETIC CONSIDERATIONS
Adult Polycystic Kidney Disease

- Approximately 90% of cases are inherited as an autosomal dominant trait; the remaining 10% are due to spontaneous mutations.
- Type 1, due to mutation of a gene on chromosome 16, accounts for approximately 85% of cases. It tends to have an earlier onset of symptoms and renal failure.
- Type 2, due to gene mutation on chromosome 4, is responsible for most remaining cases. The onset of manifestations and renal failure is later with type 2 polycystic kidney disease (Kasper et al., 2005).
- In both types, the genetic mutation affects production of *polycystin* membrane proteins and tubular epithelial cell growth and differentiation (Porth, 2005).

Pathophysiology

Renal cysts are fluid-filled sacs affecting the nephron, the functional unit of the kidneys. The cysts may range in size from microscopic to several centimeters in diameter and affect the renal cortex and medulla of both kidneys. As the cysts fill, enlarge, and multiply, the kidneys also enlarge. Renal blood vessels and nephrons are compressed and obstructed, and functional tissue destroyed (Figure 29–1 ■). The renal parenchyma atrophies and becomes fibrotic and scarred (Kasper et al., 2005).

People affected by polycystic kidney disease often develop cysts elsewhere in the body, including the liver, spleen, pancreas, and other organs. Diverticular disease of the colon is common, and may lead to perforation of the bowel (Kasper et al., 2005). Up to 10% of people affected experience subarachnoid hemorrhage from a type of congenital intracranial aneurysm. About 25% of people with polycystic kidney disease have mitral valve prolapse ("floppy" mitral valves). There also is an increased incidence of aortic and tricuspid valve insufficiency in people with polycystic kidney disease (Kasper et al., 2005).

Figure 29–1 ■ A polycystic kidney. The functional tissue of the kidneys is gradually destroyed and replaced with fluid-filled cysts.
Source: A. Glauberman/Photo Researchers, Inc.

Manifestations

Polycystic kidney disease is slowly progressive. Symptoms usually develop by age 40 to 50. Common manifestations include flank pain, microscopic or gross **hematuria** (blood in the urine), **proteinuria** (proteins in the urine), and *polyuria* and *nocturia,* as the concentrating ability of the kidney is impaired. Urinary tract infection and renal calculi are common, as cysts interfere with normal urine drainage. Most clients develop hypertension from disruption of renal vessels. The kidneys become palpable, enlarged, and knobby. Symptoms of renal insufficiency and chronic renal failure typically develop by age 60 to 70. The progression to ESRD tends to occur more rapidly in men than in women.

INTERDISCIPLINARY CARE

Diagnostic tests used to determine the extent of polycystic kidney disease include the following:

- *Renal ultrasonography* is the diagnostic procedure of choice for polycystic kidney disease.
- *Intravenous pyelography (IVP)* is used to detect cysts, evaluate their size, and determine the extent of kidney involvement.
- *Computed tomography (CT) scan* of the kidney is used to detect and differentiate renal masses such as cystic disease or tumors.

See the Diagnostic Tests table in Chapter 27 ∞ for the nursing implications of these tests.

Management of adult polycystic kidney disease is largely supportive. Care is taken to avoid further renal damage by nephrotoxic substances, UTI, obstruction, or hypertension. A fluid intake of 2000 to 2500 mL per day is encouraged to help prevent UTI and lithiasis. Hypertension associated with polycystic disease is generally controlled using angiotensin-converting enzyme (ACE) inhibitors or other antihypertensive agents (see Chapter 35 ∞). Ultimately, dialysis or renal transplantation is required. Clients with polycystic kidney disease are typically good candidates for transplantation because of the absence of associated systemic disease.

NURSING CARE

For those with adult polycystic kidney disease, an autosomal dominant disorder, discuss genetic counseling and screening of family members for evidence of the disease. This is particularly important if renal transplantation is contemplated and family members are potential donors. Consider the following nursing diagnoses when planning care for the client with polycystic kidney disease:

- *Excess Fluid Volume* related to impaired renal function
- *Anticipatory Grieving* related to potential loss of kidney function
- *Deficient Knowledge* regarding measures to help preserve kidney function
- *Risk for Ineffective Coping* related to potential genetic transmission of the disorder to offspring.

Teach the client with polycystic kidney disease about the disease, its genetic nature, and usual course. Discuss measures to maintain optimal renal function. Instruct to maintain a fluid intake of at least 2500 mL per day. Include additional information about preventing UTI (such as hygiene measures) and early manifestations of UTI. Stress the importance of seeking treatment to prevent further kidney damage. Advise to avoid drugs that are potentially toxic to the kidneys and to check with the primary care provider before taking any new drug. Discuss the potential benefits of genetic counseling with the client and family.

THE CLIENT WITH A GLOMERULAR DISORDER

Disorders and diseases involving the glomerulus are the leading cause of chronic renal failure in the United States. They are the underlying disease process for more than half of those people needing dialysis and result in a significant number of deaths per year (NKUDIC, 2004).

Glomerular disorders may be either primary, involving mainly the kidney, or secondary to a multisystem disease or hereditary condition. Primary glomerular disease is often immunologic or idiopathic in origin. Diabetes mellitus, systemic lupus erythematosus (SLE), and Goodpasture's syndrome are frequently implicated in secondary glomerular disorders.

FAST FACTS

- Glomerular disorders and diseases are the leading cause of chronic renal failure in the United States.
- Hematuria, proteinuria, and hypertension often are early manifestations of glomerular disorders.
- Acute poststreptococcal glomerulonephritis (also called acute proliferative glomerulonephritis) is the most common primary glomerular disorder.
- Diabetes mellitus and SLE are common causes of secondary glomerulonephritis.

Physiology Review

The glomerulus is a tuft of capillaries surrounded by a thin, double-walled capsule (Bowman's capsule). (See Figure 27–3.)

About 20% of the resting cardiac output flows through the glomeruli of the kidneys, forming approximately 180 L of plasma ultrafiltrate. More than 99% of this filtrate is reabsorbed in the renal tubules. The rate of glomerular filtration is controlled by opposing forces: The pressure and amount of blood flowing through the glomeruli promote filtration, and the pressure in Bowman's capsule and colloid osmotic (*oncotic*) pressure of the blood oppose it. The total surface area of glomerular capillaries also affects the GFR. The glomerular capillary membrane has three layers: the capillary endothelial layer, the basement membrane, and the capsule epithelial layer. Water and the smallest solutes (such as electrolytes) pass freely across this membrane, whereas larger molecules (such as plasma proteins) are retained in the blood.

Pathophysiology

Glomerular disease affects both the structure and function of the glomerulus, disrupting glomerular filtration. The capillary membrane becomes more permeable to plasma proteins and blood cells. This increased permeability in the glomerulus causes the manifestations common to glomerular disorders: hematuria, proteinuria, and edema. The GFR falls, leading to **azotemia** (increased blood levels of nitrogenous waste products) and hypertension. Glomerular involvement may be diffuse, involving all glomeruli, or focal, involving some glomeruli while others remain essentially normal.

Both hematuria and proteinuria are caused by glomerular capillary membrane damage, which allows blood cells and proteins to escape from the blood into the glomerular filtrate. Hematuria may be either gross or microscopic. Proteinuria is considered to be the most important indicator of glomerular injury, because it increases progressively with increased glomerular damage. Loss of plasma proteins leads to *hypoalbuminemia* (low serum albumin levels), which in turn reduces the plasma oncotic pressure (osmotic pressure created by plasma proteins), leading to edema.

As plasma proteins are lost, the forces opposing filtration diminish, and the amount of filtrate increases. The increased flow of filtrate stimulates the renin–angiotensin–aldosterone mechanism (see Chapter 27 ∞), producing vasoconstriction and a fall in GFR. Increased aldosterone production causes salt and water retention, which further contribute to edema. As the GFR falls, filtration and elimination of nitrogenous wastes, including urea, decrease, causing azotemia. **Oliguria**, urine output of less than 400 mL in 24 hours, may result from the decreased GFR. Hypertension results from fluid retention and disruption of the renin–angiotensin system, a key regulator of blood pressure.

The major primary glomerular disorders include acute glomerulonephritis, rapidly progressive glomerulonephritis, nephrotic syndrome, and chronic glomerulonephritis. Diabetic nephropathy and lupus nephritis are the most common secondary forms of glomerular disease.

Acute Proliferative Glomerulonephritis

Glomerulonephritis is inflammation of the glomerular capillary membrane. Acute glomerulonephritis can result from systemic diseases or primary glomerular diseases, but acute poststreptococcal glomerulonephritis (also known as acute proliferative glomerulonephritis) is the most common form. Infection of the pharynx or skin with group A beta-hemolytic streptococcus is the usual initiating event for this disorder. Staphylococcal or viral infections, such as hepatitis B, mumps, or varicella (chickenpox), can lead to a similar postinfectious acute glomerulonephritis (Porth, 2005). This primarily childhood disease also can affect adults.

In acute glomerulonephritis, circulating antigen–antibody immune complexes formed during the primary infection become trapped in the glomerular membrane, leading to an inflammatory response. The complement system is activated, and vasoactive substances and inflammatory mediators are released. Endothelial cells proliferate, and the glomerular membrane swells and becomes permeable to plasma proteins and blood cells (Figure 29–2 ■). Renal involvement is diffuse, spread throughout the kidneys. See *Pathophysiology Illustrated: Acute Glomerulonephritis* on the next page.

MANIFESTATIONS AND COMPLICATIONS Acute proliferative glomerulonephritis is characterized by an abrupt onset of hematuria, proteinuria, salt and water retention, and evidence of azotemia occurring 10 to 14 days after the initial infection. The urine often appears brown or cola-colored. Salt and water retention increase extracellular fluid volume, leading to hypertension and edema. The edema is primarily noted in the face, particularly around the eyes (*periorbital* edema). Dependent edema, affecting the hands and upper extremities in particular, may also be noted. Other manifestations may include fatigue, anorexia, nausea

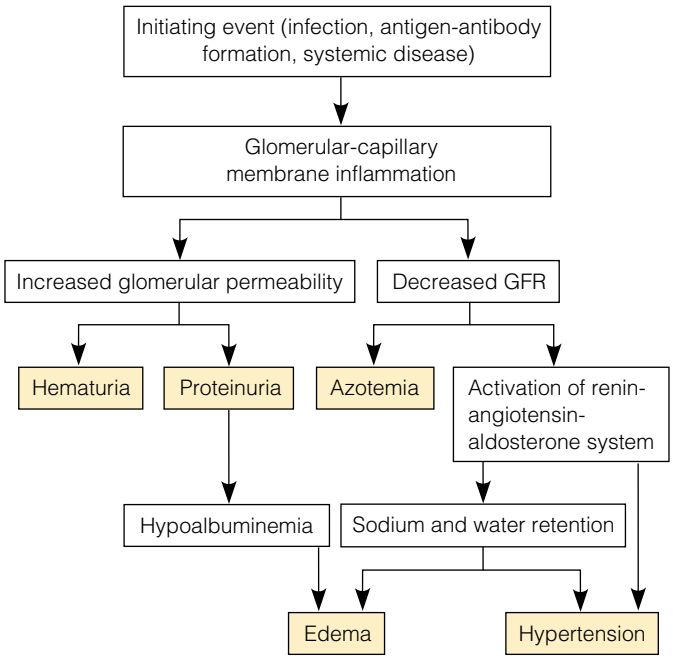
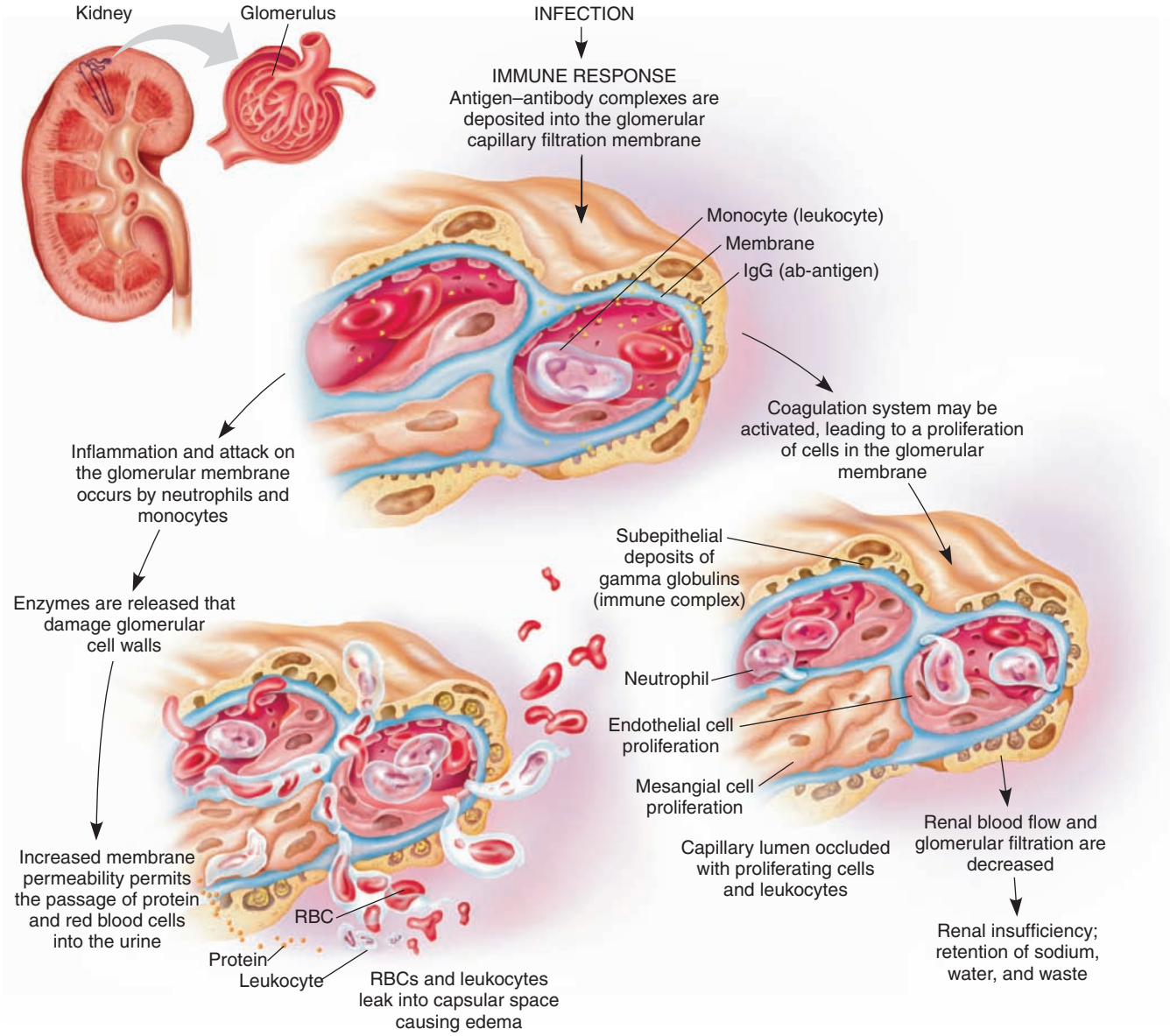


Figure 29–2 ■ The pathogenesis of glomerulonephritis.

Acute Glomerulonephritis

Infection from group A beta-hemolytic Streptococcus causes an immune response that causes inflammation and damage to the glomeruli. Protein and red blood cells are allowed to pass through the glomeruli. Blood flow to the glomeruli is reduced due to obstruction with damaged cells and renal insufficiency results, leading to the retention of sodium, water, and waste.



and vomiting, and headache (see the Manifestations box on the next page).

The older adult may have less apparent symptoms. Nausea, malaise, arthralgias, and proteinuria are common manifestations; hypertension and edema are seen less often. Pulmonary infiltrates may occur early in the disorder, often due to worsening of a preexisting condition such as heart failure.

The prognosis for adults with acute glomerulonephritis is less favorable than it is for children. The symptoms may resolve spontaneously within 10 to 14 days. Full recovery is usual in children, whereas 60% or more affected adults recover completely. The remainder have persistent symptoms, and some have permanent kidney damage (Porth, 2005).

Rapidly Progressive Glomerulonephritis

Rapidly progressive glomerulonephritis (RPGN) is characterized by manifestations of severe glomerular injury without a specific, identifiable cause. This type of

MANIFESTATIONS of Acute Glomerulonephritis

- Hematuria, cola-colored urine
- Proteinuria
- Salt and water retention
- Edema, periorbital and facial, dependent
- Hypertension
- Azotemia
- Fatigue
- Anorexia, nausea, and vomiting
- Headache

glomerulonephritis often progresses to renal failure within months. It may be idiopathic (primary), or secondary to a systemic disorder such as SLE or Goodpasture's syndrome. It affects people of all ages.

In RPGN, glomerular cells proliferate and, together with macrophages, form crescent-shaped lesions that obliterate Bowman's space (Porth, 2005). Glomerular damage is diffuse, leading to a rapid, progressive decline in renal function. Irreversible renal failure often develops over weeks to months (Kasper et al., 2005).

Clients with RPGN typically present with complaints of weakness, nausea, and vomiting. Some may relate a history of a flulike illness preceding the onset of the glomerulonephritis. Other symptoms include oliguria and abdominal or flank pain. Moderate hypertension may develop. On urinalysis, hematuria and massive proteinuria are noted.

GOODPASTURE'S SYNDROME *Goodpasture's syndrome* is a rare autoimmune disorder of unknown etiology. It is characterized by formation of antibodies to the glomerular basement membrane. These antibodies also may bind to alveolar basement membranes, damaging alveoli and causing pulmonary hemorrhage. Goodpasture's syndrome usually affects young men between ages 18 and 35, although it can occur at any age and affect women as well.

Although the glomeruli may be nearly normal in appearance and function in Goodpasture's syndrome, extensive cell proliferation and crescent formation characteristic of rapidly progressive glomerulonephritis are more common. Renal manifestations include hematuria, proteinuria, and edema. Rapid progression to renal failure may occur. Alveolar membrane damage can lead to mild or life-threatening pulmonary hemorrhage. Cough, shortness of breath, and hemoptysis (bloody sputum) are early respiratory manifestations.

Nephrotic Syndrome

Nephrotic syndrome is a group of clinical findings as opposed to a specific disorder. It is characterized by massive proteinuria, hypoalbuminemia, hyperlipidemia, and edema. A number of disorders can affect the glomerular capillary membrane, changing its porosity and allowing plasma proteins to escape into the urine.

Minimal change disease (MCD) is the most common cause of nephrotic syndrome in children and accounts for 20% of adults with nephrotic syndrome (Kasper et al., 2005). In MCD, the size and form of glomeruli appear normal by light microscopy. The prognosis for MCD is good.

In adults, *membranous glomerulonephropathy* is the most common cause of idiopathic nephrotic syndrome. The glomerular basement membrane thickens, although no inflammation is present. This form of nephrotic syndrome also occurs with some systemic diseases such as SLE and hepatitis B, and with drugs such as gold or penicillamine. *Focal sclerosis,* in which scarring (sclerosis) of glomeruli occurs, and *membranoproliferative glomerulonephritis,* caused by thickening and proliferation of glomerular basement membrane cells, are additional forms of nephrotic syndrome.

With plasma protein loss in the urine and resulting hypoalbuminemia, the oncotic pressure of the plasma falls. Fluid shifts from the vascular compartment to interstitial spaces, causing the edema characteristic of nephrotic syndrome. Salt and water retention, possibly due to activation of the renin–angiotensin system, contribute to the edema. Edema may be severe, affecting the face and periorbital area as well as dependent tissues (Figure 29–3 ■).

Loss of plasma proteins stimulates the liver to increase albumin production and lipoprotein synthesis. As a result, serum triglyceride and low-density lipoprotein (LDL) levels increase, as do urine lipids (*lipiduria*). Hyperlipidemia increases the risk for atherosclerosis in clients with nephrotic syndrome.

Thromboemboli (mobilized blood clots) are a relatively common complication of nephrotic syndrome. Loss of clotting and anticlotting factors along with plasma proteins are thought to disrupt the coagulation system, increasing the risk for renal venous thrombosis, deep venous thrombosis, and pulmonary embolism. Renal venous thrombosis can cause flank or groin pain on one or both sides, gross hematuria, and a reduced GFR (Porth, 2005).

Nephrotic syndrome usually resolves without long-term effects in children. The prognosis for adults is less optimistic because the syndrome often occurs secondarily to another disorder. Many adults do not recover completely, experiencing persistent proteinuria and potentially, progressive renal impairment.

Chronic Glomerulonephritis

Chronic glomerulonephritis is typically the end stage of other glomerular disorders such as RPGN, lupus nephritis, or diabetic nephropathy. In many cases, however, no previous glomerular disease has been identified.

Slow, progressive destruction of the glomeruli and a gradual decline in renal function are characteristic of chronic glomerulonephritis. The kidneys decrease in size symmetrically, and their surfaces become granular or roughened. Eventually, entire nephrons are lost.

Symptoms develop insidiously, and the disease is often not recognized until signs of renal failure develop. Chronic glomerulonephritis may also be diagnosed when hypertension and impaired renal function are found coincidentally during a routine physical examination or treatment for an unrelated disorder. Viral

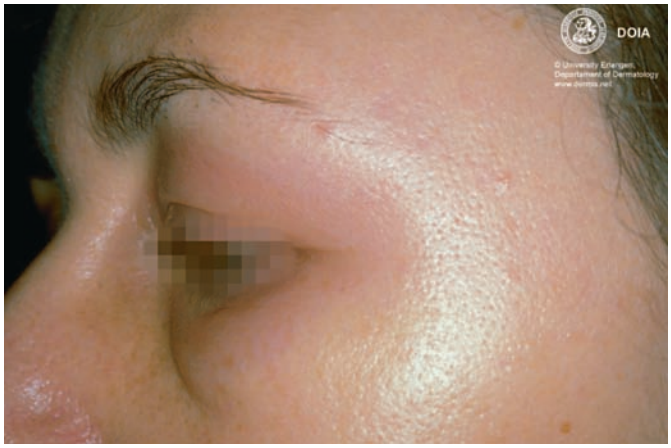


Figure 29–3 ■ Severe edema characteristic of nephrotic syndrome.

Source: Science Photo Library/Library Researcher, Inc.

or bacterial infectious diseases can exacerbate the disorder, prompting its diagnosis.

The course of chronic glomerulonephritis varies, with years to decades between the diagnosis and the development of end-stage renal failure.

Diabetic Nephropathy

Diabetic nephropathy, kidney disease common in the later stages of diabetes mellitus (DM), is the leading cause of ESRD in North America. Thirty percent of clients with type 1 DM and about 20% of type 2 diabetic clients develop nephropathy. ESRD resulting from diabetic nephropathy is seen more frequently in blacks with type 2 DM and in whites with type 1 DM (Kasper et al., 2005).

Initial evidence of microproteinuria indicating renal damage is typically seen within 10 to 15 years after the onset of diabetes. Overt proteinuria and nephropathy generally develop within 15 to 20 years of the initial diagnosis.

The characteristic lesion of diabetic nephropathy is glomerulosclerosis and thickening of the glomerular basement membrane. As the disease progresses, the glomerular capillary lumen narrows, reducing the surface area for glomerular filtration. Arteriosclerosis, a common feature of long-term diabetes and hypertension, contributes to the disease, as do nephritis and tubular lesions. Pyelonephritis, inflammation of the kidney, is also implicated in the development of diabetic nephropathy. A further discussion is found in Chapter 20 ∞.

Lupus Nephritis

Systemic lupus erythematosus (SLE) is an inflammatory autoimmune disorder affecting the connective tissue of the body. Between 40% and 85% of clients with SLE develop manifestations of nephritis (Kasper et al., 2005). Immune complexes that form within the glomerular capillary wall are the usual trigger for glomerular injury in SLE. Manifestations of lupus nephritis range from microscopic hematuria to massive proteinuria. Its progression may be slow and chronic or *fulminant,* with a sudden onset and the rapid development of renal failure. Most clients with minimal or mild lesions survive for at least 10 years. Improved management of the underlying disease, immunotherapy, dialysis, and renal transplantation have significantly improved the prognosis in recent years.

INTERDISCIPLINARY CARE

Management of all types of glomerulonephritis, acute and chronic, primary and secondary, focuses on identifying the underlying disease process and preserving kidney function. In most glomerular disorders, there is no specific treatment to achieve a cure. Treatment goals are to maintain renal function, prevent complications, and support the healing process.

Diagnosis

Laboratory and diagnostic testing are valuable to identify the cause of glomerulonephritis and evaluate kidney function.

The following studies may be ordered to help identify the underlying cause or etiology:

- *Throat or skin cultures* detect infection by group A beta-hemolytic streptococci. Although poststreptococcal glomerulonephritis typically follows the acute infection by 1 to 2 weeks, treatment to eradicate any remaining organisms is initiated to minimize antibody production.
- *Antistreptolysin O (ASO) titer* and other tests detect streptococcal *exoenzymes* (bacterial enzymes that stimulate the immune response in acute poststreptococcal glomerulonephritis). Other titers such as antistreptokinase (ASK) or antideoxyribonuclease B (ADNAase B) may be obtained as well.
- *Erythrocyte sedimentation rate (ESR)* is a general indicator of inflammatory response. It may be elevated in acute poststreptococcal glomerulonephritis and in lupus nephritis.
- *KUB* (kidney, ureter, bladder) *abdominal x-ray* may be done to evaluate kidney size and rule out other causes of the client's manifestations. The kidneys may be enlarged in acute glomerulonephritis, whereas bilateral small kidneys are typical of late chronic glomerulonephritis.
- *Kidney scan,* a nuclear medicine procedure, allows visualization of the kidney after intravenous administration of a radioisotope. In glomerular diseases, the uptake and excretion of the radioactive material are delayed.
- *Biopsy,* microscopic examination of kidney tissue, is the most reliable diagnostic procedure for glomerular disorders. Biopsy helps determine the type of glomerulonephritis, the prognosis, and appropriate treatment. Renal biopsy is usually done percutaneously, by inserting a biopsy needle through the skin into the kidney to obtain a tissue sample. Open biopsy, which requires surgery, may also be done.

See Chapter 27 ∞ for the nursing implications of diagnostic tests used to evaluate glomerular disorders.

The following studies are used to evaluate kidney function:

- *Blood urea nitrogen (BUN)* measures urea nitrogen, the end product of protein metabolism. It is created by the breakdown

MEDIALINK Case Study: Acute Glomerulonephritis

and metabolism of both dietary and body proteins. Urea is eliminated from the body by filtration in the glomerulus; minimal amounts are reabsorbed in the renal tubules. Glomerular diseases interfere with filtration and elimination of urea nitrogen, causing blood levels to rise. Increased protein catabolism (destruction), which may occur with GI bleeding or tissue breakdown, can also raise the BUN. Normal BUN values are listed in Table 29–2. Levels up to 50 mg/dL or 17.7 mmol/L indicate mild azotemia, and levels higher than 100 mg/dL or 35.7 mmol/L indicate severe renal impairment.

- *Serum creatinine* measures the amount of creatinine in the blood. Creatinine is also a metabolic by-product, produced in relatively constant amounts by skeletal muscles. It is excreted entirely by the kidneys, making the serum creatinine a good indicator of kidney function. Normal values (see Table 29–2) are lower in the older adult because of decreased muscle mass. Levels greater than 4 mg/dL indicate serious impairment of renal function.
- *Urine creatinine* also is an indicator of renal function and the GFR. Urine creatinine levels decrease when renal function is impaired because it is not effectively eliminated from the body.
- *Creatinine clearance* is a specific indicator of renal function used to evaluate the GFR. The *clearance,* or amount of blood cleared of creatinine in 1 minute, depends on the amount and pressure of blood being filtered and the filtering ability of the glomeruli. Levels normally decline with aging as the GFR decreases in the older adult. Disorders such as glomerulonephritis affect glomerular filtration, decreasing the creatinine clearance.
- *Serum electrolytes* are evaluated because impaired kidney function alters their excretion. Monitoring serum electrolytes is particularly important to prevent complications associated with imbalances.
- *Urinalysis* often shows red blood cells (RBCs) and proteins in the urine of clients with a glomerular disorder. These substances, normally too large to enter glomerular filtrate, escape due to increased porosity of glomerular capillaries in glomerular disorders. A 24-hour urine specimen is used to determine the amount of protein in the urine.

Medications

Although no drugs are available to cure glomerular disorders, medications are used to treat underlying disorders, reduce inflammation, and manage the symptoms.

Antibiotics are prescribed for the client with poststreptococcal glomerulonephritis to eradicate any remaining bacteria, removing the stimulus for antibody production. Nephrotoxic antibiotics, such as the aminoglycoside antibiotics, streptomycin, and some cephalosporins, are avoided.

Aggressive immunosuppressive therapy is used to treat acute inflammatory processes such as rapidly progressive glomerulonephritis, Goodpasture's syndrome, and exacerbations of SLE. When begun early, immunosuppressive therapy significantly reduces the risk of end-stage renal disease and renal failure. Prednisone, a glucocorticoid, is prescribed in relatively large doses of 1 mg per kilogram of body weight per day (e.g., a 160-pound man would receive 70 to 75 mg per day). Other immunosuppressive agents such as cyclophosphamide (Cytoxan) or azathioprine (Imuran) are prescribed in

TABLE 29–2 Changes in Laboratory Values Associated with Kidney Disease

TEST	NORMAL VALUE	VALUE IN RENAL DISEASE
Blood urea nitrogen (BUN)	5–20 mg/dL Slightly higher in older adult	20–50 mg/dL or higher
Creatinine, serum	Female: 0.5–1.1 mg/dL Male: 0.6–1.2 mg/dL Slightly lower in older adult	Elevated; levels >4 mg/dL indicate severe impairment of renal function
Creatinine clearance	Female: 88–128 mL/min Male: 97–137 mL/min Values decline in older adult	Reduced renal reserve: 32.5–90.0 mL/min Renal insufficiency: 6.5–32.5 mL/min Renal failure: <6.5 mL/min
Serum albumin	3.2–5 g/dL; 3.2–4.8 g/dL in older adult	Decreased in nephrotic syndrome
Serum electrolytes	Potassium: 3.5–5.0 mEq/L Sodium: 136–145 mEq/L Calcium: 4.5–5.5 mEq/L or 8.2–10.5 mg/dL Phosphorus: 3.0–4.5 mg/dL	Increased in renal insufficiency Decreased in nephrotic syndrome Decreased in renal failure Increased in renal failure
Red blood cell count	Female: 4.0–5.5 million/mm^3 Male: 4.5–6.2 million/mm^3	Decreased in chronic renal failure
Urine creatinine	Female: 600–1800 mg/24 hours Male: 800–2000 mg/24 hours	Decreased in disorders of impaired renal function
Urine protein	Resting: 50–80 mg/24 hours Ambulatory: <150–250 mg/24 hours	Increased in disorders of impaired renal function
Urine red blood cells	<2–3/HPF; no RBC casts	Present in glomerular disorders

conjunction with corticosteroids. Corticosteroid use in poststreptococcal glomerulonephritis may actually worsen the condition, so it is avoided.

Oral glucocorticoids such as prednisone also are used in high doses to induce remission of nephrotic syndrome. When glucocorticoids alone are ineffective, other immunosuppressive agents such as cyclophosphamide or chlorambucil (Leukeran) may be used to induce or maintain remission. See Chapter 13 for more information about corticosteroids and other immunosuppressive drugs.

ACE inhibitors may be ordered to reduce protein loss associated with nephrotic syndrome. These drugs reduce proteinuria and slow the progression of renal failure. They have a protective effect on the kidney in clients with diabetic nephropathy. Nonsteroidal anti-inflammatory drugs (NSAIDs) also reduce proteinuria in some clients, but can increase salt and water retention (Kasper et al., 2005).

Antihypertensives may be prescribed to maintain the blood pressure within normal levels. Blood pressure management is important because systemic and renal hypertension are associated with a poorer prognosis in clients with glomerular disorders.

Treatments

Bed rest may be ordered during the acute phase of poststreptococcal glomerulonephritis. When the edema of nephrotic syndrome is significant or the client is hypertensive, sodium intake may be restricted to 1 to 2 g per day. Dietary protein may be restricted if azotemia is present. When proteins are restricted, those included in the diet should be complete or high-value proteins. Complete proteins supply the essential amino acids required for growth and tissue maintenance. Complete and incomplete proteins are compared in Table 29–3.

Plasma exchange therapy (**plasmapheresis**), a procedure to remove damaging antibodies from the plasma, is used in conjunction with immunosuppressive therapy to treat RPGN and Goodpasture's syndrome. Plasma and glomerular-damaging antibodies are removed using a blood cell separator. The RBCs are then returned to the client along with albumin or human plasma to replace the plasma removed. This procedure is usually done in a series of treatments. It is not without risk, and informed consent is required. Potential complications of plasma exchange therapy include those associated with intravenous catheters, fluid volume shifts, and altered coagulation.

Renal failure resulting from a glomerular disorder may necessitate dialysis to restore fluid and electrolyte balance and remove waste products from the body. Dialysis procedures and related nursing care are explained in the acute renal failure section later in this chapter.

TABLE 29–3 Complete and Incomplete Protein Sources

	COMPLETE PROTEINS	INCOMPLETE PROTEINS
Definition	Provide all essential amino acids needed for growth and tissue maintenance	Lack one or more essential amino acids or contain inadequate proportions
Examples	Milk, eggs, cheese, meats, poultry, fish, and soy	Vegetables, breads, cereals and grains, legumes, seeds, and nuts

NURSING CARE

Health Promotion

Discuss the importance of effectively treating streptococcal infections in all age groups to help reduce the risk for acute glomerulonephritis. Stress the importance of completing the full course of antibiotic therapy to eradicate the infecting bacteria. Teach clients with diabetes mellitus and SLE about potential renal effects of their disease. Discuss measures to reduce the risk of associated nephritis, such as effectively managing the disease, treating hypertension, and avoiding drugs and substances that are potentially toxic to the kidneys.

Assessment

Review Chapter 27 for complete assessment of the renal and urinary systems. Focused assessment data related to glomerular disorders include the following:

- *Health history:* Complaints of facial or peripheral edema or weight gain, fatigue, nausea and vomiting, headache, general malaise, abdominal or flank pain; cough or shortness of breath; changes in amount, color, or character of urine (e.g., frothy urine); history of skin or pharyngeal streptococcal infection, diabetes, SLE, or kidney disease; current medications.
- *Physical examination:* General appearance; vital signs; weight; presence of periorbital, facial, or peripheral edema; skin for lesions, infection; inspect throat, obtain culture as indicated; urine specimen for color, character, odor.

Nursing Diagnoses and Interventions

Nursing care is supportive and educational. Monitoring renal function and fluid volume status are key components of care, as is protecting the client from infection. Both manifestations of glomerular disorders and their treatment can interfere with a client's ability to maintain usual roles and responsibilities. For additional potential nursing diagnoses and interventions, see the Nursing Care Plan that follows.

Excess Fluid Volume

Excess fluid volume and resulting edema are common manifestations of glomerular disorders. When proteins are lost in the urine, the oncotic pressure of plasma falls, and fluid shifts into the interstitial spaces. The body responds to this fluid shift by retaining sodium and water to maintain intravascular volume, leading to excess fluid volume.

MEDIALINK Care Plan Activity: Acute Glomerulonephritis

NURSING CARE PLAN A Client with Acute Glomerulonephritis

Jung-Lin Chang is a 23-year-old graduate student in biology. He presents at the university health center with brown and foamy urine. The physician there admits him to the infirmary and orders a throat culture, ASO titer, CBC, BUN, serum creatinine, and urinalysis.

ASSESSMENT

Connie King, the nurse admitting Mr. Chang, notes that his history is essentially negative for past kidney or urinary problems. He relates having had a "pretty bad" sore throat a couple of weeks before admission. However, it was during midterms, so he took a few antibiotics he had from a previous bout of strep throat, increased his fluids, and did not see a doctor. The sore throat resolved, and he felt well until noticing the change in his urine. He admits that his eyes seemed a little puffy, but he thought this was due to lack of sleep and fatigue. He has eaten little the past 2 days, but was not alarmed because his food intake is irregular most of the time.

Physical assessment findings include T 98.8°F (37.1°C) PO, P 98, R 18, and BP 136/90. Weight 165 pounds (75 kg), up from his normal of 160 (72.5 kg). Moderate periorbital edema and edema of hands and fingers noted.

Throat culture is negative, but the ASO titer is high. CBC essentially normal. BUN 42 mg/dL, serum creatinine 2.1 mg/dL. Urinalysis reveals the presence of protein, red blood cells, and RBC casts. A subsequent 24-hour urine protein analysis shows 1025 mg of protein (normal 30 to 150 mg/24 hours).

The physician diagnoses acute poststreptococcal glomerulonephritis and places Mr. Chang on bed rest with bathroom privileges. He orders fluid restriction (1200 mL/day) and a restricted sodium and protein diet.

DIAGNOSES

- *Excess Fluid Volume* related to plasma protein deficit and sodium and water retention
- *Risk for Imbalanced Nutrition: Less than Body Requirements* related to anorexia
- *Anxiety* related to prescribed activity restriction
- *Risk for Ineffective Therapeutic Regimen Management* related to lack of information about glomerulonephritis and treatment

EXPECTED OUTCOMES

- Maintain blood pressure within normal limits.
- Return to usual weight with no evidence of edema.
- Consume adequate calories following prescribed dietary limitations.
- Verbalize reduced anxiety regarding ability to continue studies.
- Demonstrate an understanding of acute glomerulonephritis and prescribed treatment regimen.

PLANNING AND IMPLEMENTATION

- Vital signs every 4 hours; notify physician of significant changes.
- Weigh daily; intake and output every 8 hours.
- Schedule fluids allowing 650 mL on day shift, 450 mL on evening shift, and 100 mL on night shift.
- Arrange dietary consultation to plan a diet that includes preferred foods as allowed.
- Provide small meals with high-carbohydrate between-meal snacks.
- Encourage Mr. Chang to talk about his condition and its potential effects.
- Assist with problem solving and exploring options for maintaining studies.
- Enlist friends and family to listen and provide support.
- Teach Mr. Chang and his family about acute glomerulonephritis and prescribed treatment.
- Instruct in appropriate antibiotic use.

EVALUATION

Mr. Chang is released from the infirmary after 4 days. He decides to return to his parents' home for the 6 to 12 weeks of convalescence prescribed by his doctor. Mr. Chang's renal function gradually returns to normal with no further azotemia and minimal proteinuria after 4 months. He verbalizes understanding of the relationship between the strep throat, his inappropriate use of antibiotics, and the glomerulonephritis. He says, "I may not always remember to take every pill on time in the future, but I sure won't save them for the next time again!"

CRITICAL THINKING IN THE NURSING PROCESS

1. How did Mr. Chang's use of "a few" previously prescribed antibiotics to treat his sore throat affect his risk for developing poststreptococcal glomerulonephritis?
2. What additional risk factors did Mr. Chang have for developing glomerulonephritis?
3. The initial manifestations of acute poststreptococcal glomerulonephritis and rapidly progressive glomerulonephritis are very similar. What diagnostic test would the physician use to make the differential diagnosis? Develop a plan of care for a client undergoing this examination.

See Evaluating Your Response in Appendix C.

- Monitor vital signs, including blood pressure, apical pulse, respirations, and breath sounds, at least every 4 hours. Report significant changes. *Excess fluid increases the cardiac workload and the blood pressure. Tachycardia may result. Associated electrolyte imbalances can cause dysrhythmias. Increased pulmonary vascular pressure can lead to pulmonary edema, tachypnea, dyspnea, and crackles (rales) in the lungs.*
- Record intake and output every 4 to 8 hours, or more frequently as indicated. *Accurate intake and output records help determine fluid volume status.*

PRACTICE ALERT

Weigh daily, using consistent technique (time of day, scale, and clothing). Accurate daily weights are the best indicator of approximate fluid balance (Wise et al., 2000).

- Monitor serum electrolytes, hemoglobin and hematocrit, BUN, and creatinine. *Glomerular disorders affect fluid balance and may alter electrolyte balance as well, potentially leading to complications such as cardiac dysrhythmias (see Chapter 10 ∞). Increased intravascular volume can result in low hemoglobin and hematocrit values. BUN and creatinine provide information about renal function.*
- Maintain fluid restriction as ordered. Offer ice chips (in limited and measured amounts) and frequent mouth care to relieve thirst. With the client, develop a fluid intake schedule. *Fluids may be restricted to reduce fluid overload, edema, and hypertension. Ice chips and frequent mouth care moisten mucous membranes and help relieve thirst while maintaining oral tissue integrity. Including the client in planning fluid intake promotes a sense of control and understanding of the treatment regimen.*

PRACTICE ALERT
Carefully monitor and regulate intravenous infusions; include fluid used to dilute IV medications as intake. Significant "hidden" fluid intake can occur with intravenous medication administration.

- Arrange dietary consultation regarding sodium or protein-restricted diets. *Including the client and dietitian in planning allows individualization of the diet to client preferences. The glomerular disorder may reduce appetite; considering food preferences can help maintain adequate nutrition.*
- Monitor for desired and adverse effects of prescribed medications. *Diuretic therapy helps reduce excess fluid volume; however, glomerular disorders can affect the client's response to treatment. In addition, diuretics can exacerbate the electrolyte imbalances and muscle weakness often associated with glomerular disorders.*
- Provide frequent position changes and good skin care. *Perfusion may be altered by tissue edema, increasing the risk of breakdown.*

Fatigue

Fatigue is a common manifestation of glomerular disorders. Anemia, loss of plasma proteins, headache, anorexia, and nausea compound this fatigue. The ability to maintain usual physical and mental activities may be impaired.

- Document energy level. *As glomerular function improves, fatigue begins to resolve, and energy increases.*
- Schedule activities and procedures to provide adequate rest and energy conservation. Prevent unnecessary fatigue. *Adequate rest and energy conservation reduce fatigue and improve the client's ability to tolerate and cope with required treatments and activities.*
- Assist with ADLs as needed. *The goal is to conserve limited energy reserves.*
- Discuss the relationship between fatigue and the disease process with client and family. *Understanding the nature of the disease and associated fatigue helps the client and family cope with reduced energy and comply with prescribed rest.*
- Reduce energy demands with frequent, small meals and short periods of activity. Limit the number of visitors and visit length. *Small, frequent meals reduce the energy needed for eating and digestion. Limiting visitors and visit length helps conserve energy. In addition, nurses can assist the fatigued client who may be reluctant to ask visitors to leave.*

Ineffective Protection

The effects of both the glomerular disorder and treatment with anti-inflammatory and cytotoxic drugs can depress the immune system, increasing the risk for infection. The anti-inflammatory effect of corticosteroids may also mask early manifestations of infection.

PRACTICE ALERT
Monitor vital signs, temperature, and mental status every 4 hours. An elevated temperature may indicate infection; anti-inflammatory drugs may moderate this response, however. Tachycardia, increasing lethargy, or confusion may be the initial signs of infection.

- Assess frequently for other signs of infection such as purulent wound drainage, productive cough, adventitious breath sounds, and red or inflamed lesions. Monitor for manifestations of UTI, such as dysuria, frequency and urgency, and cloudy, foul-smelling urine. *Early identification and treatment of infection is important to prevent systemic complications in the susceptible client.*
- Monitor CBC, focusing on the WBC and differential. *An elevated WBC and increased numbers of immature WBCs in the blood (left shift) may be early indicators of infection.*
- Use good hand washing technique. Protect from cross-infection by providing a private room and restricting ill visitors. *Clients with decreased resistance to infection need increased protection.*
- Avoid or minimize invasive procedures. *Maintaining the protective skin barrier is especially important for the client with altered immune status.*
- If catheterization is required, use sterile intermittent straight catheterization or maintain a closed drainage system for an indwelling catheter. Prevent urine reflux from the drainage system to the bladder or the bladder to the kidneys by ensuring a patent, gravity flow system. *The urinary tract is a frequent entry point for infection, particularly in the hospitalized or institutionalized client. Maintaining strict asepsis during catheterization is vital. Intermittent catheterization is associated with a lower risk of UTI than an indwelling catheter.*
- Provide a nutritionally sound diet with complete proteins. *A well-balanced, nutritionally sound diet is important to maintain nutritional status and support immune function.*
- Teach measures to prevent infection. *Care often is provided in the home, requiring the client and family to use appropriate infection control measures.*

Ineffective Role Performance

The manifestations and treatment of glomerular disorders can affect the ability to maintain usual roles and activities. Fatigue and muscle weakness may limit physical and social activities. Bed rest or activity limitations may be ordered to minimize the degree of proteinuria. If azotemia is present, malaise, nausea, and mental status changes can interfere with role function. Facial and periorbital edema affect the client's self-esteem and may lead to isolation.

- Establish a strong therapeutic relationship. *It is important to gain the client's trust and confidence.*
- Encourage self-care and participation in decision making. *Increased autonomy helps restore self-confidence and reduce powerlessness.*
- Provide time for verbalization of thoughts and feelings; listen actively, acknowledging and accepting fears and concerns. *Adequate time and active listening encourage expression of concerns and the effect of the disease or treatments on daily life. This helps the client deal with the illness, its treatment, and associated losses.*
- Support coping skills, helping the client identify personal strengths. *This support helps the client gain confidence.*
- When possible, enlist the support of family, other clients, and friends. *These people can provide physical, psychologic, emotional, and social support.*
- Discuss the effect of the disease and treatments on roles and relationships, helping identify potential changes in roles, relationships, and lifestyle. Help the client and family develop a plan for alternative behaviors and relationships, encouraging the client to maintain usual roles to the extent possible. *Developing a plan helps reduce the strain of role changes and maintain a sense of dignity and control.*
- Provide accurate and optimistic information about the disorder and its short- and long-term effects. *The client and family need accurate information to plan for the future.*
- Evaluate the need for additional support and social services for the client and family. Provide referrals as indicated. *Depending on client and family strengths, the severity of the disorder, and its treatment and prognosis, ongoing social support services may be necessary to facilitate coping and adaptation.*

Community-Based Care

Glomerular disorders may be self-limited or progressive. In either case, the course is lengthy, ranging from months to years. Self-management is essential. Provide instructions for the client and family, including the following topics:

- Information about the disease and the prognosis
- Prescribed treatment, including activity and diet restrictions; the use and potential effects, both beneficial and adverse, of all medications
- Risks, manifestations, prevention, and management of complications such as edema and infection
- Signs, symptoms, and implications of improving or declining renal function
- Measures to prevent further kidney damage, such as nephrotoxic drugs to avoid
- Community resources, such as home care providers and support groups.

THE CLIENT WITH A VASCULAR KIDNEY DISORDER

Renal function is dependent on an adequate supply of blood. Blood supports renal cell metabolism and is vital to kidney function, the nephron in particular. The kidney can regulate fluid, electrolyte, and acid–base balance and serve as a major organ of excretion only when its blood supply is sufficient. Vascular disorders, therefore, can have a significant impact on renal function.

Hypertension

Hypertension, sustained elevation of the systemic blood pressure, can result from or cause kidney disease.

Prolonged hypertension damages the walls of arterioles and accelerates the process of atherosclerosis. This damage primarily affects the heart, brain, kidneys, eyes, and major blood vessels. In the kidney, arteriosclerotic lesions develop in the *afferent* (leading into) and *efferent* (going out of) arterioles and the glomerular capillaries. The glomerular filtration rate declines and tubular function is affected, resulting in proteinuria and microscopic hematuria. Approximately 10% of deaths attributed to hypertension result from renal failure (Kasper et al., 2005).

Malignant hypertension is a rapidly progressive form of hypertension that can develop in clients with untreated primary hypertension. The diastolic pressure is in excess of 120 mmHg and may be as high as 150 to 170 mmHg. Malignant hypertension affects less than 1% of hypertensive clients; it is more common in African Americans than in people of European ancestry. Untreated, malignant hypertension causes a rapid decline in renal function due to vessel changes, renal ischemia, and infarction.

Approximately 5% to 10% of hypertensive clients have *secondary hypertension,* which is actually a manifestation of an underlying disease. Renal vascular disease and diseases of the renal parenchyma, such as diabetic nephropathy, are commonly associated with secondary hypertension.

Management of hypertension to maintain the blood pressure within normal limits is vital to prevent kidney damage. When hypertension is secondary to kidney disease, adequate blood pressure control can slow the decline in renal function. Hypertension and its management is discussed in depth in Chapter 35 ∞.

Renal Artery Occlusion

Renal arteries can be occluded by either a primary process affecting the renal vessels or by emboli, clots, or other foreign material. Risk factors for *acute renal artery thrombosis* (formation of a blood clot in the renal artery) include severe abdominal trauma, vessel trauma from surgery or angiography, aortic or renal artery aneurysms, and severe aortic or renal artery atherosclerosis. Emboli from the left side of the heart can travel via the aorta to occlude the renal artery. Emboli may form as a result of atrial fibrillation (irregular and uncoordinated electrical activity of the atria), following myocardial infarction, as vegetative growths on heart valves associated with bacterial endocarditis, or from fatty plaque in the aorta.

Renal arterial occlusion may be asymptomatic when the occlusion develops slowly and the affected vessels are small.

Acute occlusion leading to ischemia and infarction typically causes sudden, severe localized flank pain, nausea and vomiting, fever, and hypertension. Hematuria and oliguria may occur. In the older client, the new onset of hypertension or worsening of previously controlled hypertension may signal renal artery thrombosis.

Laboratory studies reveal leukocytosis (elevated WBC), and elevated renal enzyme levels, including aspartate transaminase (AST) and lactic dehydrogenase (LDH). These enzymes, normally present in renal cells, are released into the circulation when cells necrose and die. With bilateral arterial occlusion and infarction, renal function deteriorates rapidly, leading to acute renal failure (Kasper et al., 2005).

Surgery to restore blood flow to the affected kidney may be indicated for acute occlusion. Management usually is more conservative, using anticoagulant therapy, hypertension control, and supportive treatment.

Renal Vein Occlusion

A thrombus (clot) formed in a renal vein can occlude the vessel. The cause of the thrombus often is unclear. In adults, renal venous thrombosis usually occurs with nephrotic syndrome. Other predisposing factors include pregnancy, oral contraceptive use, and certain malignancies.

Gradual or acute deterioration of renal function may be the only manifestation of renal vein occlusion. If the thrombus breaks loose, it can become a pulmonary embolism. The definitive diagnosis is made by visualizing the thrombus through renal venography.

Thrombolytic drugs such as streptokinase or tissue plasminogen activator (tPA) may be given to dissolve or break up the thrombus. Anticoagulant therapy also is used to prevent further clotting and pulmonary emboli. Renal function often improves with treatment.

Renal Artery Stenosis

Renal artery stenosis (narrowing) causes 2% to 5% of all cases of hypertension (Kasper et al., 2005). It can affect one or both kidneys.

Atherosclerosis with gradual occlusion of the renal artery lumen by plaque is the primary cause of renal artery stenosis in men. In younger women, the most common cause is fibromuscular dysplasia, structural abnormalities involving the intimal, medial, or adventitial layers of the arterial wall.

Renal artery stenosis is suspected when hypertension develops before age 30 or after age 50 with no prior history of high blood pressure. An epigastric bruit (murmur) and other manifestations of vascular insufficiency may also be present. The affected kidney appears small and atrophied on renal ultrasound. The captopril test for renin activity and renal angiography are used to confirm the diagnosis. Clients with renovascular hypertension show higher levels of renin activity following administration of captopril, an ACE inhibitor drug (see Chapter 35 ∞), than clients with essential or primary hypertension (Kasper et al., 2005). Renal angiography uses radiologic contrast dye injected into the renal arteries to allow visualization of renal blood vessels.

The preferred treatment for renal artery stenosis is dilation of the stenotic vessel by percutaneous transluminal angioplasty. In this procedure, a balloon-tipped catheter is inserted via the femoral artery and aorta to dilate the renal artery. It relieves symptoms immediately in 90% of clients with fibromuscular dysplasia. One year after treatment, 60% remain symptom free. Some clients require a bypass graft of the renal artery. A section of saphenous vein or hypogastric artery is grafted from the aorta to the renal artery beyond the stenosis. The blood pressure may not return to normal following treatment, but is more easily managed using medications.

THE CLIENT WITH KIDNEY TRAUMA

The kidneys are relatively well protected by the rib cage and back muscles, but trauma due to blunt force or penetrating injury may inflict damage. Many renal injuries heal uneventfully, but prompt diagnosis and immediate treatment can be lifesaving in the event of major damage.

Pathophysiology and Manifestations

Blunt force is the most common cause of kidney injury. Falls, motor vehicle accidents, and sports injuries can damage the kidney. The injury may be minor, causing a contusion or small hematoma, or more serious, resulting in laceration or other damage. The kidney may fragment or "shatter," causing significant blood loss and urine extravasation. Tearing of the renal artery or vein may cause rapid hemorrhage, with shock and possible death.

Gunshot wounds, knife wounds, impalement injuries, and fractured ribs can penetrate the kidney. Minor penetrating injuries may lacerate the capsule or renal cortex. Major injuries include laceration or destruction of renal parenchyma or the vascular supply. Renal artery, renal vein, and renal pelvis lacerations are critical injuries.

The primary manifestations of kidney trauma are hematuria (gross or microscopic), flank or abdominal pain, and oliguria or anuria. There may be localized swelling, tenderness, or ecchymoses in the flank region. Retroperitoneal bleeding from the kidney may cause Turner's sign, a bluish discoloration of the flank. Signs of shock may be present, including hypotension, tachycardia, tachypnea, cool and pale skin, and an altered level of consciousness.

INTERDISCIPLINARY CARE

Hemoglobin and hematocrit levels fall in significant renal injury with hemorrhage. Hematuria is typically noted on urinalysis. AST levels rise within 12 hours of significant renal trauma. Renal ultrasonography is used to diagnose bleeding and kidney damage. A CT scan and IVP to visualize renal structures may be done to establish a definitive diagnosis. Renal arteriography is used when major injury is suspected and surgery is anticipated.

Treatment of minor kidney injuries is generally conservative, including bed rest and observation. In these injuries, bleeding is typically minor and self-limiting. With major or critical trauma, immediate treatment focuses on controlling hemorrhage and treating or preventing shock. Surgery or percutaneous arterial

embolization during angiography may be required to stop the bleeding. Major lacerations may require surgical repair, partial nephrectomy, or total **nephrectomy** (removal) of the affected kidney.

NURSING CARE

Nursing care for the client who has experienced renal trauma focuses on timely and accurate assessment and appropriate intervention to preserve life and prevent complications. Obtain a urine specimen for analysis when kidney trauma is suspected. Monitor level of consciousness, vital signs, skin color and temperature, and urine output for possible signs of shock. See Chapter 11 ∞ for additional nursing care measures for the client who has had a traumatic injury or who develops shock.

THE CLIENT WITH A RENAL TUMOR

Renal tumors may be either benign or malignant, primary or metastatic. Benign renal tumors are infrequent and are often found only on autopsy. Primary renal malignancies account for about 2% of adult cancers and approximately 12,600 deaths per year (American Cancer Society, 2005). Most primary renal tumors arise from renal cells; a primary tumor also may develop in the renal pelvis, although less frequently. Metastatic lesions to the kidney are associated with lung and breast cancer, melanoma, and malignant lymphoma.

Males are affected by renal cancer more than females by a 2:1 ratio. The highest incidence is seen in people over the age of 55 years. Smoking and obesity are risk factors; chronic irritation associated with renal calculi may also contribute. Some renal cancers are associated with genetic factors. Clients with ESRD also may develop renal cancer.

Pathophysiology and Manifestations

Most (85% to 95%) primary renal tumors are renal cell carcinomas (Kasper et al., 2005; Porth, 2005). These tumors arise from tubular epithelium and can occur anywhere in the kidney. The tumor, which can range in size up to several centimeters, has clearly defined margins and contains areas of ischemia, necrosis, and hemorrhage. Renal tumors tend to invade the renal vein, and often have metastasized when first identified. Metastases tend to occur in the lungs, bone, lymph nodes, liver, and brain.

Renal tumors are often silent, with few manifestations. The classic triad of symptoms, gross hematuria, flank pain, and a palpable abdominal mass, is seen in only about 10% of people with renal cell carcinoma. Hematuria, often microscopic, is the most consistent symptom. Systemic manifestations include fever without infection, fatigue, and weight loss. See the box on this page.

The tumor may produce hormones or hormone-like substances, including parathyroid hormone, prostaglandins, prolactin, renin, gonadotropins, and glucocorticoids. These substances produce *paraneoplastic syndromes,* with additional manifestations such as hypercalcemia, hypertension, and hyperglycemia. The progression of renal cell carcinomas varies from prolonged periods of stable disease to very aggressive. Table 29–4 outlines the staging and prognosis for renal cell cancers.

INTERDISCIPLINARY CARE

Hematuria is often the only initial manifestation of renal cancer; its presence indicates a need for further diagnostic studies, including:

- *Renal ultrasonography* to detect renal masses and differentiate cystic kidney disease from renal carcinoma
- *CT scan* to determine tumor density, local extension of the tumor, and regional lymph node or vascular involvement
- *IVP* and *magnetic resonance imaging* (MRI) may be done to evaluate renal structure and function
- *Renal angiography, aortography,* and *inferior venacavography* may be used to evaluate the extent of vascular involvement prior to surgery
- *Chest x-ray, bone scan,* and *liver function studies* to identify potential metastases.

Radical nephrectomy is the treatment of choice for kidney tumors. In a radical nephrectomy, the adrenal gland, upper ureter, fat and fascia surrounding the kidney, as well as the entire kidney, are removed. Regional lymph nodes may also be resected. Although nephrectomy can be done using a laparoscopic approach, laparotomy primarily is used for radi-

MANIFESTATIONS of Renal Tumors

- Microscopic or gross hematuria
- Flank pain
- Palpable abdominal mass
- Fever
- Fatigue
- Weight loss
- Anemia or polycythemia

TABLE 29–4 **Renal Cell Cancer Staging**

STAGE	EXTENT OF TUMOR	PROGNOSIS
I	Confined to the kidney capsule	66% 5-year survival
II	Invasion through the capsule but confined to local fascia	64% 5-year survival
III	Regional lymph node, ipsilateral renal vein, or inferior vena cava involvement	42% 5-year survival
IV	Locally invasive or distant metastases	11% or less 5-year survival

Source: Adapted from *Harrison's Principles of Internal Medicine* (16th ed.) by D. L. Kasper et al. (Eds.), 2005, New York: McGraw-Hill.

cal nephrectomy. Nursing care for the client having a nephrectomy is summarized in the box below.

No effective treatment is available for advanced renal carcinoma with metastases. Biologic therapies such as interferon or interleukin-2 have been used, but rarely achieve a durable effect. No chemotherapy drug consistently causes tumor regression in more than 20% of clients (Kasper et al., 2005).

NURSING CARE

Nursing Diagnoses and Interventions

Nursing care for the client with renal cancer focuses on needs related to the cancer diagnosis and to the surgical intervention. Postoperative pain may be significant and the risk for respiratory complications is high. The remaining kidney must be protected from damage to preserve renal function. Psychologically, the client may grieve the loss of a major organ and the diagnosis of cancer.

Pain

The size and location of the incision used for a radical nephrectomy (Figure 29–4 ■) make pain management a challenge. Intercostal blocks, patient-controlled analgesia (PCA), or routine analgesic administration can effectively relieve the discomfort. Nursing care focuses on assessing pain relief, providing supportive measures to enhance analgesia, and ensuring that pain or the fear of pain does not lead to respiratory complications.

- Assess frequently for adequate pain relief. Use a standard pain scale and nonverbal signs such as grimacing, tense body position, apparent dozing, elevated pulse, change of blood pressure, or rapid, shallow respirations. Notify the physician of inadequate pain relief. *The client may assume that pain is to be expected or may fear becoming addicted to analgesics. Careful questioning and assessment allow effective pain management. Responses to analgesics are individual, and the prescribed dose may need to be adjusted.*
- Assess the incision for inflammation or swelling and drainage catheters and tubes for patency. *An obstructed catheter can lead to hydronephrosis, hematoma, or abscess, increasing incisional pain.*

PRACTICE ALERT

Assess for abdominal distention, tenderness, and bowel sounds. Intra-abdominal bleeding, peritonitis, or paralytic ileus can cause pain that may be confused with incisional pain.

- Use adjunctive pain relief measures such as positioning, diversional activities, management of environmental stimuli,

NURSING CARE OF THE CLIENT HAVING A Nephrectomy

PREOPERATIVE CARE

- Provide routine preoperative care as outlined in Chapter 4 ∞.
- Report abnormal laboratory values to the surgeon. *Bacteriuria, blood coagulation abnormalities, or other significant abnormal values may affect surgery and postoperative care.*
- Discuss operative and postoperative expectations as indicated, including the location of the incision (Figure 29–4) and anticipated tubes, stents, and drains. *Preoperative teaching about postoperative expectations reduces anxiety for the client and family during the early postoperative period.*

POSTOPERATIVE CARE

- Provide routine postoperative care as described in Chapter 4 ∞.
- Frequently assess urine color, amount, and character, noting any hematuria, pyuria, or sediment. Promptly report oliguria or anuria, as well as changes in urine color or clarity. *Preserving function of the remaining kidney is critical; frequent assessment allows early intervention for potential problems.*
- Note the placement, status, and drainage from ureteral catheters, stents, nephrostomy tubes, or drains. Label each clearly. Maintain gravity drainage; irrigate only as ordered. *Maintaining drainage tube patency is vital to prevent potential hydronephrosis. Bright bleeding or unexpected drainage may indicate a surgical complication.*
- Support the grieving process and adjustment to the loss of a kidney. *Loss of a major organ leads to a body image change and grief response. When renal cancer is the underlying diagnosis, the client may also grieve the loss of health and potential loss of life.*
- Provide the following home care instructions for the client and family:
  a. The importance of protecting the remaining kidney by preventing UTI, renal calculi, and trauma. See Chapter 28 ∞ for measures to prevent UTI and calculi. *Damage to the remaining kidney by UTI, renal calculi, or trauma can lead to renal failure.*
  b. Maintain a fluid intake of 2000 to 2500 mL per day. *This important measure helps prevent dehydration and maintain good urine flow.*
  c. Gradually increase exercise to tolerance, avoiding heavy lifting for a year after surgery. Participation in contact sports is not recommended to reduce the risk of injury to the remaining kidney. *Lifting is avoided to allow full tissue healing. Trauma to the remaining kidney could seriously jeopardize renal function.*
  d. Care of the incision and any remaining drainage tubes, catheters, or stents. *This routine postoperative instruction is vital to prepare the client for self-care and prevent complications.*
  e. Report signs and symptoms to the physician, including manifestations of UTI (dysuria, frequency, urgency, nocturia, cloudy, malodorous urine) or systemic infection (fever, general malaise, fatigue), redness, swelling, pain, or drainage from the incision or any catheter or drain tube site. *Prompt treatment of postoperative infection is vital to allow continued healing and prevent compromise of the remaining kidney.*

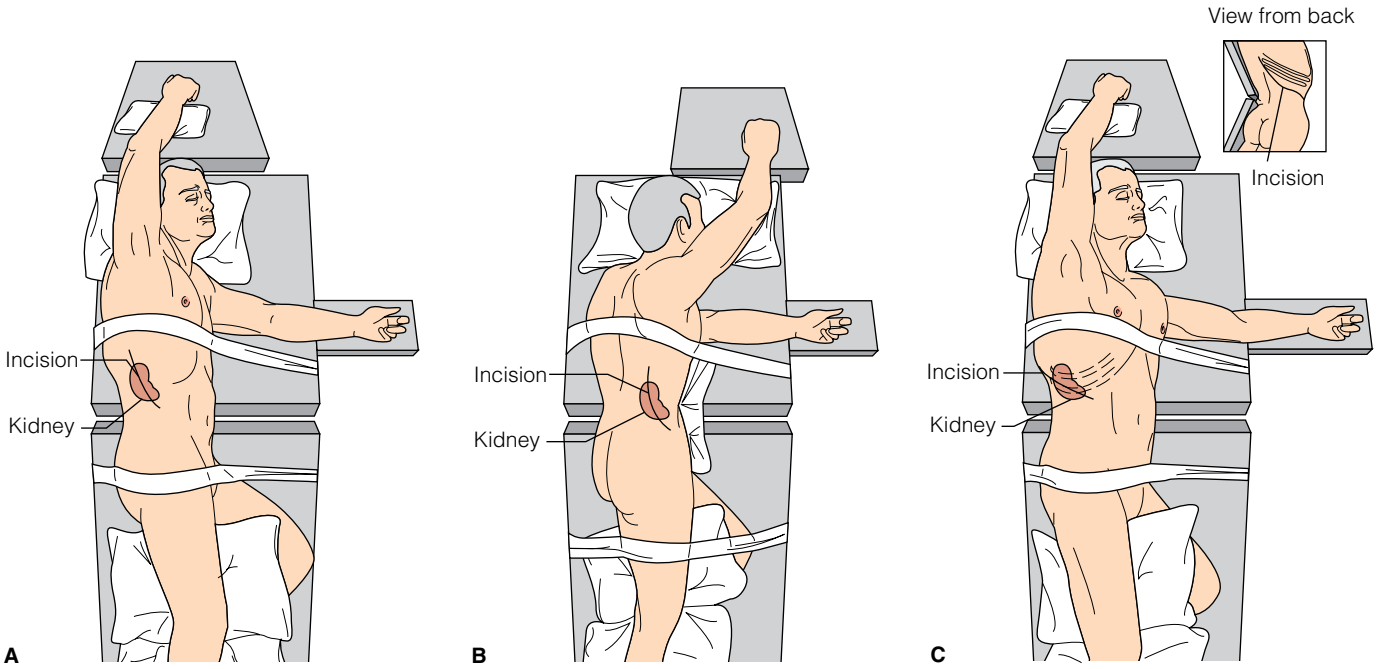


Figure 29–4 ■ Incisions used for kidney surgery: *A,* flank; *B,* lumbar; and *C,* thoracoabdominal.

guided imagery, and relaxation techniques. *These can enhance the effects of analgesia.*

Ineffective Breathing Pattern

The location of the incision combined with the respiratory depressant effects of narcotic analgesics increases the risk for respiratory complications in the client who has had a nephrectomy.

- Position to promote respiratory excursion, using semi-Fowler's position and side-lying positions as allowed and tolerated. *Lung expansion is improved in semi-Fowler's and Fowler's positions.*

PRACTICE ALERT

Assess respiratory status frequently, including rate and depth, cough, breath sounds, oxygen saturation, and temperature. Pneumothorax on the operative side is common. Early identification and intervention can prevent major respiratory complications.

- Change position frequently, ambulate as soon as possible. *These measures promote lung expansion and the movement of mucus out of airways.*
- Encourage frequent (every 1 to 2 hours) deep breathing, spirometer use, and coughing. Assist to splint the incision. *These measures promote alveolar ventilation, gas exchange, and airway clearance.*

Risk for Impaired Urinary Elimination

Surgery involving the urinary tract increases the risk for altered renal function and urine elimination. In addition, removal of one kidney dictates extra caution to maintain renal circulation, a sterile urinary tract, and free urine flow.

- Monitor vital signs, CVP, and urine output every 1 to 2 hours initially, then every 4 hours. *Hypovolemia due to hemorrhage, diuresis, or fluid sequestering (third spacing) reduces blood flow to the kidney and increases the risk of renal ischemia with possible acute tubular necrosis and acute renal failure.*
- Frequently assess the amount and nature of drainage on surgical dressings and from drainage tubes, stents, and catheters. Measure and record output from each drain or catheter separately. *Frequent and accurate assessment of drainage helps to identify excess bleeding, abnormal fluid loss, infection, or other potential surgical complications.*

PRACTICE ALERT

Prevent kinking, twisting, or tension on drains and tubes. Do not clamp. Irrigate carefully and only with a physician's order. Notify the physician immediately if any tube becomes dislodged. It is vital to maintain the patency of drains, particularly any affecting the remaining kidney, to prevent the excess pressure of hydronephrosis.

- Maintain fluid intake with intravenous fluids until oral intake is resumed. Encourage an intake of 2000 to 2500 mL per day as soon as the client tolerates oral liquids. *A liberal fluid intake prevents dehydration, helps to dilute any nephrotoxic substances, and promotes good urinary output.*
- Use strict aseptic technique in caring for all urinary catheters, tubes, stents, drains, and incisions. *Asepsis is vital to prevent infection and possible compromise of the remaining kidney.*
- Following catheter removal, assess frequently for urinary retention. Notify the physician if the client is unable to void within 4 to 6 hours or if manifestations of retention (distended bladder, discomfort, urinary dribbling) develop. *Maintenance of urine output is vital to prevent stasis and possible complications such as infection and hydronephrosis.*
- Monitor laboratory results, including urinalysis, BUN, serum creatinine, and serum electrolytes. Report abnormal findings

to the physician. *Abnormal values may indicate early acute renal failure; prompt intervention is necessary to preserve renal function.*

Anticipatory Grieving

The client having a radical nephrectomy for renal cancer not only loses a major organ but also has to adjust to the diagnosis of cancer. Although the prognosis for recovery may be good, many people perceive cancer as always fatal. Providing support for the client and family during the initial stages of grieving can improve physical recovery, psychologic coping, and eventual adaptation.

- Work to develop a trusting relationship with the client and family. *Trust increases the nurse's effectiveness in helping them work through the process of grieving.*
- Listen actively, encouraging the client and family to express fears and concerns. *As they begin to express their concerns, client and family can begin to deal more effectively with them.*
- Assist the client and family to identify strengths, past experiences, and support systems. *These resources can be employed in working through the grieving process.*
- Demonstrate respect for cultural, spiritual, and religious values and beliefs; encourage use of these resources to cope with losses. *Value and belief systems can provide a structure and form for dealing with the grieving process.*
- Encourage discussion of the potential impact of loss on the client and the family structure and function. Assist family members to share concerns with one another. *Sharing of fears and concerns among family members promotes involvement and support of the entire family unit so that the individual is not left to cope alone.*
- Refer to cancer support groups, social services, or counseling as appropriate. *Support groups and counseling services provide additional resources for coping.*

Community-Based Care

If renal cancer was detected at an early stage and cure is anticipated, teaching for home care focuses on protecting the remaining kidney. Include the following measures to prevent infection, renal calculi, hydronephrosis, and trauma:

- Maintain a fluid intake of 2000 to 2500 mL per day, increasing the amount during hot weather or strenuous exercise.
- Urinate when the urge is perceived, and before and after sexual intercourse.
- Properly clean the perineal area.
- Watch for manifestations of UTI, and understand the importance of early and appropriate evaluation and intervention.
- If the client is an older adult male, he should watch for manifestations of prostatic hypertrophy, a major cause of urinary tract obstruction. Stress the importance of routine screening examinations.
- Avoid contact sports such as football or hockey; use measures to prevent motor vehicle accidents and falls, which could damage the kidney.

RENAL FAILURE

Renal failure is a condition in which the kidneys are unable to remove accumulated metabolites from the blood, leading to altered fluid, electrolyte, and acid–base balance. The cause may be a primary kidney disorder, or renal failure may be secondary to a systemic disease or other urologic defects. Renal failure may be either acute or chronic. **Acute renal failure** has an abrupt onset and with prompt intervention is often reversible. **Chronic renal failure** is a silent disease, developing slowly and insidiously, with few symptoms until the kidneys are severely damaged and unable to meet the excretory needs of the body. Both forms of renal failure are characterized by azotemia, increased levels of nitrogenous wastes in the blood.

FAST FACTS

- Acute renal failure has an abrupt onset and often is reversible with prompt treatment.
- Chronic renal failure is the end stage of long-term kidney disease. It is irreversible; renal replacement therapies (transplant or dialysis) are necessary to sustain life.
- Both acute and chronic renal failure are characterized by azotemia, accumulation of nitrogenous (protein) waste products in the blood.

Renal failure is common and costly. In 2001, more than 93,000 new clients began receiving treatment for **end-stage renal disease (ESRD)**. Annually, nearly 287,500 clients with ESRD undergo dialysis, about 15,300 have kidney transplants, and another 59,000 are awaiting kidney transplants. The annual cost of ESRD treatment (in 2001 dollars) is $22.8 billion. The cost is also measured in lives and lifestyle. The 5-year survival rate for clients undergoing dialysis is 31.9% (NKUDIC, 2004). Although many clients report satisfaction with their quality of life, often clients on dialysis are unable to work, and the family structure may disintegrate under the strain of treatment.

THE CLIENT WITH ACUTE RENAL FAILURE

Acute renal failure (ARF) is a rapid decline in renal function with azotemia and fluid and electrolyte imbalances. The most common causes of acute renal failure are ischemia and nephrotoxins. The kidney is particularly vulnerable to both because of the amount of blood that passes through it. A fall in blood pressure or volume can cause ischemia of kidney tissues. Nephrotoxins in the blood damage renal tissue directly.

Incidence and Risk Factors

Approximately 5% of all hospitalized clients develop ARF; the incidence jumps to as much as 30% in critical and special care units (Kasper et al., 2005). The mortality rate for ARF in seriously ill clients is up to 75%. This high death rate is probably more related to the populations affected by ARF—

older clients and the critically ill—than to the disorder itself (Porth, 2005).

Major trauma or surgery, infection, hemorrhage, severe heart failure, severe liver disease, and lower urinary tract obstruction are risk factors for ARF. Drugs and radiologic contrast media that are toxic to the kidney (*nephrotoxic*) also increase the risk for ARF. Older adults develop ARF more frequently due to their higher incidence of serious illness, hypotension, major surgeries, diagnostic procedures, and treatment with nephrotoxic drugs. The older adult also may have some degree of preexisting renal insufficiency associated with aging.

Physiology Review

The functional unit of the kidneys, the nephron (see Figure 27–3 ∞), produces urine through three processes: glomerular filtration, tubular reabsorption, and tubular secretion. In the *glomerulus,* a filtrate of water and small solutes is formed. The solute concentration of this filtrate is equal to that of plasma, with the exception of large molecules such as plasma proteins and blood cells. The GFR, the amount of filtrate formed per minute, is affected by blood volume and pressure, the autonomic nervous system, and other factors. From the glomerulus, the filtrate flows into the *tubules,* where its composition is changed by the processes of *tubular reabsorption* and *tubular secretion.* Most water and many filtered solutes such as electrolytes and glucose are reabsorbed. Metabolic waste products such as urea, hydrogen ion, ammonia, and some creatinine are secreted into the tubule for elimination. By the time urine exits the collecting duct into the renal pelvis, 99% of the filtrate has been reabsorbed.

Pathophysiology

The causes and pathophysiology of acute renal failure are commonly categorized as prerenal, intrinsic, and postrenal ARF. Prerenal ARF is the most common, accounting for about 55% of the total. In *prerenal ARF*, hypoperfusion leads to acute renal failure without directly affecting the integrity of kidney tissues. *Intrinsic* (or *intrarenal*) *ARF*, due to direct damage to functional kidney tissue, is responsible for another 40%. Urinary tract obstruction with resulting kidney damage is the precipitating factor for *postrenal ARF*, the least common form (~5%). Table 29–5 summarizes the causes of acute renal failure. See *Pathophysiology Illustrated: Acute Renal Failure* on the next page.

Prerenal ARF

Prerenal ARF results from conditions that affect renal blood flow and perfusion. Any disorder that significantly decreases vascular volume, cardiac output, or systemic vascular resistance can affect renal blood flow. The kidneys normally receive 20% to 25% of the cardiac output to maintain the GFR. A drop in renal blood flow to less than 20% of normal causes the GFR to fall. As the filtration of substances by the glomeruli is reduced, less reabsorption of substances in the tubule is required. As a result, kidney cells require less energy and oxygen, and their metabolism slows. Prerenal ARF is rapidly reversed when blood flow is restored, and the renal parenchyma remains undamaged. Continued ischemia can lead to tubular cell necrosis and significant nephron damage (Kasper et al., 2005; Porth, 2005). Intrinsic ARF due to ischemic injury may result.

FAST FACTS

- Prerenal ARF is common, particularly in trauma, surgical, and critically ill clients.
- Restoration of blood pressure and blood flow to the kidneys rapidly reverses prerenal ARF.
- If not promptly identified and treated, prerenal ARF leads to ischemic acute tubular necrosis and intrarenal or intrinsic ARF.

Postrenal ARF

Obstructive causes of acute renal failure are classified as postrenal. Any condition that prevents urine excretion can lead to postrenal ARF. Benign prostatic hypertrophy is the most

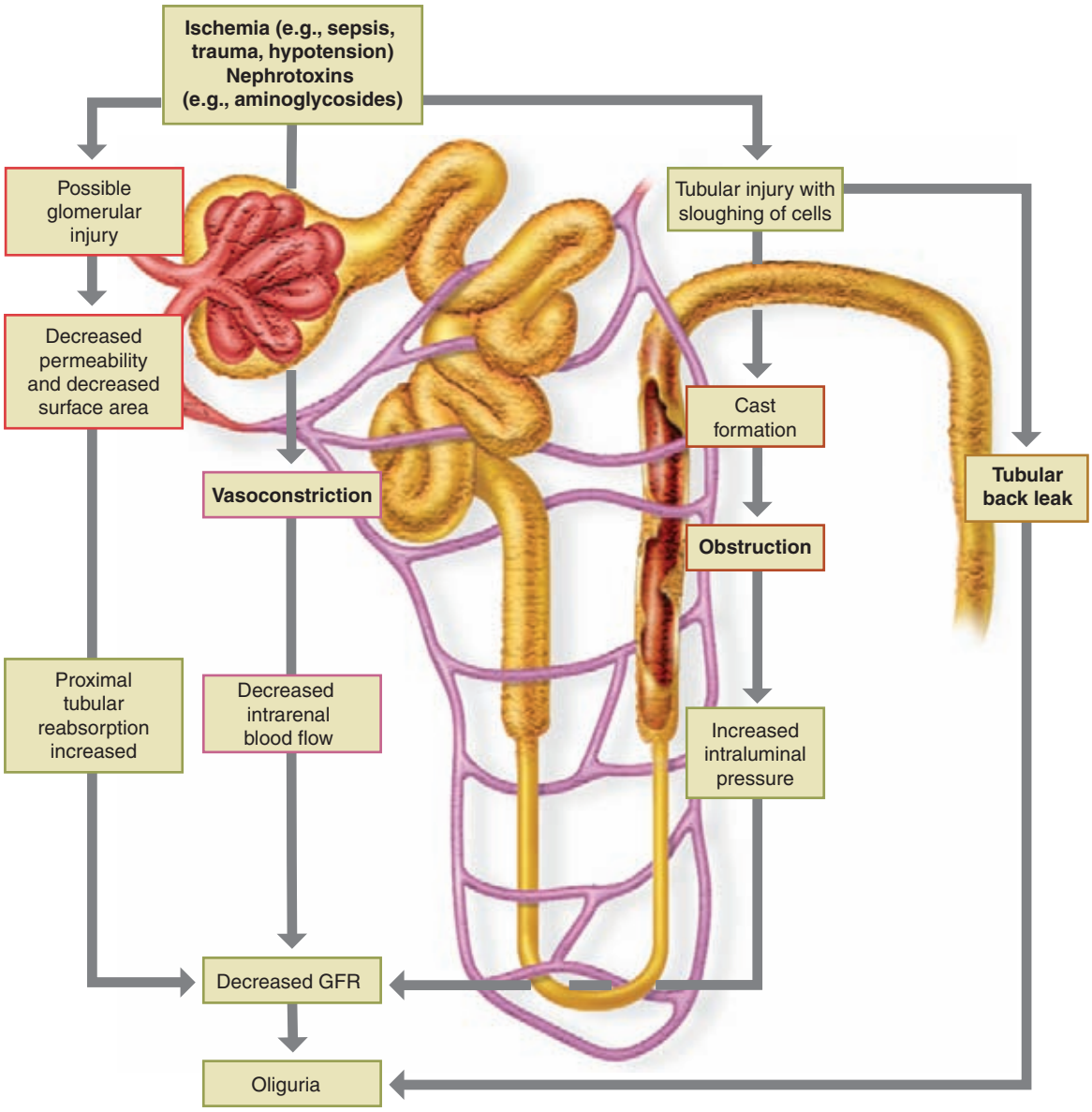
TABLE 29–5 **Causes of Acute Renal Failure**

	CAUSE	EXAMPLES
Prerenal	Hypovolemia	Hemorrhage, dehydration, excess fluid loss from GI tract, burns, wounds
	Low cardiac output	Heart failure, cardiogenic shock
	Altered vascular resistance	Sepsis, anaphylaxis, vasoactive drugs
Intrarenal	Glomerular/ microvascular injury	Glomerulonephritis, DIC, vasculitis, hypertension, toxemia of pregnancy, hemolytic uremic syndrome
	Acute tubular necrosis	Ischemia due to conditions associated with prerenal failure; toxins such as drugs, heavy metals; hemolysis, rhabdomyolysis (muscle cell breakdown)
	Interstitial nephritis	Acute pyelonephritis, toxins, metabolic imbalances, idiopathic
Postrenal	Ureteral obstruction	Calculi, cancer, external compression
	Urethral obstruction	Prostatic enlargement, calculi, cancer, stricture, blood clot

PATHOPHYSIOLOGY ILLUSTRATED

Acute Renal Failure

The initial kidney injury is usually associated with an acute condition such as sepsis, trauma, and hypotension, or the result of treatment for an acute condition with a nephrotoxic medication. Injury to the kidney can occur because of glomerular injury, vasoconstriction of capillaries, or tubular injury. All consequences of injury lead to decreased glomerular filtration and oliguria.



common precipitating factor. Others include renal or urinary tract calculi and tumors. See Chapter 28 ∞ for further discussion of urinary tract obstruction.

Intrinsic (Intrarenal) ARF

Intrinsic or intrarenal failure is characterized by acute damage to the renal parenchyma and nephrons. Intrarenal causes include diseases of the kidney itself and acute tubular necrosis, the most common intrarenal cause of ARF.

In acute glomerulonephritis, glomerular inflammation can reduce renal blood flow and cause ARF. Vascular disorders affecting the kidney, such as vasculitis (inflammation of the blood vessels), malignant hypertension, and arterial or venous occlusion, can damage nephrons sufficiently to result in acute renal failure.

ACUTE TUBULAR NECROSIS Nephrons are especially susceptible to injury from ischemia or exposure to nephrotoxins. **Acute tubular necrosis (ATN)**, destruction of tubular epithelial cells,

causes an abrupt and progressive decline of renal function. Prolonged ischemia is the primary cause of ATN. When ischemia and nephrotoxin exposure occur concurrently, the risk for ATN and tubular dysfunction is especially high. See Figure 29–5 ■ for the pathogenesis of acute renal failure due to ATN. Risk factors for ischemic ATN include major surgery, severe hypovolemia, sepsis, trauma, and burns. The impact of ischemia resulting from vasodilation and fluid loss in sepsis, trauma, and burns often is compounded by toxins released by bacteria or from damaged tissue.

Ischemia lasting more than 2 hours causes severe and irreversible damage to kidney tubules with patchy cellular necrosis and sloughing. The GFR is significantly reduced as a result of (1) ischemia, (2) activation of the renin–angiotensin system, and (3) tubular obstruction by cellular debris, which raises the pressure in the glomerular capsule.

Common nephrotoxins associated with ATN include the aminoglycoside antibiotics and radiologic contrast media. Many other drugs (e.g., NSAIDs and some chemotherapy drugs), heavy metals such as mercury and gold, and some common chemicals such as ethylene glycol (antifreeze) are also potentially toxic to the renal tubule. The risk for ATN is higher when nephrotoxic drugs are given to older clients or clients with preexisting renal insufficiency, and when used in combination with other nephrotoxins. Dehydration increases the risk by increasing the toxin concentration in nephrons.

Nephrotoxins destroy tubular cells by both direct and indirect effects. As tubular cells are damaged and lost through necrosis and sloughing, the tubule becomes more permeable. This increased permeability results in filtrate reabsorption, further reducing the ability of the nephron to eliminate wastes.

Rhabdomyolysis may account for 7% to 15% of all cases of ARF (Criddle, 2003; Russell, 2005). It is caused by release of excess myoglobin from injured skeletal muscles. Myoglobin is a protein that acts as the oxygen reservoir for muscle fibers, much as hemoglobin does for the blood. Muscle trauma, strenuous exercise, hyperthermia or hypothermia, drug overdose, infection, and other factors can precipitate rhabdomyolysis. The myoglobin clogs renal tubules causing ischemic injury, and contains an iron pigment that directly damages the tubules. *Hemolysis,* red blood cell destruction, releases hemoglobin into the circulation, with much the same effect as rhabdomyolysis.

Course and Manifestations

The course of acute renal failure due to ATN typically includes three phases: initiation, maintenance, and recovery.

Initiation Phase

The *initiation phase* may last hours to days. It begins with the initiating event (e.g., hemorrhage) and ends when tubular injury occurs. If ARF is recognized and the initiating event is effectively treated during this phase, the prognosis is good. The initiation phase of ARF has few manifestations; in fact, it is often identified only when manifestations of the maintenance phase develop.

Maintenance Phase

The *maintenance phase* of ARF is characterized by a significant fall in GFR and tubular necrosis. Oliguria may develop, although many clients continue to produce normal or near-normal amounts of urine (nonoliguric ARF). Even though urine may be produced, the kidney cannot efficiently eliminate metabolic wastes, water, electrolytes, and acids from the body during the maintenance phase of ARF. Azotemia, fluid retention, electrolyte imbalances, and metabolic acidosis develop. These abnormalities are more severe in the oliguric client than in the nonoliguric one, leading to a poorer prognosis with oliguria.

During the maintenance phase, salt and water retention cause edema, increasing the risk for heart failure and pulmonary edema. Impaired potassium excretion leads to hyperkalemia. When the serum potassium level is greater than 6.0 to 6.5 mEq/L, manifestations of its effect on neuromuscular function develop. These include muscle weakness, nausea and diarrhea, electrocardiographic changes, and possible cardiac arrest. Other electrolyte imbalances include hyperphosphatemia and hypocalcemia. Metabolic acidosis results from impaired hydrogen ion elimination by the kidneys.

Anemia develops after several days of ARF due to suppressed erythropoietin secretion by the kidneys. Immune function may be impaired, increasing the risk for infection. Other manifestations of the maintenance phase include:

- Edema and hypertension due to salt and water retention
- Confusion, disorientation, agitation or lethargy, hyperreflexia, and possible seizures or coma due to azotemia and electrolyte and acid–base imbalances
- Anorexia, nausea, vomiting, and decreased or absent bowel sounds
- Uremic syndrome if ARF is prolonged (see the section on chronic renal failure that follows).

Recovery Phase

The recovery phase of ARF is characterized by a process of tubule cell repair and regeneration and gradual return of the GFR to normal or pre-ARF levels. Diuresis may occur as the nephrons and GFR recover, and retained salt, water, and solutes are excreted. Serum creatinine, BUN, potassium, and phosphate levels remain high and may continue to rise in spite of increasing urine output. Renal function improves rapidly during the first 5 to 25 days of the recovery phase, and continues to improve for up to 1 year.

INTERDISCIPLINARY CARE

Preventing acute renal failure is a goal in caring for all clients, especially those in high-risk groups. Maintaining an adequate vascular volume, cardiac output, and blood pressure is vital to preserve kidney perfusion. Nephrotoxic drugs are avoided if possible. When a nephrotoxic drug or substance must be used, the risk of ARF can be reduced by us-

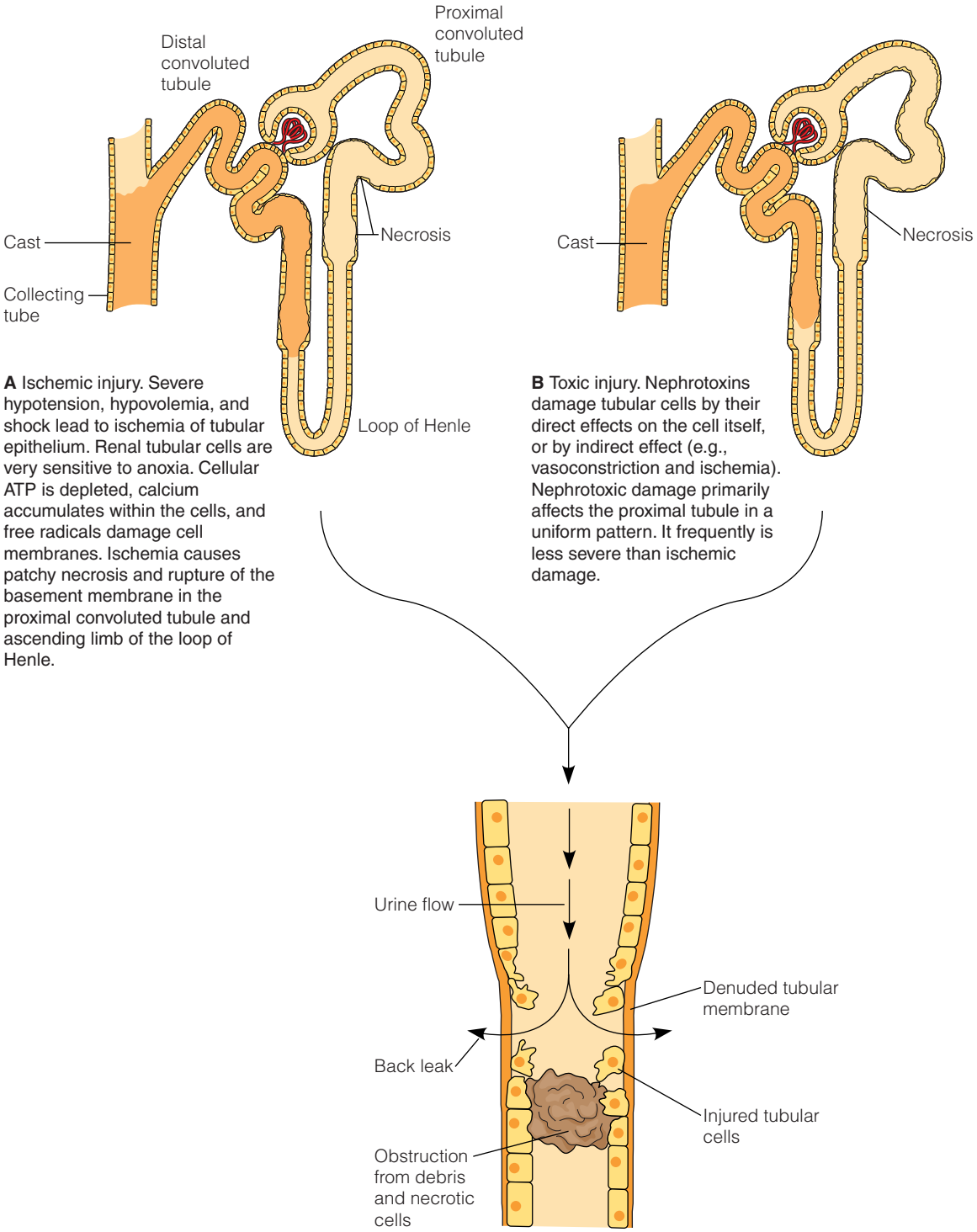


Figure 29–5 ■ Acute tubular necrosis. In ATN, tubular epithelial cells are destroyed by either ischemic or toxic injury.

ing the minimum effective dose, maintaining hydration, and eliminating other known nephrotoxins from the medication regimen.

Treatment goals for acute renal failure are to (1) identify and correct the underlying cause, (2) prevent additional kidney damage, (3) restore the urine output and kidney function, and

(4) compensate for renal impairment until kidney function is restored. Fluid and electrolyte balance is a key component in managing ARF.

The client's history and physical assessment can provide clues about the initiating event for ARF. Impaired perfusion for as few as 30 minutes may cause significant renal ischemia.

Diagnosis

Diagnostic tests are used to identify the cause of acute renal failure and monitor its effects on homeostasis:

- *Urinalysis* often shows the following abnormal findings in acute renal failure:
  a. A fixed specific gravity of 1.010 (equal to the specific gravity of plasma) because the tubules are unable to concentrate the filtrate
  b. Proteinuria if glomerular damage is the cause of ARF
  c. The presence of RBCs (due to glomerular dysfunction), WBCs (related to inflammation), and renal tubular epithelial cells (indicating ATN)
  d. Cell casts, which are protein and cellular debris molded in the shape of the tubular lumen (in ARF, RBCs, WBCs, and renal tubular epithelial casts may be present. Brownish pigmented casts and positive tests for occult blood indicate hemoglobinuria or myoglobinuria.)
- *Serum creatinine* and *BUN* are used to evaluate renal function. In ARF, serum creatinine levels increase rapidly, within 24 to 48 hours of the onset. Creatinine levels generally peak within 5 to 10 days. Creatinine and BUN levels tend to increase more slowly when urine output is maintained. The onset of recovery is marked by a halt in the rise of the serum creatinine and BUN.
- *Serum electrolytes* are monitored to evaluate the fluid and electrolyte status. The serum potassium rises at a moderate rate and is often used to indicate the need for dialysis. Hyponatremia is common, due to the water excess associated with ARF.
- *Arterial blood gases* often show a metabolic acidosis due to the kidneys' inability to adequately eliminate metabolic wastes and hydrogen ions (see Chapter 10 ∞).
- *CBC* shows reduced RBCs, moderate anemia, and a low hematocrit. ARF affects erythropoietin secretion and RBC production. Iron and folate absorption may also be impaired, further contributing to anemia.

Laboratory findings associated with kidney disease are summarized in Table 29–2.

- *Renal ultrasonography* is used to identify obstructive causes of renal failure, and to differentiate acute renal failure from end-stage chronic renal failure. In ARF, the kidneys may be enlarged, whereas they typically appear small and shrunken in chronic renal failure.
- *CT scan* also may be done to evaluate kidney size and identify possible obstructions.
- *IVP, retrograde pyelography,* or *antegrade pyelography* may also be used to evaluate kidney structure and function. Radiologic contrast media are used with extreme caution because of their potential nephrotoxicity. Retrograde pyelography, in which contrast dye is injected into the ureters, and antegrade pyelography, in which the contrast medium is injected percutaneously into the renal pelvis, are preferred because they have fewer nephrotoxic effects than IVP.
- *Renal biopsy* may be necessary to differentiate between acute and chronic renal failure.

See Chapter 27 ∞ for nursing implications of tests used to identify causes of renal failure.

Medications

The primary focus in drug management for acute renal failure is to restore and maintain renal perfusion and to eliminate drugs that are nephrotoxic from the treatment regimen.

Intravenous fluids and blood volume expanders are given as needed to restore renal perfusion. Dopamine (Intropin), administered in low doses by intravenous infusion, increases renal blood flow. Dopamine is a sympathetic neurotransmitter that improves cardiac output and dilates blood vessels of the mesentery and kidneys when given in low therapeutic doses.

If restoration of renal blood flow does not improve urinary output, a potent loop diuretic such as furosemide (Lasix) or an osmotic diuretic such as mannitol may be given with intravenous fluids. The purpose is twofold. First, if nephrotoxins are present, the combination of fluids and potent diuretics may, in effect, "wash out" the nephrons, reducing toxin concentration. Second, establishing urine output may prevent oliguria, and reduce the degree of azotemia and fluid and electrolyte imbalances. Furosemide also may be used to manage salt and water retention associated with ARF.

Aggressive hypertension management limits renal injury when ARF is associated with disorders such as toxemia and pregnancy-induced hypertension. ACE inhibitors or other antihypertensive medications are used to control arterial pressures.

All drugs that are either directly nephrotoxic or that may interfere with renal perfusion (such as potent vasoconstrictors) are discontinued. NSAIDs, nephrotoxic antibiotics, and other potentially harmful drugs are avoided throughout the course of acute renal failure.

The client in acute renal failure has an increased risk of gastrointestinal bleeding, probably related to the stress response and impaired platelet function. Regular doses of antacids, histamine H_2-receptor antagonists (e.g., famotidine or ranitidine), or a proton-pump inhibitor such as omeprazole (Prilosec) are often ordered to prevent GI hemorrhage.

Hyperkalemia may require active intervention as well as restricted potassium intake. Serum levels of greater than 6.5 mEq/L are treated to prevent cardiac effects of hyperkalemia. With significant hyperkalemia, calcium chloride, bicarbonate, and insulin and glucose may be given intravenously to reduce serum potassium levels by moving potassium into the cells. A potassium-binding exchange resin such as sodium polystyrene sulfonate (Kayexalate, SPS Suspension) may be given orally or by enema. This agent removes potassium from the body by exchanging sodium for potassium, primarily in the large intestine. When given orally, it is often combined with sorbitol to prevent constipation. Rectally, it is instilled as a retention enema, allowed to remain in the bowel for approximately 30 to 60 minutes, and then irrigated out using a tap-water enema.

Aluminum hydroxide (AlternaGEL, Amphojel, Nephrox), an antacid, is used to control hyperphosphatemia in renal failure. It binds with phosphates in the GI tract, which are then excreted in the feces.

Because many drugs are eliminated from the body by the kidney, drug dosages may need to be adjusted. Doses within the usual range can lead to potentially toxic blood levels, because their elimination is slowed and half-life prolonged. Nursing implications for medications commonly prescribed for the client in ARF are summarized in the Medication Administration box below.

Fluid Management

Once vascular volume and renal perfusion are restored, fluid intake is usually restricted. The allowed daily fluid intake is calculated by allowing 500 mL for insensible losses (respiration, perspiration, bowel losses) and adding the amount excreted as urine (or lost in vomitus) during the previous 24 hours. For example, if a client with ARF excretes 325 mL of urine in 24 hours, the client is allowed a fluid intake (including oral and intravenous fluids) of 825 mL for the next 24 hours. Fluid balance is carefully monitored, using accurate weight measurements and the serum sodium as the primary indicators.

MEDICATION ADMINISTRATION The Client with Acute Renal Failure

LOOP DIURETICS

Bumetanide (Bumex)
Ethacrynic acid (Edecrin)
Furosemide (Lasix)
Torsemide (Demadex)

The loop diuretics, named for their primary site of action in the loop of Henle, are *high-ceiling diuretics:* The response increases with increasing doses. These are highly effective diuretics used in early ARF to reestablish urine flow and convert oliguric renal failure to nonoliguric renal failure. Loop diuretics may be given with intravenous dopamine to promote renal blood flow. In ATN due to a nephrotoxin, loop diuretics are used to clear the toxin from the nephrons more rapidly. Loop diuretics cause potassium wasting, which is generally not a concern in ARF because renal failure impairs normal potassium elimination.

Nursing Responsibilities

- Assess weight and vital signs for baseline data.
- Monitor intake and output, daily weight (or more frequently as ordered), vital signs, skin turgor, and other indicators of fluid volume status frequently.
- Assess for orthostatic hypotension because these potent diuretics can lead to hypovolemia.
- Monitor laboratory results, especially serum electrolyte, glucose, BUN, and creatinine levels.
- Administer by mouth or, if ordered, by intravenous injection:
  a. Furosemide undiluted at a rate of no more than 20 mg per minute
  b. Ethacrynic acid 50 mg diluted with 50 mL of normal saline at a rate of no more than 10 mg per minute
  c. Bumetanide undiluted over at least 1 minute or diluted in lactated Ringer's solution, normal saline, or 5% dextrose in water for infusion
  d. Torsemide undiluted over at least 2 minutes.
- Assess response. Urine output typically increases within 10 minutes after intravenous administration.
- Monitor hearing and for complaints such as tinnitus. High doses of loop diuretics increase the risk of ototoxicity, especially with ethacrynic acid. These effects may be reversible if detected early and the drug is discontinued.
- Avoid administering concurrently with other ototoxic agents, such as aminoglycoside antibiotics and cisplatin.

Health Education for the Client and Family

- Unless contraindicated, maintain a fluid intake of 2 to 3 quarts per day.
- Rise slowly from lying or sitting positions, because a fall in blood pressure may cause light-headedness.
- Take in the morning and, if ordered twice a day, late afternoon to avoid sleep disturbance.
- Take with food or milk to prevent gastric distress.
- NSAIDs interfere with the effectiveness of loop diuretics and should be avoided.

OSMOTIC DIURETICS

Mannitol (Osmitrol, Isotol)
Urea (Ureaphil)

The osmotic diuretics act by increasing the osmotic draw in the blood and urine. In the blood, the effect is to pull extracellular water into the vascular system, increasing the GFR. These substances are then freely filtered in the glomerulus and increase the osmotic draw of the urine, inhibiting water reabsorption. The effect is to increase urine volume and flow. In addition, osmotic diuretics dilute waste products in the urine, decreasing the risk of renal damage due to excess concentrations.

Nursing Responsibilities

- Assess urine output. Osmotic diuretics are used in early renal failure to maintain urine output but are contraindicated in anuria. A test dose may be administered; urine output of 30 mL per hour following the test dose shows an adequate response.
- Do not give these diuretics to clients who have heart failure or who are severely dehydrated. They increase vascular volume and may worsen heart failure. These drugs are not effective unless extracellular volume is adequate.
- Administer mannitol intravenously, diluting before use if indicated. Check solution for crystallization. Dissolve crystals by warming the solution slightly. Infuse 15% to 25% mannitol solutions through a filter over 30 to 90 minutes.
- Administer urea intravenously, diluting in 100 mL of 5% or 10% dextrose in water for every 30 g of urea. Administer no faster than 4 mL per minute through a filter.
- Monitor vital signs, breath sounds, and urinary output.
- Discontinue the drug if signs of heart failure or pulmonary edema develop or if renal function continues to decline.

Health Education for the Client and Family

- Report shortness of breath, headache, chest pain, or dizziness immediately.

ELECTROLYTES AND ELECTROLYTE MODIFIERS

Calcium chloride
Calcium gluconate

MEDIALINK Furosemide Animation

(continued)

MEDICATION ADMINISTRATION **The Client with Acute Renal Failure (continued)**

Sodium bicarbonate
Sodium polystyrene sulfonate (Kayexalate)

Calcium chloride or gluconate and sodium bicarbonate are administered intravenously in the initial management of hyperkalemia. Calcium is also administered to correct hypocalcemia and reduce hyperphosphatemia (calcium and phosphate have a reciprocal relationship in the body; as the level of one rises, the level of the other falls). Sodium bicarbonate helps correct acidosis and move potassium back into the intracellular space. Sodium polystyrene sulfonate is not used to replace an electrolyte, but to remove excess potassium from the body by exchanging sodium for potassium in the large intestine.

Nursing Responsibilities

- Assess serum electrolyte levels prior to and during therapy. Report rapid shifts or adverse responses to the physician.
- Administer as appropriate:
  a. Intravenous calcium chloride at less than 1 mL per minute; intravenous calcium gluconate at 0.5 mL per minute. Inject into a large vein through a small-bore needle; avoid infiltration because extravasation of intravenous solution will cause tissue necrosis.
  b. Intravenous sodium bicarbonate infusion over 4 to 8 hours; oral tablets as prescribed.
  c. Sodium polystyrene sulfonate as an oral solution mixed with sorbitol to prevent constipation, or as a retention enema mixed with warm water. Leave in the bowel for 30 to 60 minutes, irrigate using a small tap-water enema.
- Monitor for adverse reactions, such as dysrhythmias, electrolyte imbalances, and metabolic alkalosis.

Health Education for the Client and Family

- Intravenous calcium may make you light-headed; remain in bed for at least 30 minutes after administration.
- Chew sodium bicarbonate tablets and follow with 8 ounces of water. Do not take with milk.
- Retain the sodium polystyrene sulfonate enema as long as possible.

Nutrition

Renal insufficiency and the underlying disease process increase the rate of *catabolism* (the breakdown of body proteins) and decrease the rate of *anabolism* (body tissue repair). The client with ARF needs adequate nutrients and calories to prevent catabolism. Proteins are limited to 0.6 g per kilogram of body weight per day to minimize the degree of azotemia. Dietary proteins should be of high biologic value (rich in essential amino acids). Carbohydrates are increased to maintain adequate calorie intake and provide a protein-sparing effect.

Parenteral nutrition providing amino acids, concentrated carbohydrates, and fats may be instituted when the client cannot consume an adequate diet (e.g., due to nausea, vomiting, or underlying critical illness). The disadvantages of parenteral nutrition in the client with ARF are the high volume of fluid required and the risk for infection through the venous line.

Renal Replacement Therapy: Dialysis

Manifestations of **uremia**, organ dysfunction due to accumulated metabolic wastes, severe fluid overload, hyperkalemia, or metabolic acidosis in a client with renal failure, indicate a need to replace renal function. **Dialysis** is the diffusion of solute molecules across a semipermeable membrane from an area of higher solute concentration to one of lower concentration. It is used to remove excess fluid and metabolic waste products in renal failure. Early use of dialysis can reduce the rate of complications. Dialysis may also be used to rapidly remove nephrotoxins in acute tubular necrosis. While dialysis compensates for lost renal elimination functions, it does not replace lost erythropoietin production. Anemia is a continuing problem for the client receiving dialysis.

In dialysis, blood is separated from a dialysis solution (**dialysate**) by a semipermeable membrane. Either **hemodialysis**, a procedure in which blood passes through a semipermeable membrane filter outside the body, or **peritoneal dialysis**, which uses the peritoneum surrounding the abdominal cavity as the dialyzing membrane, may be used for the client with ARF. **Continuous renal replacement therapy (CRRT)**, in which blood is continuously circulated through a highly porous hemofilter from artery to vein or vein to vein, is a newer form of dialysis that may be used to treat ARF.

HEMODIALYSIS Hemodialysis uses the principles of diffusion and ultrafiltration to remove electrolytes, waste products, and excess water from the body. Blood is taken from the client via a vascular access and pumped to the dialyzer (Figure 29–6 ■). The porous membranes of the dialyzer unit allow small molecules such as water, glucose, and electrolytes to pass through, but block larger molecules such as serum proteins and blood cells. The dialysate, a solution of approximately the same composition and temperature as normal extracellular fluid, passes along the other side of the membrane. Small solute molecules move freely across the membrane by diffusion. The direction of movement for any substance is determined by the concentrations of that substance in the blood and the dialysate. Electrolytes and waste products such as urea and creatinine diffuse from the blood into the dialysate. If it is necessary to add something to the blood, such as calcium to replace depleted stores, it can be added to the dialysate to diffuse into the blood. Excess water is removed by creating a higher hydrostatic pressure of the blood moving through the dialyzer than of the dialysate, which flows in the opposite direction. This process is known as **ultrafiltration**.

Initially, clients with ARF typically undergo daily hemodialysis, then three to four sessions per week as indicated. Hemodialysis is not used if the client is hemodynamically unstable (e.g., with hypotension or low cardiac output). Following are complications associated with hemodialysis:

- Hypotension, the most frequent complication during hemodialysis, is related to changes in serum osmolality, rapid removal of fluid from the vascular compartment, vasodilation, and other factors.
- Bleeding is related to altered platelet function associated with uremia and the use of heparin during dialysis.

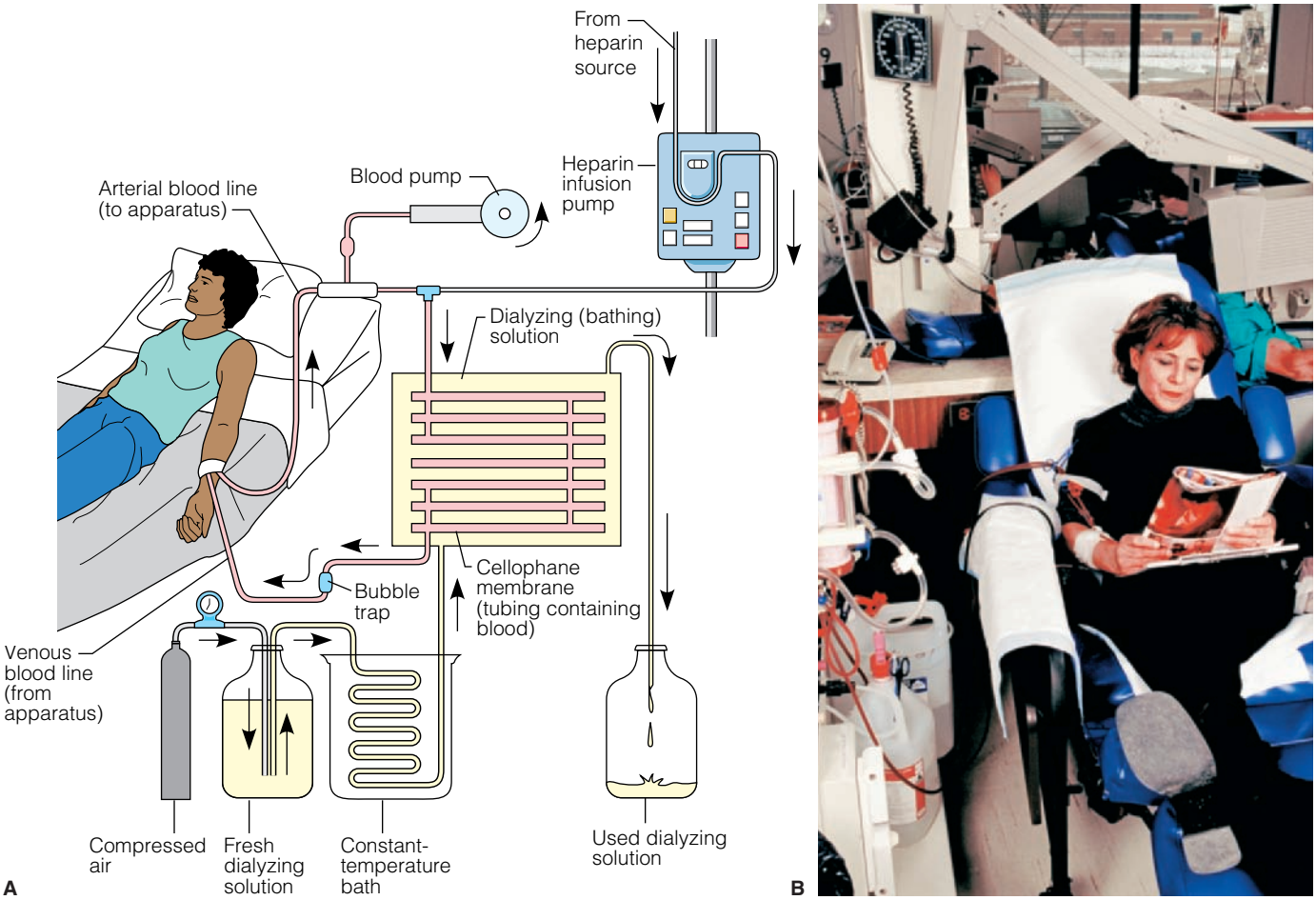


Figure 29–6 ■ *A,* The components of a hemodialysis system. *B*, A woman receiving kidney dialysis.

■ Infection (local or systemic) is related to WBC damage and immune system suppression. *Staphylococcus aureus* septicemia is commonly associated with contamination of the vascular access site. Clients on chronic hemodialysis have higher rates of hepatitis B, hepatitis C, cytomegalovirus, and HIV infection than the general population.

See the box on the next page for nursing care for the client undergoing hemodialysis.

CONTINUOUS RENAL REPLACEMENT THERAPY Clients with acute renal failure may be unable to tolerate hemodialysis and rapid fluid removal if their cardiovascular status is unstable (e.g., due to trauma, major surgery, heart failure). Continuous renal replacement therapy, which allows more gradual fluid and solute removal, often is used for these clients. In CRRT, blood is continuously circulated from an artery to a vein or a vein to a vein through a highly porous hemofilter for a period of 12 or more hours. Excess water and solutes such as electrolytes, urea, creatinine, uric acid, and glucose drain into a collection device. Fluid may be replaced with normal saline or a balanced electrolyte solution as needed during CRRT. This slower process helps maintain hemodynamic stability and avoid complications associated with rapid changes in ECF composition. The most common CRRT techniques are outlined in Table 29–6.

TABLE 29–6 **Continuous Renal Replacement Therapies**

TYPE	INDICATIONS	DESCRIPTION
Continuous arteriovenous hemofiltration (CAVH)	Remove fluid and some solutes	Arterial blood circulates through a hemofilter, then returns to client through venous line; ultrafiltrate collects in a drainage bag.
Continuous arteriovenous hemodialysis (CAVHD)	Remove fluid and waste products	Arterial blood circulates through a hemofilter surrounded by dialysate, then returns to client through venous line; ultrafiltrate collects in a drainage bag.
Continuous venovenous hemodialysis (CVVHD)	Remove fluid and waste products	Venous blood circulates through a hemofilter surrounded by dialysate, then returns to client through double-lumen venous catheter; ultrafiltrate collects in a drainage bag.

NURSING CARE OF THE CLIENT UNDERGOING Hemodialysis

PREDIALYSIS CARE

- Assess vital signs, including orthostatic blood pressures (lying, sitting, and standing), apical pulse, respirations, and lung sounds. *These data provide baseline information to help evaluate the effects of hemodialysis. Hypertension may indicate excess fluid volume. The client who is hypotensive may not tolerate rapid fluid volume changes during dialysis. Abnormal heart sounds (e.g., a gallop or murmur) and changes in heart rate or rhythm may indicate excess fluid volume or electrolyte imbalance. Fluid overload may also cause dyspnea, tachypnea, and rales or crackles in the lungs.*
- Record weight. *Weight changes are an effective indicator of fluid volume.*
- Assess vascular access site for a palpable pulsation or vibration and an audible bruit and for inflammation. *Infection and thrombus formation are the most common problems affecting the access site in hemodialysis clients.*
- Alert all personnel to avoid using the extremity with the vascular access site (or the nondominant arm, if long-term access has not been established) for blood pressures or venipuncture. *These procedures may damage vessels and lead to failure of the arteriovenous fistula.*

POSTDIALYSIS CARE

- Assess and document vital signs, weight, and vascular access site condition. *Rapid fluid and solute removal during dialysis may lead to orthostatic hypotension, cardiopulmonary changes, and weight loss.*
- Monitor BUN, serum creatinine, serum electrolyte, and hematocrit levels between dialysis treatments. *These values help determine the effectiveness of the treatment, the need for fluid and diet restrictions, and the timing of future dialysis sessions. The anemia associated with renal failure does not improve with dialysis, and iron and folate supplements or periodic blood transfusions may be needed.*
- Assess for dialysis disequilibrium syndrome, with headache, nausea and vomiting, altered level of consciousness; and hypertension. *Rapid changes in BUN, pH, and electrolyte levels during dialysis may lead to cerebral edema and increased intracranial pressure.*
- Assess for other adverse responses to dialysis, such as dehydration, nausea and vomiting, muscle cramps, or seizure activity. Treat as ordered. *Excess fluid removal and rapid changes in electrolyte balance can cause fluid deficit, nausea, vomiting, and seizure activity.*
- Assess for bleeding at the access site or elsewhere. Use standard precautions at all times. *Renal failure and heparinization during dialysis increase the risk for bleeding. Frequent exposure to blood and blood products increase the risk for hepatitis B or C or other bloodborne diseases.*
- If a transfusion is given during dialysis, monitor for possible transfusion reaction (e.g., chills and fever; dyspnea; chest, back, or arm pain; and urticaria or itching). *Clients in renal failure may receive multiple transfusions, increasing the risk of transfusion reaction. Close monitoring during and after the transfusion is important to identify early signs of a reaction.*
- Provide psychologic support and listen actively. Address concerns and accept responses such as anger, depression, and noncompliance. Reinforce client and family strengths in coping with renal failure and hemodialysis. *Grieving is a normal response to loss of organ function. The client may feel hopeless or helpless and resent dependence on a machine. The nurse can help the client and family work through these responses and focus on positive aspects of living.*
- Refer to social services and counseling as indicated. *Clients with renal failure may need additional support services to help them adapt to and live with their disease.*

CRRT is typically performed in an intensive care unit or specialized nephrology unit. Both arterial and venous lines are required for some types of CRRT (Figure 29–7 ■); for others, a double-lumen venous catheter is used. Strict aseptic technique is vital in caring for vascular access sites to reduce the risk of infection.

VASCULAR ACCESS Acute or temporary vascular access for hemodialysis or CRRT usually is gained by inserting a double-lumen catheter into the subclavian, jugular, or femoral vein. The double-lumen catheter has a central partition separating the blood withdrawal side of the catheter from the return side. Blood is drawn into the catheter through small openings in the proximal portion of the catheter, and returned to the circulation through an opening in the distal end of the catheter to avoid withdrawing the blood that has just been dialyzed.

For longer term vascular access, an *arteriovenous (AV) fistula* (Figure 29–8 ■) is created. In preparation for fistula formation, the nondominant arm is not used for venipuncture or blood pressure measurement during renal failure. The fistula is created by surgical anastomosis of an artery and vein, usually the radial artery and cephalic vein. It takes about a month for the fistula to mature so that it can be used for taking and replacing blood during dialysis. A functional AV fistula has a palpable pulsation and a bruit on auscultation. Venipunctures and blood pressures are avoided on the arm with the fistula.

In chronic renal failure, an *arteriovenous graft* is most often used for vascular access. The graft, a tube made of Gortex, is surgically implanted and connects the artery and the vein. Blood flows through the graft from the artery to the vein. Occasionally, an *external AV shunt* connecting a peripheral artery with a peripheral vein is used for vascular access.

The rate of complications and mortality associated with catheter access is higher than with AV fistulas or grafts. Ideally, an AV fistula or graft is created as soon as the potential need for long-term renal replacement therapies is identified (Dinwiddie, 2004). Localized AV fistula, graft, or shunt problems can occur, however. Infection and clotting or thrombosis are the most common shunt problems. Aneurysms may also

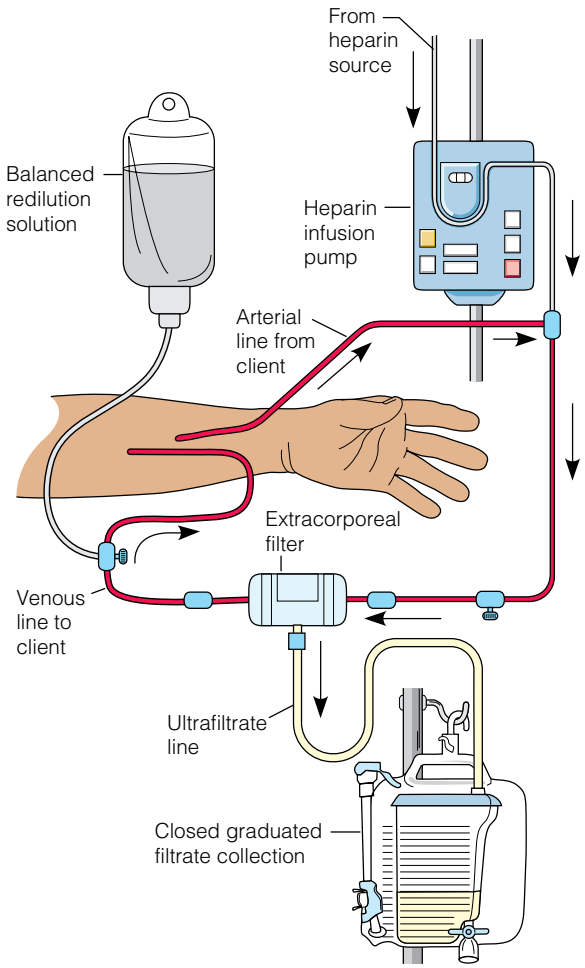


Figure 29–7 ■ Continuous arteriovenous hemofiltration (CAVH).

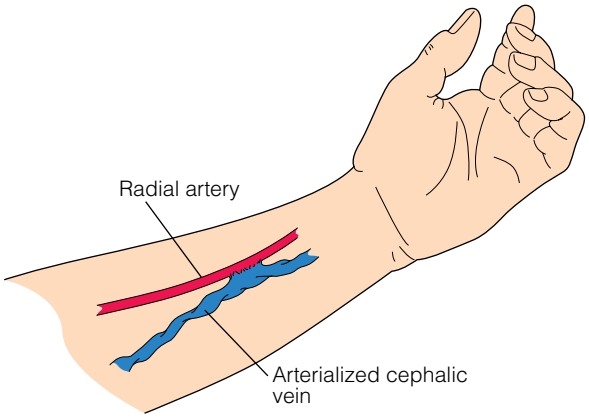


Figure 29–8 ■ An arteriovenous fistula.

develop. Both infection and thrombosis can lead to systemic manifestations such as septicemia and embolization. These local complications may cause the fistula or graft to fail, necessitating development of a new site. The psychologic impact of AV fistula or graft failure is significant, often causing depression and low self-esteem.

PERITONEAL DIALYSIS In peritoneal dialysis, the highly vascular peritoneal membrane serves as the dialyzing surface (Figure 29–9 ■). Warmed sterile dialysate is instilled into the peritoneal cavity through a catheter inserted into the peritoneal cavity. Metabolic waste products and excess electrolytes diffuse into the dialysate while it remains in the abdomen. Water movement is controlled using dextrose as an osmotic agent to draw it into the dialysate. The fluid is then drained by gravity out of the peritoneal cavity into a sterile bag. This process of

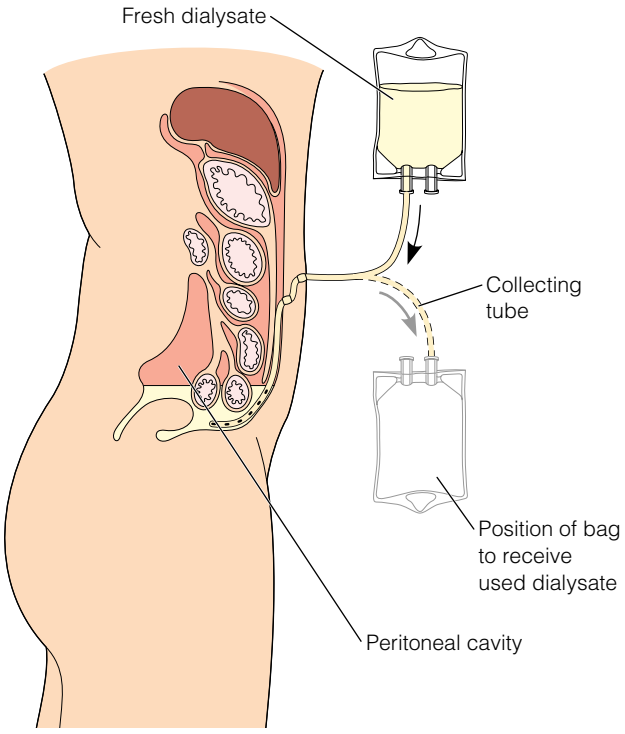


Figure 29–9 ■ *A,* Peritoneal dialysis. *B,* Woman receiving peritoneal dialysis.

dialysate infusion, dwell time of the solution in the abdomen, and drainage is repeated at prescribed intervals.

Because excess fluid and solutes are removed more gradually in peritoneal dialysis, it poses less risk for the unstable client; however, this slower rate of metabolite removal can be a disadvantage in ARF. Peritoneal dialysis increases the risk for developing peritonitis. It is contraindicated for clients who have had recent abdominal surgery, significant lung disease, or peritonitis. See the box below for nursing care for the client having peritoneal dialysis.

NURSING CARE

Health Promotion

Acute renal failure often can be prevented by measures that maintain fluid volume and cardiac output and reduce the risk of exposure to nephrotoxins. Carefully monitor critically ill, postoperative, and other at-risk clients for early signs of hypovolemia (low urine output, altered mental status, changes in vital signs, skin color or temperature). Promptly report a fall in

NURSING CARE OF THE CLIENT UNDERGOING **Peritoneal Dialysis**

PREDIALYSIS CARE

- Document vital signs including temperature, orthostatic blood pressures (lying, sitting, and standing), apical pulse, respirations, and lung sounds. *These baseline data help assess fluid volume status and tolerance of the dialysis procedure. Hypertension, abnormal heart or lung sounds, or dyspnea may indicate excess fluid volume. Poor respiratory function may affect the ability to tolerate peritoneal dialysis. Temperature measurement is vital, because infection is the most common complication of peritoneal dialysis.*
- Weigh daily or between dialysis runs as indicated. *Weight is an accurate indicator of fluid volume status.*
- Note BUN, serum electrolyte, creatinine, pH, and hematocrit levels prior to peritoneal dialysis and periodically during the procedure. *These values are used to assess the efficacy of treatment.*
- Measure and record abdominal girth. *Increasing abdominal girth may indicate retained dialysate, excess fluid volume, or early peritonitis.*
- Maintain fluid and dietary restrictions as ordered. *Fluid and diet restrictions help reduce hypervolemia and control azotemia.*
- Have the client empty the bladder prior to catheter insertion. *Emptying the bladder reduces the risk of inadvertent puncture.*
- Warm the prescribed dialysate solution to body temperature (98.6°F or 37°C) using a warm water bath or heating pad on low setting. *Dialysate is warmed to prevent hypothermia.*
- Explain all procedures and expected sensations. *Knowledge helps reduce anxiety and elicit cooperation.*

INTRADIALYSIS CARE

- Use strict aseptic technique during the dialysis procedure and when caring for the peritoneal catheter. *Peritonitis is a common complication of peritoneal dialysis; sterile technique reduces the risk.*
- Add prescribed medications to the dialysate; prime the tubing with solution and connect it to the peritoneal catheter, taping connections securely and avoiding kinks. *This allows dialysate to flow freely into the abdominal cavity and prevents leaking or contamination.*
- Instill dialysate into the abdominal cavity over a period of approximately 10 minutes. Clamp tubing and allow the dialysate to remain in the abdomen for the prescribed dwell time. Keep drainage tubing clamped at all times during instillation and dwell time. *Dialysate should flow freely into the abdomen if the peritoneal catheter is patent. Dialysis, the exchange of solutes and water between the blood and dialysate, occurs across the peritoneal membrane during the dwell time.*
- During instillation and dwell time, observe closely for signs of respiratory distress, such as dyspnea, tachypnea, or crackles. Place in Fowler's or semi-Fowler's position and slow the rate of instillation slightly to relieve respiratory distress if it develops. *Respiratory compromise may result from overly rapid filling or overfilling of the abdomen or from a diaphragmatic defect that allows fluid to enter the thoracic cavity.*
- After prescribed dwell time, open drainage tubing clamps and allow dialysate to drain by gravity into a sterile container. Note the clarity, color, and odor of returned dialysate. *Blood or feces in the dialysate may indicate organ or bowel perforation; cloudy or malodorous dialysate may indicate an infection.*
- Accurately record amount and type of dialysate instilled (including any added medications), dwell time, and amount and character of the drainage. *When more dialysate drains than has been instilled, excess fluid has been lost (output). If less dialysate is returned than has been instilled, a fluid gain has occurred (intake).*
- Monitor BUN, serum electrolyte, and creatinine levels. *These values are used to assess the effectiveness of dialysis.*
- Troubleshoot for possible problems during dialysis:
  a. Slow dialysate instillation. Increase the height of the container and reposition the client. Check tubing and catheter for kinks. Check abdominal dressing for wetness, indicating leakage around the catheter. *Slow dialysate flow may be related to a partially obstructed tube or catheter.*
  b. Excess dwell time. *Prolonged dwell time may lead to water depletion or hyperglycemia.*
  c. Poor dialysate drainage. Lower the drainage container, reposition, check for tubing kinks. Check abdominal dressing. *Tubing or catheter obstruction can also interfere with dialysate drainage.*

POSTDIALYSIS CARE

- Assess vital signs, including temperature. *Comparison of pre- and postdialysis vital signs helps identify beneficial and adverse effects of the procedure.*
- Time meals to correspond with dialysis outflow. *Scheduling meals while the abdomen is empty of dialysate enhances intake and reduces nausea.*
- Teach the client and family about the procedure. *The client may elect to use peritoneal dialysis at home to manage end-stage renal disease and prevent uremia.*

urine output to less than 30 mL per hour and other evidence of decreased cardiac output. Maintain intravenous fluids as ordered. Alert the physician if the client is receiving more than one nephrotoxic drug or if a nephrotoxic drug is ordered for a dehydrated client. Closely observe clients receiving blood or blood cells for early signs of transfusion reaction and intervene appropriately.

Assessment

Both subjective and objective data are useful when assessing the client with acute renal failure:

- *Health history:* Complaints of anorexia, nausea, weight gain, or edema; recent exposure to a nephrotoxin such as an aminoglycoside antibiotic or radiologic procedure using an injected contrast medium; previous transfusion reaction; chronic diseases such as diabetes, heart failure, or kidney disease.
- *Physical examination:* Vital signs including temperature; urine output (amount, color, clarity, specific gravity, presence of blood cells or protein); weight; skin color, peripheral pulses; presence of edema (periorbital or dependent); lung sounds, heart sounds, and bowel tones.

Nursing Diagnoses and Interventions

The client with acute renal failure has numerous nursing care needs related not only to the renal failure but also to the underlying condition that precipitated it. Priority nursing care needs relate to fluid volume alterations, appetite and nutrition, and teaching/learning. For additional nursing diagnoses and interventions, see the Nursing Care Plan on the next page.

Excess Fluid Volume

In acute renal failure, the kidneys often cannot excrete adequate urine to maintain a normal extracellular fluid balance. Fluid retention is greater in oliguric renal failure than in nonoliguric failure. Rapid weight gain and edema indicate fluid retention. In addition, heart failure and pulmonary edema may develop. In the older adult or severely debilitated client, fluid retention can present a significant management problem.

- Maintain hourly intake and output records. *Accurate intake and output records help guide therapy, especially fluid restrictions.*
- Weigh daily or more frequently, as ordered. Use standard technique (same scale, clothing, or coverings) to ensure accuracy. *Rapid weight changes are an accurate indicator of fluid volume status, particularly in the oliguric client.*
- Assess vital signs at least every 4 hours. *Hypertension, tachycardia, and tachypnea may indicate excess fluid volume.*

PRACTICE ALERT

Frequently assess breath and heart sounds, neck veins for distention, and back and extremities for edema. Report abnormal findings. Adventitious breath sounds (crackles), abnormal heart sounds such as an S_3 or S_4 gallop, distended neck veins, and peripheral edema may indicate hypervolemia, heart failure, or pulmonary edema.

- If not contraindicated, place in semi-Fowler's position *to enhance cardiac and respiratory function.*
- Report abnormal serum electrolyte values and manifestations of electrolyte imbalance. The client with ARF is at particular risk for the following electrolyte imbalances:
  a. *Hyperkalemia* due to impaired potassium excretion. Manifestations include irritability, nausea, diarrhea, abdominal cramping, cardiac dysrhythmias, and ECG changes.
  b. *Hyponatremia* due to water retention. Manifestations include nausea, vomiting, and headache, with possible central nervous system (CNS) manifestations of lethargy, confusion, seizures, and coma.
  c. *Hyperphosphatemia* due to decreased phosphate excretion. Manifestations include hyperreflexia, paresthesias, and possible tetany.

  ARF impairs electrolyte and water excretion, causing multiple electrolyte imbalances.
- Restrict fluids as ordered. Provide frequent mouth care and encourage using hard candies to decrease thirst. If ice chips are allowed, include the water content (approximately one-half of the total volume) as intake. *Fluids are restricted to minimize fluid retention and complications of fluid volume excess.*
- Administer medications with meals. *Giving oral medications with meals minimizes ingestion of excess fluids.*
- Turn frequently and provide good skin care. *Edema decreases tissue perfusion and increases the risk of skin breakdown, especially in the older or debilitated client.*

Imbalanced Nutrition: Less than Body Requirements

Anorexia and nausea associated with renal failure often interfere with food intake and nutrition. In addition, the disease process leading to ARF may contribute to increased nutritional needs for healing and decreased food intake.

- Monitor and record food intake, including the amount and type of food consumed. *A detailed intake record helps guide decisions about nutritional status and necessary supplements.*
- Weigh daily. *Weight changes over time (days to weeks) reflect nutritional status, while rapid weight changes are more reflective of fluid volume status. In ARF, weight may remain stable or increase due to fluid retention even though tissue mass is being lost.*
- Arrange for dietary consultation to plan meals within prescribed limitations that consider the client's food preferences. *Diets restricted in protein, salt, and potassium can be unpalatable; intake and appetite improve when preferred foods are included as allowed.*
- Engage the client in planning daily menus. *Participation in meal planning increases the client's sense of control and autonomy.*
- Allow family members to prepare meals within dietary restrictions. Encourage family members to eat with the client. *Familiar foods and social interaction encourage eating and increase enjoyment of meals.*
- Provide frequent, small meals or between-meal snacks. *These measures promote food intake in the fatigued or anorectic client.*

NURSING CARE PLAN A Client with Acute Renal Failure

Judy Devak is driving home late one evening when she loses control of her car trying to avoid hitting a deer in the road. Her car strikes a tree and rolls into a deep ditch beside the road, out of sight of passing cars. The wreck is not discovered until 2 hours later. On arrival at the accident scene, the paramedics find Ms. Devak hypotensive: BP 90/60, P 120, and R 24. She is alert and in severe pain, with a fractured right femur. After immobilizing Ms. Devak's neck and back and extricating her from the car, they apply a traction splint to her leg and transport her to the local hospital.

ASSESSMENT

Katie Leaper, RN, obtains a nursing history on Ms. Devak's admission to the intensive care unit. Ms. Devak indicates that she has been healthy, having experienced only minor illnesses and chickenpox as a child. She has never been hospitalized, and knows of no allergies to medications. Ms. Devak is not currently taking prescription or nonprescription drugs. Physical assessment findings include T 97.4°F (36.3°C) PO, P 100, R 18, and BP 124/68. Skin pale, cool, and dry, with multiple scrapes, minor abrasions, and bruises on face and extremities. A linear bruise is noted on her chest and abdomen from the seat belt. Lung sounds clear, heart tones normal, and abdomen tender but soft to palpation. Right leg alignment maintained with skeletal traction. One unit of whole blood was infused prior to ICU admission, a second unit is currently infusing. An indwelling urinary catheter and a nasogastric tube are in place.

During the first few hours after admission, Ms. Leaper notes that Ms. Devak's hourly output has dropped from 55 mL to 45 to 28 mL of clear yellow urine. The physician orders a 500-mL intravenous fluid challenge, STAT urinalysis, BUN, and serum creatinine. The fluid challenge elicits only a slight increase in urine output. Urinalysis results show a specific gravity of 1.010 and the presence of WBCs, red and white cell casts, and tubular epithelial cells in the sediment. Ms. Devak's BUN is 28 mg/dL; her serum creatinine, 1.5 mg/dL. The physician diagnoses probable acute renal failure and orders a nephrology consultation. In addition, the physician orders aluminum hydroxide, 10 mL every 2 hours per nasogastric tube, and ranitidine 50 mg intravenously every 8 hours.

DIAGNOSES

- *Acute Pain* related to injuries sustained in accident
- *Anxiety* related to being in the intensive care unit
- *Risk for Excess Fluid Volume* related to impaired renal function
- *Impaired Physical Mobility* related to skeletal traction
- *Ineffective Protection* related to injuries and invasive procedures

EXPECTED OUTCOMES

- Report adequate pain control.
- Verbalize reduced anxiety.
- Maintain stable weight and vital signs within normal range.
- Maintain skin integrity.
- Use the trapeze appropriately to adjust position in bed while maintaining body alignment.
- Remain free of infection, bleeding, or respiratory distress.

PLANNING AND IMPLEMENTATION

- Maintain PCA.
- Assess frequently for pain control and response to analgesia.
- Encourage expression of thoughts, feelings, and fears about condition and placement in ICU.
- Document vital signs and heart and lung sounds at least every 4 hours.
- Weigh every 12 hours.
- Document hourly intake and output.
- Restrict fluids as ordered, including diluent for all intravenous medications as intake.
- Assist with mouth care every 3 to 4 hours; allow frequent rinsing of mouth and ice chips as allowed.
- Assist with position changes at least every 2 hours; teach use of the overhead trapeze.
- Monitor frequently for signs of infection, bleeding, or respiratory distress.

EVALUATION

After just over 3 days of oliguria, Ms. Devak's urine output increases. By the end of the fourth day she is excreting 60 to 80 mL/h of urine. Although her BUN, serum creatinine, and potassium levels remain high, they never reach a critical point, and dialysis is not required. She is transferred from the ICU on the fifth day after admission. When Ms. Devak is able to begin eating, she is placed on a low-potassium diet, restricted to 50 g of protein. Her renal function gradually improves. By discharge, results of her renal function studies, including BUN and serum creatinine, are nearly normal. Ms. Devak verbalizes an understanding of the need to avoid nephrotoxins such as NSAIDs until allowed by her physician.

CRITICAL THINKING IN THE NURSING PROCESS

1. What was the most likely specific precipitating factor for Ms. Devak's acute renal failure? Did anything else contribute to her risk?
2. Why did the physician prescribe aluminum hydroxide and ranitidine? Consider both the acute renal failure and Ms. Devak's placement in the intensive care unit.
3. Ms. Devak is at risk for respiratory distress related to potential fluid volume excess. How does her fractured femur further contribute to risk for respiratory distress?
4. Develop a care plan for Ms. Devak for the nursing diagnosis of *Deficient Diversional Activity.*
   See Evaluating Your Response in Appendix C.

- Administer antiemetics as ordered and provide mouth care prior to meals. *Nausea and a metallic taste in the mouth, common manifestations of uremia, can decrease food intake.*
- Administer parenteral nutrition as ordered if the client is unable to eat or tolerate enteral nutrition. *Preventing or slowing tissue catabolism is important for the client with ARF.*

PRACTICE ALERT

Intravenous lines and parenteral nutrition solutions can increase the risk for infection. Monitor sites carefully for signs of infection or inflammation.

Deficient Knowledge

The client with ARF has multiple learning needs. These include information about ARF, diagnostic and laboratory studies, management strategies, and implications for the recovery period.

- Assess anxiety level and ability to comprehend instruction. Tailor information and presentation to developmental level and physical, mental, and emotional status. *The client with ARF may be critically ill or have uremic effects that hinder learning. During the initial stages of ARF it may be necessary to limit information to immediate concerns.*
- Assess knowledge and understanding. *To enhance understanding and retention, relate information presented to previous learning.*
- Teach about diagnostic tests and therapeutic procedures. *Teaching reduces anxiety and improves understanding and cooperation.*
- Discuss dietary and fluid restrictions. *These measures may be continued after discharge.*
- If the client is discharged prior to the recovery phase of ARF, teach the signs and symptoms of complications, such as fluid volume excess or deficit, heart failure, and electrolyte imbalances. *As kidney function returns, urine output increases, but the concentrating ability of the nephrons and electrolyte excretion remain impaired. This impaired function increases the risk of excess fluid loss, possible dehydration, orthostatic hypotension, and electrolyte imbalance.*
- Teach how to monitor weight, blood pressure, and pulse. *These are important means of assessing fluid status.*
- Instruct to avoid nephrotoxic drugs and chemicals for up to 1 year following an episode of ARF. *During recovery, nephrons are vulnerable to damage by nephrotoxins such as NSAIDs, some antibiotics, radiologic contrast media, and heavy metals. Because alcohol can increase the nephrotoxicity of some materials, discourage alcohol ingestion.*

Linking NANDA, NIC, and NOC

Chart 29–1 shows links between NANDA nursing diagnoses, NIC, and NOC for the client with ARF.

Community-Based Care

Often the client is critically ill when ARF develops. Critical illness and the resulting state of client and family crisis can impair learning and retention of information. Include family members in teaching during the initial stages to promote understanding of what is happening and the reasons for specific treatment measures. Inclusion of the family reduces their anxiety, and provides a valuable resource for reinforcing client teaching about care after discharge.

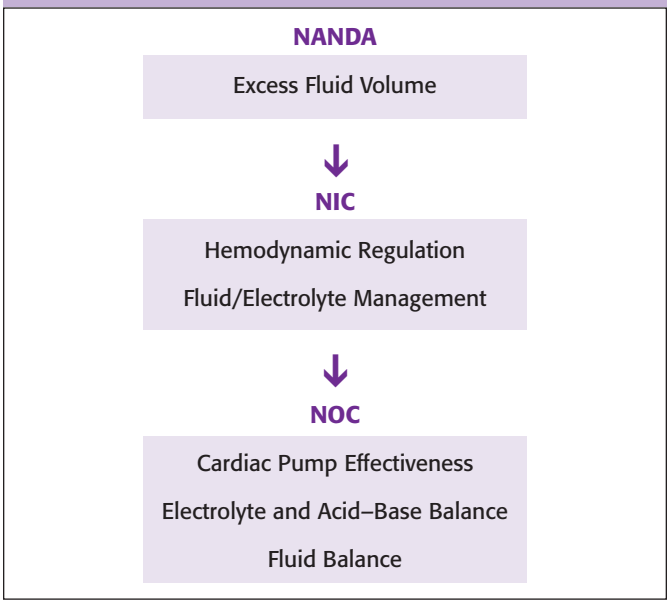
Client teaching needs for home care include:

- Avoiding exposure to nephrotoxins, particularly those in over-the-counter products
- Preventing infection and other major stressors that can slow healing
- Monitoring weight, blood pressure, and pulse
- Manifestations of relapse
- Continuing dietary restrictions
- Knowing when to contact the physician.

See the box on the next page for home care assessment of the older adult with ARF.

NANDA, NIC, AND NOC LINKAGES

CHART 29–1 The Client with Acute Renal Failure



Data from *NANDA's Nursing Diagnoses: Definitions & Classification 2005–2006* by NANDA International (2005), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

THE CLIENT WITH CHRONIC RENAL FAILURE

Although the kidneys usually recover from acute injury, many chronic conditions can lead to progressive renal tissue destruction and loss of function. Nephron units are lost and renal mass decreases, with progressive deterioration of glomerular filtration, tubular secretion, and reabsorption. This process of chronic renal failure (CRF) may progress slowly for many years without being recognized. Eventually, the kidneys are unable to excrete metabolic wastes and regulate fluid and electrolyte balance adequately, a condition known as end-stage renal disease, the final stage of CRF.

The incidence of ESRD is increasing, particularly in older adults. In 2001, more than 93,000 people started treatment for ESRD for a total of about 392,000 people undergoing ESRD treatment (NKUDIC, 2004). African Americans have the highest incidence of ESRD, followed by Native Americans, Asians, and European Americans (USRDS, 2005).

Conditions causing CRF typically involve diffuse, bilateral disease of the kidneys with progressive destruction and scarring of the entire nephron. As indicated in Figure 29–10 ■, diabetes is the leading cause of ESRD in all population groups

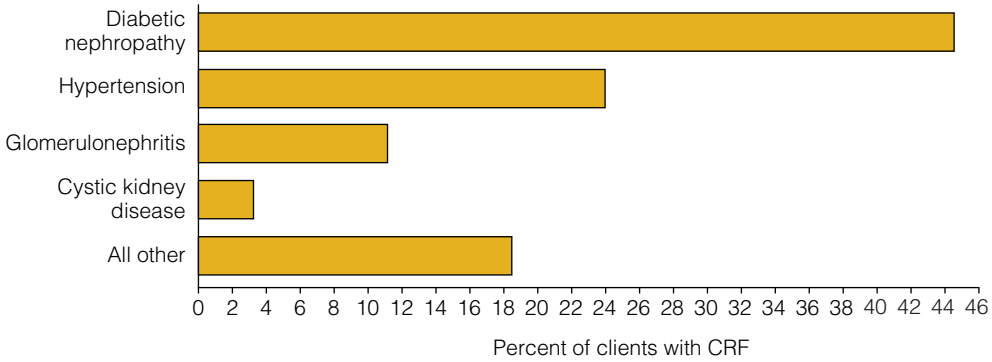


Figure 29–10 ■ The most common causes of chronic renal failure (USRDS, 2005).

in the United States. Hypertension closely follows diabetes as a major cause of ESRD; in many clients, these disorders coexist (USRDS, 2005).

FAST FACTS

- Acute renal failure develops abruptly and often can be reversed with appropriate treatment.
- Chronic renal failure is the end stage of progressive destruction of the kidneys and cannot be reversed.
- Diabetes is the leading cause of chronic renal failure, followed by hypertension, glomerulonephritis, cystic kidney disease, and all other causes.

Pathophysiology

The pathophysiology of CRF involves a gradual loss of entire nephron units. In the early stages, as nephrons are destroyed, remaining functional nephrons hypertrophy. Glomerular capillary flow and pressure increase in these nephrons, and more solute particles are filtered to compensate for lost renal mass. This increased demand predisposes the remaining nephrons to glomerular sclerosis (scarring), resulting in their eventual destruction. This process of continued loss of nephron function may continue even after the initial disease process has resolved (Kasper et al., 2005).Table 29–7 outlines common pathologic processes leading to nephron destruction and ESRD.

NURSING CARE OF THE OLDER ADULT **Renal Failure**

Structural and functional changes occur in the aging kidney. Structurally, the number of nephrons decreases. The GFR decreases, resulting in decreased renal clearance of drugs. Urine-concentrating ability decreases, and the kidney is less able to conserve sodium. Renal compensation for acid–base imbalances takes longer. Despite these changes, the kidney retains its ability to regulate fluid and electrolyte homeostasis remarkably well unless additional stresses are added. Any additional stressors such as hypotension, exposure to nephrotoxic drugs, or an inflammatory process such as glomerulonephritis may precipitate renal failure in the older adult.

The manifestations of renal failure often are missed in aging clients (e.g., edema may be attributed to heart failure or high blood pressure to preexisting hypertension). Serum creatinine levels may rise slowly. Because older adults have less muscle mass, they produce less creatinine, a by-product of muscle cell metabolism. Likewise, the BUN may remain within normal limits.

The same measures are used to treat renal failure in older adults as in younger people. Hemodialysis, peritoneal dialysis, and renal transplantation are appropriate if necessary. Treatment options (including conservative treatment or no treatment) and their potential benefits and ramifications should be clearly explained.

Assessing for Home Care

A number of factors should be considered in assessing the older adult's ability to manage treatment such as dialysis at home:

- Does the client have reasonable access to a dialysis center or outpatient unit? Is transportation available?
- Would home hemodialysis be appropriate? Is a caregiver available to be trained to manage dialysis? Does the client's home have appropriate electrical and plumbing fixtures?
- Would continuous ambulatory peritoneal dialysis be appropriate? Does the client have the manual dexterity, will, and cognitive ability to manage dialysis infusions? If not, would intermittent peritoneal dialysis using a dialyzing machine be more appropriate?
- Are family members or other support persons available to provide assistance to the client as needed?

Resources for Home Care

The following resources may be useful for clients with kidney disease:

- American Association of Kidney Patients
  800-749-2257
  813-636-8100
  www.aakp.org
- American Kidney Fund
  800-638-8299
  866-300-2900 (Spanish help line)
  www.kidneyfund.org
- National Kidney Foundation
  800-622-9010
  www.kidney.org

TABLE 29–7 **Pathophysiology of Chronic Renal Failure**

CAUSE	EXAMPLES
Diabetic nephropathy	Changes in the glomerular basement membrane, chronic pyelonephritis, and ischemia lead to sclerosis of the glomerulus and gradual destruction of the nephron.
Hypertensive nephrosclerosis	Long-standing hypertension leads to renal arteriosclerosis and ischemia resulting in glomerular destruction and tubular atrophy.
Chronic glomerulonephritis	Bilateral inflammatory process of the glomeruli leads to ischemia, nephron loss, and shrinkage of the kidney.
Chronic pyelonephritis	Chronic infection commonly associated with an obstructive or neurologic process and vesicoureteral reflux leads to reflux nephropathy (renal scarring, atrophy, and dilated calyces).
Polycystic kidney disease	Multiple bilateral cysts gradually destroy normal renal tissue by compression.
Systemic lupus erythematosus	Basement membrane damage by circulating immune complexes leads to focal, local, or diffuse glomerulonephritis.

The course of CRF is variable, progressing over a period of months to many years. In the early stage, known as *decreased renal reserve,* unaffected nephrons compensate for the lost nephrons. The GFR is about 50% of normal, and the client is asymptomatic with normal BUN and serum creatinine levels. As the disease progresses and the GFR falls to 20% to 50% of normal, azotemia and some manifestations of *renal insufficiency* may be seen. Any further insult to the kidneys at this stage (such as infection, dehydration, exposure to nephrotoxins, or urinary tract obstruction) can further reduce function and precipitate the onset of *renal failure* or overt uremia. This stage is characterized by a GFR of less than 20% of normal. The serum creatinine and BUN levels rise sharply (Figure 29–11 ■), the client becomes oliguric, and manifestations of uremia are seen. In ESRD, the final stage of CRF, the GFR is less than 5% of normal and renal replacement therapy is necessary to sustain life (Kasper et al., 2005; Porth, 2005). Table 29–8 summarizes the stages of chronic renal failure.

Manifestations and Complications

Chronic renal failure often is not identified until its final, uremic stage is reached. **Uremia,** which literally means "urine in the blood," refers to the syndrome or group of symptoms associated with ESRD. In uremia, fluid and electrolyte balance is altered, the regulatory and endocrine functions of the kidney are impaired, and accumulated metabolic waste products affect essentially every other organ system (Kasper et al., 2005; Porth, 2005).

Early manifestations of uremia include nausea, apathy, weakness, and fatigue, symptoms that are dismissed as a viral infection or influenza. As the condition progresses, frequent vomiting, increasing weakness, lethargy, and confusion develop (Porth, 2005). The *Multisystem Effects of Uremia* are illustrated on page 917.

Fluid and Electrolyte Effects

Loss of functional kidney tissue impairs its ability to regulate fluid, electrolyte, and acid–base balance. In the early stages of

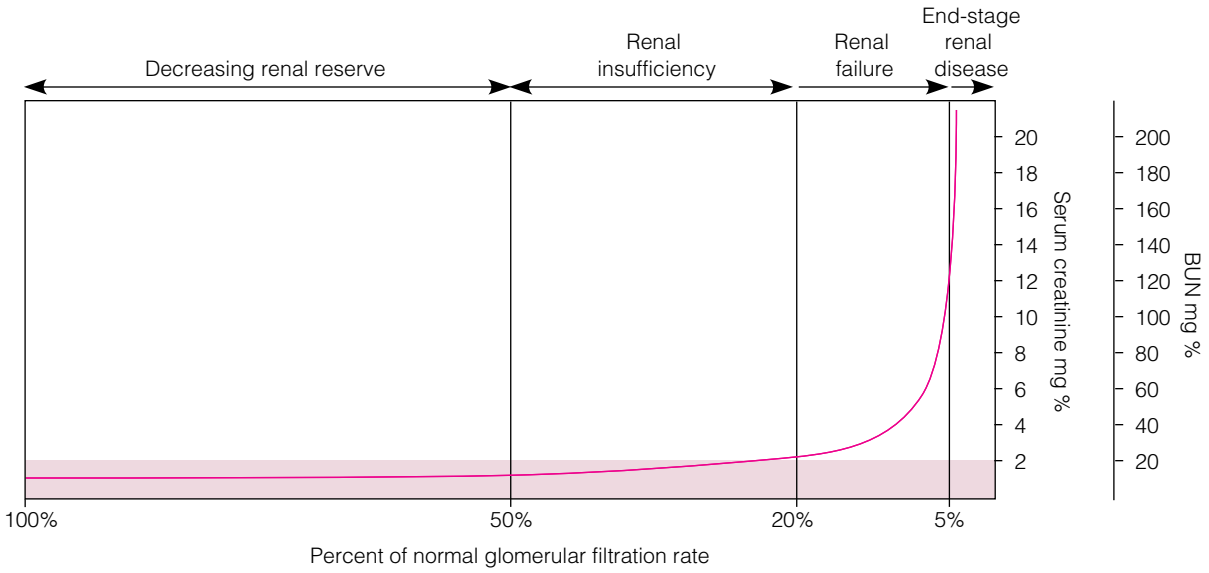


Figure 29–11 ■ The relationship of renal function to BUN and serum creatinine values through the course of chronic renal failure.

TABLE 29–8 Stages of Chronic Renal Failure

STAGE	GLOMERULAR FILTRATION RATE	MANIFESTATIONS
Decreased renal reserve	Approximately 50% of normal	None; normal BUN and creatinine
Renal insufficiency	20% to 50% of normal	Polyuria with low, fixed specific gravity; azotemia; anemia; hypertension
Renal failure	< 20% of normal	Increasing azotemia; edema, metabolic acidosis, hypercalcemia; possible uremia
End-stage renal disease	< 5% of normal	Kidney atrophy and fibrosis; overt uremia

CRF, impaired filtration and reabsorption lead to proteinuria, hematuria, and decreased urine-concentrating ability. Salt and water are poorly conserved, and risk for dehydration increases. Polyuria, nocturia, and a fixed specific gravity of 1.008 to 1.012 are common (Porth, 2005). As the GFR decreases and renal function deteriorates further, sodium and water retention are common, necessitating salt and water restrictions.

Hyperkalemia develops as renal failure progresses. Manifestations of hyperkalemia, such as muscle weakness, paresthesias, and ECG changes, are not usually seen until the GFR is less than 5 mL/min. Phosphate excretion is also impaired, leading to hyperphosphatemia and hypocalcemia. Reduced calcium absorption due to impaired vitamin D activation also contributes to hypocalcemia. Hypermagnesemia develops with advancing renal failure; magnesium-containing antacids are avoided for this reason.

As renal failure advances, hydrogen-ion excretion and buffer production are impaired, leading to metabolic acidosis. Respiratory rate and depth increase (Kussmaul's respirations) to compensate for metabolic acidosis. Although metabolic acidosis is often asymptomatic, other possible manifestations include general malaise, weakness, headache, nausea and vomiting, and abdominal pain (see Chapter 10 ∞).

Cardiovascular Effects

Cardiovascular disease is a common cause of death in ESRD, and results from accelerated atherosclerosis. Hypertension, hyperlipidemia, and glucose intolerance all contribute to the process. Cerebral and peripheral vascular manifestations of atherosclerosis are also seen.

Systemic hypertension is a common complication of ESRD. Hypertension results from excess fluid volume, increased renin–angiotensin activity, increased peripheral vascular resistance, and decreased prostaglandins. Increased extracellular fluid volume also can lead to edema and heart failure. Pulmonary edema may result from heart failure and increased permeability of the alveolar capillary membrane.

Retained metabolic toxins can irritate the pericardial sac, causing an inflammatory response and signs of pericarditis. *Cardiac tamponade,* a potential complication of pericarditis, occurs when inflammatory fluid in the pericardial sac interferes with ventricular filling and cardiac output. Once a common complication of uremia, pericarditis is less common when dialysis is initiated early.

Hematologic Effects

Anemia is common in uremia, caused by multiple factors. The kidneys produce erythropoietin, a hormone that controls RBC production. In renal failure, erythropoietin production declines. Retained metabolic toxins further suppress RBC production, and contribute to a shortened RBC life span. Nutritional deficiencies (iron and folate) and increased risk for blood loss from the GI tract also contribute to anemia.

Anemia contributes to manifestations such as fatigue, weakness, depression, and impaired cognition. It also affects cardiovascular function, and may be a major contributing factor to coronary heart disease and heart failure associated with ESRD (Porth, 2005).

Renal failure impairs platelet function, increasing the risk of bleeding disorders such as epistaxis and GI bleeding. The mechanism of impaired platelet function associated with renal failure is poorly understood.

Immune System Effects

Uremia increases the risk for infection. High levels of urea and retained metabolic wastes impair all aspects of inflammation and immune function. The WBC declines, humoral and cell-mediated immunity are impaired, and phagocyte function is defective. Both the acute inflammatory response and delayed hypersensitivity responses are affected (Porth, 2005). Fever is suppressed, often delaying the diagnosis of infection.

Gastrointestinal Effects

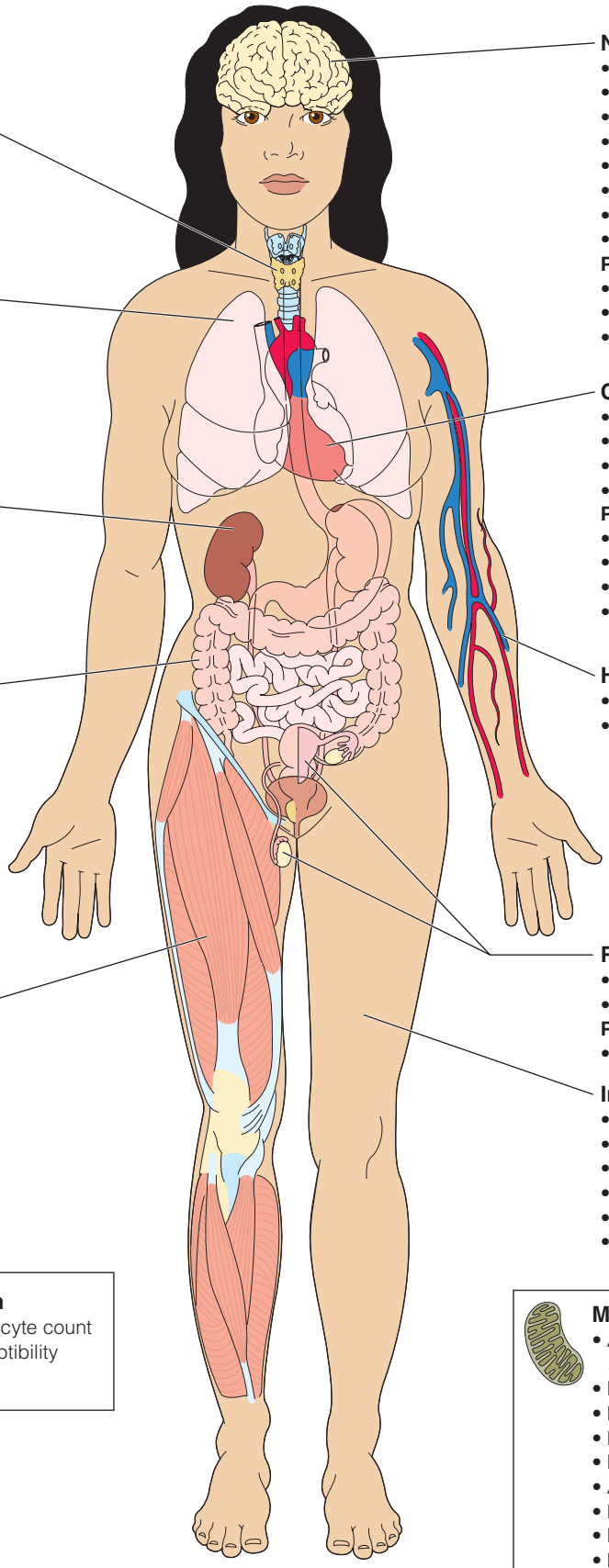
Anorexia, nausea, and vomiting are the most common early symptoms of uremia. Hiccups also are commonly experienced. Gastroenteritis is frequent. Ulcerations may affect any level of the GI tract and contribute to an increased risk of GI bleeding. Peptic ulcer disease is particularly common in uremic clients. *Uremic fetor,* a urine-like breath odor often associated with a metallic taste in the mouth, may develop. Uremic fetor can further contribute to anorexia.

Neurologic Effects

Uremia alters both central and peripheral nervous system function. CNS manifestations occur early and include changes in mentation, difficulty concentrating, fatigue, and insomnia. Psychotic symptoms, seizures, and coma are associated with advanced uremic encephalopathy.

Peripheral neuropathy is also common in advanced uremia. Both the sensory and motor tracts are involved. The lower limbs are initially affected. "Restless leg syndrome," sensations of crawling or creeping, prickling, or itching of the lower legs with frequent leg movement, increases during rest. Paresthesias and sensory loss typically occur in a "stocking-glove" pattern. As uremia progresses, motor function is also impaired,

MULTISYSTEM EFFECTS OF **Uremia**



Endocrine
- Hyperparathyroidism
- Glucose intolerance

Respiratory
- Pulmonary edema
- Pleuritis
- Kussmaul's respirations

Urinary
- Proteinuria
- Hematuria
- Fixed specific gravity
- Nocturia
- Oliguria, anuria

Gastrointestinal
- Anorexia
- Nausea and vomiting
- Gastroenteritis
- Hiccups
- Abdominal pain
- Uremic fetor

Potential Complications
- Peptic ulcer
- GI bleeding

Musculoskeletal
- Osteodystrophy
- Bone pain
- Spontaneous fractures

Neurologic
- Apathy
- Lethargy
- Headache
- Impaired cognition
- Insomnia
- Restless leg syndrome
- Gait disturbances
- Paresthesias

Potential Complications
- Seizures
- Decreased LOC
- Coma

Cardiovascular
- Hypertension
- Edema
- Coronary heart disease
- Dysrhythmias

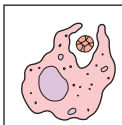
Potential Complications
- Pericarditis
- Pericardial effusion
- Cerebrovascular disease
- Heart failure

Hematologic
- Anemias
- Impaired clotting

Reproductive
- Amenorrhea (female)
- Impotence (male)

Potential Complication
- Spontaneous abortion

Integumentary
- Pallor
- Uremic skin color (yellow-green)
- Dry skin, poor turgor
- Pruritis
- Ecchymoses
- Uremic "frost"



Immune System
- Diminished leukocyte count
- Increased susceptibility to infection

Metabolic Processes
- Azotemia (↑ BUN and serum creatinine)
- Hyperkalemia
- Hyperphosphatemia
- Hypocalcemia
- Hypermagnesia
- Acidosis
- Hyperlipidemia
- Hyperuricemia
- Malnutrition

causing muscle weakness, decreased deep tendon reflexes, and gait disturbances.

Musculoskeletal Effects

Hyperphosphatemia and hypocalcemia associated with uremia stimulate parathyroid hormone secretion. Parathyroid hormone causes increased calcium resorption from bone. In addition, osteoblast (bone-forming) and osteoclast (bone-destructing) cell activity is affected. This bone resorption and remodeling, combined with decreased vitamin D synthesis and decreased calcium absorption from the GI tract, lead to *renal osteodystrophy,* also known as renal rickets. Osteodystrophy is characterized by *osteomalacia,* softening of the bones, and *osteoporosis,* decreased bone mass. Bone cysts may develop. Manifestations of osteodystrophy include bone tenderness, pain, and muscle weakness. The client is at increased risk for spontaneous fractures (Porth, 2005).

Endocrine and Metabolic Effects

Accumulated waste products of protein metabolism are a primary factor involved in the effects and manifestations of uremia. Serum creatinine and BUN levels are significantly elevated. Uric acid levels are increased, contributing to an increased risk of gout.

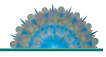
Tissues become resistant to the effects of insulin in uremia, leading to glucose intolerance. High blood triglyceride levels and lower than normal high-density lipoprotein (HDL) levels contribute to the accelerated atherosclerotic process.

Reproductive function is affected. Pregnancies are rarely carried to term, and menstrual irregularities are common. Reduced testosterone levels, low sperm counts, and impotence affect the male client with ESRD.

Dermatologic Effects

Anemia and retained pigmented metabolites cause pallor and a yellowish hue to the skin in uremia. Dry skin with poor turgor, a result of dehydration and sweat gland atrophy, is common. Bruising and excoriations are frequently seen. Metabolic wastes not eliminated by the kidneys may be deposited in the skin, contributing to itching or pruritus. In advanced uremia, high levels of urea in the sweat may result in *uremic frost,* crystallized deposits of urea on the skin.

INTERDISCIPLINARY CARE



Early management of CRF focuses on eliminating factors that may further decrease renal function and measures to slow the progression of the disease to ESRD. Additional treatment goals are to:

- Maintain nutritional status while minimizing the accumulation of toxic waste products and manifestations of uremia.
- Identify and treat complications of CRF.
- Prepare for renal replacement therapies such as dialysis or renal transplant.

Diagnosis

Diagnostic testing is used both to identify CRF and to monitor kidney function. A number of tests may be performed to determine the underlying renal disorder. Once the diagnosis is established, renal function is monitored primarily through blood levels of metabolic wastes and electrolytes. See Chapter 27 ∞ for the nursing implications of selected tests.

- *Urinalysis* is done to measure urine specific gravity and detect abnormal urine components. In CRF, the specific gravity may be fixed at approximately 1.010, equivalent to that of plasma. This fixed specific gravity is due to impaired tubular secretion, reabsorption, and urine concentrating ability. Abnormal proteins, blood cells, and cellular casts may also be noted in the urine.
- *Urine culture* is ordered to identify any urinary tract infection that may hasten the progress of CRF.
- *BUN* and *serum creatinine* are obtained to evaluate kidney function in eliminating nitrogenous waste products. Levels of both are monitored to assess the progress of renal failure. A BUN of 20 to 50 mg/dL signals mild azotemia; levels greater than 100 mg/dL indicate severe renal impairment. Uremic symptoms are seen when the BUN is around 200 mg/dL or higher. Serum creatinine levels of greater than 4 mg/dL indicate serious renal impairment.
- *Creatinine clearance* evaluates the GFR and renal function. In early CRF (renal insufficiency), the GFR is more than 20% of normal and the creatinine clearance 30 mL/min or greater. As the disease progresses and the stage of renal failure is reached, the GFR is reduced to less than 20% of normal and the creatinine clearance to 15 to 29 mL/min. In ESRD, the GFR is less than 5% of normal and the creatinine clearance is less than 15 mL/min (Kasper et al., 2005).
- *Serum electrolytes* are monitored throughout the course of CRF. The serum sodium may be within normal limits or low because of water retention. Potassium levels are elevated but usually remain below 6.5 mEq/L. Serum phosphate is elevated, and the calcium level is decreased. Metabolic acidosis is identified by a low pH, low CO_2, and low bicarbonate levels.
- *CBC* reveals moderately severe anemia with a hematocrit of 20% to 30% and a low hemoglobin. The number of RBCs and platelets is reduced.
- *Renal ultrasonography* is done to evaluate kidney size. In CRF, kidney size decreases as nephrons are destroyed and kidney mass is reduced.
- *Kidney biopsy* may be done to identify the underlying disease process if this is unclear. It is also used to differentiate acute from chronic failure. Kidney biopsy may be performed in surgery or done percutaneously using needle biopsy.

Medications

Chronic renal failure affects both the pharmacokinetic and pharmacodynamic effects of drug therapy. Most medications are excreted primarily by the kidney. The half-life and plasma levels of many drugs increase in chronic renal failure. Drug absorption may be decreased when phosphate-binding agents are administered concurrently. Proteinuria can significantly reduce plasma protein levels, leading to manifestations of toxicity when highly protein-bound drugs are given. In addition, any potentially nephrotoxic agent is avoided or used with extreme

caution. Drugs such as meperidine, metformin (Glucophage), and other oral hypoglycemic agents eliminated by the kidney are avoided entirely (Kasper et al., 2005).

Diuretics such as furosemide or other loop diuretics may be prescribed to reduce extracellular fluid volume and edema. Diuretic therapy also can reduce hypertension and cause potassium wasting, lowering serum potassium levels. Other antihypertensive agents are used to maintain the blood pressure within normal levels, slow the progress of renal failure, and prevent complications of coronary heart disease and cerebral vascular disease. ACE inhibitors are preferred, although any class of antihypertensive agent may be prescribed (see Chapter 35 ∞).

Other drugs may be used to manage electrolyte imbalances and acidosis. Sodium bicarbonate or calcium carbonate may be used to correct mild acidosis. Oral phosphorus binding agents such as calcium carbonate or calcium acetate are given to lower serum phosphate levels and normalize serum calcium levels. Aluminum hydroxide may be used in acute treatment of hyperphosphatemia. Its use is limited to short term by complications such as encephalopathy and osteodystrophy associated with long-term administration of aluminum-containing preparations (Tierney et al., 2005). Vitamin D supplements may be given to improve calcium absorption.

If the serum potassium rises to dangerously high levels, a combination of bicarbonate, insulin, and glucose may be given intravenously to promote potassium movement into the cells. Sodium polystyrene sulfonate (Kayexalate), a potassium-ion exchange resin, can be given either orally or rectally (as an enema).

Folic acid and iron supplements are given to combat anemia associated with chronic renal failure. A multiple vitamin preparation is also often prescribed, because anorexia, nausea, and dietary restrictions may limit nutrient intake.

Nutrition and Fluid Management

As renal function declines, the elimination of water, solutes, and metabolic wastes is impaired. Accumulation of these wastes in the body leads to uremic symptoms. Instituted early in the course of CRF, dietary modifications can slow the progress of nephron destruction, reduce uremic symptoms, and help prevent complications.

Unlike carbohydrates and fats, the body is unable to store excess proteins. Unused dietary proteins are degraded into urea and other nitrogenous wastes, which are then eliminated by the kidneys. Protein-rich foods also contain inorganic ions such as hydrogen ion, phosphate, and sulfites that are eliminated by the kidneys. Research has shown that restricting dietary protein intake slows the progression of CRF and reduces uremic symptoms (Kasper et al., 2005). A daily protein intake of 0.6 g/kg of body weight, or approximately 40 g/day for an average male client, provides the amino acids necessary for tissue repair. Proteins should be of high biologic value, rich in the essential amino acids. Carbohydrate intake is increased to maintain energy requirements and provide approximately 35 kcal/kg per day.

Water and sodium intake are regulated to maintain the extracellular fluid volume at normal levels. Water intake of 1 to 2 L per day is generally recommended to maintain water balance. Sodium is restricted to 2 g per day initially. More stringent water and sodium restrictions may be necessary as renal failure progresses. The client is instructed to monitor weight daily and report any weight gain in excess of 5 pounds over a 2-day period.

When the GFR falls to less than 10 to 20 mL/min, potassium and phosphorous intake are also restricted. Potassium intake is limited to less than 60 to 70 mEq/day (normal intake is about 100 mEq/day) (Tierney et al., 2005). The client is cautioned to avoid using salt substitutes, which typically contain high levels of potassium chloride. Foods high in phosphorus include eggs, dairy products, and meat.

Renal Replacement Therapies

When pharmacologic and dietary management strategies are no longer effective to maintain fluid and electrolyte balance and prevent uremia, dialysis or kidney transplantation is considered.

A number of considerations affect the choice of long-term treatment. Hemodialysis and peritoneal dialysis each have advantages and disadvantages. Establishing vascular access for hemodialysis may take several months. Planning ahead to develop the access before dialysis is necessary can ease the transition to dialysis. Established access is not a consideration for peritoneal dialysis. The peritoneal catheter can be placed and treatment initiated as soon as it is indicated. When dialysis treatments will be performed at home, initiating instruction before it is required can result in more effective learning. If a family member will serve as a dialysis helper, training begins prior to the onset of uremia.

If transplantation is considered, tissue typing and identification of potential living related donors can be done prior to the onset of ESRD. To make an informed decision, both the client and the potential donor need to understand the risks, benefits, and options available. If the decision for transplant is made early, dialysis can potentially be avoided. The client's age, concurrent health problems, donor availability, and personal preference influence the choice of renal replacement therapy.

DIALYSIS Approximately 80% of all people being treated for ESRD in the United States are receiving dialysis at an average maintenance cost of about $65,000 per year (USRDS, 2005). For the client who is not a candidate for renal transplantation or who has had a transplant failure, dialysis is life sustaining.

The most common therapies for ESRD in the United States are hemodialysis performed in a dialysis center, followed by peritoneal dialysis and kidney transplant (NKUDIC, 2004). Both hemodialysis and peritoneal dialysis can be done in the home, but few clients use home hemodialysis. Of the two, peritoneal dialysis is typically the choice for at-home treatment. Because the morbidity and mortality for each are comparable, factors such as the desire and ability to manage home care, employment, and availability of a dialysis center become the primary factors influencing the choice of hemodialysis or peritoneal dialysis.

Clients on long-term dialysis have a higher risk for complications and death than the general population. Many have other severe diseases along with ESRD. Infection and cardiovascular disease are common causes of illness and death. The 1-year

survival rate for clients receiving dialysis is nearly 78%; long-term survival, however, falls to 32% at 5 years and about 9% at 10 years (NKUDIC, 2004).

The decision to initiate dialysis is not easy. Like insulin therapy for the diabetic, dialysis manages the symptoms of ESRD but does not cure it. Dialysis is a constant factor of life, requiring thinking and planning ahead at all times. Clients on dialysis may not be able to maintain a job. Families often fall apart with the day-to-day stress. Even with dialysis, the client may have constant flulike symptoms, never feeling truly well. Clients on hemodialysis may feel powerless because of their dependence on others for treatment. On the other hand, home peritoneal dialysis places a continuing burden on the client to maintain treatment. In the end, the client may choose to discontinue treatment, preferring death over continued dialysis.

Hemodialysis for ESRD typically is done three times a week for a total of 9 to 12 hours. The amount of dialysis needed (or *dialysis dose*) is individually determined by factors such as body size and residual renal function, dietary intake, and concurrent illness. Hypotension and muscle cramps are common complications during hemodialysis treatments. Infection and vascular access problems are common long-term complications of hemodialysis. Cardiovascular disease is the leading cause of death for clients receiving hemodialysis. The death rate from cardiovascular disease is higher in clients on hemodialysis than those on peritoneal dialysis or who have had a kidney transplant for reasons that are unclear (Kasper et al., 2005). See the previous section on ARF and the box on page 908 for more information about hemodialysis and related nursing care.

Peritoneal dialysis is currently used by approximately 10% of people who require long-term dialysis in the United States. In Canada and Europe, 35% to 45% of clients with ESRD are treated with peritoneal dialysis. In Third World countries, peritoneal dialysis is used to treat the majority of clients with ESRD.

Continuous ambulatory peritoneal dialysis (CAPD) is the most common form of peritoneal dialysis used. Dialysate (2 L) is instilled into the peritoneal cavity, and the catheter is sealed. The client can then continue normal daily activities, emptying the peritoneal cavity and replacing the dialysate every 4 to 6 hours. No special equipment is needed. A variation of CAPD is *continuous cyclic peritoneal dialysis (CCPD),* which uses a delivery device during nighttime hours and a continuous dwell during the day. CAPD can be performed anywhere, and CCPD allows for home treatment at night, leaving the client free during the day.

Peritoneal dialysis has several advantages over hemodialysis. Heparinization and vascular complications associated with an AV fistula are avoided. The clearance of metabolic wastes is slower but more continuous, avoiding rapid fluctuations in extracellular fluid composition and associated symptoms. More liberal intake of fluids and nutrients is often allowed for the client on CAPD. While glucose absorbed from dialysate can increase blood glucose levels in the diabetic, regular insulin can be added to the infusion to manage hyperglycemia. The client on peritoneal dialysis is better able to self-manage the treatment regimen, reducing feelings of helplessness.

The major disadvantages of peritoneal dialysis include less effective metabolite elimination and risk of infection (peritonitis). Peritoneal dialysis may not be effective for large clients with no residual kidney function. Serum triglyceride levels increase with peritoneal dialysis. Finally, the presence of an indwelling peritoneal catheter may cause a body image disturbance. See the previous section of this chapter and the box on page 910 for more information about dialysis and nursing care for the client undergoing peritoneal dialysis.

KIDNEY TRANSPLANT Kidney transplant has become the treatment of choice for many clients with ESRD. Kidneys are the solid organ most commonly transplanted, and to date kidney transplantation is the most successful of transplantation procedures. The first kidney transplant was performed in 1954; the donor and recipient were identical twins. Kidney transplant as a treatment for ESRD is limited primarily by availability of organs. In 2004, more than 16,000 people received a kidney transplant; however, there currently are nearly 62,000 awaiting a transplant (Organ Procurement and Transplantation Network, 2005; United Network for Organ Sharing, 2005).

Kidney transplant improves both survival and quality of life for the client with ESRD. The client on dialysis has a 62.9% probability of surviving after 2 years of dialysis; the transplant recipient has a greater than 91.6% probability of survival after 2 years. At 5 years, the difference is even greater: 31.9% for dialysis compared with 80.6% for transplant (NKUDIC, 2004). The transplant client is no longer tethered to a dialysis catheter, machine, or center. Dietary and fluid restrictions are reduced, and the body image is more "whole."

Most transplanted kidneys are obtained from cadavers; however, transplants from living donors are increasing. In 2004, of transplanted kidneys, 41.5% came from living donors, most of whom were related to the recipient (UNOS, 2005). With both cadaver and living donor transplants, a close match between blood and tissue type is desired. Human leukocyte antigens (HLAs) are compared between the donor and recipient; six antigens in common is considered to be a "perfect" match. The success of well-matched living-donor transplants is better than for cadaver organ transplants, with a 1-year graft survival of 97.6% compared to 93.7% for cadaver transplants (NKUDIC, 2004). Close tissue matching probably accounts for the better outcome with living donors. People with normal kidneys who are in good physical health may donate a kidney. Predonation counseling is vital: Nephrectomy is major surgery and involves a risk of trauma or disease damaging the remaining kidney in the future. If the transplant fails, the psychologic impact on the donor can be significant. Nursing care of the client having a nephrectomy is summarized in the box on page 897.

Cadaver kidneys are obtained from people who meet the criteria for brain death, are less than 65 years old, and are free of systemic disease, malignancy, or infection, including HIV and hepatitis B or C. Kidneys are removed after brain death has been determined, and are preserved by hypothermia or a technique called continuous hypothermic pulsatile perfusion. A kidney preserved by hypothermia is transplanted within 24 to 48 hours. Continuous pulsatile perfusion allows up to 3 days

BOX 29–1 How Cadaver Kidneys Are Allocated for Transplant

The scarcity of organs for transplant raises questions about how cadaver kidneys are allocated—who receives a kidney and who does not. Past inequities in the allocation process (e.g., more men than women, more Caucasians than people of color, more rich than poor, and more young than old) led to the development of the United Network for Organ Sharing (UNOS) in 1986. UNOS has policies for organ distribution, including kidneys, hearts, livers, and other transplanted organs.

UNOS maintains national, regional, and local lists of clients awaiting transplants. When an organ becomes available, donor information is entered into the UNOS computer. The computer then runs a match program, generating a list of clients ranked by criteria such as blood and tissue type, organ size, and medical urgency of the client. Factors such as time on the waiting list and distance between the donor and the transplant center also are considered. A candidate with a perfect match (six HLAs in common) and compatible blood type gets priority for the kidney, regardless of region or geographic area. Otherwise, the list of clients in the local area is checked first, then the regional list of clients awaiting transplant. If no match is found in the region, the organ becomes available to clients nationwide.

The UNOS allocation system, standardized fees, and Medicare coverage for transplantation have done much to ensure equitable access to available kidneys. Still, controversy exists. Clients with resources for travel may register in several different regions for an organ. Up to 10% of clients receiving a transplant in any center may be foreign nationals competing with U.S. citizens for scarce organ resources. A transplant center can accept or reject a candidate for transplant who has lost a kidney because of noncompliance.

As long as the demand for kidneys exceeds the supply of donor organs, it is likely that controversy will exist regarding their allocation. Nurses can help by identifying potential donors and contacting the transplant coordinator. In addition, nurses can inform the public about organ donation and the allocation system, and encourage donation.

before transplantation. The system used to allocate cadaver kidneys for transplantation is outlined in Box 29–1.

The donor kidney is placed in the lower abdominal cavity of the recipient, and the renal artery, vein, and ureter are anastomosed (Figure 29–12 ■). The renal artery of the donor kidney is connected to the hypogastric artery, and the renal vein to the iliac vein. The ureter is connected to one of the recipient's ureters or directly to the bladder, using a tunnel technique to prevent reflux. Nursing care for the client having a kidney transplant is outlined in the box on the next page.

Unless the donor and recipient are identical twins, the grafted organ stimulates an immune response to reject the transplanted organ. Immunosuppressive drugs minimize this response. Azathioprine or mycophenolate mofetil are commonly used, often in combination with prednisone, a corticosteroid. Cyclosporine, a potent immunosuppressive, also may be used. These drugs suppress a portion of the immune system and the inflammatory response, increasing the risk for infections and cancers with long-term therapy. The nursing implications of immunosuppressive therapy are outlined in Chapter 13 ∞.

Glucocorticoids such as prednisone and methylprednisolone are used for both maintenance immunosuppression and to treat acute rejection episodes. Side effects of long-term corticosteroid use include impaired wound healing, emotional disturbances, osteoporosis, and cushingoid effects on glucose, protein, and fat metabolism.

Azathioprine inhibits both cellular and humoral immunity. Because this drug is rapidly metabolized by the liver, the dose may not need to be altered in the presence of renal failure. Bone marrow suppression, abnormalities of liver function, and alopecia are the primary significant adverse effects for azathioprine. The action of mycophenolate mofetil is similar to that of azathioprine. Its advantages are minimal bone marrow suppression and increased potency in preventing or reversing rejection of the transplanted organ (Kasper et al., 2005).

Cyclosporine primarily affects cellular immunity, the helper T cells in particular. Among its many adverse effects, which include hepatotoxicity and hirsutism, nephrotoxicity is a primary concern for the kidney transplant client.

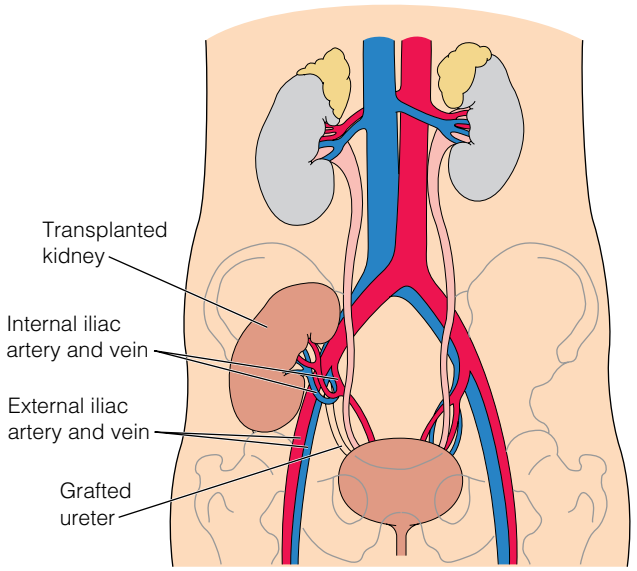


Figure 29–12 ■ Placement of a transplanted kidney in the iliac fossa with anastomosis to the hypogastric artery, iliac vein, and bladder.

Even with immunosuppressive therapy, the transplanted kidney can be rejected at any time. Either acute or chronic rejection may develop. *Acute rejection* develops within months of the transplant. It is caused by a cellular immune response with T-lymphocyte proliferation (Porth, 2005). Few manifestations may be apparent other than a rise in serum creatinine and possible oliguria. Methylprednisolone, a glucocorticoid, and OKT3 monoclonal antibody (see Chapter 13 ∞) are used to manage acute rejection episodes. OKT3 can cause severe systemic reactions, including chills, fever, hypotension, headache, and possible pulmonary edema (Kasper et al., 2005). *Chronic rejection,* which may develop months to years following the transplant, is a major

MEDIALINK Case Study: Kidney Transplant

NURSING CARE OF THE CLIENT HAVING A KIDNEY TRANSPLANT

PREOPERATIVE CARE

- Provide routine preoperative care as outlined in Chapter 4 ∞.
- Assess knowledge and feelings about the procedure, answering questions and clarifying information as needed. Listen and address concerns about surgery, the source of the donor organ, and possible complications. *Addressing concerns and reducing preoperative anxiety improve postoperative recovery.*
- Continue dialysis as ordered. *Continued renal replacement therapy is necessary to manage fluid and electrolyte balance and prevent uremia prior to surgery.*
- Administer immunosuppressive drugs as ordered before surgery. *Immunosuppression is initiated before transplantation to prevent immediate graft rejection.*

POSTOPERATIVE CARE

- Provide routine postoperative care as outlined in Chapter 4 ∞.
- Maintain urinary catheter patency and a closed system. *Catheter patency is vital to keep the bladder decompressed and prevent pressure on suture lines. A closed drainage system minimizes the risk for urinary tract infection.*
- Measure urine output every 30 to 60 minutes initially. *Careful assessment of urine output helps determine fluid balance and transplant function. Acute tubular necrosis is a common early complication, usually due to tissue ischemia during the period between removal of the kidney from the donor and transplantation. Oliguria is an early sign.*
- Monitor vital signs and hemodynamic pressures closely. *Diuresis may occur immediately, resulting in hypovolemia, low cardiac output, and impaired perfusion of the transplanted kidney.*
- Maintain fluid replacement, generally calculated to replace urine output over the previous 30 or 60 minutes, milliliter for milliliter. *Fluid replacement is vital to maintain vascular volume and tissue perfusion.*
- Administer diuretics as ordered. *Loop and/or osmotic diuretics such as furosemide or mannitol may be used to promote postoperative diuresis.*
- Remove the catheter within 2 to 3 days or as ordered. Encourage to void every 1 to 2 hours and assess frequently for signs of urinary retention following catheter removal. *The bladder may have atrophied prior to surgery, reducing its capacity. Urinary retention places stress on suture lines and increases the risk of infection.*
- Monitor serum electrolytes and renal function tests. *These tests are used to monitor graft function and fluid and electrolyte status. Electrolyte imbalances may develop as the transplanted kidney begins to function and diuresis occurs. Elevated serum creatinine and BUN levels may be early signs of rejection or graft failure.*
- Monitor for possible complications:
  a. *Hemorrhage* from an arterial or venous anastomosis can be either acute or insidious. Indicators include swelling at the operative site, increased abdominal girth, and signs of shock, including changes in vital signs and level of consciousness. *Hemorrhage is a surgical emergency, requiring prompt recognition and treatment to preserve the graft.*
  b. *Ureteral anastomosis failure* causes urine leakage into the peritoneal cavity. It may be marked by decreased urine output with abdominal swelling and tenderness. *Failure of the ureteral anastomosis requires surgical intervention.*
  c. *Renal artery thrombosis* is characterized by an abrupt onset of hypertension and reduced GFR. *Renal artery thrombosis can result in transplant failure.*
  d. *Infection* due to immunosuppression is an immediate and continuing risk. The inflammatory response is blunted, and infection may not significantly elevate the temperature. Monitor for signs such as change in level of consciousness, cloudy or malodorous urine, or purulent drainage from the incision. *Prevention and prompt treatment of infections is particularly important in the immunosuppressed client.*
- Include the following in predischarge teaching for the client and family:
  a. The use and effects of prescribed medications, including antihypertensive medications, immunosuppressive agents, prophylactic antibiotics, and others as ordered.
  b. Monitoring vital signs (including temperature) and weight.
  c. Manifestations of organ rejection, such as swelling and tenderness over the graft site, fever, joint aching, weight gain, and decreased urinary output. Stress the importance of promptly reporting signs and symptoms to the physician.
  d. Ordered or recommended dietary restrictions such as restricted carbohydrate and sodium intake, and increased protein intake.
  e. Measures to prevent infection, such as avoiding crowds and obviously ill individuals.

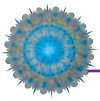
  The client and family will manage care after discharge, and therefore need a good understanding of what to expect, how to monitor graft status, and measures to reduce the adverse effects of medications.
- Provide psychologic support, address concerns, and provide information as needed. *The client knows that transplant success is not guaranteed. In addition, the client has often been managing a chronic disease independently and is used to having a degree of control. Providing information and allowing the client to retain control relieves anxiety and improves recovery.*

cause of graft loss. Both humoral and cellular immune responses are involved in chronic rejection. It does not respond to increased immunosuppression. The presenting manifestations of chronic rejection—progressive azotemia, proteinuria, and hypertension—are those of progressive renal failure.

Hypertension is a possible complication of kidney transplant, resulting from graft rejection, renal artery stenosis, or renal vasoconstriction. Clients may develop glomerular lesions and manifestations of nephrosis. Hypertension and altered blood lipids (increased LDLs and decreased HDLs) increase the risk of death from myocardial infarction and stroke following transplant (Kasper et al., 2005).

Long-term immunosuppression has adverse effects as well. Infection is a continuing threat. Bacterial and viral infections

may develop, as well as fungal infections of the blood, lungs, and CNS. Tumors are also common, with carcinoma *in situ* of the cervix, lymphomas, and skin cancers most prevalent. The risk of congenital anomalies is increased in infants whose mothers have undergone immunosuppressive therapy. Corticosteroid use may lead to bone problems, gastrointestinal disorders such as peptic ulcer disease, and cataract formation.



NURSING CARE

Health Promotion

Measures to reduce the risk of CRF focus on preventing kidney disease and appropriately managing diabetes and hypertension. Promote early and effective treatment of all infections, particularly skin and pharyngeal infections caused by streptococcal bacteria. Discuss measures to reduce the risk for urinary tract infections, and stress the importance of prompt treatment to eradicate the infecting organism. Discuss the relationship between diabetes, hypertension, and kidney disease. Emphasize that maintaining blood glucose levels and the blood pressure within the recommended ranges reduces the risk of adverse effects on the kidneys. Ensure that all clients with less than optimal renal function are well hydrated, particularly when a nephrotoxic drug is prescribed or anticipated. Finally, encourage the client with ESRD to investigate options for early transplantation to avoid long-term dialysis.

Assessment

Both subjective and objective data are used to assess the client with CRF:

- *Health history:* Complaints of anorexia, nausea, weight gain, or edema; current treatment (if any), including type and frequency of dialysis or previous kidney transplant; chronic diseases such as diabetes, heart failure, or kidney disease.
- *Physical examination:* Mental status; vital signs including temperature, heart and lung sounds, and peripheral pulses; urine output (if any); weight; skin color, moisture, condition; presence of edema (periorbital or dependent); bowel tones; presence and location of an AV fistula, shunt, or graft, or peritoneal catheter.

See the box on page 914 ∞ for assessment of the older adult with ESRD.

Nursing Diagnoses and Interventions

Whether the client with ESRD is facing long-term dialysis or renal transplantation, a number of nursing care needs can be identified. This section focuses on nursing care related to impaired renal function, nutritional deficits due to dietary restrictions and nausea, increased risk for infection, and changes in body image. Also see the Nursing Care Plan on the next page for additional potential nursing diagnoses and interventions for the client with chronic renal failure.

Ineffective Tissue Perfusion: Renal

Capillaries are an integral part of the nephron. As nephrons are destroyed, kidney perfusion progressively declines. As renal perfusion and nephron function fall, the kidney is less able to maintain fluid and electrolyte balance and eliminate waste products from the body.

- Monitor intake and output, vital signs including orthostatic blood pressures, and weight. *These provide important data to identify changes in fluid volume.*

PRACTICE ALERT

Weight changes are a more accurate indicator of fluid volume status in the oliguric or anuric client than intake and output measurements.

- Restrict fluids as ordered. *As renal function declines, the ability to eliminate excess fluid is impaired.*
- Monitor respiratory status, including lung sounds, every 4 to 8 hours. *Fluid volume overload may lead to heart failure and possible pulmonary edema.*
- Monitor BUN, serum creatinine, pH, electrolytes, and CBC. Report significant changes. *As renal function declines, progressive azotemia with increasing BUN and serum creatinine is seen. Metabolic acidosis develops as the kidney is unable to eliminate hydrogen ions and conserve bicarbonate. Hyponatremia, hyperkalemia, hyperphosphatemia, and hypocalcemia are associated with renal failure. The RBC count, hemoglobin, and hematocrit decline due to deficient erythropoietin to stimulate cell production in the bone marrow. An acute fall in hemoglobin and hematocrit may indicate GI bleeding, a risk in clients with ESRD.*
- Report manifestations of electrolyte imbalances, such as cardiac dysrhythmias and other ECG changes, muscle tremors and possible tetany, and Kussmaul's respirations. *Manifestations of electrolyte imbalance may indicate the need for intervention.*
- Administer medications to treat electrolyte imbalances as ordered. *Medications may be prescribed to help maintain electrolyte and acid–base balance and prevent adverse effects of imbalances.*

PRACTICE ALERT

Monitor carefully for desired and adverse effects of all medications. Impaired renal function affects drug elimination and increases the risk for toxic effects.

- Administer antihypertensive medications as ordered. *Hypertension management is an important factor in slowing the progression of CRF.*
- Time activities and procedures to allow rest periods. *The anemia associated with CRF may cause significant fatigue and activity intolerance.*

Imbalanced Nutrition: Less than Body Requirements

Anorexia, nausea, and vomiting are common manifestations of ESRD and uremia. The client often has a metallic taste and bad breath, which also diminish appetite. A diet restricted in protein and sodium will compound these problems. Food intake may be insufficient to meet metabolic needs. Catabolism, the breakdown of body proteins to meet energy needs, exacerbates azotemia and uremia.

NURSING CARE PLAN A Client with End-Stage Renal Disease

Walter Cohen, 45 years old, is the print shop manager at a local community college. He has been a type 1 diabetic since the age of 20, and was diagnosed with diabetic nephropathy 10 years ago. Despite blood pressure control with antihypertensive medications and frequent blood glucose monitoring with insulin coverage, he developed overt proteinuria 5 years ago and has now progressed to end-stage renal disease. He enters the nephrology unit for temporary hemodialysis to relieve uremic symptoms. While there, a CAPD catheter will be inserted. Mr. Cohen's desire to continue working is the primary factor in his choice of CAPD over hemodialysis.

ASSESSMENT

Richard Gonzalez, Mr. Cohen's care manager, obtains a nursing assessment. Mr. Cohen states that his diabetes has always been difficult to control. He has had numerous hypoglycemic episodes and has been hospitalized "four or five times" for ketoacidosis. Recently he has developed symptoms of peripheral neuropathy and increasing retinopathy. He attributed his lack of appetite, nausea, vomiting, and fatigue over the past month to "a touch of the flu." His weight remained stable, so he did not worry about not eating much.

Physical assessment findings include T 97.8°F (36.5°C) PO, P 96, R 20, and BP 178/100. Skin cool and dry, with minor excoriations on forearms and lower legs. Breath odor fetid. Scattered fine rales noted in bilateral lung bases. Soft S_3 gallop noted at cardiac apex. Bilateral pitting edema of lower extremities to just below the knees; fingers and hands also edematous. Abdominal assessment essentially normal, with hypoactive bowel sounds. Urinalysis shows a specific gravity of 1.011, gross proteinuria, and multiple cell casts. CBC results: RBC 2.9 million/mm^3; hemoglobin 9.4 g/dL; hematocrit 28%. Blood chemistry abnormalities include BUN 198 mg/dL; creatinine 18.5 mg/dL; sodium 125 mEq/L; potassium 5.7 mEq/L; calcium 7.1 mg/dL; phosphate 6.8 mg/dL. A temporary jugular venous catheter will be placed for hemodialysis the next day, followed by peritoneal catheter insertion later in the week.

DIAGNOSES

- *Excess Fluid Volume* related to failure of kidneys to eliminate excess body fluid
- *Imbalanced Nutrition: Less than Body Requirements* related to effects of uremia
- *Impaired Skin Integrity* of lower extremities related to dry skin and itching
- *Risk for Infection* related to invasive catheters and impaired immune function

EXPECTED OUTCOMES

- Adhere to the prescribed fluid restriction of 750 mL per day.
- Demonstrate reduced extracellular fluid volume by weight loss, decreased peripheral edema, clear lung sounds, and normal heart sounds.
- Consume and retain 100% of prescribed diet, including snacks.
- Demonstrate healing of lower extremity skin lesions.
- Remain free of infection.
- Demonstrate appropriate peritoneal catheter care and CAPD.

PLANNING AND IMPLEMENTATION

- Space fluids, allowing 400 mL from 0700 to 1500, 200 mL from 1500 to 2300, and 100 mL from 2300 to 0700.
- Provide mouth care at least every 4 hours and before every meal.
- Keep sugarless hard candy and ice chips at the bedside; include ice consumed as fluid intake.
- Weigh daily before breakfast; monitor vital signs, and heart and lung sounds, every 4 hours.
- Document intake and output every 4 hours.
- Arrange dietary consultation for menu planning.
- Administer prescribed antiemetic 1 hour before meals.
- Monitor food intake, noting percentage and types of food consumed.
- Clean lesions on lower extremities every 8 hours and assess healing.
- Teach CAPD procedure and peritoneal catheter care.
- Assist to identify strengths and needs in health regimen management.

EVALUATION

Mr. Cohen was hospitalized for 2 weeks, undergoing four hemodialysis sessions to reduce uremic symptoms. An arteriovenous fistula has been created in his left arm in case he should need hemodialysis in the future. He begins peritoneal dialysis the second week, and by discharge he is able to manage the catheter care and dialysis runs with the help of his wife. His heart and lung sounds are normal, and he has minimal peripheral edema on discharge. The excoriations on his legs have healed. His temperature is normal, and no evidence of infection is noted. Mr. Cohen remains anorectic and slightly nauseated, but is eating most of his prescribed diet and snacks. He has lost 10 pounds with excess fluid removal by dialysis, but his weight remains stable during the second week. Mr. Cohen and his wife have been introduced to another client who has been on CAPD for several years and promises to help them with problem solving.

CRITICAL THINKING IN THE NURSING PROCESS

1. How does diabetes mellitus damage the kidneys and lead to ESRD? Why is this more significant for a client with type 1 diabetes than for someone with type 2 diabetes (see Chapter 20 ∞)?
2. Why do high levels of urea in the blood often cause changes in cognition and mental status? What manifestations of encephalopathy would you expect to see?
3. How might Mr. Cohen's insulin dosage and diet need to be changed with the institution of peritoneal dialysis? Why?
4. Develop a care plan for the nursing diagnosis *Disturbed Body Image.*

See Evaluating Your Response in Appendix C.

- Monitor food and nutrient intake as well as episodes of vomiting. *Careful monitoring helps determine the adequacy of intake.*
- Weigh daily before breakfast. *This provides the most accurate measurement. Remember that a gain of 2 pounds or more over a 24-hour period is more likely to reflect fluid retention than a gain in body mass.*
- Administer antiemetic agents 30 to 60 minutes before eating. *Antiemetics reduce nausea and the risk of vomiting with food intake.*
- Assist with mouth care prior to meals and at bedtime. *Mouth care improves taste, stimulates the appetite, and maintains the integrity of oral mucous membranes.*
- Serve small meals and provide between-meal snacks. *Small meals are less likely to prompt nausea and help improve food intake.*
- Arrange for a dietary consultation. Provide preferred foods to the extent possible, and involve the client in planning daily menus. Encourage family members to bring food as dietary restrictions allow. *Providing preferred foods within restrictions promotes intake.*
- Monitor nutritional status by tracking weight, laboratory values such as serum albumin and BUN, and anthropometric measurements (see Chapters 21 and 22). *Indicators of impaired nutrition develop gradually and may be subtle. Careful assessment is important.*
- Administer parenteral nutrition as prescribed. Routinely monitor blood glucose levels, and use strict aseptic technique when handling the solution and venous access site. *Parenteral nutrition may be necessary to prevent catabolism and increasing azotemia. Hyperglycemia and infection are risks associated with parenteral nutrition (see* Chapter 22*). Immune system suppression associated with renal failure further increases the risk for infection.*

Risk for Infection

Chronic renal failure affects the immune system and leukocyte function, increasing susceptibility to infection. Invasive devices required for hemodialysis or peritoneal dialysis add to this risk. The client who has had a kidney transplant remains on immunosuppressive therapy for life, further depressing the immune system and increasing the risk for infection.

- Use standard precautions and good hand washing technique at all times. *Hand washing is a primary means of preventing the transfer of organisms. Clients who are on hemodialysis or who have had multiple blood transfusions to treat anemia have an increased risk for hepatitis B, hepatitis C, and HIV infection.*

PRACTICE ALERT

Use strict aseptic technique when managing ports, catheters, and incisions, to reduce the risk of introducing infectious organisms when immune responses are impaired.

- Monitor temperature and vital signs at least every 4 hours. *A low-grade fever or increased pulse rate may indicate an infection in the immunosuppressed client.*
- Monitor WBC count and differential. *Increased WBCs may indicate a bacterial infection; decreased WBCs may indicate viral infection. A shift in the differential showing more immature WBCs (bands) in circulation is another indicator of infection.*
- Culture urine, peritoneal dialysis fluid, and other drainage as indicated. *Culture is done to verify the presence of pathogens.*

PRACTICE ALERT

Monitor clarity of dialysate return. Dialysate should return clear in the client undergoing peritoneal dialysis. Cloudy dialysate may indicate peritonitis, the most common complication of peritoneal dialysis, and should be reported and cultured.

- Provide good respiratory hygiene including position changes, coughing, and deep breathing. *These measures improve clearance of respiratory secretions, reducing the risk for infection.*
- Restrict visits from obviously ill people. Teach the client and family about the risk for infection and measures to reduce the spread of infection. *The client's resistance to infection is impaired, necessitating extra caution in preventing unnecessary exposures.*

Disturbed Body Image

Chronic disease and impaired kidney function can affect the client's body image. Hemodialysis requires an arteriovenous fistula or shunt; a permanent peritoneal catheter is required for peritoneal dialysis. While kidney transplant can restore an image of wholeness, a visible scar remains and the organ may be perceived as "foreign."

- Involve the client in care, including meal planning, dialysis, and catheter, port, or incision care to the extent possible. *Involvement improves acceptance and stimulates discussion about the effect of the disease and treatment measures on the client's life.* See the Nursing Research box on the next page.
- Encourage expression of feelings and concerns, accepting perceptions and feelings without criticism. *Self-expression enhances the client's self-worth and acceptance.*
- Include the client in decision making and encourage self-care. *Increased autonomy enhances the client's sense of control, independence, and self-worth.*
- Support positive gains, but do not support denial. *The client may have difficulty accepting the renal failure, but adaptation to the loss is important.*
- Help the client develop and achieve realistic goals. *Realistic goals allow the client to see progress.*
- Provide positive reinforcement and feedback. *These measures support growth and adaptation.*
- Reinforce effective coping strategies. *Reinforcement helps the client develop positive versus negative strategies for coping.*
- Facilitate contact with a support group or other community members affected by renal failure. *The client benefits by providing and receiving support in a group of people going through similar circumstances.*
- Refer for mental health counseling as indicated or desired. *Counseling can help the client develop effective coping and adaptation strategies.*

NURSING RESEARCH Evidence-Based Practice: Client on Hemodialysis

In a study of clients undergoing hemodialysis for CRF, researchers in Sweden looked at suffering on three levels: related to sickness and treatment, related to care provided, and related to the client's unique life experience and existence (Hagren et al., 2001). The study included 15 clients between ages 50 and 86.

The researchers identified dependence on the hemodialysis machine and dependence on caregivers as the primary sources of suffering in these clients. This dependence and the loss of freedom associated with hemodialysis affected marital and family relationships and social lives of the sufferers. Accepting dependence on the hemodialysis machine and being seen as an individual by caregivers, thus promoting autonomy, relieved clients' suffering.

IMPLICATIONS FOR NURSING

Treating all clients with ESRD holistically, respecting their individual and unique characteristics and experience, is vital to promote acceptance and autonomy. Listen carefully, responding to each person's concerns. Discuss the effects of the disease and its treatment on the client's life, marital and family relationships, and socialization. Suggest strategies to maintain independence and provide relief for caregivers. When appropriate, discuss alternatives to hemodialysis for treating ESRD, such as kidney transplant or peritoneal dialysis. Peritoneal dialysis may be managed independently by the client, reducing dependence on others and time spent in treatment.

CRITICAL THINKING IN CLIENT CARE

1. Identify assessment tools and data you could use to evaluate degree of suffering in the client receiving long-term hemodialysis.
2. In addition to the above, what interventions can you, as the nurse, implement to promote autonomy and acceptance of hemodialysis in the client with ESRD?
3. Develop a teaching plan for families and significant others to help them promote acceptance and autonomy in the client undergoing long-term hemodialysis.

Community-Based Care

Chronic renal failure and ESRD are long-term processes that require client management. No matter what treatment option is chosen (hemodialysis, peritoneal dialysis, or renal transplantation), day-to-day management falls to the client and family. Teaching for home care includes the following topics:

- Nature of the kidney disease and renal failure, including expected progression and effects
- Monitoring weight, vital signs, and temperature
- Prescribed dietary and fluid restrictions (Involve the client, a dietitian, and the family member usually responsible for cooking. Include strategies to manage nausea and relieve thirst within allowed fluid limits.)
- How to assess and protect a fistula or shunt for hemodialysis (or the extremity to be used if one is anticipated)
- Peritoneal catheter care and the procedure for peritoneal dialysis as indicated (Include a family member or significant other, in case the client is unable to perform the procedure independently at some time.)
- Following kidney transplant, prescribed medications, adverse effects and their management, infection prevention, graft protection, and manifestations of organ rejection.

Refer to a dietitian for diet planning and counseling. If home hemodialysis is planned, refer the designated dialysis helper for formal training. Both the National Kidney Foundation and the American Association of Kidney Patients may be able to provide support and educational materials for the client with ESRD (see the box on page 914). Local and state chapters of these organizations can provide additional support.

EXPLORE MEDIALINK

Prentice Hall Nursing MediaLink DVD-ROM

Audio Glossary
NCLEX-RN® Review

Animation/Video

Furosemide
The Kidney

COMPANION WEBSITE www.prenhall.com/lemone

Audio Glossary
NCLEX-RN® Review
Care Plan Activity: Acute Glomerulonephritis
Case Studies
Acute Glomerulonephritis
Kidney Transplant
MediaLink Applications
Kidney Disorders
Renal Insufficiency
Links to Resources

CHAPTER HIGHLIGHTS

- Congenital and acquired disorders of the kidneys can profoundly affect urinary elimination and ultimately all body systems.
- Glomerulonephritis, inflammation of the glomerulus of the kidney, leads to loss of proteins and blood cells in the urine, a decrease in the glomerular filtration rate, and severe edema.
- The renal and cardiovascular systems are closely interrelated. Vascular disorders, such as hypertension, renal artery stenosis, or obstruction of the renal artery or vein, can have serious consequences in terms of renal function.
- Renal cell malignancies, while uncommon, often are not evident until the cancer is advanced and has metastasized to other sites.
- Acute renal failure is a frequent complication of critical illnesses, typically occurring in people with no prior history of kidney disorders. Ischemic and nephrotoxic damage to the kidney are the most common precipitating factors for ARF.
- Chronic renal failure is the end stage of numerous systemic and kidney disorders, such as diabetes mellitus, systemic lupus erythematosus, and chronic glomerulonephritis.
- When the kidneys fail, renal replacement therapies are necessary to eliminate metabolic waste products and sustain life. Dialysis and kidney transplant are the primary renal replacement therapies used.

TEST YOURSELF NCLEX-RN® REVIEW

1 The physician orders digoxin 0.125 mg three times per week for an 82-year-old client with heart failure. The nurse should:
   1. question the order because older clients frequently require larger doses of the drug due to impaired ability of the kidneys to concentrate urine.
   2. administer the drug as ordered, monitoring the client for manifestations of toxicity.
   3. assess the client's urine specific gravity and pH before administering the drug at this dose.
   4. use 0.25-mg digoxin tablets, cutting the tablet in half to save money for the client.

2 A client newly diagnosed with polycystic kidney disease asks if there is anything his children need to know about their risk for getting the disorder. The appropriate response by the nurse is:
   1. because the condition was just diagnosed, it is due to a new genetic mutation, and there is no risk of passing the condition on to his children.
   2. when his children prepare to marry, they and their potential partners should undergo genetic testing to determine if their children will be at risk.
   3. the adult form of this disorder is transmitted as a dominant gene; each child has a 50% risk of having inherited the defective gene.
   4. his children would have developed symptoms of the disorder *in utero* or shortly after birth if they had inherited the defective gene.

3 In obtaining a nursing history from a 22-year-old client admitted with a diagnosis of acute glomerulonephritis, the nurse specifically asks the client about a recent history of which of the following?
   1. urinary tract infection
   2. strep throat
   3. x-ray using contrast media
   4. illicit drug use

4 The nurse evaluates his teaching for a client with acute glomerulonephritis as effective when the client:
   1. chooses soy or animal proteins for allowed grams of protein in diet.
   2. states the need to remain on bed rest until his urine returns to clear yellow.
   3. demonstrates care for the vascular shunt or peritoneal catheter.
   4. limits fluid intake to less than 1500 mL per day.

5 Appropriate postoperative nursing interventions for the client who has had a partial or total nephrectomy include:
   1. connecting all catheters and drains to a single collection device.
   2. routine irrigation of all catheters with sterile normal saline.
   3. administering cough suppressant medication as needed.
   4. labeling and securing all catheters, tubes, and drains.

6 Important nursing interventions to prevent acute renal failure in the critically ill client include:
   1. maintaining fluid volume and cardiac output.
   2. avoiding all potentially nephrotoxic drugs.
   3. administering antihypertensive drugs.
   4. assessing for a history of diabetes or systemic lupus erythematosus.

7 The nurse evaluates her teaching as effective when the client recovering from acute renal failure states that he will:
   1. limit his fluid intake to 1500 mL or less per day.
   2. consume only vegetable proteins.
   3. avoid taking drugs that may be nephrotoxic.
   4. self-catheterize for residual urine at least once a week.

8 The nurse caring for a client preparing to undergo hemodialysis includes which of the following in the plan of care? (Select all that apply.)
   1. Obtain weight and orthostatic vital signs.
   2. Assess blood pressure of extremity where fistula has been created.
   3. Monitor serum creatinine, BUN, and hematocrit levels.
   4. Determine urine specific gravity and pH.
   5. Restrict fluid and protein intake.

9 An appropriate goal of nursing care for a client with end-stage renal disease is the client will be able to:
   1. identify a live-in caregiver.
   2. state the advantages and disadvantages of hemodialysis, peritoneal dialysis, and kidney transplant as renal replacement therapies.
   3. demonstrate the ability to independently perform hemodialysis in the home.
   4. relate the hospice philosophy and identify indicators of the need for hospice care.

10 Following a kidney transplant, the nurse notes that the client's urine is cloudy. The most appropriate response by the nurse is to:
   1. record the finding.
   2. increase the intravenous flow rate.
   3. irrigate the urinary catheter.
   4. notify the physician.

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UNIT 8 BUILDING CLINICAL COMPETENCE
Responses to Altered Urinary Elimination

FUNCTIONAL HEALTH PATTERN: Elimination

Think about clients with altered urinary elimination for whom you have cared in your clinical experiences.

- What were the clients' major medical diagnoses (e.g., urinary tract infection, kidney stones, bladder cancer, acute glomerulonephritis, acute or chronic renal failure)?
- What manifestations did each of these clients have? Was the appearance or odor of their urine affected? Did the clients complain of pain or discomfort? Were other body systems affected by their primary diagnosis (e.g., neurologic or cardiovascular)? How were their manifestations similar or different?
- How did the clients' urinary tract problems interfere with their elimination status? How many times did they urinate during the day and at night? Was their ability to control urination and completely empty their bladder affected?
- Did these clients have difficulty talking about urination or the effects of their condition on social interactions? What measures did you use to assist the clients in openly expressing their concerns?

The Elimination Pattern describes the client's patterns of excretory function, including urinary and bowel elimination and perspiration. Urinary system disorders disrupt the elimination pattern because they affect urine production and elimination.

The urinary system is responsible for regulating body fluids, filtering metabolic wastes from the bloodstream, reabsorbing needed substances and water back into the bloodstream from the filtrate, and eliminating metabolic wastes and water as urine. Any alteration in the structure and function of the urinary system can potentially affect the whole body, leading to manifestations such as:

- Hematuria (inflamed or infected urinary tract tissue ▶ local release of inflammatory mediators ▶ vasodilation and increased capillary permeability ▶ escape of WBCs and RBCs into tissue, filtrate, and urine; tumor growth and tissue invasion ▶ blood vessel damage and growth of new, fragile vessels into tumor ▶ vessel rupture and bleeding into urine)
- Proteinuria (inflammation ▶ increased glomerular capillary permeability ▶ proteins escape from the bloodstream across the capillary membrane into the filtrate ▶ presence of protein in the urine)
- Pyuria (breakdown of local defense mechanisms in urinary tract mucosa ▶ bacteria invade mucosa and multiply ▶ activation of immune responses and formation of immune complexes ▶ phagocytosis of immune complexes by neutrophils and macrophages ▶ pus formation)

Priority nursing diagnoses within the Elimination Pattern that may be appropriate for clients with urinary disorders include:

- *Impaired Urinary Elimination* as evidenced by frequency, urgency, hesitancy, dysuria, and nocturia
- *Urge Urinary Incontinence* as evidenced by frequency, urgency, loss of urine before reaching toilet, and voiding in small or large amounts
- *Urinary Retention* as evidenced by sensation of bladder fullness, dribbling urine, dysuria, and bladder distention
- *Stress Urinary Incontinence* as evidenced by dribbling urine with increased abdominal pressure, urinary urgency, and urinary frequency.

Two nursing diagnoses from other functional health patterns often are of high priority for the client with urinary elimination disorders. The first nursing diagnosis is important because of the client's need to actively manage many urinary elimination disorders. Physiologic responses to the second problem can interfere with urinary elimination:

- *Ineffective Health Maintenance* (Health Perception-Health Management)
- *Acute Pain* (Cognitive-Perceptual)

Directions: Read the clinical scenario below and answer the questions that follow. To complete this exercise successfully, you will use not only knowledge of the content in this unit, but also principles related to setting priorities and maintaining client safety.

CLINICAL SCENARIO

You have been assigned to work with the following four clients for the 0700 shift on a renal medical-surgical unit. Significant data obtained during report are as follows:

- Phillip Connor is a 45-year-old who was admitted 2 days ago after a fall from a deer hunting stand. He experienced a bruised right kidney and numerous ecchymotic areas on his right side from the fall. His vital signs are T 99°F, P 98, R 28, BP 110/68. He is complaining of abdominal pain and difficulty urinating.
- Agnes Gibson is an 84-year-old who was admitted 2 hours ago with manifestations of urinary incontinence, anorexia, confusion, and lethargy. Her vital signs on admission were T 97°F, P 88, R 20, BP 148/90. The physician ordered trimethoprim-sulfamethoxazole (Bactrim) to be started as soon as possible.
- Joseph Rouse is a 45-year-old who is to undergo surgery for removal of uric acid stones after having a failed lithotripsy. His vital signs are T 99.6°F, P 94, R 24, BP 112/68. His skin is pale, cool, and clammy. He is complaining of nausea, severe left-sided flank pain with spasms, and light-headedness.
- Angela Baldwin is a 34-year-old who has a medical history of systemic lupus erythematosus. She was admitted with complaints of left flank pain and generalized edema. Urinalysis results indicate hematuria and proteinuria. Vital signs are T 100°F, P 88, R 26, BP 144/90. She is admitted for aggressive immunosuppressive therapy.

Questions

1 In what order would you visit these patients after report?

1. _____
2. _____
3. _____
4. _____

2 What top two priority nursing diagnoses would you choose for each of the clients presented above? Can you explain, if asked, the rationale for your choices?

	Priority Nursing Diagnosis #1	Priority Nursing Diagnosis #2
Phillip Connor		
Agnes Gibson		
Joseph Rouse		
Angela Baldwin		

3 The physician ordered trimethoprim-sulfamethoxazole (Bactrim) for Agnes Gibson's uncomplicated cystitis. Mrs. Smith understands the length of antibiotic therapy when she verbalizes which statement?

1. "I should be able to return home after three days of antibiotics."
2. "I can be discharged after five days of antibiotics."
3. "I need to stay in the hospital for one week to finish the antibiotics."
4. "I can stay in the hospital for five days of antibiotics and take five days of antibiotics at home."

4 The physician orders phenazopyridine (Pyridium) for relief of pain and burning with cystitis. Which does the nurse teach the client about the use of this medication?

1. Take the medication with antacids to prevent stomach upset.
2. Drink less fluid to allow the drug to concentrate in the bladder.
3. Wear a sanitary pad to protect your clothing from stains while taking this drug.
4. Stop taking the drug if nausea and diarrhea occur.

5 The client diagnosed with uric acid stones is ordered to follow a diet low in purines. Which is the meal plan lowest in purines?

1. liver with onions and potatoes
2. chicken sandwich with french fries
3. spaghetti with ground beef meat sauce
4. macaroni and cheese with stewed tomatoes

6 When assessing a client with glomerulonephritis, which manifestations are indicative of an early disease process?

1. pyuria, leukocytosis, and hyperthermia
2. hematuria, proteinuria, and hypertension
3. dysuria, hyperglycemia, and hypertension
4. oliguria, flank pain, and hypotension

7 Which are risk factors of urinary tract infections? (Select all that apply.)

1. circumcision in males
2. decreased cervicovaginal antibodies
3. sexual intercourse in women
4. short urethra in men
5. aging in men
6. urinary catheterization

8 Which is the most accurate indicator of fluid volume status in the oliguric or aneuric client?

1. intake and output
2. weight changes
3. restricted fluids
4. BUN and creatinine levels

9 The most reliable diagnostic procedure to determine glomerular disorders is which diagnostic test?

1. kidney scan
2. antistreptolysin O titer
3. kidney biopsy
4. blood urea nitrogen

10 A new postoperative client is complaining of inability to void. A bladder ultrasound indicates that there is a significant amount of urine in the bladder. Which interventions does the nurse perform?

1. Insert a urinary catheter and completely drain the bladder at once.
2. Insert a urinary catheter and drain urine in 500-mL increments.
3. Ambulate client to the bathroom to try to void and run water in the sink.
4. Give the client a glass of water to drink to encourage voiding.

11 Which nursing actions are instituted for the client with kidney trauma?

1. Monitor level of consciousness and urine output.
2. Monitor vital signs for hypotension and bradycardia.
3. Observe for hypertension and check urine for hematuria.
4. Observe urine for oliguria and proteinuria.

12 After a client has returned from surgery, the nurse needs to report which urinary output?

1. 20 mL per hour
2. 40 mL per hour
3. 300 mL per 8 hours
4. 400 mL per 8 hours

CASE STUDY

Joe Jenkins is a 45-year-old black male who has been a truck driver for the past 20 years. He is admitted to the hospital with complaints of nausea for several weeks, weakness, fatigue, and loss of appetite. He has been feeling very depressed. He has a past medical history of type 1 diabetes mellitus, hypertension, and diabetic nephropathy. On admission, his vital signs are temperature 98.7°F, P 96, R 20, BP 170/110. He has bilateral pitting edema of the lower extremities. His fingers and hands are also edematous. He complains of dry and itching skin. His urine is dark, frothy, and scanty. A specimen is collected for a urinalysis and blood work is drawn and sent to the laboratory. Results returned with the urinalysis show a specific gravity of 1.011, gross hematuria, and 3+ protein. His blood work reveals a BUN of 198 mg/dL and creatinine of 12.5 mg/dL. Based on his past medical history of diabetes, hypertension, and diabetic nephropathy, and the current findings, a medical diagnosis of chronic renal failure is established.

Based on Mr. Jenkins' assessment and past medical history, the nursing diagnosis of *Impaired Urinary Elimination* is identified as the highest priority for planning nursing care.