CHAPTER

15

Acute Kidney Injury

Kathleen Kerber, MSN, RN, ACNS-BC, CCRN

evolve WEBSITE

Many additional resources, including self-assessment exercises, are located on the Evolve companion website at http://evolve.elsevier.com/Sole.

- Review Questions
- Mosby's Nursing Skills Procedures
- Animations
- Video Clips

INTRODUCTION

The kidney is the primary regulator of the body's internal environment. With sudden cessation of renal function, all body systems are affected by the inability to maintain fluid and electrolyte balance and eliminate metabolic waste. Renal dysfunction is a common problem in the critical care unit with nearly two thirds of patients experiencing some degree of renal dysfunction.[19,36] The most severe cases requiring renal replacement therapy have a reported mortality of 50% to 60%.[19,37]

Acute kidney injury (AKI) is the internationally recognized term for renal dysfunction in acutely ill patients.[2,8,34] In contrast to acute renal failure, AKI encompasses the range of renal dysfunction from mild impairment to complete cessation of renal function. Acute kidney injury that progresses to chronic renal failure is associated with increased morbidity and mortality, and reduced quality of life.[11] Nurses play a pivotal role in promoting positive outcomes in patients with AKI. Recognition of high-risk patients, preventive measures, sharp assessment skills, and supportive nursing care are fundamental to ensure delivery of high-quality care to these challenging and complex patients. In this chapter, the pathophysiology, assessment, and collaborative management of AKI are discussed.

REVIEW OF ANATOMY AND PHYSIOLOGY

The kidneys are a pair of highly vascularized, bean-shaped organs that are located retroperitoneally on each side of the vertebral column, adjacent to the first and second lumbar vertebrae. The right kidney sits slightly lower than the left kidney because the liver lies above it. An adrenal gland sits on top of each kidney and is responsible for the production of aldosterone, a hormone that influences sodium and water balance. Each kidney is divided into two regions: an outer region, called the *cortex,* and an inner region, called the *medulla.*

The *nephron* is the basic functional unit of the kidney. A nephron is composed of a renal corpuscle (glomerulus and Bowman's capsule) and a tubular structure, as depicted in Figure 15-1. Approximately 1 to 3 million nephrons exist in each kidney. About 85% of these nephrons are found in the cortex of the kidney and have short loops of Henle. The remaining 15% of nephrons are called *juxtamedullary nephrons* because of their location just outside the medulla. Juxtamedullary nephrons have long loops of Henle and, along with the vasa recta (long capillary loops), are primarily responsible for concentration of urine.

The kidneys receive approximately 20% to 25% of the cardiac output, which computes to 1200 mL of blood per minute. Blood enters the kidneys through the renal artery, travels through a series of arterial branches, and reaches the glomerulus by way of the afferent arteriole (*afferent* meaning to carry toward). Blood leaves the glomerulus through the efferent arteriole (*efferent* meaning to carry away from), which then divides into two extensive capillary networks called the *peritubular capillaries* and the *vasa recta.* The capillaries then rejoin to form venous branches by which blood eventually exits the kidney via the renal vein. The glomerulus is a cluster of minute blood vessels that filter blood. The glomerular walls

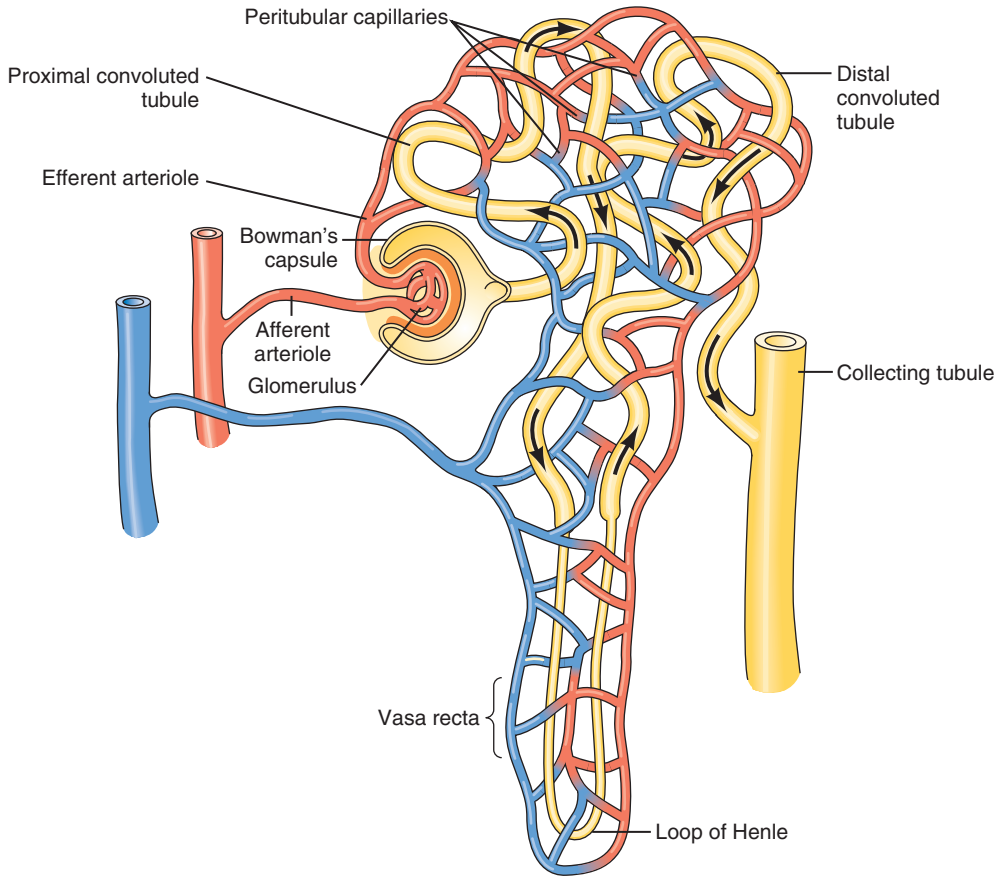

FIGURE 15-1 Anatomy of the nephron, the functional unit of the kidney. (From Banasik J. Renal function. In Copstead L, Banasik J, eds. *Pathophysiology*. 4th ed. Philadelphia: Saunders. 2010.)

BOX 15-1 FUNCTIONS OF THE KIDNEY

- Regulation of fluid volume
- Regulation of electrolyte balance
- Regulation of acid-base balance
- Regulation of blood pressure
- Excretion of nitrogenous waste products
- Regulation of erythropoiesis
- Metabolism of vitamin D
- Synthesis of prostaglandin

are composed of three layers: the endothelium, the basement membrane, and the epithelium. The epithelium of the glomerulus is continuous with the inner layer of Bowman's capsule, the sac that surrounds the glomerulus. Bowman's capsule is the entry site for filtrate leaving the glomerulus.[25]

The kidneys perform numerous functions that are essential for the maintenance of a stable internal environment. The following text provides a brief overview of key roles the kidneys perform in maintaining homeostasis. Box 15-1 lists functions of the kidney.

Regulation of Fluid and Electrolytes and Excretion of Waste Products

As blood flows through each glomerulus, water, electrolytes, and waste products are filtered out of the blood across the glomerular membrane and into Bowman's capsule, to form what is known as *filtrate*. The glomerular capillary membrane is approximately 100 times more permeable than other capillaries. It acts as a high-efficiency sieve and normally allows only substances with a certain molecular weight to cross. Normal glomerular filtrate is basically protein free and contains electrolytes, including sodium, chloride, and phosphate, and nitrogenous waste products, such as creatinine, urea, and uric acid, in amounts similar to those in plasma.[16,25] Red blood cells, albumin, and globulin are too large to pass through the healthy glomerular membrane.

Glomerular filtration occurs as a result of a pressure gradient, which is the difference between the forces that favor filtration and the pressures that oppose filtration. Generally, the capillary hydrostatic pressure favors glomerular filtration, whereas the colloid osmotic pressure and the hydrostatic pressure in Bowman's capsule oppose filtration (Figure 15-2). Under normal conditions, the capillary hydrostatic pressure

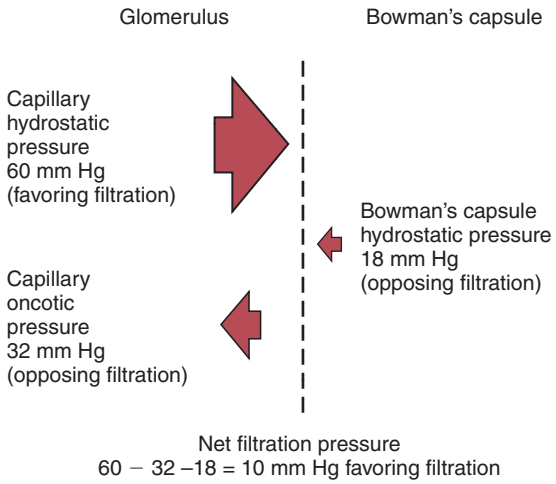

FIGURE 15-2 Average pressures involved in filtration from the glomerular capillaries.

is greater than the two opposing forces, and glomerular filtration occurs.

At a normal glomerular filtration rate (GFR) of 80 to 125 mL/min, the kidneys produce 180 L/day of filtrate. As the filtrate passes through the various components of the nephron's tubules, 99% is reabsorbed into the peritubular capillaries or vasa recta. Reabsorption is the movement of substances from the filtrate back into the capillaries. A second process that occurs in the tubules is secretion, or the movement of substances from the peritubular capillaries into the tubular network. Various electrolytes are reabsorbed or secreted at numerous points along the tubules, thus helping to regulate the electrolyte composition of the internal environment.

Aldosterone and antidiuretic hormone (ADH) play a role in water reabsorption in the distal convoluted tubule and collecting duct. Aldosterone also plays a role in sodium reabsorption and promotes the excretion of potassium. Eventually, the remaining filtrate (1% of the original 180 L/day) is excreted as urine, for an average urine output of 1 to 2 L/day.

Regulation of Acid-Base Balance

The kidneys help to maintain acid-base equilibrium in three ways: by reabsorbing filtered bicarbonate, producing new bicarbonate, and excreting small amounts of hydrogen ions (acid) buffered by phosphates and ammonia.[17] The tubular cells are capable of generating ammonia to help with excretion of hydrogen ions. This ability of the kidney to assist with ammonia production and excretion of hydrogen ions (in exchange for sodium) is the predominant adaptive response by the kidney when the patient is acidotic. When alkalosis is present, increased amounts of bicarbonate are excreted in the urine and cause the serum pH to return toward normal.

Regulation of Blood Pressure

Specialized cells in the afferent and efferent arterioles and the distal tubule are collectively known as the *juxtaglomerular apparatus.* These cells are responsible for the production of a hormone called *renin,* which plays a role in blood pressure regulation. Renin is released whenever blood flow through the afferent and efferent arterioles decreases. A decrease in the sodium ion concentration of the blood flowing past the specialized cells (e.g., in hypovolemia) also stimulates the release of renin. Renin activates the renin-angiotensin-aldosterone cascade, as depicted in Figure 15-3, which ultimately results in angiotensin II production. Angiotensin II causes vasoconstriction and release of aldosterone from the adrenal glands, thereby raising blood pressure and flow and increasing sodium and water reabsorption in the distal tubule and collecting ducts.

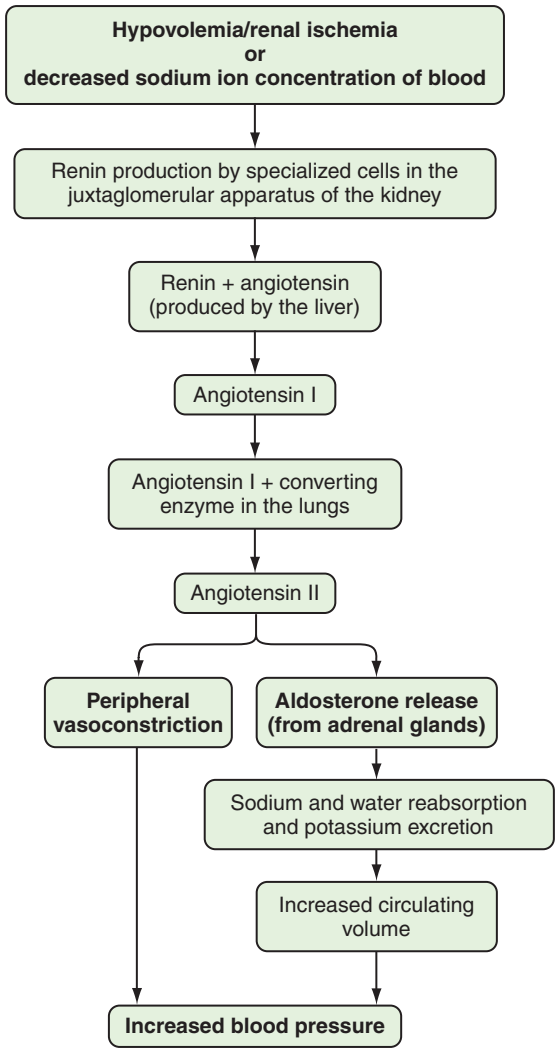

FIGURE 15-3 Renin-angiotensin-aldosterone cascade.

Effects of Aging

The most important renal physiological change that occurs with aging is a decrease in the GFR. After age 40 years, renal blood flow gradually diminishes at a rate of 10% per decade.[28] With advancing age, there is also a decrease in renal mass, the number of glomeruli, and peritubular density.[5]

Serum creatinine levels may remain the same in the elderly patient even with a declining GFR because of decreased muscle mass and hence decreased creatinine production.

The ability to concentrate and dilute urine is impaired as well, due to an inability of the renal tubules to maintain the osmotic gradient in the medullary portion of the kidney. This tubular change affects the countercurrent mechanism, significantly altering sodium conservation, especially if a salt-restricted diet is being followed. Other tubular changes include a diminished ability to excrete drugs, including radiocontrast dyes used in diagnostic testing, which necessitates a decrease in drug dosing to prevent nephrotoxicity. Many medications, including antibiotics require dose adjustments as kidney function declines. Drug databases are available for appropriate dosing.

Age-related changes in renin and aldosterone levels also occur that can lead to fluid and electrolyte abnormalities. Renin levels are decreased by 30% to 50% in the elderly, resulting in less angiotensin II production and lower aldosterone levels. Together these can cause an increased risk of hyperkalemia (with possible cardiac conduction abnormalities), a decreased ability to conserve sodium, and a tendency to develop volume depletion and dehydration. The aging kidney is also slower to correct an increase in acids, causing a prolonged metabolic acidosis and the subsequent shifting of potassium out of cells and worsening hyperkalemia. There is a slight increase in ADH production with aging, but an associated decreased responsiveness to ADH may exacerbate volume depletion and dehydration.[28]

PATHOPHYSIOLOGY OF ACUTE KIDNEY INJURY

Definition

Acute kidney injury is defined as the sudden decline in kidney function causing disturbances in fluid, electrolyte, and acid-base balance because of a loss in small solute clearance and decreased glomerular filtration rate.[10] The cardinal features of AKI are azotemia and oliguria. Azotemia refers to increases in blood urea nitrogen and serum creatinine. Oliguria is defined as urine output less than 0.5 mL/kg/hr. Two international consensus groups have worked to define and stage AKI based on serum creatinine levels and urine output. The Acute Dialysis Quality Initiative (ADQI) created the RIFLE classification system.[2] The RIFLE criteria are shown in Figure 15-4. This staging system identifies five levels with three grades of severity (risk, injury, and failure) and two outcomes (loss and end-stage renal disease). Each grade of severity is based on changes from baseline serum creatinine level or urine output over time. The Acute Kidney Injury Network (AKIN) identified three stages that correspond to the RIFLE system (risk, injury, and failure) but assess changes over 48 hours (Table 15-1).[27]

The Kidney Disease Improving Global Outcomes (KDIGO) is an international program of the National Kidney Foundation. In 2012, the KDIGO Clinical Practice Guidelines for Acute Kidney Injury were published that focus on the prevention, recognition, and management of AKI.[24] These guidelines combine both RIFLE and AKIN criteria to define and diagnose AKI (see Table 15-1).[19]

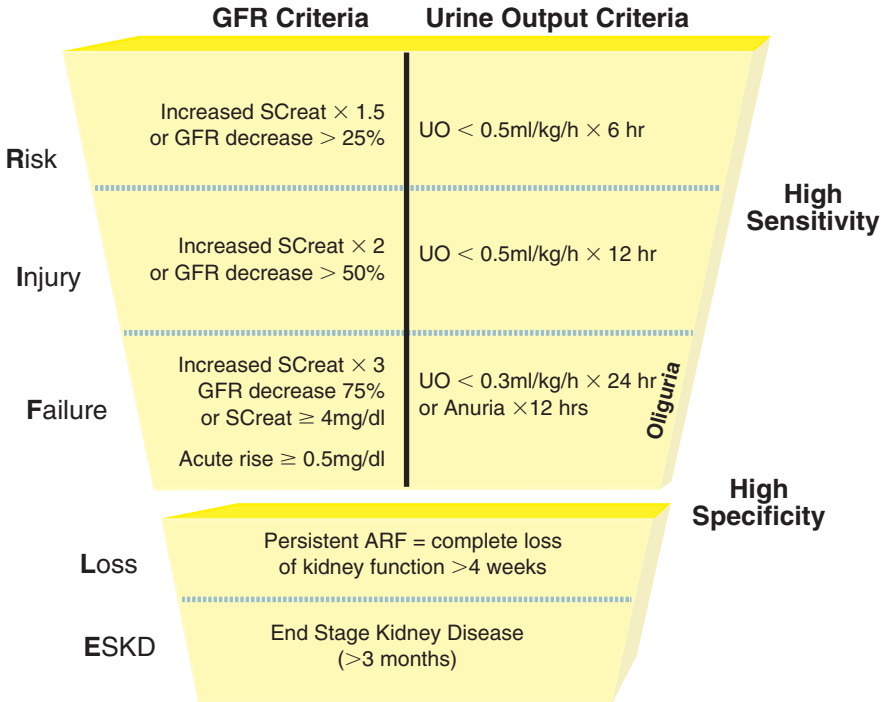

FIGURE 15-4 RIFLE classification system. *ARF,* Acute renal failure; *GFR,* glomerular filtration rate; *SCreat,* serum creatinine; *UO,* urine output. Note: The RIFLE classification system was developed before the terminology was changed from *acute renal failure* to *acute kidney injury*. (From Bellomo R, Ronc C, Kellum J, et al. Acute renal failure-definition outcome measures, animal models, fluid therapy and information technology needs: The Second International Consensus Conference of the Acute Dialysis Quality Initiative [ADHQI] Group, *Critical Care*. 2004;8[4]:R204-R212.)

TABLE 15-1 ACUTE KIDNEY INJURY NETWORK STAGING

STAGE	CRITERIA
1	Creatinine increases >0.3 mg/dL or more than or equal to 150%-200% (1.5-2.0 times baseline) >12 hours Urine output <0.5 mL/kg/hr for more than 6 hours
2	Creatinine increase 2-3 times baseline Urine output <0.5 mL/kg/hr for more than 12 hours
3	Creatinine increase 3 times baseline or >4 mg/dL with acute rise of 0.5 mg/dL Urine output <0.3 mL/kg/hr × 24 hours, or anuria for 12 hours

From Mehta RL, Kellum JA, Shah SV, Molitoris BA, Ronco C, Warnock DG & Acute Kidney Injury Network. (2007). Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. *Critical Care,* 11(2), R31.

Etiology

The etiology of AKI in critically ill patients is often multifactorial and develops from a combination of hypovolemia, sepsis, medications, and hemodynamic instability.[10] Sepsis is the most common cause of AKI.[37] The etiology of AKI is classified as either prerenal, postrenal, or intrarenal. Classification depends on where the precipitating factor exerts its pathophysiological effect on the kidney.

Prerenal Causes of Acute Kidney Injury

Conditions that result in AKI by interfering with renal perfusion are classified as *prerenal.* Most prerenal causes of AKI are related to intravascular volume depletion, decreased cardiac output, renal vasoconstriction, or pharmacological agents that impair autoregulation and GFR (Box 15-2).[8] These conditions reduce the glomerular perfusion and the GFR, and the kidneys are hypoperfused. For example, major abdominal surgery can cause hypoperfusion of the kidney as a result of blood loss during surgery, or as a result of excess vomiting or nasogastric suction during the postoperative period. The body attempts to normalize renal perfusion by reabsorbing sodium and water. If adequate blood flow is restored to the kidney, normal renal function resumes. Most forms of prerenal AKI can be reversed by treating the cause. However, if the prerenal situation is prolonged or severe, it can progress to intrarenal damage, acute tubular necrosis (ATN), or acute cortical necrosis.[23] Implementation of preventive measures, recognition of the condition, and prompt treatment of prerenal conditions are extremely important.

Postrenal Causes of Acute Kidney Injury

Acute kidney injury resulting from obstruction of the flow of urine is classified as *postrenal,* or obstructive renal injury. Obstruction can occur at any point along the urinary system (Box 15-3). With postrenal conditions, increased intratubular pressure results in a decrease in the GFR and abnormal nephron function. The presence of hydronephrosis on renal

BOX 15-2 PRERENAL CAUSES OF ACUTE KIDNEY INJURY

Intravascular Volume Depletion
- Hemorrhage
- Trauma
- Surgery
- Intraabdominal compartment syndrome
- Gastrointestinal loss
- Renal loss
- Diuretics
- Osmotic diuresis
- Diabetes insipidus
- Volume shifts
- Burns

Vasodilation
- Sepsis
- Anaphylaxis
- Medications
  - Antihypertensives
  - Afterload reducing agents
- Anesthesia

Decreased Cardiac Output
- Heart failure
- Myocardial infarction
- Cardiogenic shock
- Dysrhythmias
- Pulmonary embolism
- Pulmonary hypertension
- Positive-pressure ventilation
- Pericardial tamponade

Pharmacological Agents that Impair Autoregulation and Glomerular Filtration
- Angiotensin-converting enzyme inhibitors in renal artery stenosis
- Inhibition of prostaglandins by nonsteroidal antiinflammatory drug use during renal hypoperfusion
- Norepinephrine
- Ergotamine
- Hypercalcemia

BOX 15-3 POSTRENAL CAUSES OF ACUTE KIDNEY INJURY

- Benign prostatic hypertrophy
- Blood clots
- Renal stones or crystals
- Tumors
- Postoperative edema
- Drugs
  - Tricyclic antidepressants
  - Ganglionic blocking agents
- Foley catheter obstruction
- Ligation of ureter during surgery

ultrasound or a postvoid residual volume greater than 100 mL is suggestive of postrenal obstruction. The location of the obstruction in the urinary tract determines the method by which the obstruction is treated and may include bladder catheterization, ureteral stenting, or the placement of nephrostomy tubes.

Intrarenal Causes of Acute Kidney Injury

Conditions that produce AKI by directly acting on functioning kidney tissue (either the glomerulus or the renal tubules) are classified as *intrarenal.* The most common intrarenal condition is ATN.[8] This condition may occur after prolonged ischemia (prerenal), exposure to nephrotoxic substances, or a combination of these. Ischemic ATN usually occurs when perfusion to the kidney is considerably reduced. The renal ischemia overwhelms the normal autoregulatory defenses of the kidneys and thus initiates cell injury that may lead to cell death. Some patients have ATN after only several minutes of hypotension or hypovolemia, whereas others can tolerate hours of renal ischemia without having any apparent tubular damage

Nephrotoxic agents (particularly aminoglycosides and radiographic contrast media) damage the tubular epithelium as a result of direct drug toxicity, intrarenal vasoconstriction, and intratubular obstruction. AKI does not occur in all patients who receive nephrotoxic agents; however, predisposing factors such as advanced age, diabetes mellitus, and dehydration enhance susceptibility to intrinsic damage.[13,33] Other intrarenal causes of AKI are listed in Box 15-4.

Multiple mechanisms are involved in the pathophysiology of ATN. Figure 15-5 is a detailed schematic of some of the mechanisms that play a role in the ATN cascade resulting in a reduced GFR. Mechanisms include alterations in renal hemodynamics, tubular function, and tubular cellular metabolism.

Decreases in cardiac output, intravascular volume, or renal blood flow activate the renin-angiotensin-aldosterone cascade. Angiotensin II causes further renal vasoconstriction and decreased glomerular capillary pressure, resulting in a decreased GFR. The decreased GFR and renal blood flow lead to tubular dysfunction. In addition, administration of medications that cause vasoconstriction of the renal vessels can precipitate ATN, including nonsteroidal antiinflammatory drugs, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, cyclosporine, and tacrolimus.[8,23] Endogenous substances that have been implicated in both causing and maintaining renal vessel vasoconstriction include endothelin-1, prostaglandins, adenosine, angiotensin II, and nitric oxide. A deficiency of renal vasodilators (prostaglandins, atrial natriuretic peptide, and endothelium-derived nitric oxide) has also been implicated.[8]

The renal tubules in the medulla are very susceptible to ischemia. The medulla receives only 20% of the renal blood flow but is very sensitive to any reduction in blood flow. When the tubules are damaged, necrotic endothelial cells and other cellular debris accumulate and can obstruct the lumen of the tubule. This intratubular obstruction increases the intratubular pressure, which decreases the GFR and leads to tubular dysfunction. In addition, the tubular damage often produces alterations in the tubular structure that permit the glomerular filtrate to leak out of the tubular lumen and back into the plasma, resulting in oliguria.[8]

Ischemic episodes result in decreased energy supplies, including adenosine triphosphate (ATP). Oxygen deprivation results in a rapid breakdown of ATP. The proximal tubule is very dependent on ATP, which explains why it is the most commonly injured portion of the renal tubule. Without ATP, the sodium-potassium ATPase of the cell membrane is not able to effectively transport electrolytes across the membrane. This leads to increased intracellular calcium levels, free

BOX 15-4 INTRARENAL CAUSES OF ACUTE KIDNEY INJURY

Glomerular, Vascular, or Hematological Problems

- Glomerulonephritis (poststreptococcal)
- Vasculitis
- Malignant hypertension
- Systemic lupus erythematosus
- Hemolytic uremic syndrome
- Disseminated intravascular coagulation
- Scleroderma
- Bacterial endocarditis
- Hypertension of pregnancy
- Thrombosis of renal artery or vein

Tubular Problem (Acute Tubular Necrosis or Acute Interstitial Nephritis)

- Ischemia
- Causes of prerenal azotemia (see Box 15-2)
- Hypotension from any cause
- Hypovolemia from any cause
- Obstetric complications (hemorrhage, abruptio placentae, placenta previa)
- Medications (see Box 15-5)
- Radiocontrast media (large volume; multiple procedures)
- Transfusion reaction causing hemoglobinuria
- Tumor lysis syndrome
- Rhabdomyolysis
- Miscellaneous: heavy metals (mercury, arsenic), paraquat, snake bites, organic solvents (ethylene glycol, toluene, carbon tetrachloride), pesticides, fungicides
- Preexisting renal impairment
- Diabetes mellitus
- Hypertension
- Volume depletion
- Severe heart failure
- Advanced age

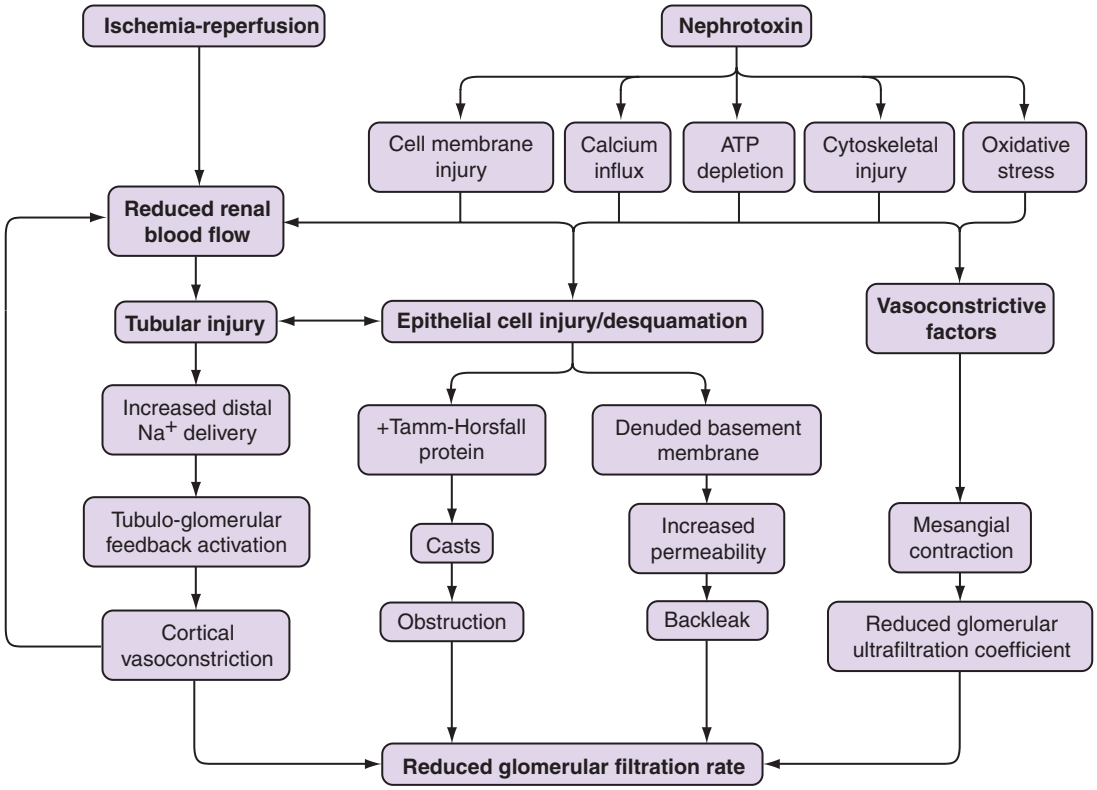

FIGURE 15-5 Schematic of loss of glomerular filtration seen in ischemic and nephrotoxic acute tubular necrosis. *ATP,* adenosine triphosphate; *Na^+*, sodium. (From Woolfson R, Hillman K. Causes of acute renal failure. In Johnson R, Feehally, eds. *Comprehensive Clinical Nephrology.* 2nd ed. London: Mosby. 2003.)

radical formation (which produces toxic effects), and breakdown of phospholipids. Cellular edema occurs and further decreases renal blood flow, damages the tubules, and ultimately leads to tubular dysfunction and oliguria.[8]

Contrast-induced nephropathy. Though the administration of contrast is generally considered safe for the individual with normal kidney function, contrast-induced nephropathy (CIN) is the third leading cause of AKI in the hospitalized patient[3,21] (see box, "Evidence-Based Practice"). *Contrast-induced nephropathy* is defined as the sudden, rapid deterioration of kidney function resulting from parenteral contrast administration in the absence of another clinical explanation.[21] Contrast-induced kidney injury is diagnosed by an increase in serum creatinine of 25% or more, or a value of 0.5 mg/dL or more, occurring within 48 to 72 hours following the administration of contrast.[33] Urine output usually remains normal; however, in severe cases oliguria may be seen.

Two pathological mechanisms contribute to the development of contrast-induced AKI. The first mechanism is by the direct toxic effect of the contrast media on the cells lining the renal tubule.[31,33] The second mechanism of injury is the result of reduced medullary blood flow.

Contrast media is suspected to initiate vasodilation of renal blood vessels followed by an intense and persistent vasoconstriction.[8] Oxygen delivery to the renal cells is reduced, precipitating cell injury. In addition, contrast agents stimulate the influx of extracellular calcium, which may lead to a loss of medullary autoregulation as well as a direct toxic effect on the renal tubules.[33] Patients with chronic renal insufficiency are at the greatest risk for developing CIN.[8] Other risk factors include diabetes, dehydration, advancing age, heart failure, ongoing treatment with nephrotoxic drugs, and vascular disease.[3,33]

Reduced medullary blood flow from cholesterol embolism or atheromatous emboli are common causes of AKI after an interventional radiology procedure. Any arterial angiographic procedure, such as cardiac catheterization, can dislodge atheromatous emboli, which can lodge in small renal arteries and produce an occlusion of the vessel, ischemia, and tubular dysfunction. A decline in renal function typically occurs over a period of 3 to 8 weeks, rather than the rapid decline seen with CIN. Patients also typically have evidence of embolization to other areas of the body, including the skin (digital necrosis and gangrene), central nervous system (stroke, blindness), or gastrointestinal system (pancreatitis).

EVIDENCE-BASED PRACTICE

Acute Kidney Injury Related to Contrast Media

Problem

Contrast-induced nephropathy is the third leading cause of AKI in hospitalized patients and is associated with significant patient morbidity, prolongation of hospital stays, and increased healthcare costs. Critically ill patients are at increased risk for contrast-induced nephropathy because of hemodynamic instability, volume depletion, multiple organ dysfunction, and the use of nephrotoxic medications. Critically ill diabetic patients receiving radiological contrast have multiple risk factors for contrast-induced nephropathy. Preventive measures are needed to reduce the risk of contrast-induced nephropathy in high risk populations.

Clinical Question

What are the most effective interventions for preventing contrast-induced AKI?

Evidence

Many studies have been conducted to evaluate interventions to reduce the risk of contrast-induced nephropathy; however, results have been inconsistent. Hydration is the intervention that has demonstrated benefit in most randomized controlled trials. Data are controversial on which intravenous fluid is best for hydration. Although isotonic saline has been identified as effective, intravenous administration of a 154 mEq/L solution of sodium bicarbonate has been proposed as an effective method of hydration that offers additional protection from the alkalinizing properties of contrast agents.

The PREVENT Trial compared the ability of sodium bicarbonate plus *N*-acetylcysteine (NAC) versus sodium chloride plus NAC to prevent contrast-induced nephropathy in 382 diabetic patients with impaired renal function undergoing coronary or endovascular angiography or interventions.[2] The findings of this trial indicated hydration with sodium bicarbonate was not superior to hydration with sodium chloride in preventing contrast-induced nephropathy in the study population.

The CIN Consensus Working Panel recommends that adequate intravenous volume expansion with isotonic crystalloid (normal saline [0.9%], 1.0-1.5 mL/kg/hr) for 3 to 12 hours before the procedure and continue for 6 to 24 hours afterward can lessen the probability of contrast-induced nephropathy in patients at risk.[3]

Implications for Nursing

The findings of this study support current recommendations for the use of normal saline for hydration. High-risk hospitalized patients can begin intravenous hydration 12 hours before the procedure, and the infusion can be continued for at least 6 to 12 hours afterward. For outpatients, especially those with risk factors for contrast-induced nephropathy, intravenous hydration can be started 3 hours before the procedure and continued for 6 or more hours afterwards. The recommended fluid administration rate is 1 mL/kg/hr.[1] In some circumstances, the physician may request a rate of 2 mL/kg/hr for the first 2 hours followed by a rate of 1 mL/kg/hr.[1] Ongoing clinical trials will determine additional preventive strategies in hopes of reducing the incidence of contrast-induced nephropathy. Nurses must assist in identifying patients at risk for contrast-induced nephropathy and advocating for early and adequate hydration.

Level of Evidence

B—Controlled studies with consistent results

References

1. Ellis J & Cohan R. Prevention of contrast-induced nephropathy: an overview. *Radiologic Clinics North America,* 2009;47, 801-811.
2. Lee S, Kim W, Kim Y, Park S, Park D, Yun S, Lee J, Kang S, Lee C, Park S. Preventive strategies of renal insufficiency in patient with diabetes undergoing intervention or arteriography (the PREVENT trial). *American Journal of Cardiology*, 2011;107(10), 1447-1452.
3. Kellum J, Lamiere N, Aspelin P, Barsoum R, Burdman E, Goldstein S, et al. (2012). KDIGO Clinical practice guidelines for acute kidney injury. *Kidney International*, 2(1) (Supp.l) S1-138.

Course of Acute Kidney Injury

The patient with AKI progresses through three phases of the disease process: the initiation phase, the maintenance phase, and the recovery phase.[31]

Initiation Phase

The initiation phase is the period that elapses from the occurrence of the precipitating event to the beginning of the change in urine output. This phase spans several hours to 2 days, during which time the normal renal processes begin to deteriorate, but actual intrinsic renal damage has not yet occurred. The patient is unable to compensate for the diminished renal function and exhibits clinical signs and symptoms that reflect the chemical imbalances. Renal dysfunction is potentially reversible during the initiation phase.

Maintenance Phase

During the maintenance phase, intrinsic renal damage is established, and the GFR stabilizes at approximately 5 to 10 mL/min. Urine volume is usually at its lowest point during the maintenance phase; however, patients may be nonoliguric, with urine outputs greater than 400 mL in 24 hours. This phase usually lasts 8 to 14 days, but it may last up to 11 months. The longer a patient remains in this stage, the slower the recovery and

the greater the chance of permanent renal damage. Complications resulting from uremia, including hyperkalemia and infection, occur during this phase.

Recovery Phase

This phase is the period during which the renal tissue recovers and repairs itself. A gradual increase in urine output and an improvement in laboratory values occur. Some patients may experience diuresis during this phase. This diuresis reflects excretion of salt and water accumulated during the maintenance phase, the osmotic diuresis induced by filtered urea and other solutes, and the administration of diuretics to enhance salt and water elimination.[8] However, with early and aggressive use of dialytic therapy, many patients are maintained in a relative "dry" or volume-depleted state and do have a large post-ATN diuresis. Recovery may take as long as 4 to 6 months.

ASSESSMENT

Patient History

Obtaining a thorough patient history is important. Renal-related symptoms provide valuable clues to assist the clinician in focusing the assessment. For example, dysuria, frequency, incontinence, nocturia, pyuria, and hematuria can be indicative of urinary tract infection. The history provides clues about medical conditions that predispose the patient to AKI, including diabetes mellitus, hypertension, immunological diseases, and any hereditary disorders, such as polycystic disease. The medical record is reviewed to elicit additional risk factors, such as hypotensive episodes or any surgical or radiographic procedures performed. Information regarding exposure to potential nephrotoxins is extremely important. Common nephrotoxins include antibiotics such as aminoglycosides. Risk factors for development of aminoglycoside nephrotoxicity include volume depletion, prolonged use of the drug (>10 days), hypokalemia, sepsis, preexisting renal disease, high trough concentrations, concurrent use of other nephrotoxic drugs, and older age.[31] Symptoms of AKI are usually seen about 1 to 2 weeks after exposure. Because of this delay, the patient must be questioned about any recent medical visits (clinic or emergency department) for which an aminoglycoside may have been prescribed. In addition, a history of over-the-counter medication use, including nonsteroidal antiinflammatory medications, is important. Box 15-5 lists medications that are associated with AKI.

Vital Signs

Changes in blood pressure are common in AKI. Patients with kidney injury from prerenal causes may be hypotensive and tachycardic as a result of volume deficits. ATN, particularly if associated with oliguria, often causes hypertension. Patients may hyperventilate as the lungs attempt to compensate for the metabolic acidosis often seen in AKI. Body temperature may be decreased (as a result of the antipyretic effect of the uremic toxins), normal, or increased (as a result of infection).

BOX 15-5 COMMON NEPHROTOXIC MEDICATIONS

- Aminoglycosides
- Amphotericin B
- Penicillins
- Acyclovir
- Vancomycin
- Pentamidine
- Rifampin
- Cephalosporins
- Cyclosporine
- Tacrolimus
- Methotrexate
- Cisplatin
- Fluorouracil (5-FU)
- Nonsteroidal antiinflammatory drugs (NSAIDs)
- Angiotensin-converting enzyme (ACE) inhibitors
- Angiotensin receptor blockers (ARBs)
- Interferon
- Indinavir
- Ritonavir
- Adefovir

Physical Assessment

The patient's general appearance is assessed for signs of *uremia* (retention of nitrogenous substances normally excreted by the kidneys) such as malaise, fatigue, disorientation, and drowsiness. The skin is assessed for color, texture, bruising, petechiae, and edema. The patient's hydration status is also carefully assessed. Current and admission body weight and intake and output information are evaluated. Skin turgor, mucous membranes, breath sounds, presence of edema, neck vein distention, and vital signs (blood pressure and heart rate) are all key indicators of fluid balance. An oliguric patient with weight loss, tachycardia, hypotension, dry mucous membranes, flat neck veins, and poor skin turgor may be volume depleted (prerenal cause). Weight gain, edema, distended neck veins, and hypertension in the presence of oliguria suggest an intrarenal cause. Table 15-2 summarizes the systemic manifestations of AKI according to body system and also lists the pathophysiological mechanisms involved.

Evaluation of Laboratory Values

Alteration in renal function is associated with changes in serum and urine laboratory values. The serum creatinine level is often used to evaluate kidney function. Creatinine is a by-product of muscle metabolism and is produced at a relatively constant rate, then cleared by the kidneys. With stable kidney function, creatinine production and excretion are fairly equal, and serum creatinine levels remain constant. When kidney function decreases, creatinine levels rapidly rise, indicating a decline in function or a decrease in the GFR. The serum

TABLE 15-2 SYSTEMIC MANIFESTATIONS OF ACUTE KIDNEY INJURY

SYSTEM	MANIFESTATION	PATHOPHYSIOLOGICAL MECHANISM
Cardiovascular	Heart failure	Fluid overload and hypertension
	Pulmonary edema	↑ Pulmonary capillary permeability
		Fluid overload
		Left ventricular dysfunction
	Dysrhythmias	Electrolyte imbalances (especially hyperkalemia and hypocalcemia)
	Peripheral edema	Fluid overload
		Right ventricular dysfunction
	Hypertension	Fluid overload
		↑ Sodium retention
Hematological	Anemia	↓ Erythropoietin secretion
		Loss of RBCs through GI tract, mucous membranes, or dialysis
		↓ RBC survival time
		Uremic toxins' interference with folic acid secretion
	Alterations in coagulation	Platelet dysfunction
	↑ Susceptibility to infection	↓ Neutrophil phagocytosis
Electrolyte imbalances	Metabolic acidosis	↓ Hydrogen ion excretion
		↓ Bicarbonate ion reabsorption and generation
		↓ Excretion of phosphate salts or titratable acids
		↓ Ammonia synthesis and ammonium excretion
Respiratory	Pneumonia	Thick tenacious sputum from ↓ oral intake
		Depressed cough reflex
		↓ Pulmonary macrophage activity
	Pulmonary edema	Fluid overload
		Left ventricular dysfunction
		↑ Pulmonary capillary permeability
Gastrointestinal	Anorexia, nausea, vomiting	Uremic toxins
		Decomposition of urea releasing ammonia that irritates mucosa
	Stomatitis and uremic halitosis	Uremic toxins
		Decomposition of urea releasing ammonia that irritates oral mucosa
	Gastritis and bleeding	Uremic toxins
		Decomposition of urea releasing ammonia that irritates mucosa, causing ulcerations and increased capillary fragility
Neuromuscular	Drowsiness, confusion, irritability, and coma	Uremic toxins produce encephalopathy
		Metabolic acidosis
		Electrolyte imbalances
	Tremors, twitching, and convulsions	Uremic toxins produce encephalopathy
		↓ Nerve conduction from uremic toxins
Psychosocial	Decreased mentation, decreased concentration, and altered perceptions	Uremic toxins produce encephalopathy
		Electrolyte imbalances
		Metabolic acidosis
		Tendency to develop cerebral edema
Integumentary	Pallor	Anemia
	Yellowness	Retained urochrome pigment
	Dryness	↓ Secretions from oil and sweat glands
	Pruritus	Dry skin
		Calcium and/or phosphate deposits in skin
		Uremic toxins' effect on nerve endings
	Purpura	↑ Capillary fragility
		Platelet dysfunction
	Uremic frost (rarely seen)	Urea or urate crystal excretion
Endocrine	Glucose intolerance (usually not clinically significant)	Peripheral insensitivity to insulin
		Prolonged insulin half-life from ↓ renal metabolism
Skeletal	Hypocalcemia	Hyperphosphatemia from ↓ excretion of phosphates ↓
		↓ GI absorption of vitamin D
		Deposition of calcium phosphate crystals in soft tissues

GI, Gastrointestinal; *RBC,* red blood cell.

! CLINICAL ALERT

Serum Creatinine

The same serum creatinine level can reflect very different glomerular filtration rates in patients because of differences in muscle mass. For example, a 25-year-old man weighing 220 lb with a serum creatinine level of 1.2 mg/dL has an estimated glomerular filtration rate of 133 mL/hr (normal), whereas a 75-year-old woman weighing 121 lb with the same serum creatinine level of 1.2 mg/dL has an estimated glomerular filtration rate of 35 mL/hr (markedly decreased).

creatinine level should not be the only measure used to assess kidney function (see box, "Clinical Alert: Serum Creatinine"). When evaluating the serum creatinine level, it is helpful to review past values to determine whether an elevated level is due to an acute insult or a progressive loss of renal function. If past creatinine levels are not available, it is often difficult to distinguish acute from chronic kidney failure.

Although the serum blood urea nitrogen (BUN) level is also used to evaluate kidney function, the BUN level is not a reliable indicator of kidney function because the rate of protein metabolism (urea is a by-product of protein metabolism) is not constant. Extrarenal factors including dehydration, a high-protein diet, starvation, blood in the gastrointestinal tract, corticosteroids, and fever all can elevate the BUN level. For example, when a patient has gastrointestinal bleeding, the blood in the gut breaks down and results in an increased protein load and hence an elevated BUN level.

The *BUN/creatinine ratio* provides useful information. The normal BUN/creatinine ratio is 10:1 to 20:1 (e.g., BUN level, 20 mg/dL, and creatinine level, 1.0 mg/dL). If the ratio is greater than 20:1 (e.g., BUN level, 60 mg/dL, and creatinine level, 1.0 mg/dL), problems other than kidney failure should be suspected. In prerenal conditions, an increased BUN/creatinine ratio is typically noted. There is a decrease in the GFR and hence a drop in urine flow through the renal tubules. This allows more time for urea to be reabsorbed from the renal tubules back into the blood. Creatinine is not readily reabsorbed; therefore the serum BUN level rises out of proportion to the serum creatinine level. A normal BUN/creatinine ratio is present in ATN, where there is actual injury to the renal tubules and a rapid decline in the GFR. Hence urea and creatinine levels both rise proportionally from increased reabsorption and decreased clearance.[8]

Assessment of the urine is important in the evaluation of AKI. Historically, timed 24-hour urine collections have been used to evaluate GFR or creatinine clearance. Timed urine collections are cumbersome and time-consuming, and are susceptible to multiple errors in collection. To measure creatinine clearance accurately, the nurse and patient must rigidly adhere to the following procedure:

1. The patient empties his or her bladder, the exact time is recorded, and the specimen is discarded.
2. All urine for the next 24 hours is saved in a container and stored in a refrigerator.
3. Exactly 24 hours after the start of the procedure, the patient voids again, and the specimen is saved.
4. The serum creatinine level is assessed at the end of 24 hours.
5. The 24-hour urine collection is sent to the laboratory for testing. (Urine can also be obtained from an indwelling urinary catheter.)

Urinary *creatinine clearance* is calculated with the following formula:

$$U_c \times V/P_c = C_{cr}$$

U_c = concentration of creatinine in the urine
V = volume of urine per unit of time
P_c = concentration of creatinine in the plasma
C_{cr} = creatinine clearance

Creatinine clearance is an estimate of GFR and is measured in mL/min. Thus, given the following set of patient data,

U_c = 175 mg/100 mL
V = 288 mL/1440 min (24 hours = 1440 min)
P_c = 17.5 mg/100 mL

the patient's creatinine clearance would be calculated as follows:

$$\frac{175 \text{ mg/100 mL} \times 288 \text{ mL/1440 min}}{17.5 \text{ mg/100 mL}} = 2 \text{ mL/min}$$

Because a normal creatinine clearance is about 84 to 138 mL/min, the clinician recognizes this patient's creatinine clearance as being consistent with severe renal dysfunction.

If a reliable 24-hour urine collection is not possible, the Cockcroft and Gault formula may be used to determine the creatinine clearance from a serum creatinine value[23,26]:

$$C_{cr} = \frac{(140 - \text{Age [yr]}) \times (\text{Lean body weight [kg]})}{72 \times \text{Serum creatinine (mg/dL)}}$$

For women, the calculated result is multiplied by 0.85 to account for the smaller muscle mass as compared to men.

Analysis of urinary sediment and electrolyte levels is helpful in distinguishing among the various causes of AKI. Urine is inspected for the presence of cells, casts, and crystals. In prerenal conditions, the urine typically has no cells but may contain hyaline casts. Casts are cylindrical bodies that form when proteins precipitate in the distal tubules and collecting ducts. Postrenal conditions may present with stones, crystals, sediment, bacteria, and clots from the obstruction. Coarse, muddy brown granular casts are classic findings in ATN.[8] Microscopic hematuria and a small amount of protein may also be seen on a random urine specimen. If a 24-hour urine specimen is collected, microalbumin levels are usually less than 30 mg/L, but vary with many factors such as age, activity, and infection.

Urine electrolyte levels help to discriminate between prerenal causes and ATN. The nurse obtains urine samples (often called spot urine levels) for electrolyte determinations before diuretics are administered because these drugs alter the urine results for up to 24 hours. Urinary sodium concentrations of less than 10 mEq/L are seen in prerenal conditions, as the kidneys attempt to conserve sodium and water to compensate for the hypoperfusion state. Urine sodium concentrations are greater than 40 mEq/L in

ATN as a result of impaired reabsorption in the diseased tubules.[8]

The fractional excretion of sodium (FE_{Na}) is a useful test for assessing how well the kidney can concentrate urine and conserve sodium. To determine the FE_{Na}, the following formula is used:

$$FE_{Na} = \frac{\text{(Urine sodium) (Serum creatinine)} \times 100}{\text{(Urine creatinine) (Serum sodium)}}$$

In prerenal conditions, the FE_{Na} is less than 1%, whereas ATN presents with a FE_{Na} of greater than 1%.[8,11] Table 15-3 summarizes laboratory data useful in differentiating among the three categories of AKI.

Urine specific gravity and osmolality have a limited role in the diagnosis of AKI, especially in older adults, because the body's ability to concentrate urine decreases with age (see box, "Geriatric Considerations").[5,29] In general, prerenal conditions cause concentrated urine (high specific gravity and osmolality), whereas intrinsic azotemia causes dilute urine (low specific gravity and osmolality). The volume of urine output is also not a good indicator of renal function. Although patients with nonoliguric AKI excrete large volumes of fluid with little solute, they still have renal dysfunction and azotemia. In an older adult, assessment parameters are modified when assessing for acute renal failure.

TABLE 15-3 LABORATORY FINDINGS USEFUL IN DIFFERENTIATING CAUSES OF ACUTE KIDNEY INJURY

TYPE OF ACUTE KIDNEY INJURY	SPECIFIC GRAVITY	URINE OSMOLALITY	URINE SODIUM	MICROSCOPIC EXAMINATION	BUN/CR RATIO	FE_{NA}
Prerenal	>1.020	>500 mOsm/L	<10 mEq/L	Few hyaline casts possible	Elevated	<1%
Intrarenal	1.010	<350 mOsm/L	>20 mEq/L	Epithelial casts, red blood cell casts, pigmented granular casts	Normal	>1%
Postrenal	Normal to 1.010	Variable	Normal to 40 mEq/L	May have stones, crystals, sediment, clots, or bacteria	Normal	>1%

BUN, Blood urea nitrogen; *CR,* creatinine; *FE_{Na},* fractional excretion of sodium.

GERIATRIC CONSIDERATIONS

Management of Acute Kidney Injury

- Older adults are at increased risk for AKI related to comorbidities such as diabetes mellitus, hypertension, and from polypharmacy. Commonly prescribed classes of medications that have adverse effects on renal blood flow are nonsteroidal antiinflammatory drugs and angiotensin-converting enzyme inhibitors.[31]
- The aging kidney is more susceptible to nephrotoxic and ischemic injury. Monitor drug dosages carefully, adjust drug dosages for underlying renal insufficiency, and use nephrotoxic agents judiciously.
- The primary risk factor for contrast-induced nephropathy is a preexisting decline in renal function, which places the elderly patient at risk. Monitor radiographic contrast media usage closely, using only as necessary. Maintain adequate hydration if radiographic contrast media must be used.
- Older adults are prone to developing volume depletion (prerenal conditions) because of a decreased ability to concentrate urine and conserve sodium. Volume status is difficult to assess because of altered skin turgor and decreased skin elasticity, decreased baroreceptor reflexes, and mouth dryness caused by mouth breathing. Be sure fluids are easily within reach of older adults not on fluid restriction. Offer fluids frequently if not on fluid restriction (diminished thirst response and may not feel thirsty). Provide intravenous fluids to maintain adequate hydration as prescribed.
- Urinary indices are of limited value in assessment of older adults because of impaired ability to concentrate urine.
- Older patients tend to exhibit uremic symptoms at lower levels of serum blood urea nitrogen and creatinine than do younger patients. The typical signs and symptoms of AKI may be attributed to other disorders associated with aging, thus delaying prompt diagnosis and treatment.
- Atypical signs and symptoms of uremia may be seen, such as an unexplained exacerbation of well-controlled heart failure, unexplained mental status changes, or personality changes.
- Older adults often have poor nutritional status before AKI and require early and adequate nutrition.
- Older adults have special needs in regard to renal replacement therapies. They may need dialysis or continuous renal replacement therapy earlier than younger patients, because they become symptomatic with lower serum creatinine and blood urea nitrogen levels. They are at an increased risk for vascular access problems secondary to diabetes mellitus and peripheral vascular disease. Keep ultrafiltration rate less than 1 L/hr because decreased cardiac reserve and autonomic dysfunction make ultrafiltration difficult.
- Supply supplemental oxygen if needed to offset the hypoxemia that often develops at the start of dialysis. Monitor for increased risk of complications associated with systemic heparinization, including subdural hematomas from falls and gastritis.
- Older adults are more prone to infection because of a compromised immune system. Use meticulous technique for all procedures. Avoid indwelling urinary catheters especially if the patient is anuric; use intermittent catheterization as necessary.

TABLE 15-4 INVASIVE DIAGNOSTIC PROCEDURES FOR ASSESSING THE RENAL SYSTEM

PROCEDURE	PURPOSE	POTENTIAL PROBLEMS
Intravenous pyelography	To visualize the renal parenchyma, calyces, renal pelvis, ureters, and bladder to obtain information regarding size, shape, position, and function of the kidneys	Hypersensitivity reaction to contrast media Acute kidney injury
Computed tomography	To visualize the renal parenchyma to obtain data regarding the size, shape, and presence of lesions, cysts, masses, calculi, obstructions, congenital anomalies, and abnormal accumulations of fluid	Hypersensitivity reaction to contrast media (if used)
Renal angiography	To visualize the arterial tree, capillaries, and venous drainage of the kidneys to obtain data regarding the presence of tumors, cysts, stenosis infarction, aneurysms, hematomas, lacerations, and abscesses	Hypersensitivity reaction to contrast media Hemorrhage or hematoma at the catheter insertion site Acute kidney injury
Renal scanning	To determine renal function by visualizing the appearance and disappearance of the radioisotopes within the kidney; also provides some anatomical information	Hypersensitivity reaction to contrast media
Renal biopsy	To obtain data for making a histological diagnosis to determine the extent of the pathology, the appropriate therapy, and the possible prognosis	Hemorrhage Postbiopsy hematoma

DIAGNOSTIC PROCEDURES

Various diagnostic procedures are used to evaluate renal function. Noninvasive diagnostic procedures are usually performed before any invasive diagnostic procedures are conducted. Noninvasive diagnostic procedures that assess the renal system are radiography of the kidneys, ureters, and bladder (KUB), renal ultrasonography, and magnetic resonance imaging (MRI). A KUB x-ray delineates the size, shape, and position of the kidneys. It may also detect abnormalities such as calculi, hydronephrosis (dilatation of the renal pelvis), cysts, or tumors. Renal ultrasound is helpful in evaluating for obstruction, which is manifest by hydronephrosis or hydroureter (dilatation of the ureters). Ultrasound can also document the size of the kidneys, which may be helpful in differentiating acute from chronic renal conditions. The kidneys are often small (<10 cm) in chronic kidney disease. Real-time ultrasound is used during renal biopsy and during placement of percutaneous nephrostomy tubes (often placed for hydronephrosis). MRI provides anatomical information about renal structures.

Invasive diagnostic procedures for assessing the renal system include intravenous pyelography, computed tomography, renal angiography, renal scanning, and renal biopsy.[3] These procedures are summarized in Table 15-4.

As for all diagnostic procedures, the nurse instructs the patient, assists with the procedures, and monitors the patient after the procedure. When workup is done for AKI, it is also important to assess for allergies to contrast media and provide appropriate fluids to the patient to maintain hydration before and after the procedures. Urinary output is closely monitored after the procedure.

NURSING DIAGNOSES

Nursing care of the patient with acute kidney injury is complex. Multiple nursing diagnoses must be dealt with in these often critically ill patients. The Nursing Care Plan for the Patient with Acute Kidney Injury (see box) addresses nursing diagnoses, patient outcomes, and interventions.

NURSING CARE PLAN

for the Patient with Acute Kidney Injury

NURSING DIAGNOSIS

Excess Fluid Volume related to sodium and water retention and excess intake

PATIENT OUTCOMES

Stable fluid balance

- Body weight within 2 lb of dry weight
- Intake and output balanced; bilateral breath sounds clear; vital signs normal

NURSING INTERVENTIONS	RATIONALES
• Obtain daily weights	• Weight gain is best indicator of fluid gain
• Maintain accurate intake and output records	• Identify imbalances
• Monitor respiratory status, including respiratory rate and crackles	• Assess volume overload
• Assess heart rate, blood pressure, and respiratory rate	• Hypertension, tachycardia, and tachypnea indicate volume overload
• Administer all fluids and medications in the least amount of fluid possible	• Minimize intake
• Monitor blood and urine laboratory tests	• Levels are altered in acute kidney injury

NURSING DIAGNOSIS

Risk for Infection related to depressed immune response secondary to uremia and Impaired Skin Integrity

PATIENT OUTCOMES

Absence of infection

- Infection is absent
- Patient is afebrile
- WBC count and differential are normal
- All cultures are negative

NURSING INTERVENTIONS	RATIONALES
• Monitor WBC count and culture results	• Detect infection early
• Monitor temperature	• Fever may indicate infection
• Avoid invasive equipment whenever possible, such as indwelling urinary catheters and central lines	• Prevent infection
• Use good hand-washing technique	• Prevent infection
• Use aseptic technique for all procedures	• Prevent infection
• Perform pulmonary preventive techniques (turn, cough, deep breathing)	• Mobilize secretions to prevent pneumonia
• Assess potential sites of infection (urinary, pulmonary, wound, intravenous catheters)	• Detect early signs of infection

NURSING DIAGNOSIS

Imbalanced Nutrition: Less Than Body Requirements related to uremia, altered oral mucous membranes, and dietary restrictions

PATIENT OUTCOMES

Adequate nutritional and caloric intake

- Body weight at patient's baseline
- Energy level appropriate
- Verbalizes comfort of oral cavity and ability to taste food normally

NURSING INTERVENTIONS	RATIONALES
• Monitor body weight and caloric intake daily	• Identify deficits in nutritional intake and response to nutritional therapy
• Collaborate with dietitian about nutritional needs	• Provide optimal nutritional support
• Provide diet with essential nutrients but within restrictions	• Prevent nutritional deficits; prevent electrolyte imbalances and fluid overload
• Provide oral hygiene every 2 to 4 hours	• Minimize dryness of oral mucosa and promote patient comfort
• Remove noxious stimuli from room	• Reduce nausea, vomiting, and anorexia

Continued

NURSING CARE PLAN

for the Patient with Acute Kidney Injury—cont'd

NURSING DIAGNOSIS

Anxiety related to diagnosis, treatment plan, prognosis, and unfamiliar environment

PATIENT OUTCOME

Anxiety levels reduced

- Effective coping mechanisms
- Participation in treatment plan

NURSING INTERVENTIONS	RATIONALES
• Monitor for signs of anxiety: tachycardia, muscle tension, inappropriate behaviors	• Recognize anxiety
• Explain all procedures; provide calm, relaxing environment	• Reduce anxiety by providing factual information
• Implement measures to reduce fear and anxiety	• Facilitate relaxation
• Allow patient to make choices	• Promote feelings of control to reduce anxiety
• Assess for ineffective coping (depression, withdrawal)	• Assess need for counseling and/or medications
• Administer antianxiety medications as prescribed	• Reduce anxiety

NURSING DIAGNOSIS

Deficient Knowledge related to disease process and therapeutic regimen

PATIENT OUTCOME

Adequate knowledge of disease and treatment

- Patient and family have sufficient, accurate information related to condition to be informed participants in the care

NURSING INTERVENTIONS	RATIONALES
• Provide specific, factual information on acute kidney injury, impact on the patient, and treatment plan	• Knowledge will enhance patient understanding
• Encourage patient and family to ask questions	• Promote increased knowledge
• Encourage patient and family members to participate in care	• Facilitate self-care management

WBC, White blood cell.

Based on data from Gulanick M and Myers JL. *Nursing Care Plans: Diagnoses, Interventions, and Outcomes,* 7th ed. St. Louis: Mosby; 2011.

NURSING INTERVENTIONS

Accurate measurement of intake and output and determination of daily weights are two vital nursing interventions. A urine meter or other type of accurate measuring device is essential for recording urinary output. Normal urine output is 0.5 to 1 mL/kg/hr. Oral fluid intake must also be carefully monitored. Fluid intake levels are often restricted to the amount of urine output in a 24-hour period plus insensible losses (approximately 600 to 1000 mL/day).[38] Administration of intravenous fluids as prescribed before procedures in which radiocontrast media will be given is critical.[13]

Assessment of daily weights is one of the most useful noninvasive diagnostic tools. The daily weight is used to validate intake and output measurements. A 1-kg gain in body weight is equal to a 1000-mL fluid gain. Weight should be obtained at the same time each day with the same scale. Many critical care beds have built-in scales, which simplify the procedure. When the patient is weighed, the nurse ensures that the scale is properly calibrated and that the same number of bed linens and pillows are weighed with the patient each time. The nurse must recognize signs and symptoms of fluid volume overload, which can lead to pulmonary edema and severe respiratory distress (see box, "Clinical Alert: Fluid Volume Overload").

! CLINICAL ALERT

Fluid Volume Overload

Signs and symptoms of fluid volume overload include hypertension, edema, crackles, dyspnea, neck vein distention, weight gain, increased pulmonary artery pressures, decreased urine output, decreased hematocrit, and presence of an S_3 heart sound.

Infection is the most common and serious complication of AKI and accounts for up to 75% of deaths in patients with AKI.[8] Nurses play a key role in preventing infections. Indwelling urinary catheters should not routinely be inserted, because they increase the risk of infection, and many patients remain oliguric for 8 to 14 days. Strict aseptic technique with all intravenous lines (central and peripheral), including temporary access devices used for dialysis, is also of extreme importance, both at the time of insertion and during daily maintenance.

Another important role of the nurse in preventing AKI, as well as delaying its progression, is monitoring trough drug levels. Nurses are responsible for scheduling and obtaining the trough blood levels at the appropriate times to ensure accurate results. Drug dosage adjustments must be made to prevent accumulation of the drug and toxic side effects. For example, aminoglycoside doses are based on drug levels and the patient's estimated creatinine clearance. If the drug level is too high, either the dose of the aminoglycoside can be kept constant and the interval between doses increased, or the interval can be kept constant and the dose is decreased. A trough level is drawn just before the next dose is given and is an indicator of how the body has cleared the drug.

MEDICAL MANAGEMENT OF ACUTE KIDNEY INJURY

Prerenal Causes

Acute kidney injury from prerenal conditions is usually reversible if renal perfusion is quickly restored; therefore early recognition and prompt treatment are essential. However, prevention of prerenal conditions is just as important as early recognition and aggressive management. Prompt replacement of extracellular fluids and aggressive treatment of shock may help prevent AKI. Hypovolemia is treated in various ways, depending on the cause. Blood loss may necessitate blood transfusions, whereas patients with pancreatitis and peritonitis are usually treated with isotonic solutions such as normal saline. Hypovolemia resulting from large urine or gastrointestinal losses often requires the administration of a hypotonic solution, such as 0.45% saline. Patients with cardiac instability usually require positive inotropic agents, antidysrhythmic agents, preload or afterload reducers, or an intraaortic balloon pump. Hypovolemia from intense vasodilation may require vasoconstrictor medications, isotonic fluid replacement, and antibiotics (if the patient has sepsis) until the underlying problem has been resolved. Invasive hemodynamic monitoring with a central venous catheter or pulmonary artery catheter may be considered in the management of fluid balance.

Postrenal Causes

Postrenal obstruction should be suspected whenever a patient has an unexpected decrease in urine volume. Postrenal conditions are usually resolved with the insertion of an indwelling bladder catheter, either transurethral or suprapubic. Occasionally, a ureteral stent may have to be placed if the obstruction is caused by calculi or carcinoma.

Intrarenal Causes: Acute Tubular Necrosis

Common interventions for the patient with ATN include drug therapy, dietary management such as protein and electrolyte restrictions, management of fluid and electrolyte imbalances, and renal replacement therapies such as intermittent hemodialysis or continuous renal replacement therapy (CRRT).

Considering the detrimental impact of AKI, nurses must focus on efforts aimed at prevention. The most important preventive strategies include identification of patients at risk and elimination of potential contributing factors. Aggressive treatment must begin at the earliest sign of renal dysfunction.

In general, maintenance of cardiovascular function and adequate intravascular volume are the two key goals in the prevention of AKI. Box 15-6 summarizes important measures for preventing AKI.

Pharmacological Management

Diuretics. Diuretic therapy in the treatment of patients with AKI is controversial. In clinical practice, diuretics may be used to manage volume overload. Although it is believed

BOX 15-6 MEASURES TO PREVENT ACUTE KIDNEY INJURY

Avoid Nephrotoxins
- Use iso-osmolar radiocontrast media (e.g.,iodixanol)
- Limit contrast volume to <100 mL
- Use antibiotics cautiously with appropriate dose modification
- Monitor drug levels (aminoglycosides)
- Stop certain medications (NSAIDs, ACE inhibitors, ARBs) before high-risk procedures

Optimize Volume Status Before Surgery or Invasive Procedures
- Aim for urinary output >40 mL/hr
- Keep mean arterial pressure >80 mm Hg
- Hydrate with normal saline before and after procedures requiring radiocontrast media
- Hold diuretics day before and day of procedure

Reduce Incidence of Nosocomial Infections
- Use indwelling urinary catheters judiciously
- Remove indwelling urinary catheters when no longer needed
- Use strict aseptic technique with all intravenous lines

Implement Tight Glycemic Control in the Critically Ill

Aggressively Investigate and Treat Sepsis

ACE, Angiotensin-converting enzyme; *ARBs,* angiotensin receptor blockers; *NSAIDs,* nonsteroidal antiinflammatory drugs.

that diuretics increase renal blood flow and GFR (thereby increasing urine output), and reduce tubular dysfunction and obstruction, evidence suggests that they may cause excess diuresis and renal hypoperfusion, compromising an already insulted renal system.[8] Diuretics may increase the risk of AKI from volume depletion when they are given before procedures requiring radiological contrast media or if the patient is hypovolemic. Adequate hydration before administration of diuretics is essential. The widespread use of diuretics is currently being discouraged.[8,10]

If diuretic therapy is implemented, a loop diuretic is commonly ordered. Large doses of furosemide are often needed to induce diuresis. This may lead to excessive diuresis and volume depletion. High doses of furosemide have been associated with deafness, which may become permanent.[8]

Mannitol, an osmotic diuretic often used in AKI caused by rhabdomyolysis, increases plasma volume and is believed to protect the kidney by minimizing postischemic swelling. Patients may be at risk for the development of pulmonary edema due to the rapid expansion of intravascular volume triggered by mannitol.

Dopamine. The role of dopamine is controversial in the treatment of AKI. Low-dose dopamine continues to be ordered for patients with AKI despite numerous studies that fail to show any benefit. Dopamine in low doses (1 to 3 mcg/kg/min) may cause a transient increase renal blood flow and GFR by stimulating the dopaminergic receptors in the kidney.[10] However, there is broad consensus that dopamine is potentially harmful and its use for renal perfusion should be avoided.[8,10,18]

N-Acetylcysteine. Multiple studies have been conducted using prophylactic *N*-acetylcysteine (Mucomyst) in patients at risk of contrast-induced AKI. *N*-Acetylcysteine, an antioxidant, in conjunction with intravenous fluids has been thought to reduce the incidence of contrast-induced AKI. The mechanism of action is unclear, but *N*-acetylcysteine is thought to act by scavenging oxygen free radicals or enhancing the vasodilatory effects of nitric oxide.[13,33] Prophylactic administration of *N*-acetylcysteine (600 mg orally twice a day on the day before and on the day the contrast is given), along with hydration (half-normal [0.45%] saline at 1 mL/kg/hr overnight before procedure) is hypothesized to reduce the amount of acute renal damage in high-risk patients undergoing procedures requiring contrast agents.[13,24,33] However, current data on the administration of acetylcysteine remains inconclusive.[13,33]

Fenoldopam. Another agent that is postulated to protect against contrast-induced AKI is fenoldopam, a dopamine-1 receptor agonist (DA-1). Fenoldopam (Corlopam) acts as a vasodilator of peripheral arteries (reducing blood pressure) and as a potent renal vasodilator (increasing renal blood flow). It is six times more potent than dopamine in increasing renal blood flow, especially to critical regions in the renal medulla. Fenoldopam is given via intravenous infusion several hours before the contrast agent is given and is continued for a minimum of 4 hours after the procedure. Ongoing studies are focused on the use of fenoldopam in the prevention of contrast-induced nephropathy; however, no consistent outcome has been noted.[13,18,33]

Miscellaneous agents. Multiple miscellaneous agents have been administered in an attempt to attenuate the course of AKI. None, however, has consistently proved effective. Many of these drugs are administered in an attempt to improve renal blood flow through vasodilation (atrial natriuretic peptide, endothelin-1 receptor antagonists, prostaglandin E_1), prevent accumulation of intracellular calcium as occurs in ischemic azotemia (calcium channel blockers), protect renal tubule cells during ischemia (glycine, magnesium adenosine triphosphate dichloride) or stimulate renal cell regeneration (epidermal growth factor, growth hormone, insulin-like growth factor). Many of these agents and numerous others have shown beneficial results in experimental models, but results are inconsistent in the clinical setting.

Prostaglandin E_1 has a vasodilatory effect and has been shown in small studies to counteract the vasoconstriction from radiocontrast media that may cause AKI in high-risk patients. Administration of an intravenous sodium bicarbonate solution before and after the procedure is also thought to prevent CIN. It is speculated that alkalinizing the urine may reduce the nephrotoxic potential of the radiocontrast media in the renal capillaries or tubules. However, trials comparing administration of normal saline with sodium bicarbonate solutions are inconclusive.[8,13,24] Ongoing studies are being conducted on a variety of agents in the prevention and treatment of AKI.[3,8,10]

Pharmacological management considerations. Drug therapy for the patient with AKI poses a challenge because two thirds of all drugs or their metabolites are eliminated from the body by the kidneys. Substantial alterations in drug dosages are often necessary to prevent toxic levels and adverse reactions. Assessment of renal function by creatinine clearance is often used to assist with drug dosing. The pharmacokinetic characteristics of the drug to be given, the route of elimination, and the extent of protein binding are also considered. Clinical pharmacists assist in determining optimum drug dosages for critically ill patients.

Many drugs are removed by dialysis, and extra doses are often required to avoid suboptimal drug levels. Drugs that are primarily water soluble, such as vitamins, cimetidine, and phenobarbital should be administered after dialysis. Drugs that become bound to proteins or lipids or are metabolized by the liver, such as phenytoin, lidocaine, and vancomycin, are not removed by dialysis and can be given at any time.[8] Box 15-7 is a partial list of drugs that are removed by dialysis and should be administered after dialysis.

Dietary Management

Dietary management in patients with AKI is important. Energy expenditure in catabolic patients with acute kidney injury is much higher than normal. Dialysis also contributes to protein catabolism. The loss of amino acids and water-soluble vitamins in the dialysate solution constitutes another drain on the patient's nutritional stores. The overall goal of dietary management for acute kidney injury is provision of

BOX 15-7 COMMON DRUGS REMOVED BY HEMODIALYSIS*

- Aminoglycosides (gentamicin, tobramycin)
- Aspirin
- Cephalosporins (including cefoxitin and ceftazidime)
- Cimetidine
- Enalapril
- Erythromycin
- Folic acid
- Isoniazid
- Lisinopril
- Lithium carbonate
- Metformin
- Methyldopa
- Metoprolol
- Nitroprusside
- Penicillins (piperacillin, penicillin G)
- Phenobarbital
- Procainamide
- Quinidine
- Ranitidine
- Sulfonamides (sulfamethoxazole, sulfisoxazole)
- Trimethoprim-sulfamethoxazole
- Water-soluble vitamins

*If possible, hold daily doses until after dialysis; supplemental doses may be required for many of these agents.

adequate energy, protein, and micronutrients to maintain homeostasis in patients who may be extremely catabolic. Nutritional recommendations include the following[9]:

- Caloric intake of 25 to 35 kcal/kg of ideal body weight per day
- Protein intake of no less than 0.8 g/kg. Patients who are extremely catabolic should receive 1.5 to 2.0 g/kg of ideal body weight per day—75% to 80% of which contains all the required essential amino acids.
- Sodium intake of 0.5 to 1.0 g/day
- Potassium intake of 20 to 50 mEq/day
- Calcium intake of 800 to 1200 mg/day
- Fluid intake equal to the volume of the patient's urine output plus an additional 600 to 1000 mL/day

In addition, patients undergoing dialysis usually receive multivitamins, folic acid, and occasionally an iron supplement to replace the water-soluble vitamins and other essential elements lost during dialysis. If the patient is unable to ingest or tolerate an adequate oral nutritional intake, enteral feedings or total parenteral nutrition are prescribed. Nutritional support must supply the patient with sufficient nonprotein glucose calories, essential amino acids, fluids, electrolytes, and essential vitamins. Adequate nutrition not only prevents further catabolism, negative nitrogen balance, muscle wasting, and other uremic complications, but also enhances the patient's tubular regenerating capacity, resistance to infection, and ability to combat other multisystem dysfunctions. The physician may also prescribe early renal replacement therapy to treat the increased fluid volume the patient receives from enteral or total parenteral nutrition.

Management of Fluid, Electrolyte, and Acid-Base Imbalances

Fluid imbalance. Volume overload is managed by dietary restriction of salt and water and administration of diuretics. In addition, dialysis or other renal replacement therapies may be indicated for fluid control. These modalities are discussed later in this chapter.

Electrolyte imbalance. Common electrolyte imbalances in AKI are listed in the box, "Laboratory Alert," along with their "critical" values and the significance of the laboratory alert. The nurse immediately notifies the physician once a critical laboratory value is known. Hyperkalemia is common in AKI, especially if the patient is hypercatabolic. Hyperkalemia occurs when potassium excretion is reduced as a result of the decrease in GFR. Sudden changes in the serum potassium level can cause dysrhythmias, which may be fatal. Figure 15-6 shows the electrocardiographic changes commonly seen in hyperkalemia.

Three approaches are used to treat hyperkalemia: (1) reduce the body potassium content, (2) shift the potassium from outside the cell to inside the cell and (3) antagonize the membrane effect of the hyperkalemia. Only dialysis and administration of cation exchange resins (sodium polystyrene sulfonate [Kayexalate]) actually reduce plasma potassium levels and total

! LABORATORY ALERT

Acute Kidney Injury

LABORATORY TEST	CRITICAL VALUE	SIGNIFICANCE
Potassium (K^+)	>6.6 mEq/L	**Hyperkalemia:** potential for heart blocks, asystole, ventricular fibrillation; may cause muscle weakness, diarrhea, and abdominal cramps
Sodium (Na^+)	≤110 mEq/L	**Hyponatremia:** potential for lethargy, confusion, coma, or seizures; may cause nausea, vomiting, and headaches
Total calcium (Ca^{++})	<7.0 mg/dL	**Hypocalcemia:** potential for seizures, laryngospasm, stridor, tetany, heart blocks, and cardiac arrest; may see positive Chvostek or Trousseau sign
Magnesium (Mg^{++})	>3.0 mg/dL	**Hypermagnesemia:** potential for bradycardia and heart blocks, lethargy, coma, hypotension, hypoventilation, and weak-to-absent deep tendon reflexes

QRS complex	Approximate serum K^+ (mEq/L)	ECG change
P wave T wave	4	Normal
	6-7	Peaked T waves
	7-8	Flattened P wave Prolonged PR interval Depressed ST segment Peaked T wave
	8-9	Atrial standstill Prolonged QRS duration Further peaking T waves
	> 9	Sine-wave pattern

FIGURE 15-6 Electrocardiographic (ECG) changes seen in hyperkalemia. (From Weiner D, Linas S, Wingo C. Disorders of potassium metabolism. In Feehally J, Floege J, Johnson R, eds. *Comprehensive Clinical Nephrology*. Philadelphia: Mosby. 2007.)

body potassium content in a patient with renal dysfunction. In the past, sorbitol has been combined with sodium polystyrene sulfonate powder for administration. The concomitant use of sorbitol with sodium polystyrene sulfonate has been implicated in cases of colonic intestinal necrosis and therefore this combination is not recommended.[22] Other treatments only "protect" the patient for a short time until dialysis or cation exchange resins can be instituted. Table 15-5 summarizes medications used in the treatment of hyperkalemia. Commonly prescribed treatments for hyperkalemia consists of the following[32]:

- Calcium gluconate, 10 mL of a 10% solution given intravenously over 5 minutes
- Regular insulin, 10 units given intravenously with glucose (50 mL of 50% dextrose) intravenously
- Albuterol 10 to 20 mg given by nebulized inhalation over 15 minutes
- Sodium bicarbonate, 50 mEq/L given intravenously in patients with severe acidosis with pH less than 7.2 or serum HCO_3^- less than 15 mEq/L

Hyponatremia generally occurs from water overload. However, as nephrons are progressively damaged, the ability to conserve sodium is lost, and major salt-wasting states can develop, causing hyponatremia. Hyponatremia is treated with fluid restriction, specifically restriction of free water intake. Alterations in the serum calcium and phosphorus levels occur frequently in AKI as a result of abnormalities in excretion, absorption, and metabolism of the electrolytes. Mild degrees of hypermagnesemia are common in AKI secondary to decreased renal excretion.

Acid-base imbalance. Metabolic acidosis is the primary acid-base imbalance seen in AKI. Box 15-8 summarizes the etiology and the signs and symptoms of metabolic acidosis in AKI. Treatment of metabolic acidosis depends on its severity. In mild metabolic acidosis, the lungs compensate by excreting carbon dioxide. Patients with a serum bicarbonate level of less than 15 mEq/L and a pH of less than 7.20 are usually treated with intravenous sodium bicarbonate. The goal of treatment is to raise the pH to a value greater than 7.20. Rapid correction of the acidosis should be avoided, because tetany may occur as a result of hypocalcemia. The pH determines how much ionized calcium is present in the serum; the more acidic the serum, the more ionized calcium is present. If the metabolic acidosis is rapidly corrected, the serum ionized calcium level decreases as the calcium binds with albumin and other substances such as phosphate and sulfate. For this reason, intravenous calcium gluconate may be prescribed. Renal replacement therapies also may correct metabolic acidosis because it removes excess hydrogen ions, and bicarbonate is added to the dialysate and replacement solutions.

Renal Replacement Therapy

Renal replacement therapy is the primary treatment for the patient with AKI. The decision to initiate renal replacement therapy is a clinical decision based on the fluid, electrolyte, and metabolic status of each patient. Renal replacement therapy options include intermittent hemodialysis, CRRT, or peritoneal dialysis.

TABLE 15-5 PHARMACOLOGY

Medications to Treat Hyperkalemia

MEDICATION	ACTION/USE	DOSAGE/ROUTE	SIDE EFFECTS	NURSING IMPLICATIONS
Sodium polystyrene sulfonate (Kayexalate)	↑ Fecal excretion of potassium by exchanging sodium ions for potassium ions	*Oral:* 15 g 1-4 times daily by mouth *Rectal:* 30-50 g via enema every 6 hours	Constipation, hypokalemia, hypernatremia, nausea and vomiting, fecal impaction in the elderly	Available as a powder or suspension Mix powder with full glass of liquid and chill to increase palatability Do not mix oral powder with orange juice Do not mix with sorbitol
Furosemide (Lasix)	↑ Renal excretion of potassium	*Oral:* 20-80 mg daily twice a day *IV:* 20-40 mg/dose every 6-12 hours *Continuous infusion:* 10-40 mg/hr	Orthostatic hypotension, hypokalemia, urinary frequency, dizziness, ototoxicity	Administer IV dose over several minutes; ototoxicity is associated with rapid administration Assess for allergy to sulfonylurea before giving Monitor for dehydration, hypokalemia, hypotension Diuretics only work if the patient is nonoliguric
Insulin/ dextrose	Shifts potassium temporarily from the extracellular fluid (blood) into the intracellular fluid; the dextrose helps prevent hypoglycemia	*IV:* 10 units regular insulin and 50 mL of 50% dextrose IV push	Hyperglycemia, hypoglycemia, hypokalemia	If the serum glucose is >300 mg/dL, the physician may order only the insulin
Sodium bicarbonate	Shifts potassium temporarily from the extracellular fluid (blood) to the intracellular fluid	*IV:* 50 mEq/L IV push	Hypernatremia, hypokalemia, pulmonary edema	Do not mix with any other medications to prevent precipitation Helpful if patient has a severe metabolic acidosis
Albuterol	Adrenergic agonist ↑ plasma insulin concentration; shifts potassium to intracellular space	*Inhalation:* 10-20 mg over 10 minutes *IV:* 0.5 mg over 15 minutes	Tachycardia, angina, palpitations, hypertension, nervousness, irritability	Note that the dose used is much higher than that used in treating pulmonary conditions Use concentrated form (5 mg/mL) so the volume to be inhaled is minimized
Calcium gluconate	Electrolyte replacement	*IV:* 10 mL of 10% solution IV push over 5 minutes	Bradycardia, hypotension, syncope, necrosis if infiltrated	Has no effect on actually lowering serum potassium Will see almost immediate effect on ECG appearance Be sure IV is patent; prevent extravasation

ECG, Electrocardiogram; *IV,* intravenous.

BOX 15-8 METABOLIC ACIDOSIS IN ACUTE KIDNEY INJURY

Etiology

- Inability of kidney to excrete hydrogen ions; decreased production of ammonia by the kidney (normally assists with hydrogen ion excretion)
- Retention of acid end-products of metabolism, which use available buffers in the body; inability of kidney to synthesize bicarbonate

Signs and Symptoms

- Low pH of arterial blood (pH <7.35)
- Low serum bicarbonate
- Increased rate and depth of respirations to excrete carbon dioxide from the lungs (compensatory mechanism); known as Kussmaul's respiration
- Low $PaCO_2$
- Lethargy and coma if severe

$PaCO_2$, Partial pressure of carbon dioxide in arterial blood.

Definition. *Dialysis* is defined as the separation of solutes by differential diffusion through a porous or semipermeable membrane that is placed between two solutions. The various dialysis methods are distinguished by the type of semipermeable membrane and the two solutions that are used.

Indications for dialysis. The most common reasons for initiating dialysis in AKI include acidosis, hyperkalemia, volume overload, and uremia. Dialysis is usually started early in the course of the renal dysfunction before uremic complications occur. In addition, dialysis is may be started for fluid management when total parenteral nutrition is administered in patients with impaired renal function.[7]

Principles and mechanisms. Dialysis therapy is based on two physical principles that operate simultaneously: diffusion and ultrafiltration. *Diffusion* (or clearance) is the movement of solutes such as urea from the patient's blood to the dialysate cleansing fluid, across a semipermeable membrane (the hemofilter). Substances such as bicarbonate may also cross in the opposite direction, from the dialysate through the semipermeable membrane into the patient's blood. Movement of solutes across the semipermeable membrane depends on the following:

- The amount of solutes on each side of the semipermeable membrane; typically, the patient's blood has larger amounts of solutes such as urea, creatinine, and potassium
- The surface area of the semipermeable membrane (the size of the hemofilter)
- The permeability of the semipermeable membrane
- The size and charge of the solutes
- The rate of blood flowing through the hemofilter
- The rate of dialysate cleansing fluid flowing through the hemofilter

Ultrafiltration is the removal of plasma water and some low–molecular weight particles by using a pressure or osmotic gradient. Ultrafiltration is primarily aimed at controlling fluid volume, whereas dialysis is aimed at decreasing waste products and treating fluid and electrolyte imbalances.[6]

Vascular access. An essential component of all the renal replacement therapies is adequate, easy access to the patient's bloodstream. Various types of vascular access devices (Figures 15-7 and 15-8) are used for hemodialysis: percutaneous venous catheters, arteriovenous fistulas, and arteriovenous grafts.

Temporary percutaneous catheters are commonly used in patients with AKI because they can be used immediately. The typical catheter has a single or double lumen and is designed only for short-term renal replacement therapy during acute situations. Though these catheters can be inserted into the subclavian, jugular, or femoral veins, the femoral site is discouraged because it carries an increased risk of infection.[30] The subclavian site should also be avoided in patients with advanced kidney disease because of the risk of subclavian vein stenosis.[30] Routine replacement of hemodialysis catheters to prevent infection is not recommended.[30] The decision to remove or replace the catheter is based on clinical need and/or signs and symptoms of infection.[30] Occasionally a percutaneous tunneled catheter is placed if the patient needs ongoing hemodialysis. These catheters are usually inserted in the operating room or in an interventional radiology area. Examples of tunneled hemodialysis catheters include the Permacath and Tesio twin catheters.

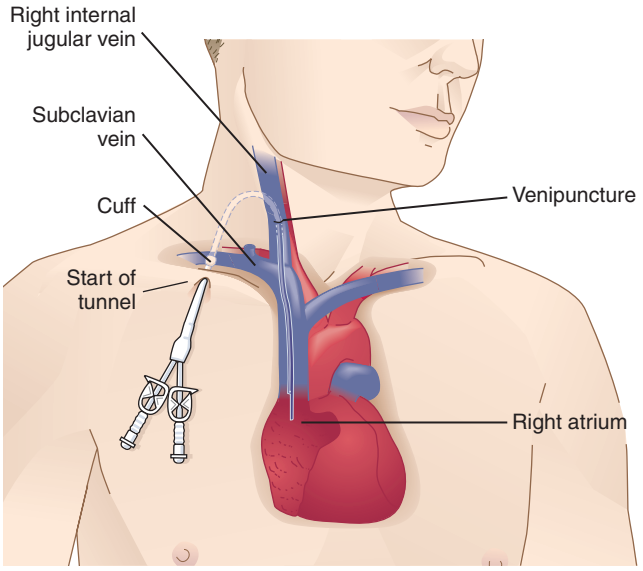

FIGURE 15-7 Central venous catheter used for hemodialysis. (From Headley CM. Acute kidney injury and chronic kidney disease. In Lewis SL, Dirksen SR, Heitkemper MM, et al, eds. *Medical-Surgical Nursing: Assessment and Management of Clinical Problems*. 8th ed. St. Louis: Mosby, 2011.)

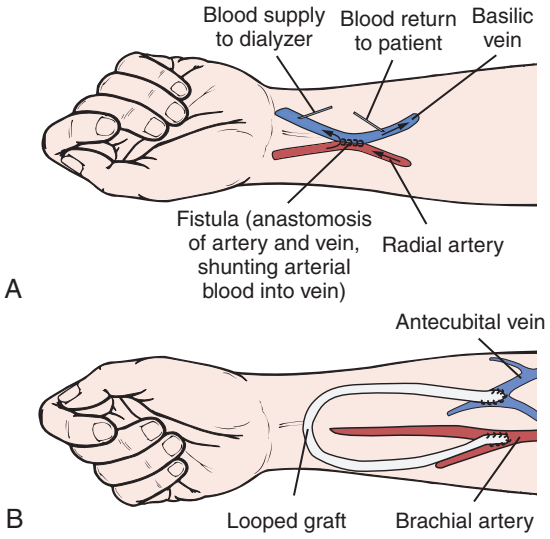

FIGURE 15-8 Hemodialysis access devices. **A,** Arteriovenous fistula. **B,** Arteriovenous graft. (From Headley CM. Acute kidney injury and chronic kidney disease. In Lewis SL, Dirksen SR, Heitkemper MM, et al, eds. *Medical-Surgical Nursing: Assessment and Management of Clinical Problems*. 8th ed. St. Louis: Mosby, 2011.)

An *arteriovenous fistula* is an internal, surgically created communication between an artery and a vein. The most frequently created fistula is the Brescia-Cimino fistula, which

involves anastomosing the radial artery and cephalic vein in a side-to-side or end-to-side manner. The anastomosis permits blood to bypass the capillaries and to flow directly from the artery into the vein. As a result, the vein is forced to dilate to accommodate the increased pressure that accompanies the arterial blood. This method produces a vessel that is easy to cannulate but requires 4 to 6 weeks before it is mature enough to use.

Arteriovenous grafts are created by using different types of prosthetic materials. Most commonly, polytetrafluoroethylene (Teflon) grafts are placed under the skin and are surgically anastomosed between an artery (usually brachial) and a vein (usually antecubital). The graft site usually heals within 2 to 4 weeks.

Nursing care of arteriovenous fistula or graft. The nurse must protect the vascular access site. An arteriovenous fistula or graft should be auscultated for a bruit and palpated for the presence of a thrill or buzz every 8 hours. The extremity that has a fistula or graft must never be used for drawing blood specimens, obtaining blood pressure measurements, or administering intravenous therapy or intramuscular injections. Such activities produce pressure changes within the altered vessels that could result in clotting or rupture. The nurse must alert other healthcare personnel of the presence of the fistula or graft by posting a large sign at the head of the patient's bed that indicates which arm should be used. The presence and strength of the pulse distal to the fistula or graft are evaluated at least every 8 hours. Inadequate collateral circulation past the fistula or graft may result in loss of this pulse. The physician is notified immediately if no bruit is auscultated, no thrill is palpated, or the distal pulse is absent.

Nursing care of percutaneous catheters. Strict aseptic technique must be applied to any percutaneous catheter placed for dialysis. Transparent, semipermeable polyurethane dressings are recommended because they allow continuous visualization for assessment of signs of infection.[30] Replace transparent dressings on temporary percutaneous catheters at least every 7 days and no more than once a week for tunneled percutaneous catheters unless the dressing is soiled or loose.[30] Monitor the catheter site visually when changing the dressing or by palpation through an intact dressing. Tenderness at the insertion site, swelling, erythema or drainage should be reported to the physician. To prevent accidental dislodging, minimize manipulation of the catheter. The catheter is not used to administer fluids or medications or to sample blood unless a specific order is obtained to do so. Dialysis personnel may instill medication in the catheter to maintain patency, and clamp the catheter when not in use.

Hemodialysis. Intermittent hemodialysis is the most frequently used renal replacement therapy for treating AKI. Hemodialysis consists of simply cleansing the patient's blood through a hemofilter by the use of diffusion and ultrafiltration. Water and waste products of metabolism are easily removed. Hemodialysis is efficient and corrects biochemical disturbances quickly. Treatments are typically 3 to 4 hours long and are performed in the critical care unit at the patient's bedside. Patients with AKI may be hemodynamically unstable and unable to tolerate intermittent hemodialysis. In those instances, other methods of renal replacement therapy such as peritoneal dialysis or CRRT are considered.

Complications. Several complications are associated with hemodialysis. Hypotension is common and is usually the result of preexisting hypovolemia, excessive amounts of fluid removal, or excessively rapid fluid removal.[15] Other factors that contribute to hypotension include left ventricular dysfunction from preexisting heart disease or medications, autonomic dysfunction resulting from medication or diabetes, and inappropriate vasodilation resulting from sepsis or antihypertensive drug therapy. Dialyzer membrane incompatibility may also cause hypotension.

Dysrhythmias may occur during dialysis. Causes of dysrhythmias include a rapid shift in the serum potassium level, clearance of antidysrhythmic medications, preexisting coronary artery disease, hypoxemia, or hypercalcemia from rapid influx of calcium from the dialysate solution.

Muscle cramps may occur during dialysis, but they occur more commonly in chronic renal failure. Cramping is thought to be caused by ischemia of the skeletal muscles resulting from aggressive fluid removal. The cramps typically involve the legs, feet, and hands and occur most often during the last half of the dialysis treatment.

A decrease in the arterial oxygen content of the blood can occur in patients undergoing hemodialysis. Usually the decrease ranges from 5 to 35 mm Hg (mean, 15 mm Hg) and is not clinically significant except in the unstable critically ill patient. Several theories have been offered to explain the hypoxemia, including leukocyte interactions with the hemofilter and a decrease in carbon dioxide levels, resulting from either an acetate dialysate solution or a loss of carbon dioxide across the semipermeable membrane.

Dialysis disequilibrium syndrome often occurs after the first or second dialysis treatment or in patients who have had sudden, large decreases in BUN and creatinine levels as a result of the hemodialysis. Because of the blood-brain barrier, dialysis does not deplete the concentrations of BUN, creatinine, and other uremic toxins in the brain as rapidly as it decreases those substances in the extracellular fluid. An osmotic concentration gradient established in the brain allows fluid to enter until the concentration levels equal those of the extracellular fluid. The extra fluid in the brain tissue creates a state of cerebral edema for the patient, which results in severe headaches, nausea and vomiting, twitching, mental confusion, and occasionally seizures. The incidence of dialysis disequilibrium syndrome may be decreased by the use of shorter, more frequent dialysis treatments.

Infectious complications associated with hemodialysis include vascular access infections and hepatitis C. Vascular access infections are usually caused by a break in sterile technique, whereas hepatitis C is usually acquired through transfusion.

Hemolysis, air embolism, and hyperthermia are rare complications of hemodialysis. Hemolysis can occur when the patient's blood is exposed to incorrectly mixed dialysate solution or hypotonic chemicals (formaldehyde and bleach). An air embolism can occur when air is introduced into the bloodstream through a break in the dialysis circuit. Hyperthermia may result if the temperature control devices on the dialysis machine malfunction. Complications of hemodialysis are summarized in Box 15-9.

Nursing care of the patient. The patient receiving hemodialysis requires specialized monitoring and interventions by the critical care nurse. Laboratory values are monitored and abnormal results reported to the nephrologist and dialysis staff. The patient is weighed daily to monitor fluid status. On the day of dialysis, dialyzable (water-soluble) medications are not given until after treatment. The dialysis nurse or pharmacist can be consulted to determine which medications to withhold or administer. Supplemental doses are administered as ordered after dialysis. Administration of antihypertensive agents is avoided for 4 to 6 hours before treatment, if possible. Doses of other medications that lower blood pressure (narcotics, sedatives) are reduced, if possible. The percutaneous catheter, fistula, or graft is assessed frequently; unusual findings such as loss of bruit, redness, or drainage at the site must be reported. After dialysis, the patient is assessed for signs of bleeding, hypovolemia, and dialysis disequilibrium syndrome.

Continuous renal replacement therapy. CRRT is a continuous extracorporeal blood purification system managed by the bedside critical care nurse. It is similar to conventional intermittent hemodialysis in that a hemofilter is used to facilitate the processes of ultrafiltration and diffusion. It differs in that CRRT provides a slow removal of solutes and water as compared to the rapid removal of water and solutes that occurs with intermittent hemodialysis.

Indications. The clinical indications for CRRT are similar to those for intermittent hemodialysis, including volume overload, hyperkalemia, acidosis, and uremia. It is frequently selected for patients with AKI because of the ability to provide a gentle correction of uremia and fluid imbalances while minimizing hypotension. CRRT modalities have also been thought to absorb many of the interleukins associated with inflammation and sepsis.[4,7,20]

Principles. The first CRRT systems were introduced in the 1970s. The extracorporeal circuit consisted of an arterial access catheter, hemofilter, and venous return catheter. The patient's blood pressure determined the flow rate through the circuit. Arteriovenous systems are no longer used because of therapy limitations related to patient dependent blood flow and concern for complications related to arterial cannulation. Venovenous circuits are currently the standard for renal replacement therapy.[1] Improvements in dual-lumen venous catheters, mechanical blood pumps, and user-friendly renal replacement therapy cassette circuits and monitors have increased the safety and efficiency of venovenous replacement therapies. In venovenous therapy, two venous accesses or a dual-lumen venous catheter are used. Blood is pulled from the access port of the dual-lumen dialysis catheter or one of two single-lumen venous catheters by the negative pressure gradient created by a blood pump. The blood travels through the hemofilter and returns to the patient via the return port of the dual-lumen venous dialysis catheter or a second venous catheter (Figure 15-9).

There are four types of continuous venovenous replacement therapies:

1. Slow continuous ultrafiltration (SCUF)
2. Continuous venovenous hemofiltration (CVVH)
3. Continuous venovenous hemodialysis (CVVHD)
4. Continuous venovenous hemodiafiltration (CVVHDF)

Table 15-6 outlines the various CRRT modalities.

Slow continuous ultrafiltration (SCUF) is also known as isolated ultrafiltration and is used to remove plasma water in cases of volume overload. SCUF can remove 3 to 6 liters of ultrafiltrate per day. Solute removal is minimal and therefore is not indicated for patients with conditions requiring removal of uremic toxins and correction of acidosis.

Continuous venovenous hemofiltration (CVVH) is used to remove fluids and solutes through the process of convection, which is the transfer of solutes across the semipermeable membranes of the hemofilter. As plasma moves across the membrane (ultrafiltration), it carries solute molecules. Increasing the volume of plasma water that crosses the hemofilter membranes increases the amount of solute removed. Replacement solution is added to replenish plasma water and electrolytes lost because of the high ultrafiltration rate. Replacement solutions typically are commercially prepared and contain electrolytes and a bicarbonate or lactate base. Calcium and magnesium are two electrolytes not present in bicarbonate-based replacement solutions because they will form precipitates. These two electrolytes must be administered separately. Replacement solutions can be administered before the hemofilter (predilution) or after the hemofilter (postdilution).

Continuous venovenous hemodialysis (CVVHD) is similar to CVVH in that ultrafiltration removes plasma water. It differs in

BOX 15-9 COMPLICATIONS OF DIALYSIS

- Hypotension
- Cramps
- Bleeding/clotting
- Dialyzer reaction
- Hemolysis
- Dysrhythmias
- Infections
- Hypoxemia
- Pyrogen reactions
- Dialysis disequilibrium syndrome
- Vascular access dysfunction
- Technical errors (incorrect dialysate mixture, contaminated dialysate, or air embolism)

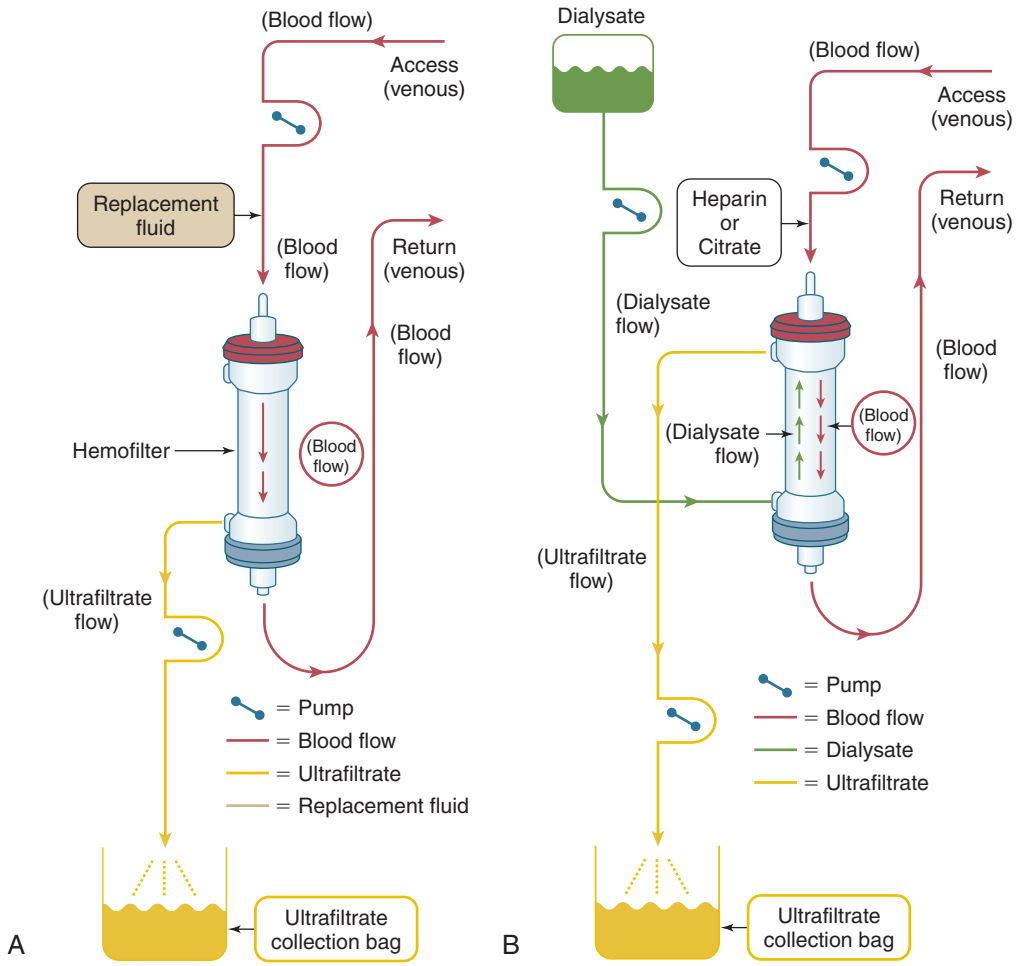

FIGURE 15-9 **A,** Schematic of continuous venovenous hemofiltration (CVVH). **B,** Schematic of continuous venovenous hemodialysis (CVVHD). (From Urden L, Stacy K, Lough M, eds. *Thelan's Critical Care Nursing: Diagnosis and Management*. 5th ed. St. Louis: Mosby, 2005.)

that dialysate solution is added around the hemofilter membranes to facilitate solute removal by the process of diffusion. Since the dialysate solution is constantly refreshed around the hemofilter membranes, the solute clearance is greater with this therapy and therefore can be used to treat both volume overload and azotemia.

Continuous venovenous hemodiafiltration (CVVHDF) combines ultrafiltration, convection, and dialysis to maximize fluid and solute removal. It is useful for the management of volume overload associated with high solute removal requirements.

Automated devices are currently marketed to facilitate the delivery of the different CRRT therapies (Figure 15-10).

Anticoagulation. The efficiency of the hemofilter can decline over time or fail suddenly because of clogging or clotting. Clogging results from the accumulation of protein and blood cells on the hemofilter membrane.[4] Filter clotting is the result of progressive loss of the hollow fibers within the hemofilter.[4] CRRT requires some form of intervention to prevent clogging and clotting. Hourly normal saline flushes may be used to extend the life of the hemofilter.

During CRRT, the patient's blood comes in contact with extracorporeal circuit and activates the coagulation cascade. Heparin is used frequently in CRRT to inhibit coagulation and extend the life of the hemofilter. However, heparin may be contraindicated if there is a risk of bleeding and heparin-induced thrombocytopenia.

An alternative to heparin during CRRT is citrate.[12,14,35] Citrate chelates calcium in the serum and inhibits activation of the coagulation cascade.[15] Systemic anticoagulation is minimal because the liver quickly converts citrate to bicarbonate. Citrate is infused into the circuit above the filter. Close monitoring of serum ionized calcium levels and calcium replacement through a separate venous line are required. Metabolic alkalosis is a concern with this therapy. Bicarbonate-based replacement solutions should not be used.

Nursing care. The critical care nurse is responsible for monitoring the patient receiving CRRT. In many critical care units, the CRRT system is set up by the dialysis staff but is maintained by critical care nurses with additional training. The patient's hemodynamic status is monitored hourly, including fluid intake and output. Temperature is monitored

TABLE 15-6 CONTINUOUS RENAL REPLACEMENT THERAPIES

ABBREVIATION	NAME	PURPOSE	VASCULAR ACCESS REQUIRED	DESCRIPTION
SCUF	Slow continuous ultrafiltration	Fluid removal	Dual-lumen venous catheter or two large venous catheters	Venous blood is circulated through a hemofilter and returned to the patient through a venous catheter: ultrafiltrate (fluid removed) is collected in a drainage bag as it exits the hemofilter
CVVH	Continuous venovenous hemofiltration	Fluid and some uremic waste product removal	Dual-lumen venous catheter or two large venous catheters	Venous blood is circulated through a hemofilter and returned to the patient through a venous catheter; replacement fluid is used to increase flow through the hemofilter; ultrafiltrate (fluid removed) is collected in a drainage bag as it exits the hemofilter
CVVHD	Continuous venovenous hemodialysis	Fluid and maximal uremic waste product removal	Dual-lumen venous catheter or two large venous catheters	Venous blood is circulated through a hemofilter (surrounded by a dialysate solution) and returned to the patient through a venous catheter; replacement solution may be used to improve convection; ultrafiltrate (fluid and waste products removed) is collected in a drainage bag as it exits the hemofilter
CVVHDF	Continuous venovenous hemodiafiltration	Maximal fluid and uremic waste product removal	Dual-lumen venous catheter or two large venous catheters	Venous blood is circulated through a hemofilter (surrounded by a dialysate solution) and returned to the patient through a venous catheter; replacement solution is used to maintain fluid balance; ultrafiltration (fluid and waste products removed) is collected in a drainage bag as it exits the hemofilter

FIGURE 15-10 Prismaflex continuous renal replacement therapy system. (Courtesy Gambro, Lakewood, CO.)

because significant heat can be lost when blood is circulating through the extracorporeal circuit. Specialized devices to warm the dialysate or replacement fluid or to rewarm the blood returning to patient are available.

Ultrafiltration volume is assessed hourly, and appropriate replacement fluid is administered. The hemofilter is assessed every 2 to 4 hours for clotting (as evidenced by dark fibers or a rapid decrease in the amount of ultrafiltration without a change in the patient's hemodynamic status). If clotting is suspected, the system is flushed with 50 to 100 mL of normal saline and observed for dark streaks or clots.[12] If present, the system may have to be changed. Results of serum chemistries, clotting studies, and other tests are monitored. The CRRT system is frequently assessed to ensure filter and lines are visible at all times, kinks are prevented, and the blood tubing is warm to the touch. The ultrafiltrate is assessed for blood (pink-tinged to frank blood), which is indicative of membrane rupture. Sterile technique is performed during vascular access dressing changes.

Peritoneal dialysis. Peritoneal dialysis is the removal of solutes and fluid by diffusion through a patient's own semipermeable membrane (the peritoneal membrane) with a dialysate solution that has been instilled into the peritoneal cavity. The peritoneal membrane surrounds the abdominal cavity and lines the organs inside the abdominal cavity. This renal replacement therapy is not commonly used for the treatment of AKI because of its comparatively slow ability to alter biochemical imbalances.

Indications. Clinical indications for peritoneal dialysis include acute and chronic kidney injury, severe water intoxication, electrolyte disorders, and drug overdose. Advantages of peritoneal dialysis include easy and rapid assembly of the equipment, relatively inexpensive cost, minimal danger of acute electrolyte imbalances or hemorrhage, and easily individualized dialysate solutions. In addition, automated peritoneal dialysis systems are available. Disadvantages of peritoneal dialysis include that it is time intensive, requiring at least 36 hours for a therapeutic effect to be achieved; biochemical disturbances are corrected slowly; access to the peritoneal cavity is sometimes difficult; and the risk of peritonitis is high.

Complications. Although rare, many complications can result from peritoneal dialysis. Complications can be divided into three categories: mechanical problems, metabolic imbalances, and inflammatory reactions. Potential complications resulting from mechanical problems include perforation of the abdominal viscera during insertion of the catheter, poor drainage in or out of the abdominal cavity as a result of catheter blockage, patient discomfort from the pressure of the fluid within the peritoneal cavity, and pulmonary complications as a result of the pressure of the fluid in the peritoneal cavity. Metabolic imbalances include hypovolemia and hypernatremia from excessively rapid removal of fluid, hypervolemia from impaired drainage of fluid, hypokalemia from the use of potassium-free dialysate, alkalosis from the use of an alkaline dialysate, disequilibrium syndrome from excessively rapid removal of fluid and waste products, and hyperglycemia from the high glucose concentration of the dialysate. Inflammatory reactions include peritoneal irritation produced by the catheter and peritonitis from bacterial infection.

Peritonitis is the most common complication of peritoneal dialysis therapy and is usually caused by contamination in the system. Aseptic technique must occur when handling the peritoneal catheter and connections. Peritonitis is manifested by abdominal pain, cloudy peritoneal fluid, fever and chills, nausea and vomiting, and difficulty in draining fluid from the peritoneal cavity.

OUTCOMES

With appropriate nursing and medical interventions, expected outcomes for the patient with AKI include:

- Fluid balance and hemodynamic status are stable.
- Body weight is within 2 lb of dry weight.
- Vital signs are stable and are consistent with baseline.
- Skin turgor is normal, and oral mucosa is intact and well hydrated.
- Serum laboratory values and arterial blood gas results are within normal limits.
- Infection is absent.
- Nutritional intake is adequate for the maintenance of the desired weight.
- The patient and family members are able to participate in the patient's care and are able to make informed decisions.

CASE STUDY

Mr. K.G. is a thin 60-year-old man admitted to the hospital for cardiac catheterization for recurrent angina. Past medical history includes hypertension, type 2 diabetes mellitus, and a previous myocardial infarction 2 years ago. Current medications are metformin (Glucophage), glipizide (Glucotrol), enteric-coated aspirin (Ecotrin), and lisinopril (Zestril). Laboratory tests on admission revealed the following: normal electrolyte levels; blood urea nitrogen (BUN), 40 mg/dL; and serum creatinine, 2.0 mg/dL. A complete blood cell count and urinalysis were unremarkable. Mr. K.G. receives intravenous fluids at 20 mL/hr on the morning of the procedure. He successfully undergoes the catheterization and returns to the telemetry unit. The day after the procedure, Mr. K.G.'s urine output decreases to less than 10 mL/hr. Mr. K.G. is given a fluid bolus of normal saline without any increase in urine output. Furosemide is administered intravenously, with a slight increase in urine output to 15 mL/hr for several hours. Laboratory studies reveal the following: potassium, 5.9 mEq/L; BUN, 70 mg/dL; serum creatinine, 7.1 mg/dL, and carbon dioxide total content, 16 mEq/L. The next day Mr. K.G. has 2+ edema and basilar crackles, and he complains of feeling short of breath. A preliminary diagnosis of AKI is made.

Questions

1. What are possible factors predisposing Mr. K.G. for AKI?
2. What laboratory studies assist in the diagnosis of AKI? Describe expected results for a patient with acute tubular necrosis.
3. What medical interventions do you anticipate for Mr. K.G.?
4. What interventions could have been taken before Mr. K.G.'s cardiac catheterization to possibly prevent his AKI?
5. Discuss the advantages and disadvantages of using diuretic therapy in patients with AKI.

SUMMARY

The patient with AKI poses many clinical challenges for healthcare personnel. Many of these patients have multisystem failure and require intensive and aggressive care. In addition, the development of AKI is an event that often catches the patient and family unprepared. Nurses play a pivotal role in promoting positive patient outcomes through prevention, sharp assessment skills, and supportive nursing care.

CRITICAL THINKING EXERCISES

1. Identify two strategies that the critical care nurse can use to help prevent AKI.
2. Describe physical examination and laboratory findings that may be seen in patients with prerenal AKI.
3. Describe patients who are at high risk for contrast-induced nephropathy and discuss medical and nursing interventions that may be used to decrease their risk.
4. You are caring for a patient with AKI postoperatively. The cardiac monitor demonstrates tall, tented T waves and a PR interval of 0.26 seconds.
   a. What electrolyte imbalance do you suspect?
   b. What medical interventions do you anticipate?
   c. Describe the mechanism of action for each medical intervention.
5. What are common indications for initiating dialysis in patients with AKI?

REFERENCES

1. Astle S. Continuous renal replacement therapies. In Wiegand D, ed. *AACN Procedure Manual for Critical Care.* 6th ed. Philadelphia: Saunders. 2011:1018-1032.
2. Bellomo R, Ronco C, Kellum J, et al. Acute renal failure-definition, outcome measures, animal models, fluid therapy and information technology needs: the Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group, *Critical Care*. 2004;8(4):R204-R212.
3. Boswell W, Jadvar H, Palmer S. Diagnostic kidney imaging. In Brenner BM, Levine SA, eds. *Brenner and Rector's The Kidney*. 8th ed. Philadelphia, PA: Saunders. 2007;839-914.
4. Boyle M, Baldwin I. Understanding the continuous renal replacement therapy circuit for acute renal failure support. *AACN Advanced Critical Care*. 2010;21(4):367-375.
5. Choudhury D, Levi M. Aging and kidney disease. In Brenner BM, Levine SA, eds. *Brenner and Rector's The Kidney*. 8th ed. Philadelphia, PA: Saunders. 2007;681-701.
6. Chrysochoou G, Marcus R, Sureshkumar K, et al. Renal replacement therapy in the critical care unit. *Critical Care Nursing Quarterly*. 2008;31(4):282-290.
7. Chung K, Juncos L, Wolf S, et al. Continuous renal replacement therapy improves survival in severely burned military casualties with acute kidney injury. *The Journal of Trauma*. 2008;64(2): S179-S187.
8. Clarkson M, Friedlewald J, Eustace J, et al. Acute kidney injury. In Brenner BM, Levine SA, eds. *Brenner and Rector's The Kidney*. 8th ed. Philadelphia, PA: Saunders. 2007;943-986.
9. Curhan GC, Mitch WE. Diet and kidney disease. In Brenner BM, Levine SA, eds. *Brenner and Rector's The Kidney.* 8th ed. Philadelphia, PA: Saunders. 2007;1817-1847:2.
10. Dennen P, Douglas I, Anderson R. Acute kidney injury in the intensive care unit: an update and primer for the intensivist. *Critical Care Medicine*. 2010;38(1):261-275.
11. Dirkes S. Acute kidney injury: not just acute renal failure anymore? *Critical Care Nurse*. 2011;31(1):37-50.
12. Dirkes S, Hodge K. Continuous renal replacement therapy in the adult intensive care unit: history and current trends. *Critical Care Nurse.* February Supplement. 2008;8-27.
13. Ellis J, Cohan R. Prevention of contrast-induced nephropathy: an overview. *Radiologic Clinics North America*. 2009;47: 801-811.
14. Faybik P, Hetz H, Mitterer G, et al. Regional citrate anticoagulation in patients with liver failure supported by a molecular adsorbent recirculating system. *Critical Care Medicine*. 2011;39(2):273-279.
15. Goldberg S, Vijayan A. Renal replacement therapy. In Koleff M, Bedient T, Isakow W, et al, eds. *The Washington Manual of Critical Care*. Philadelphia: Lippincott, Williams & Wilkins. 2008;304-309.
16. Gong R, Dworkin L, Brenner B, et al. The renal circulation and glomerular ultrafiltration. In Brenner BM, Levine SA, eds. *Brenner and Rector's The Kidney*. 8th ed. 2007;91-129.
17. Hamm L, Nakhoul N. Renal acidification. In Brenner BM, Levine SA, eds. *Brenner and Rector's The Kidney*. 8th ed. Philadelphia, PA: Saunders. 2007;248-279.
18. Himmelfarb J, Joannidis M, Molitoris B, et al. Evaluation and initial management of acute kidney injury. *Clinical Journal of American Society of Nephrology*. 2008;3:962-967.
19. Hoste E, Schurgers M. Epidemiology of acute kidney injury: how big is the problem? *Critical Care Medicine*. 2008;36 (4 Suppl.):S146-S151.
20. Ishihara S, Ward J, Tasaki O, et al. Cardiac contractility during hemofiltration in an awake mode of hyperdynamic endotoxemia. *The Journal of Trauma*. 2009;67(5):1055-1061.
21. Katzberg R, Lamba R. Contrast-induced nephropathy: after intravenous administration: fact or fiction? *Radiologic Clinics of North America*. 2009;47:789-900.
22. U.S. Food and Drug Administration. *Kayexalate (Sodium Polystyrene Sulfonate) Powder [Safety –Medwatch Fact Sheet]*. (January 2011). www.fda.gov/safety/medwatch/safetyinformation/ucm186845.htm; 2011 Accessed May 15, 2011.
23. Kellum J. Acute kidney injury. *Critical Care Medicine.* 2008;36(4 Suppl.):S141-S145.
24. Kellum J, Lamiere N, Aspelin P, et al. KDIGO Clinical practice guidelines for acute kidney injury. *Kidney International.* 2012;2(1 Suppl.):S1-138.
25. Klouche K, Amigues L, Deleuse S, et al. Preventive strategies of renal insufficiency in patients with diabetes undergoing intervention or arteriography (the PREVENT trial). *American Journal of Cardiology*. 2011;107(10):1447-1452.
26. Madsen K, Nielsen S, Tisher C. Anatomy of the kidney. In Brenner BM, Levine SA, eds. *Brenner and Rector's The Kidney*. 8th ed. Philadelphia, PA: Saunders. 2007;25-90.
27. Martin R. Acute kidney injury. *AACN Advanced Critical Care*. 2010;21(4):350-356.
28. Mehta R, Kellum J, Shah S, et al. Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. *Critical Care. 2007;*11(2):R31.

29. Miller A. Urinary function. In *Nursing for Wellness in Older Adults*. 5th ed. Urinary function. Philadelphia: Lippincott, Williams & Wilkins. 2009;390-416.
30. O'Grady N, Alexander M, Burns L, et al. Healthcare Infection Control Practices Advisory Committee. Guidelines for the prevention of intravascular catheter-related infections 2011. http://www.cdc.gov/hicpac/pdf/guidelines/bsi-guidelines-2011.pdf; 2011 Accessed March 3, 2012.
31. Ostermann M, Chang R. Acute kidney injury in the intensive care unit according to RIFLE. *Critical Care Medicine*. 2007;35(8):1837-1843.
32. Pannu N, Nadim M. An overview of drug-induced acute kidney injury. *Critical Care Medicine*. 2008;36(4 Suppl.): S216-S223.
33. Shingarev R, Allon M. A physiologic-based approach to treatment of acute hyperkalemia. *American Journal of Kidney Diseases*. 2010;56(3):578-584.
34. Solomon R. Contrast-induced acute kidney injury. *Radiologic Clinics of North America*. 2009;47:783-788.
35. Srisawat N, Hoste E, Kellum J. Modern classification of acute kidney injury. *Blood Purification*. 2010;29:300-307.
36. Tillman J. Heparin versus citrate for anticoagulation in critically ill patients treated with continuous renal replacement therapy. *Nursing in Critical Care*. 2009;14(4):191-197.
37. Uchino S, Bellomo R, Goldsmith D, et al. An assessment of the RIFLE criteria for acute renal failure in hospitalized patients. *Critical Care Medicine*. 2006;34(7):1913-1917.
38. Uchino S, Kellum J, Bellomo R, et al (2005). Acute renal failure in critically ill patients: a multinational, multicenter study. *Journal of the American Medical Association*. 2005;294(7): 813-818.

CHAPTER

16

Hematological and Immune Disorders

Patricia B. Wolff, MSN, RN, ACNS-BC, AOCNS
Jill T. Dickerson, MSN, RN, CNS-MS, FNP-BC, CWON
Marthe J. Moseley, PhD, RN, CCNS

evolve WEBSITE

Many additional resources, including self-assessment exercises, are located on the Evolve companion website at http://evolve.elsevier.com/Sole.

- Review Questions
- Mosby's Nursing Skills Procedures
- Animations
- Video Clips

INTRODUCTION

Hematological and immunological functions are necessary for gas exchange, tissue perfusion, nutrition, acid-base balance, protection against infection, and hemostasis. These complex, integrated responses are easily disrupted. Because most critically ill patients experience some abnormalities in hematological and immune function, this chapter provides a general overview of the pertinent anatomy and physiology of these organ systems and the typical alterations in red blood cells (RBCs), immune activity, and coagulation function. Table 16-1 defines key terms used in this chapter in describing hematological and immunological disorders. Guidelines are also presented for assessment and nursing care strategies needed by novice critical care nurses caring for patients at risk for these disorders.

REVIEW OF ANATOMY AND PHYSIOLOGY

Hematopoiesis

Hematopoiesis is defined as the formation and maturation of blood cells. The primary site of hematopoietic cell production is the bone marrow; however, secondary hematopoietic organs that participate in this process include the spleen, liver, thymus, lymphatic system, and lymphoid tissues. Negative feedback mechanisms within the body induce the bone marrow's pluripotent hematopoietic stem cells to differentiate into one of the three blood cells (Figure 16-1): erythrocytes (RBCs), leukocytes (white blood cells [WBCs]), or thrombocytes (platelets).[10]

In infancy, most bones are filled with blood-forming red marrow; in adulthood, productive bone marrow is found in the vertebrae, skull, mandible, thoracic cage, shoulder, pelvis, femora, and humeri.[16] The hematopoietic and immunological organs and their key functions are summarized in Figure 16-2.

Effects of Aging

Aging affects several aspects of both hematological and immune systems. For example, elderly individuals have a greater risk of infection related to alterations in immunoglobulin levels. Changes in bone marrow reserve, immune function, lean body mass, hepatic function, and renal function contribute to the challenges of caring for this rapidly expanding, vulnerable population. These changes and implications are described in the Geriatric Considerations feature.

Components and Characteristics of Blood

Blood was recognized as being essential to life as early as the 1600s, but the specific composition and characteristics of blood were not defined until the twentieth century. Blood has four major components: (1) a fluid component called plasma, (2) circulating solutes such as ions, (3) serum proteins, and (4) cells. Plasma comprises about 55% of blood volume and is the transportation medium for important serum proteins such as albumin, globulin, fibrinogen, prothrombin, and plasminogen. The hematopoietic cells comprise the remaining 45% of blood volume. Characteristics of blood and potential alterations that may be encountered in critically ill patients are shown in Table 16-2.[16]

TABLE 16-1 HEMATOLOGY-IMMUNOLOGY KEY TERMS

TERM	DEFINITION
Active immunity	A term used when the body actively produces cells and mediators that result in the destruction of the antigen
Anemia	A reduction in the number of circulating red blood cells or hemoglobin that leads to inadequate oxygenation of tissues; subtypes named by etiology (e.g., aplastic anemia means "without cells") or by cell appearance (e.g., macrocytic anemia has large cells)
Antibody	Immune globulin, created by specific lymphocytes, and designed to immunologically destroy a specific foreign antigen
Anticoagulants	Factors inhibiting the clotting process
Antigen	Any substance that is capable of stimulating an immune response in the host
Autoimmunity	Situation in which the body abnormally sees self as nonself, and an immune response is activated against those tissues
Bone marrow transplant	Replacement of defective bone marrow with marrow that is functional; described in transplant terms of the source (e.g., autologous comes from self, and allogeneic comes from another person)
Cellular immunity	Production of cytokines in response to foreign antigen
Coagulation pathway	A predetermined cascade of coagulation proteins that are stimulated by production of the platelet plug, and occurs progressively, producing a fibrin clot; there are two pathways (intrinsic and extrinsic) triggered by different events that merge into a single list of events leading to a fibrin clot; clotting may be initiated by either or both pathways
Coagulopathy	Disorder of normal clotting mechanisms; usually used to describe inappropriate bleeding more often than excess clotting, but can refer to either one
Cytokines	Cell killer substances, or mediators secreted by white blood cells; when secreted by a lymphocyte, also called lymphokine, and secretions from monocytes are called monokines
Disseminated intravascular coagulation	Disorder of hemostasis characterized by exaggerated microvascular coagulation and intravascular depletion of clotting factors, with subsequent bleeding; also called consumption coagulopathy
Ecchymosis	Blue or purplish hemorrhagic spot on skin or mucous membrane; round or irregular, nonelevated
Epistaxis	Bleeding from the nose
Erythrocyte	Red blood cell
Fibrinolysis	Breakdown of fibrin clots that naturally occurs 1-3 days after clot development
Hemarthrosis	Blood in a joint cavity
Hematemesis	Bloody emesis
Hematochezia	Blood in stool; bright red
Hematoma	Raised, hardened mass indicative of blood vessel rupture and clotting beneath the skin surface; if subcutaneous, appears as a blue-purple or purple-black area; may occur in spaces such as pleural or retroperitoneal area
Hematopoiesis	Development of the early blood cells (erythrocytes, leukocytes, thrombocytes), encompassing their maturation in the bone marrow or lymphoreticular organs
Hematuria	Blood in the urine
Hemoglobinuria	Hemoglobin in the urine
Hemoptysis	Coughing up blood from the airways or lungs
Hemorrhage	Copious, active bleeding
Hemostasis	A physiological process involving hematological and nonhematological factors to form a platelet or fibrin clot to control the loss of blood
Human immunodeficiency virus	A retrovirus that transcribes its RNA-containing genetic material into DNA of the host cell nucleus; this virus has a propensity for the immune cells, replacing the RNA of lymphocytes and macrophages, causing an immunodeficient state
Humoral immunity	Production of antibodies in response to foreign proteins
Immunocompromised	Quantitative or qualitative defects in white blood cells or immune physiology; defect may be congenital or acquired and involve a single element or multiple processes; immune incompetence leads to lack of normal inflammatory, phagocytic, antibody, or cytokine responses

Continued