Volume 1

BRUNNER & SUDDARTH'S

Textbook of Medical-Surgical Nursing

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TWELFTH EDITION

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Contents

chapter 14

Fluid and Electrolytes: Balance and Disturbance

LEARNING OBJECTIVES

On completion of this chapter, the learner will be able to:

1. Differentiate between osmosis, diffusion, filtration, and active transport.
2. Describe the role of the kidneys, lungs, and endocrine glands in regulating the body's fluid composition and volume.
3. Identify the effects of aging on fluid and electrolyte regulation.
4. Plan effective care of patients with the following imbalances: fluid volume deficit and fluid volume excess; sodium deficit (hyponatremia) and sodium excess (hypernatremia); potassium deficit (hypokalemia) and potassium excess (hyperkalemia).
5. Describe the cause, clinical manifestations, management, and nursing interventions for the following imbalances: calcium deficit (hypocalcemia) and calcium excess (hypercalcemia); magnesium deficit (hypomagnesemia) and magnesium excess (hypermagnesemia); phosphorus deficit (hypophosphatemia) and phosphorus excess (hyperphosphatemia); chloride deficit (hypochloremia) and chloride excess (hyperchloremia).
6. Explain the roles of the lungs, kidneys, and chemical buffers in maintaining acid–base balance.
7. Compare metabolic acidosis and alkalosis with regard to causes, clinical manifestations, diagnosis, and management.
8. Compare respiratory acidosis and alkalosis with regard to causes, clinical manifestations, diagnosis, and management.
9. Interpret arterial blood gas measurements.
10. Identify a safe and effective procedure of venipuncture.
11. Describe measures used for preventing complications of intravenous therapy.

GLOSSARY

acidosis: an acid–base imbalance characterized by an increase in H^+ concentration (decreased blood pH). A low arterial pH due to reduced bicarbonate concentration is called metabolic acidosis; a low arterial pH due to increased PCO_2 is respiratory acidosis

active transport: physiologic pump that moves fluid from an area of lower concentration to one of higher concentration; active transport requires adenosine triphosphate for energy

alkalosis: an acid–base imbalance characterized by a reduction in H^+ concentration (increased blood pH). A high arterial pH with increased bicarbonate concentration is called metabolic alkalosis; a high arterial pH due to reduced PCO_2 is respiratory alkalosis

diffusion: the process by which solutes move from an area of higher concentration to one of lower concentration; does not require expenditure of energy

homeostasis: maintenance of a constant internal equilibrium in a biologic system that involves positive and negative feedback mechanisms

hydrostatic pressure: the pressure created by the weight of fluid against the wall that contains it. In the body, hydrostatic pressure in blood vessels results from the weight of fluid itself and the force resulting from cardiac contraction

hypertonic solution: a solution with an osmolality higher than that of serum

hypotonic solution: a solution with an osmolality lower than that of serum

isotonic solution: a solution with the same osmolality as serum and other body fluids. Osmolality falls within normal range for serum (280 to 300 mOsm/kg)

osmolality: the number of osmoles (the standard unit of osmotic pressure) per kilogram of solution. Expressed as mOsm/kg, osmolality is used more often than the term *osmolarity* to evaluate serum and urine

osmolarity: the number of osmoles (the standard unit of osmotic pressure) per liter of solution. It is expressed as milliosmoles per liter (mOsm/L); describes the concentration of solutes or dissolved particles

osmosis: the process by which fluid moves across a semipermeable membrane from an area of low solute concentration to an area of high solute concentration; the process continues until the solute concentrations are equal on both sides of the membrane

tonicity: fluid tension within the extracellular fluid or intracellular fluid that describes the relationship between the solutes and water, primarily determined by fluid osmolality

Fluid and electrolyte balance is a dynamic process that is crucial for life and **homeostasis.** Potential and actual disorders of fluid and electrolyte balance occur in every setting, with every disorder, and with a variety of changes that affect healthy people (eg, increased fluid and sodium loss with strenuous exercise and high environmental temperature, inadequate intake of fluid and electrolytes) as well as those who are ill.

Fundamental Concepts

Nurses need an understanding of the physiology of fluid and electrolyte balance and acid–base balance to anticipate, identify, and respond to possible imbalances. Nurses also must use effective teaching and communication skills to help prevent and treat various fluid and electrolyte disturbances.

Amount and Composition of Body Fluids

Approximately 60% of the weight of a typical adult consists of fluid (water and electrolytes). Factors that influence the amount of body fluid are age, gender, and body fat. In general, younger people have a higher percentage of body fluid than older people, and men have proportionately more body fluid than women. People who are obese have less fluid than those who are thin, because fat cells contain little water. The skeleton also has a low water content. Muscle, skin, and blood have the highest amount of water.

Body fluid is located in two fluid compartments: the intracellular space (fluid in the cells) and the extracellular space (fluid outside the cells). Approximately two thirds of body fluid is in the intracellular fluid (ICF) compartment and is located primarily in the skeletal muscle mass. Approximately one third is in the extracellular fluid (ECF) compartment.

The ECF compartment is further divided into the intravascular, interstitial, and transcellular fluid spaces. Circulatory and neurologic symptoms, physical examination findings, and laboratory test results can be used to identify the compartment from which fluid is lost (McPhee, Papadakis & Tierney, 2007). The intravascular space (the fluid within the blood vessels) contains plasma, the effective circulating volume. Approximately 3 L of the average 6 L of blood volume is made up of plasma. The remaining 3 L is made up of erythrocytes, leukocytes, and thrombocytes. The interstitial space contains the fluid that surrounds the cell and totals about 11 to 12 L in an adult. Lymph is an interstitial fluid. The transcellular space is the smallest division of the ECF compartment and contains approximately 1 L. Examples of transcellular fluids include cerebrospinal, pericardial, synovial, intraocular, and pleural fluids; sweat; and digestive secretions. As the next section describes, the ECF transports electrolytes; it also carries other substances, such as enzymes and hormones.

Body fluid normally moves between the two major compartments or spaces in an effort to maintain an equilibrium between the spaces. Loss of fluid from the body can disrupt this equilibrium. Sometimes fluid is not lost from the body but is unavailable for use by either the ICF or ECF. Loss of ECF into a space that does not contribute to equilibrium between the ICF and the ECF is referred to as a third-space fluid shift, or "third spacing" for short (Holcomb, 2008).

Early evidence of a third-space fluid shift is a decrease in urine output despite adequate fluid intake. Urine output decreases because fluid shifts out of the intravascular space; the kidneys then receive less blood and attempt to compensate by decreasing urine output. Other signs and symptoms of third spacing that indicate an intravascular fluid volume deficit include increased heart rate, decreased blood pressure, decreased central venous pressure, edema, increased body weight, and imbalances in fluid intake and output (I&O). Third-space shifts occur in patients who have hypocalcemia, decreased iron intake, severe liver diseases, alcoholism, hypothyroidism, malabsorption, immobility,, burns, and cancer (Holcomb, 2008).

Electrolytes

Electrolytes in body fluids are active chemicals (cations that carry positive charges and anions that carry negative charges). The major cations in body fluid are sodium, potassium, calcium, magnesium, and hydrogen ions. The major anions are chloride, bicarbonate, phosphate, sulfate, and proteinate ions.

These chemicals unite in varying combinations. Therefore, electrolyte concentration in the body is expressed in terms of milliequivalents (mEq) per liter, a measure of chemical activity, rather than in terms of milligrams (mg), a unit of weight. More specifically, a milliequivalent is defined as being equivalent to the electrochemical activity of 1 mg of hydrogen. In a solution, cations and anions are equal in milliequivalents per liter.

Electrolyte concentrations in the ICF differ from those in the ECF, as reflected in Table 14-1. Because special tech-

Table 14-1 APPROXIMATE MAJOR ELECTROLYTE CONTENT IN BODY FLUID

Electrolytes	mEq/L
Extracellular Fluid (Plasma)	
Cations	
Sodium (Na^+)	142
Potassium (K^+)	5
Calcium (Ca^{++})	5
Magnesium (Mg^{++})	2
Total cations	154
Anions	
Chloride (Cl^-)	103
Bicarbonate (HCO_3^-)	26
Phosphate (HPO_4^-)	2
Sulfate (SO_4^-)	1
Organic acids	5
Proteinate	17
Total anions	154
Intracellular Fluid	
Cations	
Potassium (K^+)	150
Magnesium (Mg^{++})	40
Sodium (Na^+)	10
Total cations	200
Anions	
Phosphates and sulfates	150
Bicarbonate (HCO_3^-)	10
Proteinate	40
Total anions	200

niques are required to measure electrolyte concentrations in the ICF, it is customary to measure the electrolytes in the most accessible portion of the ECF, namely, the plasma.

Sodium ions, which are positively charged, far outnumber the other cations in the ECF. Because sodium concentration affects the overall concentration of the ECF, sodium is important in regulating the volume of body fluid. Retention of sodium is associated with fluid retention, and excessive loss of sodium is usually associated with decreased volume of body fluid.

As shown in Table 14-1, the major electrolytes in the ICF are potassium and phosphate. The ECF has a low concentration of potassium and can tolerate only small changes in potassium concentrations. Therefore, release of large stores of intracellular potassium, typically caused by trauma to the cells and tissues, can be extremely dangerous.

The body expends a great deal of energy maintaining the high extracellular concentration of sodium and the high intracellular concentration of potassium. It does so by means of cell membrane pumps that exchange sodium and potassium ions. Normal movement of fluids through the capillary wall into the tissues depends on **hydrostatic pressure** (the pressure exerted by the fluid on the walls of the blood vessel) at both the arterial and the venous ends of the vessel and the osmotic pressure exerted by the protein of plasma. The direction of fluid movement depends on the differences in these two opposing forces (hydrostatic versus osmotic pressure).

Regulation of Body Fluid Compartments

Osmosis and Osmolality

When two different solutions are separated by a membrane that is impermeable to the dissolved substances, fluid shifts through the membrane from the region of low solute concentration to the region of high solute concentration until the solutions are of equal concentration. This diffusion of water caused by a fluid concentration gradient is known as **osmosis** (Fig. 14-1A). The magnitude of this force depends on the number of particles dissolved in the solutions, not on their weights. The number of dissolved particles contained in a unit of fluid determines the osmolality of a solution, which influences the movement of fluid between the fluid compartments (Goertz, 2006). **Tonicity** is the ability of all the solutes to cause an osmotic driving force that promotes water movement from one compartment to another. The control of tonicity determines the normal state of cellular hydration and cell size. Sodium, mannitol, glucose, and sorbitol are effective osmoles (capable of affecting water movement). Three other terms are associated with osmosis: osmotic pressure, oncotic pressure, and osmotic diuresis.

- Osmotic pressure is the amount of hydrostatic pressure needed to stop the flow of water by osmosis. It is

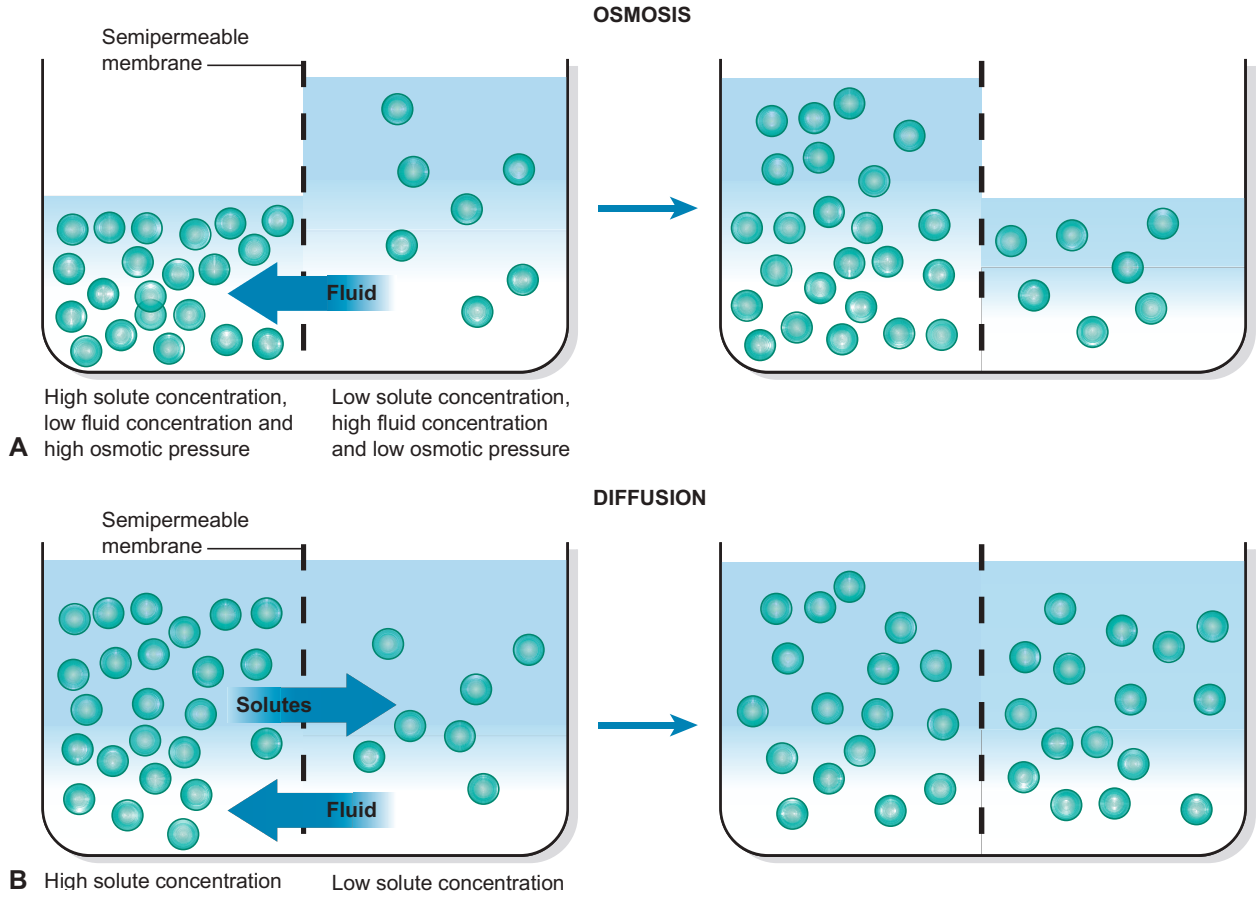

Figure 14-1 **A,** Osmosis: movement of fluid from an area of lower solute concentration to an area of higher solute concentration with eventual equalization of the solute concentrations. **B,** Diffusion: movement of solutes from an area of greater concentration to an area of lesser concentration, leading ultimately to equalization of the solute concentrations.

primarily determined by the concentration of solutes.
- Oncotic pressure is the osmotic pressure exerted by proteins (eg, albumin).
- Osmotic diuresis is the increase in urine output caused by the excretion of substances such as glucose, mannitol, or contrast agents in the urine.

Diffusion

Diffusion is the natural tendency of a substance to move from an area of higher concentration to one of lower concentration (see Fig. 14-1B). It occurs through the random movement of ions and molecules (Porth & Matfin, 2009). Examples of diffusion are the exchange of oxygen and carbon dioxide between the pulmonary capillaries and alveoli and the tendency of sodium to move from the ECF compartment, where the sodium concentration is high, to the ICF, where its concentration is low.

Filtration

Hydrostatic pressure in the capillaries tends to filter fluid out of the intravascular compartment into the interstitial fluid. Movement of water and solutes occurs from an area of high hydrostatic pressure to an area of low hydrostatic pressure. The kidneys filter approximately 180 L of plasma per day. Another example of filtration is the passage of water and electrolytes from the arterial capillary bed to the interstitial fluid; in this instance, the hydrostatic pressure results from the pumping action of the heart.

Sodium–Potassium Pump

As previously stated, the sodium concentration is greater in the ECF than in the ICF, and because of this, sodium tends to enter the cell by diffusion. This tendency is offset by the sodium–potassium pump that is maintained by the cell membrane and actively moves sodium from the cell into the ECF. Conversely, the high intracellular potassium concentration is maintained by pumping potassium into the cell. By definition, **active transport** implies that energy must be expended for the movement to occur against a concentration gradient.

Systemic Routes of Gains and Losses

Water and electrolytes are gained in various ways. Healthy people gain fluids by drinking and eating, and their daily average intake and output of water are approximately equal (Table 14-2).

Table 14-2 AVERAGE DAILY INTAKE AND OUTPUT IN AN ADULT

Intake (mL)		Output (mL)	
Oral liquids	1300	Urine	1500
Water in food	1000	Stool	200
Water produced by metabolism	300	Insensible	
		Lungs	300
		Skin	600
Total gain*	2600	Total loss*	2600

*Approximate volumes.

NURSING ALERT

When fluid balance is critical, all routes of systemic gain and loss must be recorded and all volumes compared. Organs of fluid loss include the kidneys, skin, lungs, and gastrointestinal (GI) tract.

Kidneys

The usual daily urine volume in the adult is 1 to 2 L. A general rule is that the output is approximately 1 mL of urine per kilogram of body weight per hour (1 mL/kg/h) in all age groups.

Skin

Sensible perspiration refers to visible water and electrolyte loss through the skin (sweating). The chief solutes in sweat are sodium, chloride, and potassium. Actual sweat losses can vary from 0 to 1000 mL or more every hour, depending on factors such as the environmental temperature. Continuous water loss by evaporation (approximately 600 mL/day) occurs through the skin as insensible perspiration, a nonvisible form of water loss. Fever greatly increases insensible water loss through the lungs and the skin, as does loss of the natural skin barrier (eg, through major burns).

Lungs

The lungs normally eliminate water vapor (insensible loss) at a rate of approximately 300 mL every day. The loss is much greater with increased respiratory rate or depth, or in a dry climate.

Gastrointestinal Tract

The usual loss through the GI tract is 100 to 200 mL daily, even though approximately 8 L of fluid circulates through the GI system every 24 hours. Because the bulk of fluid is normally reabsorbed in the small intestine, diarrhea and fistulas cause large losses.

Laboratory Tests for Evaluating Fluid Status

Osmolality is the concentration of fluid that affects the movement of water between fluid compartments by osmosis. Osmolality measures the solute concentration per kilogram in blood and urine. It is also a measure of a solution's ability to create osmotic pressure and affect the movement of water. Serum osmolality primarily reflects the concentration of sodium, although blood urea nitrogen (BUN) and glucose also play a major role in determining serum osmolality. Urine osmolality is determined by urea, creatinine, and uric acid. When measured with serum osmolality, urine osmolality is the most reliable indicator of urine concentration. Osmolality is reported as milliosmoles per kilogram of water (mOsm/kg) (Goertz, 2006).

In healthy adults, serum osmolality is 280 to 300 mOsm/kg, and normal urine osmolality is 200 to 800 mOsm/kg (Goertz, 2006). Sodium predominates in ECF osmolality and holds water in this compartment. Factors that increase and decrease serum and urine osmolality are identified in Chart 14-1. Serum osmolality may be measured directly through laboratory tests or estimated at the

Chart 14-1 • *Factors Affecting Serum and Urine Osmolality*

Fluid	Factors Increasing Osmolality	Factors Decreasing Osmolality
Serum (280–300 mOsm/kg water)	• Severe dehydration • Free water loss • Diabetes insipidus • Hypernatremia • Hyperglycemia • Stroke or head injury • Renal tubular necrosis • Consumption of methanol or ethylene glycol (antifreeze)	• Fluid volume excess • Syndrome of inappropriate antidiuretic hormone (SIADH) • Renal failure • Diuretic use • Adrenal insufficiency • Hyponatremia • Overhydration • Paraneoplastic syndrome associated with lung cancer
Urine (200–800 mOsm/kg water)	• Fluid volume deficit • SIADH • Congestive heart failure • Acidosis • Prerenal failure	• Fluid volume excess • Diabetes insipidus • Hyponatremia • Aldosteronism • Pyelonephritis • Acute tubular necrosis

bedside by doubling the serum sodium level or by using the following formula:

$$Na^+ \times 2 = \frac{\text{Glucose}}{18} + \frac{\text{BUN}}{3}$$
$$= \text{Approximate value of serum osmolality}$$

Osmolarity, another term that describes the concentration of solutions, is measured in milliosmoles per liter (mOsm/L). However, the term osmolality is used more often in clinical practice. The calculated value usually is within 10 mOsm of the measured osmolality.

Urine specific gravity measures the kidneys' ability to excrete or conserve water. The specific gravity of urine is compared to the weight of distilled water, which has a specific gravity of 1.000. The normal range of urine specific gravity is 1.010 to 1.025. Urine specific gravity can be measured at the bedside by placing a calibrated hydrometer or urinometer in a cylinder of approximately 20 mL of urine. Specific gravity can also be assessed with a refractometer or dipstick with a reagent for this purpose. Specific gravity varies inversely with urine volume; normally, the larger the volume of urine, the lower the specific gravity is. Specific gravity is a less reliable indicator of concentration than urine osmolality; increased glucose or protein in urine can cause a falsely elevated specific gravity. Factors that increase or decrease urine osmolality are the same as those for urine specific gravity.

BUN is made up of urea, which is an end product of the metabolism of protein (from both muscle and dietary intake) by the liver. Amino acid breakdown produces large amounts of ammonia molecules, which are absorbed into the bloodstream. Ammonia molecules are converted to urea and excreted in the urine. The normal BUN is 10 to 20 mg/dL (3.6 to 7.2 mmol/L). The BUN level varies with urine output. Factors that increase BUN include decreased renal function, GI bleeding, dehydration, increased protein intake, fever, and sepsis. Those that decrease BUN include end-stage liver disease, a low-protein diet, starvation, and any condition that results in expanded fluid volume (eg, pregnancy).

Creatinine is the end product of muscle metabolism. It is a better indicator of renal function than BUN because it does not vary with protein intake and metabolic state. The normal serum creatinine is approximately 0.7 to 1.4 mg/dL (62 to 124 mmol/L); however, its concentration depends on lean body mass and varies from person to person. Serum creatinine levels increase when renal function decreases.

Hematocrit measures the volume percentage of red blood cells (erythrocytes) in whole blood and normally ranges from 42% to 52% for males and 35% to 47% for females. Conditions that increase the hematocrit value are dehydration and polycythemia, and those that decrease hematocrit are overhydration and anemia.

Urine sodium values change with sodium intake and the status of fluid volume: As sodium intake increases, excretion increases; as the circulating fluid volume decreases, sodium is conserved. Normal urine sodium levels range from 75 to 200 mEq/24 hours (75 to 200 mmol/24 hours). A random specimen usually contains more than 40 mEq/L of sodium. Urine sodium levels are used to assess volume status and are useful in the diagnosis of hyponatremia and acute renal failure.

Homeostatic Mechanisms

The body is equipped with remarkable homeostatic mechanisms to keep the composition and volume of body fluid within narrow limits of normal. Organs involved in homeostasis include the kidneys, lungs, heart, adrenal glands, parathyroid glands, and pituitary gland (Porth & Matfin, 2009).

Kidney Functions

Vital to the regulation of fluid and electrolyte balance, the kidneys normally filter 180 L of plasma every day in the adult and excrete 1 to 2 L of urine. They act both autonomously and in response to bloodborne messengers, such as aldosterone and antidiuretic hormone (ADH) (Porth & Matfin, 2009). Major functions of the kidneys in maintaining normal fluid balance include the following:

- Regulation of ECF volume and osmolality by selective retention and excretion of body fluids

- Regulation of normal electrolyte levels in the ECF by selective electrolyte retention and excretion
- Regulation of pH of the ECF by retention of hydrogen ions
- Excretion of metabolic wastes and toxic substances

Given these functions, failure of the kidneys results in multiple fluid and electrolyte abnormalities.

Heart and Blood Vessel Functions

The pumping action of the heart circulates blood through the kidneys under sufficient pressure to allow for urine formation. Failure of this pumping action interferes with renal perfusion and thus with water and electrolyte regulation.

Lung Functions

The lungs are also vital in maintaining homeostasis. Through exhalation, the lungs remove approximately 300 mL of water daily in the normal adult. Abnormal conditions, such as hyperpnea (abnormally deep respiration) or continuous coughing, increase this loss; mechanical ventilation with excessive moisture decreases it. The lungs also play a major role in maintaining acid–base balance.

Pituitary Functions

The hypothalamus manufactures ADH, which is stored in the posterior pituitary gland and released as needed to conserve water. Functions of ADH include maintaining the osmotic pressure of the cells by controlling the retention or excretion of water by the kidneys and by regulating blood volume (Fig. 14-2).

Adrenal Functions

Aldosterone, a mineralocorticoid secreted by the zona glomerulosa (outer zone) of the adrenal cortex, has a profound effect on fluid balance. Increased secretion of aldosterone causes sodium retention (and thus water retention) and potassium loss. Conversely, decreased secretion of aldosterone causes sodium and water loss and potassium retention.

Cortisol, another adrenocortical hormone, has less mineralocorticoid action. However, when secreted in large quantities (or administered as corticosteroid therapy), it can also produce sodium and fluid retention.

Parathyroid Functions

The parathyroid glands, embedded in the thyroid gland, regulate calcium and phosphate balance by means of parathyroid hormone (PTH). PTH influences bone resorption, calcium absorption from the intestines, and calcium reabsorption from the renal tubules.

Other Mechanisms

Changes in the volume of the interstitial compartment within the ECF can occur without affecting body function. However, the vascular compartment cannot tolerate change as readily and must be carefully maintained to ensure that tissues receive adequate nutrients.

Baroreceptors

The baroreceptors are located in the left atrium and the carotid and aortic arches. These receptors respond to changes in the circulating blood volume and regulate sympathetic and parasympathetic neural activity as well as endocrine activities (Rottmann, 2007).

As arterial pressure decreases, baroreceptors transmit fewer impulses from the carotid and the aortic arches to the vasomotor center. A decrease in impulses stimulates the sympathetic nervous system and inhibits the parasympathetic nervous system. The outcome is an increase in cardiac rate, conduction, and contractility and an increase in circulating blood volume. Sympathetic stimulation constricts renal arterioles; this increases the release of aldosterone, decreases glomerular filtration, and increases sodium and water reabsorption.

Renin–Angiotensin–Aldosterone System

Renin is an enzyme that converts angiotensinogen, a substance formed by the liver, into angiotensin I (Porth & Matfin, 2009). Renin is released by the juxtaglomerular cells of the kidneys in response to decreased renal perfusion. Angiotensin-converting enzyme (ACE) converts angiotensin I to angiotensin II. Angiotensin II, with its vasoconstrictor properties, increases arterial perfusion pressure and stimulates thirst. As the sympathetic nervous system is stimulated, aldosterone is released in response to an increased release of renin. Aldosterone is a volume regulator and is also released as serum potassium increases, serum sodium decreases, or adrenocorticotropic hormone (ACTH) increases.

Antidiuretic Hormone and Thirst

ADH and the thirst mechanism have important roles in maintaining sodium concentration and oral intake of fluids. Oral intake is controlled by the thirst center located in the hypothalamus (Porth & Matfin, 2009). As serum concentration or osmolality increases or blood volume decreases, neurons in the hypothalamus are stimulated by intracellular dehydration; thirst then occurs, and the person increases his or her intake of oral fluids. Water excretion is controlled by ADH, aldosterone, and baroreceptors, as mentioned previously. The presence or absence of ADH is the most significant factor in determining whether the urine that is excreted is concentrated or dilute.

Osmoreceptors

Located on the surface of the hypothalamus, osmoreceptors sense changes in sodium concentration. As osmotic pressure increases, the neurons become dehydrated and quickly release impulses to the posterior pituitary, which increases the release of ADH, which then travels in the blood to the kidneys, where it alters permeability to water, causing increased reabsorption of water and decreased urine output. The retained water dilutes the ECF and returns its concentration to normal. Restoration of normal osmotic pressure provides feedback to the osmoreceptors to inhibit further ADH release (see Fig. 14-2).

Release of Atrial Natriuretic Peptide

Atrial natriuretic peptide (ANP), also called atrial natriuretic factor, is a peptide that is synthesized, stored, and released by muscle cells of the atria of the heart in response to several factors. These factors include increased atrial pressure, angiotensin II stimulation, endothelin (a powerful vasoconstrictor of vascular smooth muscle peptide released from damaged endothelial cells in the kidneys or other tissues), and

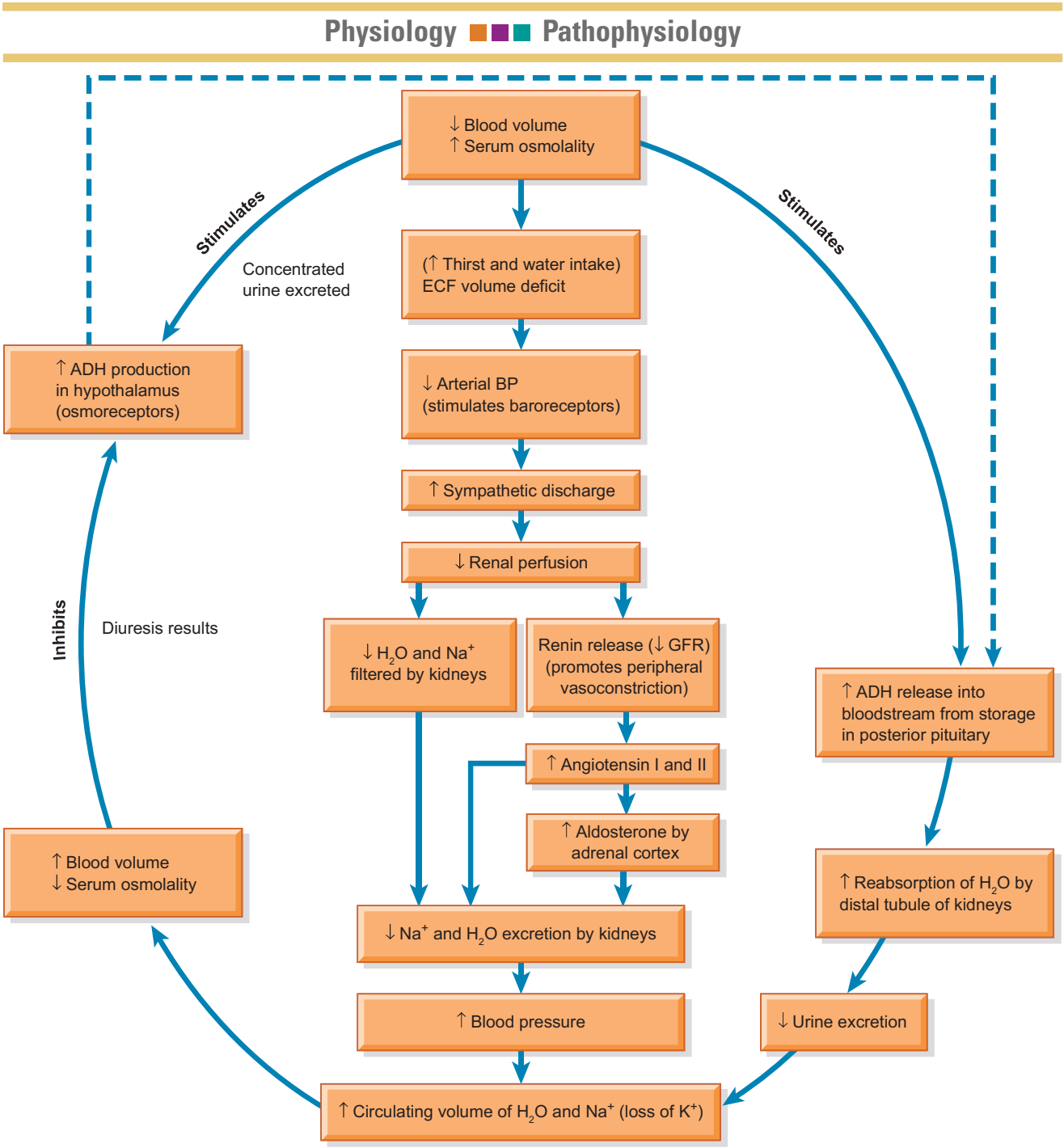

Figure 14-2 Fluid regulation cycle. ADH, antidiuretic hormone; BP, blood pressure; ECF, extracellular fluid; GFR, glomerular filtration rate.

sympathetic stimulation (Porth & Matfin, 2009). In addition, any condition that results in volume expansion (exercise, pregnancy), hypoxia, or increased cardiac filling pressures (eg, high sodium intake, heart failure, chronic renal failure, atrial tachycardia, or use of vasoconstrictor agents such as epinephrine) increases the release of ANP. The action of ANP is the direct opposite of the renin–angiotensin–aldosterone system; ANP decreases blood pressure and volume (Fig. 14-3). The ANP measured in plasma is normally 20 to 77 pg/mL (20 to 77 ng/L). This level increases in acute heart failure, paroxysmal supraventricular tachycardia, hyperthyroidism, subarachnoid hemorrhage, and small cell lung cancer. The level decreases in chronic heart failure and with the use of medications such as urea (Ureaphil) and prazosin (Minipress).

Gerontologic Considerations

Normal physiologic changes of aging, including reduced cardiac, renal, and respiratory function and reserve and alterations in the ratio of body fluids to muscle mass, may

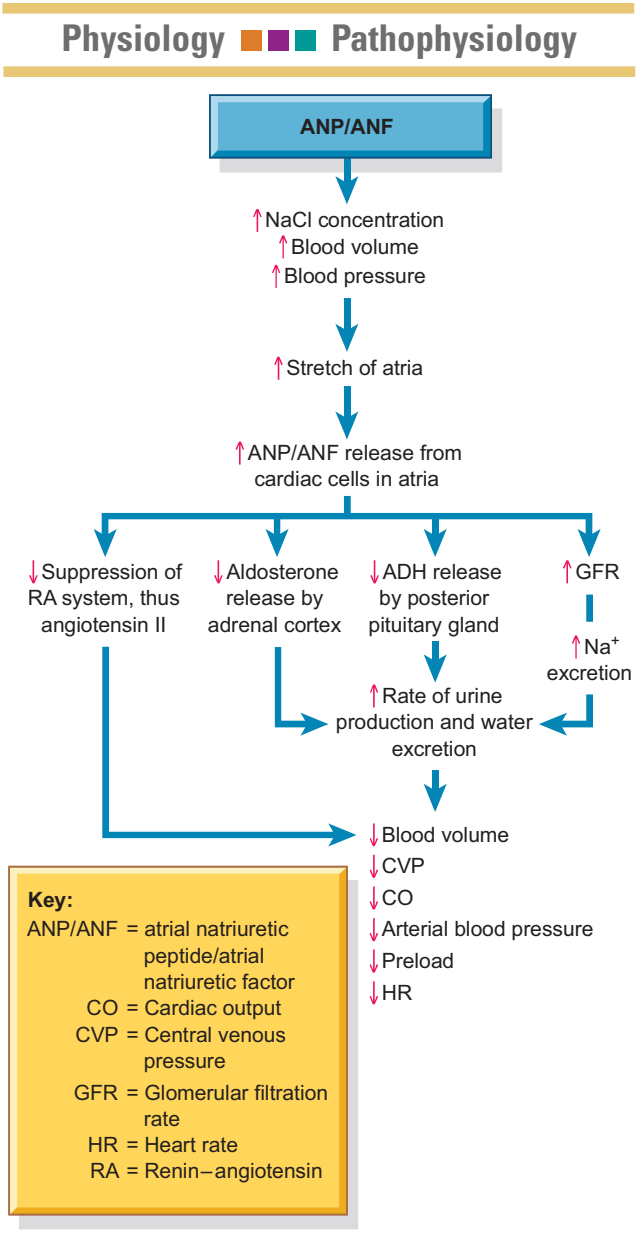

Figure 14-3 Role of ANP in maintenance of fluid balance.

alter the responses of elderly people to fluid and electrolyte changes and acid–base disturbances. Decreased respiratory function can cause impaired pH regulation in older adults with major illness or trauma. Renal function declines with age, as do muscle mass and daily exogenous creatinine production. Therefore, high-normal and minimally elevated serum creatinine values may indicate substantially reduced renal function in older adults.

In addition, the use of multiple medications by older adults can affect renal and cardiac function, thereby increasing the likelihood of fluid and electrolyte disturbances. Routine procedures, such as the vigorous administration of laxatives or enemas before colon x-ray studies, may produce a serious fluid volume deficit, necessitating the use of intravenous (IV) fluids to prevent hypotension and other effects of hypovolemia.

Alterations in fluid and electrolyte balance that may produce minor changes in young and middle-aged adults may produce profound changes in older adults. In many elderly patients, the clinical manifestations of fluid and electrolyte disturbances may be subtle or atypical. For example, fluid deficit may cause delirium in the elderly person (see Chapter 12), whereas in the young or middle-aged person the first sign commonly is increased thirst. Rapid infusion of an excessive volume of IV fluids may produce fluid overload and cardiac failure in elderly patients. These reactions are likely to occur more quickly and with the administration of smaller volumes of fluid than in healthy young and middle-aged adults because of the decreased cardiac reserve and reduced renal function that accompany aging. Dehydration in the elderly is common as a result of decreased kidney mass, decreased glomerular filtration rate, decreased renal blood flow, decreased ability to concentrate urine, inability to conserve sodium, decreased excretion of potassium, and a decrease of total body water (Powers & Daly, 2007).

FLUID VOLUME DISTURBANCES

Hypovolemia

Fluid volume deficit (FVD), or hypovolemia, occurs when loss of ECF volume exceeds the intake of fluid. It occurs when water and electrolytes are lost in the same proportion as they exist in normal body fluids, so that the ratio of serum electrolytes to water remains the same. FVD (hypovolemia) should not be confused with dehydration, which refers to loss of water alone, with increased serum sodium levels. FVD may occur alone or in combination with other imbalances. Unless other imbalances are present concurrently, serum electrolyte concentrations remain essentially unchanged.

Pathophysiology

FVD results from loss of body fluids and occurs more rapidly when coupled with decreased fluid intake. FVD can also develop with a prolonged period of inadequate intake. Causes of FVD include abnormal fluid losses, such as those resulting from vomiting, diarrhea, GI suctioning, and sweating; decreased intake, as in nausea or lack of access to fluids (Heitz & Horne, 2005); and third-space fluid shifts, or the movement of fluid from the vascular system to other body spaces (eg, with edema formation in burns, ascites with liver dysfunction). Additional causes include diabetes insipidus, adrenal insufficiency, osmotic diuresis, hemorrhage, and coma.

Clinical Manifestations

FVD can develop rapidly, and its severity depends on the degree of fluid loss. Clinical signs and symptoms include acute weight loss; decreased skin turgor; oliguria; concentrated urine; orthostatic hypotension due to volume depletion; a weak, rapid heart rate; flattened neck veins; increased temperature; thirst; decreased or delayed capillary refill; decreased central venous pressure; cool, clammy, pale skin related to peripheral vasoconstriction; anorexia; nausea; lassitude; muscle weakness; and cramps.

Assessment and Diagnostic Findings

Laboratory data useful in evaluating fluid volume status include BUN and its relation to serum creatinine concentration. A volume-depleted patient has a BUN elevated out of proportion to the serum creatinine (ratio greater than 20:1). The BUN can be elevated because of dehydration or decreased renal perfusion and function. The cause of hypovolemia may be determined through the health history and physical examination. Also, the hematocrit level is greater than normal because there is a decreased plasma volume (Powers & Daly, 2007).

Serum electrolyte changes may also exist. Potassium and sodium levels can be reduced (hypokalemia, hyponatremia) or elevated (hyperkalemia, hypernatremia).

- Hypokalemia occurs with GI and renal losses.
- Hyperkalemia occurs with adrenal insufficiency.
- Hyponatremia occurs with increased thirst and ADH release.
- Hypernatremia results from increased insensible losses and diabetes insipidus.

Urine specific gravity is increased in relation to the kidneys' attempt to conserve water and is decreased with diabetes insipidus. Aldosterone is secreted when fluid volume is low causing reabsorption of sodium and chloride, resulting in decreased urinary sodium and chloride. Urine osmolality can be greater than 450 mOsm/kg, because the kidneys try to compensate by conserving water. Normal values for laboratory data are listed in Appendix A.

Gerontologic Considerations

Increased sensitivity to fluid and electrolyte changes in elderly patients requires careful assessment of intake and output of fluids from all sources, assessment of changes in daily weight, careful monitoring of side effects and interactions of medications, and prompt reporting and management of disturbances. It is necessary to monitor skin turgor serially to detect subtle changes. However, assessment of skin turgor is not as valid in the elderly because the skin has lost some of its elasticity; therefore, other assessment measures (eg, slowness in filling of veins of the hands and feet) become more useful in detecting FVD. Skin turgor is best tested over the forehead or the sternum in elderly patients, because alterations in skin elasticity are less marked in these areas.

The nurse also performs a functional assessment of the ability of the elderly patient to determine fluid and food needs and to obtain adequate intake in addition to other assessments discussed earlier in this chapter. For example, is the patient cognitively intact, able to ambulate and to use both arms and hands to reach fluids and foods, and able to swallow? Results of this assessment have a direct bearing on how the patient will be able to meet his or her own need for fluids and foods. During an elderly patient's hospital stay, the nurse provides fluids if the patient is unable to carry out self-care activities.

The nurse should also recognize that some elderly patients deliberately restrict their fluid intake to avoid embarrassing episodes of incontinence. In this situation, the nurse identifies interventions to deal with the incontinence, such as encouraging the patient to wear protective clothing or devices, to carry a urinal in the car, or to pace fluid intake to allow access to toilet facilities during the day. Elderly people without cardiovascular or renal dysfunction should be reminded to drink adequate fluids, particularly in very warm or humid weather.

Medical Management

When planning the correction of fluid loss for the patient with FVD, the primary health care provider considers the maintenance requirements of the patient and other factors (eg, fever) that can influence fluid needs. If the deficit is not severe, the oral route is preferred, provided the patient can drink. However, if fluid losses are acute or severe, the IV route is required. Isotonic electrolyte solutions (eg, lactated Ringer's solution, 0.9% sodium chloride) are frequently used to treat the hypotensive patient with FVD because they expand plasma volume. As soon as the patient becomes normotensive, a hypotonic electrolyte solution (eg, 0.45% sodium chloride) is often used to provide both electrolytes and water for renal excretion of metabolic wastes. These and additional fluids are listed in Table 14-3.

Accurate and frequent assessments of I&O, weight, vital signs, central venous pressure, level of consciousness, breath sounds, and skin color should be performed to determine when therapy should be slowed to avoid volume overload. The rate of fluid administration is based on the severity of loss and the patient's hemodynamic response to volume replacement (Porth & Matfin, 2009).

If the patient with severe FVD is not excreting enough urine and is therefore oliguric, the primary health care provider needs to determine whether the depressed renal function is caused by reduced renal blood flow secondary to FVD (prerenal azotemia) or, more seriously, by acute tubular necrosis from prolonged FVD. The test used in this situation is referred to as a fluid challenge test. During a fluid challenge test, volumes of fluid are administered at specific rates and intervals while the patient's hemodynamic response to this treatment is monitored (ie, vital signs, breath sounds, sensorium, central venous pressure, urine output).

An example of a typical fluid challenge involves administering 100 to 200 mL of normal saline solution over 15 minutes. The goal is to provide fluids rapidly enough to attain adequate tissue perfusion without compromising the cardiovascular system. The response by a patient with FVD but normal renal function is increased urine output and an increase in blood pressure and central venous pressure.

Shock can occur when the volume of fluid lost exceeds 25% of the intravascular volume, or when fluid loss is rapid. Shock and its causes and treatment are discussed in detail in Chapter 15.

Nursing Management

To assess for FVD, the nurse monitors and measures fluid I&O at least every 8 hours, and sometimes hourly. As FVD develops, body fluid losses exceed fluid intake through excessive urination (polyuria), diarrhea, vomiting, or other mechanisms. Once FVD has developed, the kidneys attempt to conserve body fluids, leading to a urine output of less than 30 mL/h in an adult. Urine in this instance is concentrated and represents a healthy renal response. Daily body weights are monitored; an acute loss of 0.5 kg (1 lb)

Table 14-3 SELECTED WATER AND ELECTROLYTE SOLUTIONS

Solution	Comments
Isotonic Solutions	
0.9% NaCl (isotonic, also called normal saline [NS]) Na^+ 154 mEq/L Cl^- 154 mEq/L (308 mOsm/L) Also available with varying concentrations of dextrose (the most frequently used is a 5% dextrose concentration)	• An isotonic solution that expands the extracellular fluid (ECF) volume, used in hypovolemic states, resuscitative efforts, shock, diabetic ketoacidosis, metabolic alkalosis, hypercalcemia, mild Na^+ deficit • Supplies an excess of Na^+ and Cl^-; can cause fluid volume excess and hyperchloremic acidosis if used in excessive volumes, particularly in patients with compromised renal function, heart failure, or edema • Not desirable as a routine maintenance solution, as it provides only Na^+ and Cl^- (and these are provided in excessive amounts) • When mixed with 5% dextrose, the resulting solution becomes hypertonic in relation to plasma and, in addition to the above described electrolytes, provides 170 cal/L • Only solution that may be administered with blood products • Tonicity similar to plasma
Lactated Ringer's solution (Hartmann's solution) Na^+ 130 mEq/L K^+ 4 mEq/L Ca^{++} 3 mEq/L Cl^- 109 mEq/L Lactate (metabolized to bicarbonate) 28 mEq/L (274 mOsm/L) Also available with varying concentrations of dextrose (the most common is 5% dextrose)	• An isotonic solution that contains multiple electrolytes in roughly the same concentration as found in plasma (note that solution is lacking in Mg^{++}): provides 9 cal/L • Used in the treatment of hypovolemia, burns, fluid lost as bile or diarrhea, and for acute blood loss replacement • Lactate is rapidly metabolized into HCO_3^- in the body. Lactated Ringer's solution should not be used in lactic acidosis because the ability to convert lactate into HCO_3^- is impaired in this disorder. • Not to be given with a pH > 7.5 because bicarbonate is formed as lactate breaks down, causing alkalosis • Should not be used in renal failure because it contains potassium and can cause hyperkalemia • Tonicity similar to plasma
5% dextrose in water (D_5W) No electrolytes 50 g of dextrose	• An isotonic solution that supplies 170 cal/L and free water to aid in renal excretion of solutes • Used in treatment of hypernatremia, fluid loss, and dehydration • Should not be used in excessive volumes in the early postoperative period (when antidiuretic hormone secretion is increased due to stress reaction) • Should not be used solely in treatment of fluid volume deficit, because it dilutes plasma electrolyte concentrations • Contraindicated in head injury because it may cause increased intracranial pressure • Should not be used for fluid resuscitation because it can cause hyperglycemia • Should be used with caution in patients with renal or cardiac disease because of risk of fluid overload • Electrolyte-free solutions may cause peripheral circulatory collapse, anuria in patients with sodium deficiency, and increased body fluid loss. • Converts to hypotonic solution as dextrose is metabolized by body. Over time, D_5W without NaCl can cause water intoxication (intracellular fluid volume excess [FVE]) because the solution is hypotonic. • Fluid therapy for an extended period of time without electrolytes may result in hypokalemia
Hypotonic Solutions	
0.45% NaCl (half-strength saline) Na^+ 77 mEq/L Cl^- 77 mEq/L (154 mOsm/L) Also available with varying concentrations of dextrose (the most common is a 5% concentration)	• Provides Na^+, Cl^-, and free water • Free water is desirable to aid the kidneys in elimination of solute. • Lacking in electrolytes other than Na^+ and Cl^- • When mixed with 5% dextrose, the solution becomes slightly hypertonic to plasma and in addition to the above-described electrolytes provides 170 cal/L. • Used to treat hypertonic dehydration, Na^+ and Cl^- depletion, and gastric fluid loss • Not indicated for third-space fluid shifts or increased intracranial pressure • Administer cautiously, because it can cause fluid shifts from vascular system into cells, resulting in cardiovascular collapse and increased intracranial pressure.
Hypertonic Solutions	
3% NaCl (hypertonic saline) Na^+ 513 mEq/L Cl^- 513 mEq/L (1026 mOsm/L)	• Used to increase ECF volume, decrease cellular swelling • Highly hypertonic solution used only in critical situations to treat hyponatremia • Must be administered slowly and cautiously, because it can cause intravascular volume overload and pulmonary edema • Supplies no calories • Assists in removing intracellular fluid excess
5% NaCL (hypertonic solution) Na^+ 855 mEq/L Cl^- 855 mEq/L (1710 mOsm/L)	• Highly hypertonic solution used to treat symptomatic hyponatremia • Administer slowly and cautiously, because it can cause intravascular volume overload and pulmonary edema. • Supplies no calories
Colloid Solutions	
Dextran in NS or 5% D_5W Available in low-molecular-weight (Dextran 40) and high-molecular-weight (Dextran 70) forms	• Colloid solution used as volume/plasma expander for intravascular part of ECF • Affects clotting by coating platelets and decreasing ability to clot • Remains in circulatory system up to 24 h • Used to treat hypovolemia in early shock to increase pulse pressure, cardiac output, and arterial blood pressure • Improves microcirculation by decreasing red blood cell aggregation • Contraindicated in hemorrhage, thrombocytopenia, renal disease, and severe dehydration • Not a substitute for blood or blood products

represents a fluid loss of approximately 500 mL. (One liter of fluid weighs approximately 1 kg, or 2.2 lb.)

Vital signs are closely monitored. The nurse observes for a weak, rapid pulse and orthostatic hypotension (ie, a decrease in systolic pressure exceeding 15 mm Hg when the patient moves from a lying to a sitting position). A decrease in body temperature often accompanies FVD, unless there is a concurrent infection.

Skin and tongue turgor are monitored on a regular basis. In a healthy person, pinched skin immediately returns to its normal position when released. This elastic property, referred to as turgor, is partially dependent on interstitial fluid volume. In a person with FVD, the skin flattens more slowly after the pinch is released. In a person with severe FVD, the skin may remain elevated for many seconds. Tissue turgor is best measured by pinching the skin over the sternum, inner aspects of the thighs, or forehead. Tongue turgor is not affected by age (see previous Gerontologic Considerations), and evaluating this may be more valid than evaluating skin turgor. In a normal person, the tongue has one longitudinal furrow. In the person with FVD, there are additional longitudinal furrows and the tongue is smaller, because of fluid loss. The degree of oral mucous membrane moisture is also assessed; a dry mouth may indicate either FVD or mouth breathing.

Urine concentration is monitored by measuring the urine specific gravity. In a volume-depleted patient, the urine specific gravity should be greater than 1.020, indicating healthy renal conservation of fluid.

Mental function is eventually affected in severe FVD as a result of decreasing cerebral perfusion. Decreased peripheral perfusion can result in cold extremities. In patients with relatively normal cardiopulmonary function, a low central venous pressure is indicative of hypovolemia. Patients with acute cardiopulmonary decompensation require more extensive hemodynamic monitoring of pressures in both sides of the heart to determine if hypovolemia exists.

Preventing Hypovolemia

To prevent FVD, the nurse identifies patients at risk and takes measures to minimize fluid losses. For example, if the patient has diarrhea, measures should be implemented to control diarrhea and replacement fluids administered. This includes administering antidiarrheal medications and small volumes of oral fluids at frequent intervals.

Correcting Hypovolemia

When possible, oral fluids are administered to help correct FVD, with consideration given to the patient's likes and dislikes. The type of fluid the patient has lost is also considered and fluids most likely to replace the lost electrolytes are appropriate. If the patient is reluctant to drink because of oral discomfort, the nurse assists with frequent mouth care and provides nonirritating fluids. The patient may be offered small volumes of oral rehydration solutions (eg, Rehydralyte, Elete, Cytomax). These solutions provide fluid, glucose, and electrolytes in concentrations that are easily absorbed. If nausea is present, antiemetics may be needed before oral fluid replacement can be tolerated.

If the deficit cannot be corrected by oral fluids, therapy may need to be initiated by an alternative route (enteral or parenteral) until adequate circulating blood volume and renal perfusion are achieved. Isotonic fluids are prescribed to increase ECF volume.

Hypervolemia

Fluid volume excess (FVE), or hypervolemia, refers to an isotonic expansion of the ECF caused by the abnormal retention of water and sodium in approximately the same proportions in which they normally exist in the ECF. It is always secondary to an increase in the total body sodium content, which, in turn, leads to an increase in total body water. Because there is isotonic retention of body substances, the serum sodium concentration remains essentially normal.

Pathophysiology

FVE may be related to simple fluid overload or diminished function of the homeostatic mechanisms responsible for regulating fluid balance. Contributing factors can include heart failure, renal failure, and cirrhosis of the liver. Another contributing factor is consumption of excessive amounts of table or other sodium salts. Excessive administration of sodium-containing fluids in a patient with impaired regulatory mechanisms may predispose him or her to a serious FVE as well (Heitz & Horne, 2005).

Clinical Manifestations

Clinical manifestations of FVE result from expansion of the ECF and include edema, distended neck veins, and crackles (abnormal lung sounds). Other manifestations include tachycardia; increased blood pressure, pulse pressure, and central venous pressure; increased weight; increased urine output; and shortness of breath and wheezing.

Assessment and Diagnostic Findings

Laboratory data useful in diagnosing FVE include BUN and hematocrit levels. In FVE, both of these values may be decreased because of plasma dilution. Other causes of abnormalities in these values include low protein intake and anemia. In chronic renal failure, both serum osmolality and the sodium level are decreased due to excessive retention of water. The urine sodium level is increased if the kidneys are attempting to excrete excess volume. A chest x-ray may reveal pulmonary congestion. Hypervolemia occurs when aldosterone is chronically stimulated (ie, cirrhosis, heart failure, and nephrotic syndrome). Therefore, the urine sodium level does not increase in these conditions.

Medical Management

Management of FVE is directed at the causes, and if related to excessive administration of sodium-containing fluids, discontinuing the infusion may be all that is needed. Symptomatic treatment consists of administering diuretics and restricting fluids and sodium.

Pharmacologic Therapy

Diuretics are prescribed when dietary restriction of sodium alone is insufficient to reduce edema by inhibiting the reabsorption of sodium and water by the kidneys. The choice of diuretic is based on the severity of the hypervolemic state, the degree of impairment of renal function, and the potency of the diuretic. Thiazide diuretics block sodium reabsorption in the distal tubule, where only 5% to 10% of filtered sodium is reabsorbed. Loop diuretics, such as furosemide (Lasix), bumetanide (Bumex), or torsemide (Demadex), can cause a greater loss of both sodium and water because they block sodium reabsorption in the ascending limb of the loop of Henle, where 20% to 30% of filtered sodium is normally reabsorbed. Generally, thiazide diuretics, such as hydrochlorothiazide (HydroDIURIL) or metolazone (Mykrox, Zaroxolyn), are prescribed for mild to moderate hypervolemia and loop diuretics for severe hypervolemia.

Electrolyte imbalances may result from the effect of the diuretic. Hypokalemia can occur with all diuretics except those that work in the last distal tubule of the nephrons. Potassium supplements can be prescribed to avoid this complication. Hyperkalemia can occur with diuretics that work in the last distal tubule (eg, spironolactone [Aldactone]), especially in patients with decreased renal function. Hyponatremia occurs with diuresis due to increased release of ADH secondary to reduction in circulating volume. Decreased magnesium levels occur with administration of loop and thiazide diuretics due to decreased reabsorption and increased excretion of magnesium by the kidney.

Azotemia (increased nitrogen levels in the blood) can occur with FVE when urea and creatinine are not excreted due to decreased perfusion by the kidneys and decreased excretion of wastes. High uric acid levels (hyperuricemia) can also occur from increased reabsorption and decreased excretion of uric acid by the kidneys.

Dialysis

If renal function is so severely impaired that pharmacologic agents cannot act efficiently, other modalities are considered to remove sodium and fluid from the body. Hemodialysis or peritoneal dialysis may be used to remove nitrogenous wastes and control potassium and acid–base balance, and to remove sodium and fluid. Continuous renal replacement therapy may also be required. See Chapter 44 for a discussion of these treatment modalities.

Nutritional Therapy

Treatment of FVE usually involves dietary restriction of sodium. An average daily diet not restricted in sodium contains 6 to 15 g of salt, whereas low-sodium diets can range from a mild restriction to as little as 250 mg of sodium per day, depending on the patient's needs. A mild sodium-restricted diet allows only light salting of food (about half the usual amount) in cooking and at the table, and no addition of salt to commercially prepared foods that are already seasoned. Of course, foods high in sodium must be avoided. It is the sodium salt, sodium chloride, rather than sodium itself that contributes to edema. Therefore, patients are instructed to read food labels carefully to determine salt content.

Because about half of ingested sodium is in the form of seasoning, seasoning substitutes can play a major role in decreasing sodium intake. Lemon juice, onions, and garlic are excellent substitute flavorings, although some patients prefer salt substitutes. Most salt substitutes contain potassium and must therefore be used cautiously by patients taking potassium-sparing diuretics (eg, spironolactone, triamterene [Dyrenium], amiloride [Midamor]). They should not be used at all in conditions associated with potassium retention, such as advanced renal disease. Salt substitutes containing ammonium chloride can be harmful to patients with liver damage.

In some communities, the drinking water may contain too much sodium for a sodium-restricted diet. Depending on its source, water may contain as little as 1 mg or more than 1500 mg of sodium per quart. Patients may need to use distilled water if the local water supply is very high in sodium. Bottled water can have a sodium content that ranges from 0 to 1200 mg/L; therefore, if sodium is restricted, the label must be carefully examined for sodium content before purchasing and drinking bottled water. Also, patients on sodium-restricted diets should be cautioned to avoid water softeners that add sodium to water in exchange for other ions, such as calcium. Protein intake may be increased in patients who are malnourished or who have low serum protein levels in an effort to increase capillary oncotic pressure and pull fluid out of the tissues into vessels for excretion by the kidneys.

Nursing Management

To assess for FVE, the nurse measures I&O at regular intervals to identify excessive fluid retention. The patient is weighed daily, and rapid weight gain is noted. An acute weight gain of 2.2 lb (1 kg) is equivalent to a gain of approximately 1 L of fluid. Breath sounds are assessed at regular intervals in at-risk patients, particularly if parenteral fluids are being administered. The nurse monitors the degree of edema in the most dependent parts of the body, such as the feet and ankles in ambulatory patients and the sacral region in patients confined to bed. Pitting edema is assessed by pressing a finger into the affected part, creating a pit or indentation that is evaluated on a scale of 1+ (minimal) to 4+ (severe). Peripheral edema is monitored by measuring the circumference of the extremity with a tape marked in millimeters (Weber & Kelley, 2007).

Preventing Hypervolemia

Specific interventions vary with the underlying condition and the degree of FVE. However, most patients require sodium-restricted diets in some form, and adherence to the prescribed diet is encouraged. Patients are instructed to avoid over-the-counter medications without first checking with a health care provider, because these substances may contain sodium. If fluid retention persists despite adherence to a prescribed diet, hidden sources of

sodium, such as the water supply or use of water softeners, should be considered.

Detecting and Controlling Hypervolemia

It is important to detect FVE before the condition becomes severe. Interventions include promoting rest, restricting sodium intake, monitoring parenteral fluid therapy, and administering appropriate medications.

Regular rest periods may be beneficial, because bed rest favors diuresis of edema fluid. The mechanism is probably related to diminished venous pooling and the subsequent increase in effective circulating blood volume and renal perfusion. Sodium and fluid restriction should be instituted as indicated. Because most patients with FVE require diuretics, the patient's response to these agents is monitored. The rate of parenteral fluids and the patient's response to these fluids are also closely monitored. If dyspnea or orthopnea is present, the patient is placed in a semi-Fowler's position to promote lung expansion. The patient is turned and repositioned at regular intervals because edematous tissue is more prone to skin breakdown than normal tissue. Because conditions predisposing to FVE are likely to be chronic, patients are taught to monitor their response to therapy by documenting fluid I&O and body weight changes. The importance of adhering to the treatment regimen is emphasized.

Teaching Patients About Edema

Because edema is a common manifestation of FVE, patients need to recognize its symptoms and understand its importance. The nurse gives special attention to edema when teaching the patient with FVE. Edema can occur as a result of increased capillary fluid pressure, decreased capillary oncotic pressure, or increased interstitial oncotic pressure, causing expansion of the interstitial fluid compartment (Porth & Matfin, 2009). Edema can be localized (eg, in the ankle, as in rheumatoid arthritis) or generalized (as in cardiac and renal failure). Severe generalized edema is called *anasarca*.

Edema occurs when there is a change in the capillary membrane, increasing the formation of interstitial fluid or decreasing the removal of interstitial fluid. Sodium retention is a frequent cause of the increased ECF volume. Burns and infection are examples of conditions associated with increased interstitial fluid volume. Obstruction to lymphatic outflow, a plasma albumin level less than 1.5 to 2 g/dL, or a decrease in plasma oncotic pressure contributes to increased interstitial fluid volume. The kidneys retain sodium and water when there is decreased ECF volume as a result of decreased cardiac output from heart failure. A thorough medication history is necessary to identify any medications that could cause edema, such as nonsteroidal anti-inflammatory drugs (NSAIDs), estrogens, corticosteroids, and antihypertensive agents.

Ascites is a form of edema in which fluid accumulates in the peritoneal cavity; it results from nephrotic syndrome, cirrhosis, and some malignant tumors. The patient commonly reports shortness of breath and a sense of pressure because of pressure on the diaphragm.

The goal of treatment is to preserve or restore the circulating intravascular fluid volume. Thus, in addition to treating the cause of the edema, other treatments may include diuretic therapy, restriction of fluids and sodium, elevation of the extremities, application of anti-embolism stockings, paracentesis, dialysis, and continuous renal replacement therapy in cases of renal failure or life-threatening fluid volume overload (see Chapter 44).

ELECTROLYTE IMBALANCES

Disturbances in electrolyte balances are common in clinical practice and must be corrected (Table 14-4).

Sodium Imbalances

Sodium is the most abundant electrolyte in the ECF; its concentration ranges from 135 to 145 mEq/L (135 to 145 mmol/L) and it is the primary determinant of ECF volume and osmolality. Sodium has a major role in controlling water distribution throughout the body, because it does not easily cross the cell wall membrane and because of its abundance and high concentration in the body. Sodium is regulated by ADH, thirst, and the renin–angiotensin–aldosterone system. A loss or gain of sodium is usually accompanied by a loss or gain of water. Sodium also functions in establishing the electrochemical state necessary for muscle contraction and the transmission of nerve impulses (Criddle, 2006; Hayes, 2007a).

The syndrome of inappropriate secretion of antidiuretic hormone (SIADH) may be associated with sodium imbalance. When there is a decrease in the circulating plasma osmolality, blood volume, or blood pressure, arginine vasopressin (AVP) is released from the posterior pituitary. Oversecretion of AVP can cause SIADH. Patients who are at risk are the elderly, those with acquired immunodeficiency syndrome (AIDS), those on mechanical ventilation, and people taking selective serotonin reuptake inhibitors (SSRIs) (Haskal, 2007; Rottmann, 2007).

Sodium imbalance can develop under simple or complex circumstances. The two most common sodium imbalances are sodium deficit and sodium excess.

SODIUM DEFICIT (HYPONATREMIA)

Hyponatremia refers to a serum sodium level that is less than 135 mEq/L (135 mmol/L) (Criddle, 2006). Plasma sodium concentration represents the ratio of total body sodium to total body water. A decrease in this ratio can occur because of a low total body sodium with a lesser reduction in total body water, a normal total body sodium content with excess total body water, or an excess of total body sodium with an even greater excess of total body water. A hyponatremic state can be superimposed on an existing FVD or FVE.

Table 14-4 MAJOR FLUID AND ELECTROLYTE IMBALANCES

Imbalance	Contributing Factors	Signs/Symptoms and Laboratory Findings
Fluid volume deficit (hypovolemia)	Loss of water and electrolytes, as in vomiting, diarrhea, fistulas, fever, excess sweating, burns, blood loss, gastrointestinal suction, and third-space fluid shifts; and decreased intake, as in anorexia, nausea, and inability to gain access to fluid. Diabetes insipidus and uncontrolled diabetes mellitus also contribute to a depletion of extracellular fluid volume.	Acute weight loss, ↓ skin turgor, oliguria, concentrated urine, weak rapid pulse, capillary filling time prolonged, low CVP, ↓ blood pressure, flattened neck veins, dizziness, weakness, thirst and confusion, ↑ pulse, muscle cramps, sunken eyes *Labs indicate:* ↑ hemoglobin and hematocrit, ↑ serum and urine osmolality and specific gravity, ↓ urine sodium, ↑ BUN and creatinine, ↑ urine specific gravity and osmolality
Fluid volume excess (hypervolemia)	Compromised regulatory mechanisms, such as renal failure, heart failure, and cirrhosis; overzealous administration of sodium-containing fluids; and fluid shifts (ie, treatment of burns). Prolonged corticosteroid therapy, severe stress, and hyperaldosteronism augment fluid volume excess.	Acute weight gain, peripheral edema and ascites, distended jugular veins, crackles, elevated CVP, shortness of breath, ↑ blood pressure, bounding pulse and cough, ↑ respiratory rate *Labs indicate:* ↓ hemoglobin and hematocrit, ↓ serum and urine osmolality, ↓ urine sodium and specific gravity
Sodium deficit (hyponatremia) Serum sodium <135 mEq/L	Loss of sodium, as in use of diuretics, loss of GI fluids, renal disease, and adrenal insufficiency. Gain of water, as in excessive administration of D_5W and water supplements for patients receiving hypotonic tube feedings; disease states associated with SIADH such as head trauma and oat-cell lung tumor; medications associated with water retention (oxytocin and certain tranquilizers); and psychogenic polydipsia. Hyperglycemia and heart failure cause a loss of sodium.	Anorexia, nausea and vomiting, headache, lethargy, dizziness, confusion, muscle cramps and weakness, muscular twitching, seizures, papilledema, dry skin, ↑ pulse, ↓ BP, weight gain, edema *Labs indicate:* ↓ serum and urine sodium, ↓ urine specific gravity and osmolality
Sodium excess (hypernatremia) Serum sodium >145 mEq/L	Water deprivation in patients unable to drink at will, hypertonic tube feedings without adequate water supplements, diabetes insipidus, heatstroke, hyperventilation, watery diarrhea, burns, and diaphoresis. Excess corticosteroid, sodium bicarbonate, and sodium chloride administration, and salt water near-drowning victims	Thirst, elevated body temperature, swollen dry tongue and sticky mucous membranes, hallucinations, lethargy, restlessness, irritability, focal or grand mal seizures, pulmonary edema, hyperreflexia, twitching, nausea, vomiting, anorexia, ↑ pulse, and ↑ BP *Labs indicate:* ↑ serum sodium, ↓ urine sodium, ↑ urine specific gravity and osmolality, ↓ CVP
Potassium deficit (hypokalemia) Serum potassium <3.5 mEq/L	Diarrhea, vomiting, gastric suction, corticosteroid administration, hyperaldosteronism, carbenicillin, amphotericin B, bulimia, osmotic diuresis, alkalosis, starvation, diuretics, and digoxin toxicity	Fatigue, anorexia, nausea and vomiting, muscle weakness, polyuria, decreased bowel motility, ventricular asystole or fibrillation, paresthesias, leg cramps, ↓ BP, ileus, abdominal distention, hypoactive reflexes. *ECG:* flattened T waves, prominent U waves, ST depression, prolonged PR interval
Potassium excess (hyperkalemia) Serum potassium >5.0 mEq/L	Pseudohyperkalemia, oliguric renal failure, use of potassium-conserving diuretics in patients with renal insufficiency, metabolic acidosis, Addison's disease, crush injury, burns, stored bank blood transfusions, rapid IV administration of potassium, and certain medications such as ACE inhibitors, NSAIDs, cyclosporine	Muscle weakness, tachycardia → bradycardia, dysrhythmias, flaccid paralysis, paresthesias, intestinal colic, cramps, abdominal distention, irritability, anxiety. *ECG:* tall tented T waves, prolonged PR interval and QRS duration, absent P waves, ST depression
Calcium deficit (hypocalcemia) Serum calcium <8.5 mg/dL	Hypoparathyroidism (may follow thyroid surgery or radical neck dissection), malabsorption, pancreatitis, alkalosis, vitamin D deficiency, massive subcutaneous infection, generalized peritonitis, massive transfusion of citrated blood, chronic diarrhea, decreased parathyroid hormone, diuretic phase of renal failure, ↑ PO_4, fistulas, burns, alcoholism	Numbness, tingling of fingers, toes, and circumoral region; positive Trousseau's sign and Chvostek's sign; seizures, carpopedal spasms, hyperactive deep tendon reflexes, irritability, bronchospasm, anxiety, impaired clotting time, ↓ prothrombin, diarrhea, ↓ BP. *ECG:* prolonged QT interval and lengthened ST *Labs indicate:* ↓ Mg^{++}
Calcium excess (hypercalcemia) Serum calcium >10.5 mg/dL	Hyperparathyroidism, malignant neoplastic disease, prolonged immobilization, overuse of calcium supplements, vitamin D excess, oliguric phase of renal failure, acidosis, corticosteroid therapy, thiazide diuretic use, increased parathyroid hormone, and digoxin toxicity	Muscular weakness, constipation, anorexia, nausea and vomiting, polyuria and polydipsia, dehydration, hypoactive deep tendon reflexes, lethargy, deep bone pain, pathologic fractures, flank pain, calcium stones, hypertension. *ECG:* shortened ST segment and QT interval, bradycardia, heart blocks
Magnesium deficit (hypomagnesemia) Serum magnesium <1.8 mg/dL	Chronic alcoholism, hyperparathyroidism, hyperaldosteronism, diuretic phase of renal failure, malabsorptive disorders, diabetic ketoacidosis, refeeding after starvation, parenteral nutrition, chronic laxative use, diarrhea, acute myocardial infarction, heart failure, decreased serum K^+ and Ca^{++} and certain pharmacologic agents (such as gentamicin, cisplatin, and cyclosporine)	Neuromuscular irritability, positive Trousseau's and Chvostek's signs, insomnia, mood changes, anorexia, vomiting, increased tendon reflexes, and ↑ BP. *ECG:* PVCs, flat or inverted T waves, depressed ST segment, prolonged PR interval, and widened QRS

Continued

Table 14-4 MAJOR FLUID AND ELECTROLYTE IMBALANCES (Continued)

Imbalance	Contributing Factors	Signs/Symptoms and Laboratory Findings
Magnesium excess (hypermagnesemia) Serum magnesium >2.7 mg/dL	Oliguric phase of renal failure (particularly when magnesium-containing medications are administered), adrenal insufficiency, excessive IV magnesium administration, diabetic ketoacidosis, and hypothyroidism	Flushing, hypotension, muscle weakness, drowsiness, hypoactive reflexes, depressed respirations, cardiac arrest and coma, diaphoresis. *ECG:* tachycardia → bradycardia, prolonged PR interval and QRS, peaked T waves
Phosphorus deficit (hypophosphatemia) Serum phosphorus <2.5 mg/dL	Refeeding after starvation, alcohol withdrawal, diabetic ketoacidosis, respiratory and metabolic alkalosis, ↓ magnesium, ↓ potassium, hyperparathyroidism, vomiting, diarrhea, hyperventilation, vitamin D deficiency associated with malabsorptive disorders, burns, acid–base disorders, parenteral nutrition, and diuretic and antacid use	Paresthesias, muscle weakness, bone pain and tenderness, chest pain, confusion, cardiomyopathy, respiratory failure, seizures, tissue hypoxia, and increased susceptibility to infection, nystagmus
Phosphorus excess (hyperphosphatemia) Serum phosphorus >4.5 mg/dL	Acute and chronic renal failure, excessive intake of phosphorus, vitamin D excess, respiratory and metabolic acidosis, hypoparathyroidism, volume depletion, leukemia/lymphoma treated with cytotoxic agents, increased tissue breakdown, rhabdomyolysis	Tetany, tachycardia, anorexia, nausea and vomiting, muscle weakness, signs and symptoms of hypocalcemia; hyperactive reflexes; soft tissue calcifications in lungs, heart, kidneys, and cornea
Chloride deficit (hypochloremia) Serum chloride <96 mEq/L	Addison's disease, reduced chloride intake or absorption, untreated diabetic ketoacidosis, chronic respiratory acidosis, excessive sweating, vomiting, gastric suction, diarrhea, sodium and potassium deficiency, metabolic alkalosis; loop, osmotic, or thiazide diuretic use; overuse of bicarbonate, rapid removal of ascitic fluid with a high sodium content, intravenous fluids that lack chloride (dextrose and water), draining fistulas and ileostomies, heart failure, cystic fibrosis	Agitation, irritability, tremors, muscle cramps, hyperactive deep tendon reflexes, hypertonicity, tetany, slow shallow respirations, seizures, dysrhythmias, coma *Labs indicate:* ↓ serum chloride, ↓ serum sodium, ↑ pH, ↑ serum bicarbonate, ↑ total carbon dioxide content, ↓ urine chloride level, ↓ serum potassium
Chloride excess (hyperchloremia) Serum chloride >108 mEq/L	Excessive sodium chloride infusions with water loss, head injury (sodium retention), hypernatremia, renal failure, corticosteroid use, dehydration, severe diarrhea (loss of bicarbonate), respiratory alkalosis, administration of diuretics, overdose of salicylates, Kayexalate, acetazolamide, phenylbutazone and ammonium chloride use, hyperparathyroidism, metabolic acidosis	Tachypnea, lethargy, weakness, deep rapid respirations, decline in cognitive status, ↓ cardiac output, dyspnea, tachycardia, pitting edema, dysrhythmias, coma *Labs indicate:* ↑ serum chloride, ↑ serum potassium and sodium, ↓ serum pH, ↓ serum bicarbonate, normal anion gap, ↑ urinary chloride level

↑ increased; ↓ decreased; ACE, angiotensin-converting enzyme; BP, blood pressure; BUN, blood urea nitrogen; CVP, central venous pressure; D_5W, dextrose 5% in water; GI, gastrointestinal; IV, intravenous; NSAIDs, nonsteroidal anti-inflammatory drugs; PVCs, premature ventricular contractions; SIADH, syndrome of inappropriate secretion of antidiuretic hormone.

Pathophysiology

Hyponatremia primarily occurs due to an imbalance of water rather than sodium. The urine sodium assists in differentiating renal from nonrenal causes of hyponatremia. Low urine sodium occurs as the kidney retains sodium to compensate for nonrenal fluid loss (ie, vomiting, diarrhea, sweating). High urine sodium concentration is associated with renal salt wasting (ie, diuretic use). In dilutional hyponatremia, the ECF volume is increased without any edema.

A deficiency of aldosterone, as occurs in adrenal insufficiency, also predisposes to sodium deficiency. In addition, the use of certain medications, such as anticonvulsants (ie, carbamazepine [Tegretol], levetiracetam [Keppra]) and SSRIs (fluoxetine [Sarafem], sertraline [Zoloft], paroxetine [Paxil]), increases the risk of hyponatremia (McPhee, et al., 2007; Rottmann, 2007).

SIADH is seen in hyponatremia as well as hypernatremia. The physiologic disturbances include excessive ADH activity, with water retention and dilutional hyponatremia, and inappropriate urinary excretion of sodium in the presence of hyponatremia. SIADH can be the result of either sustained secretion of ADH by the hypothalamus or production of an ADH-like substance from a tumor (aberrant ADH production). Conditions affecting the central nervous system are associated with SIADH. SIADH is discussed in more detail in Chapter 42.

Clinical Manifestations

Clinical manifestations of hyponatremia depend on the cause, magnitude, and speed with which the deficit occurs. Poor skin turgor, dry mucosa, headache, decreased saliva production, orthostatic fall in blood pressure, nausea, vomiting, and abdominal cramping occur. Neurologic changes, including altered mental status, status epilepticus, and coma, are probably related to the cellular swelling and cerebral edema associated with hyponatremia. As the extracellular sodium level decreases, the cellular fluid becomes relatively more concentrated and pulls water into the cells (Fig. 14-4). In general, patients with an acute

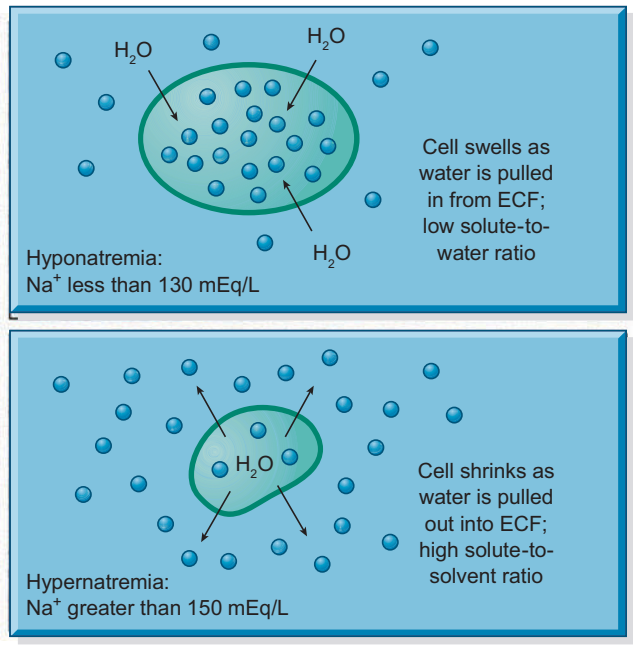

Figure 14-4 Effect of extracellular sodium level on cell size.

decrease in serum sodium levels have more cerebral edema and higher mortality rates than do those with more slowly developing hyponatremia. Acute decreases in sodium, developing in less than 48 hours, may be associated with brain herniation and compression of midbrain structures. Chronic decreases in sodium, developing over 48 hours or more, can occur in status epilepticus and cerebral pontine myelinolysis.

Features of hyponatremia associated with sodium loss and water gain include anorexia, muscle cramps, and a feeling of exhaustion. The severity of symptoms increases with the degree of hyponatremia and the speed with which it develops. When the serum sodium level decreases to less than 115 mEq/L (115 mmol/L), signs of increasing intracranial pressure, such as lethargy, confusion, muscle twitching, focal weakness, hemiparesis, papilledema, seizures, and death, may occur.

Assessment and Diagnostic Findings

Assessment includes the history and physical examination, including a focused neurologic examination; evaluation of signs and symptoms as well as laboratory test results; identification of current IV fluids, if applicable; and a review of all medications the patient is taking. Regardless of the cause of hyponatremia, the serum sodium level is less than 135 mEq/L; in SIADH, it may be lower than 100 mEq/L (100 mmol/L). Serum osmolality is also decreased, except in azotemia with the accumulation of toxins. When hyponatremia is due primarily to sodium loss, the urinary sodium content is less than 20 mEq/L (20 mmol/L), suggesting increased proximal reabsorption of sodium secondary to ECF volume depletion, and the specific gravity is low (1.002 to 1.004). However, when hyponatremia is due to SIADH, the urinary sodium content is greater than 20 mEq/L, and the urine specific gravity is usually greater than 1.012. Although the patient with SIADH retains water abnormally and therefore gains body weight, there is no peripheral edema; instead, fluid accumulates inside the cells. This phenomenon sometimes manifests as pitting edema.

Medical Management

The key to treating hyponatremia is assessment including identifying patients who are at risk and recognizing that the rapidity of the onset of hyponatremia is of primary importance (Haskal, 2007).

Sodium Replacement

The most common treatment for hyponatremia is careful administration of sodium by mouth, nasogastric tube, or a parenteral route. For patients who can eat and drink, sodium is easily replaced, because sodium is consumed abundantly in a normal diet. For those who cannot consume sodium, lactated Ringer's solution or isotonic saline (0.9% sodium chloride) solution may be prescribed. Serum sodium must not be increased by more than 12 mEq/L in 24 hours to avoid neurologic damage due to osmotic demyelination. This condition may occur when the serum sodium concentration is overcorrected (exceeding 140 mEq/L) too rapidly or in the presence of hypoxia or anoxia. It may produce lesions that show symmetric myelin destruction affecting all the fiber tracts that cause paraparesis, dysarthria, dysphagia, and coma (Abbott, Silber, Felber, et al., 2005). The usual daily sodium requirement in adults is approximately 100 mEq, provided there are not excessive losses. Selected water and electrolyte solutions are described in Table 14-3.

In SIADH, the administration of hypertonic saline solution alone cannot change the plasma sodium concentration. Excess sodium would be excreted rapidly in highly concentrated urine. With the addition of the diuretic furosemide (Lasix), urine is not concentrated and isotonic urine is excreted to effect a change in water balance. In patients with SIADH, in whom water restriction is difficult, lithium (Eskalith) or demeclocycline (Declomycin) can antagonize the osmotic effect of ADH on the medullary collecting tubule.

Water Restriction

In a patient with normal or excess fluid volume, hyponatremia is treated by restricting fluid to a total of 800 mL in 24 hours. This is far safer than sodium administration and is usually an effective treatment. However, if neurologic symptoms are severe (eg, seizures, delirium, coma), as well as in traumatic brain injury, it may be necessary to administer small volumes of a hypertonic sodium solution (see Chapter 61) (Mortimer & Jancik, 2006). Incorrect use of these fluids is extremely dangerous, because 1 L of 3% sodium chloride solution contains 513 mEq of sodium and 1 L of 5% sodium chloride solution contains 855 mEq of sodium. The prescribed volume of hypertonic saline administered depends on the patient's weight and on current and desired serum sodium levels (Mortimer & Jancik, 2006). If edema exists alone, sodium is restricted; if edema and hyponatremia occur together, both sodium and water are restricted.

NURSING ALERT

Highly hypertonic sodium solutions (2% to 23% sodium chloride) should be administered only in intensive care settings under close observation, because only small volumes are needed to elevate the serum sodium concentration from a dangerously low level. These fluids are administered slowly and in small volumes, and the patient is monitored closely.

Pharmacologic Therapy

AVP receptor antagonists are new pharmacologic agents that treat hyponatremia by stimulating free water excretion (Haskal, 2007). IV conivaptan hydrochloride (Vaprisol) use is limited to the treatment of hospitalized patients. It may be a useful therapy for those patients with moderate to severe symptomatic hyponatremia, but is contraindicated in patients with seizures, delirium, or coma, which warrant the use of hypertonic saline (Ellison & Berl, 2007; Hayes, 2007a).

Nursing Management

The nurse needs to identify and monitor patients at risk of hyponatremia. Early detection and treatment of this disorder are necessary to prevent serious consequences. For patients at risk, the nurse monitors fluid I&O as well as daily body weight. It is also necessary to note abnormal losses of sodium or gains of water, as well as GI manifestations such as anorexia, nausea, vomiting, and abdominal cramping. The nurse must be alert for central nervous system changes, such as lethargy, confusion, muscle twitching, and seizures. Neurologic signs are associated with very low sodium levels that have fallen rapidly because of fluid overloading. Serum sodium is monitored very closely in patients who are at risk for hyponatremia; when indicated, urine sodium and specific gravity are also monitored.

Hyponatremia is a frequently overlooked cause of confusion in elderly patients, who have an increased risk of hyponatremia because of decreased renal function and thus inability to excrete excess fluids. Administration of prescribed and over-the-counter (OTC) medications that cause sodium loss or water retention are predisposing factors. A diminished sense of thirst or loss of access to food or fluids may also contribute to the problem.

Detecting and Controlling Hyponatremia

For a patient with abnormal losses of sodium who can consume a general diet, the nurse encourages foods and fluids with high sodium content. For example, broth made with one beef cube contains approximately 900 mg of sodium; 8 oz of tomato juice contains approximately 700 mg of sodium. The nurse also needs to be familiar with the sodium content of parenteral fluids (see Table 14-3).

If the primary problem is water retention, it is safer to restrict fluid intake than to administer sodium. In normovolemia or hypervolemia, administration of sodium predisposes a patient to fluid volume overload. In severe hyponatremia, the aim of therapy is to elevate the serum sodium level only enough to alleviate neurologic signs and symptoms. It is generally recommended that the serum sodium concentration be increased to no greater than 125 mEq/L (125 mmol/L) with a hypertonic saline solution.

NURSING ALERT

When administering fluids to patients with cardiovascular disease, the nurse assesses for signs of circulatory overload (eg, cough, dyspnea, puffy eyelids, dependent edema, weight gain in 24 hours). The lungs are auscultated for crackles.

For the patient taking lithium, the nurse observes for lithium toxicity, particularly when sodium is lost by an abnormal route. In such instances, supplemental salt and fluid are administered. Because diuretics promote sodium loss, the patient taking lithium is instructed not to use diuretics without close medical supervision. For all patients on lithium therapy, adequate salt intake should be ensured.

Excess water supplements are avoided in patients receiving isotonic or hypotonic enteral feedings, particularly if abnormal sodium loss occurs or water is being abnormally retained (as in SIADH). Actual fluid needs are determined by evaluating fluid I&O, urine specific gravity, and serum sodium levels.

SODIUM EXCESS (HYPERNATREMIA)

Hypernatremia is a serum sodium level higher than 145 mEq/L (145 mmol/L). It can be caused by a gain of sodium in excess of water or by a loss of water in excess of sodium. It can occur in patients with normal fluid volume or in those with FVD or FVE. With a water loss, the patient loses more water than sodium; as a result, the serum sodium concentration increases and the increased concentration pulls fluid out of the cell. This is both an extracellular and an intracellular FVD. In sodium excess, the patient ingests or retains more sodium than water.

Pathophysiology

A common cause of hypernatremia is fluid deprivation in unconscious patients who cannot perceive, respond to, or communicate their thirst (Porth & Matfin, 2009). Most often affected are very old, very young, and cognitively impaired patients. Administration of hypertonic enteral feedings without adequate water supplements leads to hypernatremia, as does watery diarrhea and greatly increased insensible water loss (eg, hyperventilation, denuding effects of burns). In addition, both central and nephrogenic diabetes insipidus, which is a decreased ability to concentrate urine due to a defect in the kidney tubules that interferes with water reabsorption, result in hypernatremia if the patient does not experience or cannot respond to thirst, or if fluids are excessively restricted.

Less common causes of hypernatremia are heat stroke, near drowning in sea water (which contains a sodium concentration of approximately 500 mEq/L), and malfunction of hemodialysis or peritoneal dialysis systems. IV administration of hypertonic saline or excessive use of sodium bicarbonate also causes hypernatremia (Porth & Matfin, 2009).

Clinical Manifestations

The clinical manifestations of hypernatremia are primarily neurologic and are due to increased plasma osmolality caused by an increase in plasma sodium concentration. Water moves out of the cell into the ECF, resulting in cellular dehydration and a more concentrated ECF (see Fig. 14-4). Clinically, these changes may be manifested by restlessness and weakness in moderate hypernatremia and by disorientation, delusions, and hallucinations in severe hypernatremia. Dehydration (resulting in hypernatremia) is often overlooked as the primary reason for behavioral changes in elderly patients. If hypernatremia is severe, permanent brain damage can occur (especially in children). Brain damage is due to hemorrhages that result from brain contraction.

A primary characteristic of hypernatremia is thirst. Thirst is such a strong defender of serum sodium levels in healthy people that hypernatremia never occurs unless the person is unconscious or does not have access to water. However, ill people may have an impaired thirst mechanism. Other signs include a dry, swollen tongue and sticky mucous membranes; flushed skin; peripheral and pulmonary edema; postural hypotension; oliguria; and increased muscle tone and deep tendon reflexes (DTRs). Body temperature may increase mildly, but it returns to normal after the hypernatremia is corrected.

Assessment and Diagnostic Findings

In hypernatremia, the serum sodium level exceeds 145 mEq/L (145 mmol/L) and the serum osmolality exceeds 300 mOsm/kg (300 mmol/L). The urine specific gravity and urine osmolality are increased as the kidneys attempt to conserve water (provided the water loss is from a route other than the kidneys). Patients with nephrogenic or central diabetes insipidus have hypernatremia and produce a dilute urine with a urine osmolality less than 250 mOsm/kg.

Medical Management

Treatment of hypernatremia consists of a gradual lowering of the serum sodium level by the infusion of a hypotonic electrolyte solution (eg, 0.3% sodium chloride) or an isotonic nonsaline solution (eg, dextrose 5% in water [D_5W]). D_5W is indicated when water needs to be replaced without sodium. Clinicians consider a hypotonic sodium solution to be safer than D_5W because it allows a gradual reduction in the serum sodium level, thereby decreasing the risk of cerebral edema. It is the solution of choice in severe hyperglycemia with hypernatremia. A rapid reduction in the serum sodium level temporarily decreases the plasma osmolality below that of the fluid in the brain tissue, causing dangerous cerebral edema. Diuretics also may be prescribed to treat the sodium gain.

There is no consensus about the exact rate at which serum sodium levels should be reduced. As a general rule, the serum sodium level is reduced at a rate no faster than 0.5 to 1 mEq/L/h to allow sufficient time for readjustment through diffusion across fluid compartments. Desmopressin acetate (DDAVP), a synthetic antidiuretic hormone, may be prescribed to treat diabetes insipidus if it is the cause of hypernatremia (Porth & Matfin, 2009).

Nursing Management

As in hyponatremia, fluid losses and gains are carefully monitored in patients who are at risk for hypernatremia. The nurse should assess for abnormal losses of water or low water intake and for large gains of sodium, as might occur with ingestion of OTC medications that have a high sodium content (eg, Alka-Seltzer). In addition, the nurse obtains a medication history, because some prescription medications have a high sodium content. The nurse also notes the patient's thirst or elevated body temperature and evaluates it in relation to other clinical signs. The nurse monitors for changes in behavior, such as restlessness, disorientation, and lethargy.

Preventing Hypernatremia

The nurse attempts to prevent hypernatremia by providing fluids at regular intervals, particularly in debilitated or unconscious patients who are unable to perceive or respond to thirst. If fluid intake remains inadequate, the nurse consults with the physician to plan an alternative route for intake, either by enteral feedings or by the parenteral route. If enteral feedings are used, sufficient water should be administered to keep the serum sodium and BUN within normal limits. As a rule, the higher the osmolality of the enteral feeding, the greater is the need for water supplementation.

For patients with diabetes insipidus, adequate water intake must be ensured. If the patient is alert and has an intact thirst mechanism, merely providing access to water may be sufficient. If the patient has a decreased level of consciousness or other disability interfering with adequate fluid intake, parenteral fluid replacement may be prescribed. This therapy can be anticipated in patients with neurologic disorders, particularly in the early postoperative period.

Correcting Hypernatremia

When parenteral fluids are necessary for managing hypernatremia, the nurse monitors the patient's response to the fluids by reviewing serial serum sodium levels and by observing for changes in neurologic signs. With a gradual decrease in the serum sodium level, the neurologic signs should improve. Too-rapid reduction in the serum sodium level renders the plasma temporarily hypo-osmotic to the fluid in the brain tissue, causing movement of fluid into brain cells and dangerous cerebral edema.

Potassium Imbalances

Potassium is the major intracellular electrolyte; in fact, 98% of the body's potassium is inside the cells. The remaining 2% is in the ECF and is important in neuromuscular function. Potassium influences both skeletal and cardiac muscle activity. For example, alterations in its concentration change myocardial irritability and rhythm. Under the influence of the sodium–potassium pump, potassium is constantly moving in and out of cells. The normal serum potassium concentration ranges from 3.5 to 5.0 mEq/L (3.5 to 5 mmol/L), and even minor variations are significant. Potassium imbalances are commonly associated with various diseases, injuries, medications (eg, NSAIDs and ACE

inhibitors), and acid–base imbalances (Hayes, 2007b; McPhee, et al., 2007).

To maintain potassium balance, the renal system must function, because 80% of the potassium excreted daily leaves the body by way of the kidneys; the other 20% is lost through the bowel and in sweat. The kidneys regulate potassium balance by adjusting the amount of potassium that is excreted in the urine. As serum potassium levels increase, so does the potassium level in the renal tubular cell. A concentration gradient occurs, favoring the movement of potassium into the renal tubule and excretion of potassium in the urine. Aldosterone also increases the excretion of potassium by the kidney. Because the kidneys do not conserve potassium as well as they conserve sodium, potassium may still be lost in urine in the presence of a potassium deficit.

POTASSIUM DEFICIT (HYPOKALEMIA)

Hypokalemia (below 3.5 mEq/L [3.5 mmol/L]) usually indicates a deficit in total potassium stores. However, it may occur in patients with normal potassium stores: When **alkalosis** is present, a temporary shift of serum potassium into the cells occurs (see later discussion).

Pathophysiology

Potassium-losing diuretics, such as the thiazides and loop diuretics, can induce hypokalemia (Baumberger-Henry, 2008). Other medications that can lead to hypokalemia include corticosteroids, sodium penicillin, carbenicillin, and amphotericin B. GI loss of potassium is another common cause of potassium depletion. Vomiting and gastric suction frequently lead to hypokalemia, partly because potassium is actually lost when gastric fluid is lost and because potassium is lost through the kidneys in response to metabolic alkalosis. Because relatively large amounts of potassium are contained in intestinal fluids, potassium deficit occurs frequently with diarrhea, which may contain as much potassium as 30 mEq/L. Potassium deficit also occurs from prolonged intestinal suctioning, recent ileostomy, and villous adenoma (a tumor of the intestinal tract characterized by excretion of potassium-rich mucus).

Alterations in acid–base balance have a significant effect on potassium distribution due to shifts of hydrogen and potassium ions between the cells and the ECF. Respiratory or metabolic alkalosis promotes the transcellular shift of potassium and can have a variable and unpredictable effect on serum potassium (O'Neill, 2007). For example, hydrogen ions move out of the cells in alkalotic states to help correct the high pH, and potassium ions move in to maintain an electrically neutral state (see later discussion of acid–base balance).

Hyperaldosteronism increases renal potassium wasting and can lead to severe potassium depletion. Primary hyperaldosteronism is seen in patients with adrenal adenomas. Secondary hyperaldosteronism occurs in patients with cirrhosis, nephrotic syndrome, heart failure, or malignant hypertension (Heitz & Horne, 2005).

Because insulin promotes the entry of potassium into skeletal muscle and hepatic cells, patients with persistent insulin hypersecretion may experience hypokalemia, which is often the case in patients receiving high-carbohydrate parenteral nutrition.

Patients who do not eat a normal diet for a prolonged period are at risk for hypokalemia. This may occur in debilitated elderly people, patients with alcoholism, and patients with anorexia nervosa. In addition to poor intake, people with bulimia frequently suffer increased potassium loss through self-induced vomiting, misuse of laxatives, diuretics, and enemas. Magnesium depletion causes renal potassium loss and must be corrected first; otherwise, urine loss of potassium will continue.

Clinical Manifestations

Potassium deficiency can result in widespread derangements in physiologic function. Severe hypokalemia can cause death through cardiac or respiratory arrest. Clinical signs rarely develop before the serum potassium level has decreased to less than 3 mEq/L (3 mmol/L) unless the rate of decline has been rapid. Manifestations of hypokalemia include fatigue, anorexia, nausea, vomiting, muscle weakness, leg cramps, decreased bowel motility, paresthesias (numbness and tingling), and dysrhythmias. If prolonged, hypokalemia can lead to an inability of the kidneys to concentrate urine, causing dilute urine (resulting in polyuria, nocturia) and excessive thirst. Potassium depletion suppresses the release of insulin and results in glucose intolerance. Decreased muscle strength and DTRs can be found on physical assessment.

Assessment and Diagnostic Findings

In hypokalemia, the serum potassium concentration is less than the lower limit of normal. Electrocardiographic (ECG) changes can include flat T waves or inverted T waves or both, suggesting ischemia, and depressed ST segments (Fig. 14-5). An elevated U wave is specific to hypokalemia. Hypokalemia increases sensitivity to digitalis, predisposing the patient to digitalis toxicity at lower digitalis levels. Metabolic alkalosis is commonly associated with hypokalemia (Her, 2007). This is discussed further in the section on acid–base disturbances in this chapter.

The source of the potassium loss is usually evident from a careful history. However, if the cause of the loss is unclear, a 24-hour urinary potassium excretion test can be performed to distinguish between renal and extrarenal loss. Urinary potassium excretion exceeding 20 mEq/day with hypokalemia suggests that renal potassium loss is the cause.

Medical Management

If hypokalemia cannot be prevented by conventional measures such as increased intake in the daily diet or by oral potassium supplements for deficiencies, then it is treated cautiously with IV replacement therapy (Hayes, 2007b). Potassium loss must be corrected daily; administration of 40 to 80 mEq/day of potassium is adequate in the adult if there are no abnormal losses of potassium.

For patients who are at risk for hypokalemia, a diet containing sufficient potassium should be provided. Dietary intake of potassium in the average adult is 50 to 100 mEq/day. Foods high in potassium include most fruits and vegetables, legumes, whole grains, milk, and meat.

If dietary intake is inadequate for any reason, the physician may prescribe oral or IV potassium supplements (Muller & Bell, 2008). Many salt substitutes contain 50 to 60 mEq of potassium per teaspoon and may be sufficient to prevent hypokalemia.

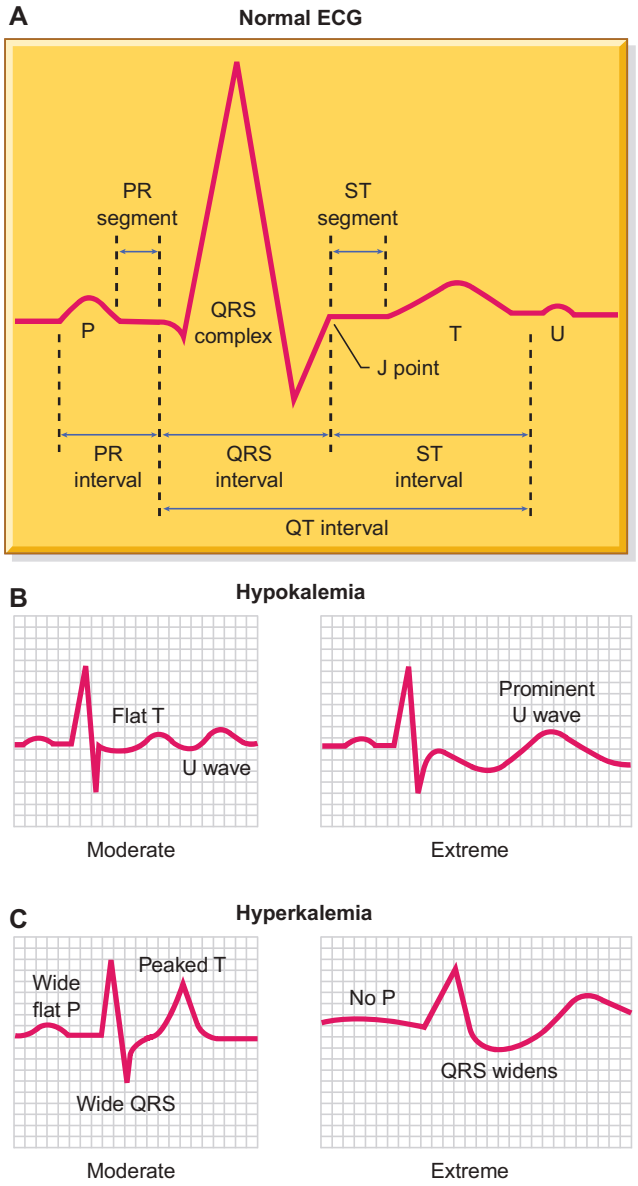

Figure 14-5 Effect of potassium on the electrocardiogram (ECG). **A,** Normal tracing. **B,** Hypokalemia: serum potassium level below normal. *Left:* Flattening of the T wave and the appearance of a U wave. *Right:* Further flattening with prominent U wave. **C,** Hyperkalemia: serum potassium level above normal. *Left:* Moderate elevation with wide, flat P wave; wide QRS complex; and peaked T wave. *Right:* ECG changes seen with extreme potassium elevation: widening of QRS complex and absence of P wave.

If oral administration of potassium is not feasible, the IV route is indicated. The IV route is mandatory for patients with severe hypokalemia (eg, serum level of 2 mEq/L). Although potassium chloride is usually used to correct potassium deficits, potassium acetate or potassium phosphate may be prescribed.

Nursing Management

Because hypokalemia can be life-threatening, the nurse needs to monitor for its early presence in patients who are at risk. Fatigue, anorexia, muscle weakness, decreased bowel motility, paresthesias, and dysrhythmias are signals that warrant assessing the serum potassium concentration. When available, the ECG may provide useful information. For example, patients receiving digitalis who are at risk for potassium deficiency should be monitored closely for signs of digitalis toxicity, because hypokalemia potentiates the action of digitalis.

Preventing Hypokalemia

Prevention may involve encouraging the patient at risk to eat foods rich in potassium (when the diet allows). Sources of potassium include fruit juices and bananas, melon, citrus fruits, fresh and frozen vegetables, fresh meats, milk, and processed foods. If the hypokalemia is caused by abuse of laxatives or diuretics, patient education may help alleviate the problem. Part of the health history and assessment should be directed at identifying problems that are amenable to prevention through education. Careful monitoring of fluid I&O is necessary, because 40 mEq of potassium is lost for every liter of urine output. The ECG is monitored for changes, and arterial blood gas values are checked for elevated bicarbonate and pH levels.

Correcting Hypokalemia

The oral route is ideal to treat a mild to moderate hypokalemia because oral potassium supplements are absorbed well. Care should be exercised when administering potassium, particularly in older adults, who have lower lean body mass and total body potassium levels and therefore lower potassium requirements. In addition, because of the physiologic loss of renal function with advancing years, potassium may be retained more readily in older than in younger people.

NURSING ALERT

Oral potassium supplements can produce small-bowel lesions; therefore, the patient must be assessed for and cautioned about abdominal distention, pain, or GI bleeding.

Administering Intravenous Potassium

Potassium should be administered only after adequate urine flow has been established. A decrease in urine volume to less than 20 mL/h for 2 consecutive hours is an indication to stop the potassium infusion until the situation is evaluated. Potassium is primarily excreted by the kidneys; when oliguria occurs, potassium administration can cause the serum potassium concentration to rise dangerously (Hayes, 2007b).

NURSING ALERT

Potassium is *never* administered by IV push or intramuscularly to avoid replacing potassium too quickly. IV potassium must be administered using an infusion pump.

Each health care facility has its own standard of care for the administration of potassium, which should be consulted; however, the maximum concentration of potassium that should be administered on a medical-surgical unit through a

peripheral IV line is 20 mEq/100 mL and the rate no faster than 10 to 20 mEq/h. Concentrations of potassium greater than 20 mEq/100 mL should be administered through a central IV catheter using an infusion pump with the patient monitored by ECG. Caution must be used when selecting the correct premixed solution of IV fluid containing potassium chloride as the concentrations range from 10 to 40 mEq/100 mL. Renal function should be monitored through BUN and creatinine levels and urine output if the patient is receiving potassium replacement. During potassium replacement, smooth muscle hyperactivity can lead to hyperactive bowel sounds, a sign of hyperkalemia (Hayes, 2007b).

POTASSIUM EXCESS (HYPERKALEMIA)

Hyperkalemia (greater than 5.0 mEq/L [5 mmol/L]) seldom occurs in patients with normal renal function (Vacca, 2008). Like hypokalemia, hyperkalemia is often caused by iatrogenic (treatment-induced) causes. Although hyperkalemia is less common than hypokalemia, it is usually more dangerous, because cardiac arrest is more frequently associated with high serum potassium levels.

Pathophysiology

The three major causes of hyperkalemia are decreased renal excretion of potassium, rapid administration of potassium, and movement of potassium from the ICF compartment to the ECF compartment. Hyperkalemia is commonly seen in patients with untreated renal failure, particularly those in whom potassium levels increase as a result of infection or excessive intake of potassium in food or medications. Patients with hypoaldosteronism or Addison's disease are at risk for hyperkalemia, because deficient adrenal hormones lead to sodium loss and potassium retention.

Medications have been identified as a probable contributing factor in more than 60% of hyperkalemic episodes. Medications commonly implicated are potassium chloride, heparin, ACE inhibitors, NSAIDs, beta-blockers, and potassium-sparing diuretics (Muller & Bell, 2008). Potassium regulation is compromised in acute and chronic renal failure, with a glomerular filtration rate less than 10% to 20% of normal.

Improper use of potassium supplements predisposes all patients to hyperkalemia, especially if salt substitutes are used. Not all patients receiving potassium-losing diuretics require potassium supplements, and patients receiving potassium-conserving diuretics should not receive supplements.

NURSING ALERT

Potassium supplements are extremely dangerous for patients who have impaired renal function and thus decreased ability to excrete potassium. Even more dangerous is the IV administration of potassium to such patients, because serum levels can rise very quickly. Aged (stored) blood should not be administered to patients with impaired renal function, because the serum potassium concentration of stored blood increases due to red blood cell deterioration. It is possible to exceed the renal tolerance of any patient with rapid IV potassium administration, as well as when large amounts of oral potassium supplements are ingested.

In **acidosis,** potassium moves out of the cells and into the ECF. This occurs as hydrogen ions enter the cells to buffer the pH of the ECF (see later discussion). An elevated ECF potassium level should be anticipated when extensive tissue trauma has occurred, as in burns, crushing injuries, or severe infections. Similarly, it can occur with lysis of malignant cells after chemotherapy (ie, tumor lysis syndrome).

Pseudohyperkalemia (a variation of hyperkalemia) has a number of causes, the most common being the use of a tight tourniquet around an exercising extremity while drawing a blood sample, producing hemolysis of the sample before analysis. Other causes include marked leukocytosis (white blood cell count exceeding 200,000/mm^3) and thrombocytosis (platelet count exceeding 1 million/mm^3); drawing blood above a site where potassium is infusing; and familial pseudohyperkalemia, in which potassium leaks out of the red blood cells while the blood is awaiting analysis. Lack of awareness of these causes of pseudohyperkalemia can lead to aggressive treatment of a nonexistent hyperkalemia, resulting in serious lowering of serum potassium levels. Therefore, measurements of grossly elevated levels should be verified by retesting.

Clinical Manifestations

The most important consequence of hyperkalemia is its effect on the myocardium. Cardiac effects of elevated serum potassium are usually not significant when the level is less than 7 mEq/L (7 mmol/L), but they are almost always present when the level is 8 mEq/L (8 mmol/L) or greater. As the plasma potassium level rises, disturbances in cardiac conduction occur. The earliest changes, often occurring at a serum potassium level greater than 6 mEq/L (6 mmol/L), are peaked, narrow T waves; ST-segment depression; and a shortened QT interval. If the serum potassium level continues to increase, the PR interval becomes prolonged and is followed by disappearance of the P waves. Finally, there is decomposition and widening of the QRS complex (see Fig. 14-5). Ventricular dysrhythmias and cardiac arrest may occur at any point in this progression.

Severe hyperkalemia causes skeletal muscle weakness and even paralysis, related to a depolarization block in muscle. Similarly, ventricular conduction is slowed. Although hyperkalemia has marked effects on the peripheral nervous system, it has little effect on the central nervous system. Rapidly ascending muscular weakness leading to flaccid quadriplegia has been reported in patients with very high serum potassium levels. Paralysis of respiratory and speech muscles can also occur. In addition, GI manifestations, such as nausea, intermittent intestinal colic, and diarrhea, may be evident.

Assessment and Diagnostic Findings

Serum potassium levels and ECG changes are crucial to the diagnosis of hyperkalemia, as discussed previously. Arterial blood gas analysis may reveal both a metabolic and respiratory acidosis. Correcting the acidosis helps correct the hyperkalemia.

Medical Management

An ECG should be obtained immediately to detect changes. Shortened repolarization and peaked T waves are seen initially. To verify results, a repeat serum potassium

level should be obtained from a vein without an IV infusing a potassium-containing solution.

In nonacute situations, restriction of dietary potassium and potassium-containing medications may correct the imbalance. For example, eliminating the use of potassium-containing salt substitutes in a patient who is taking a potassium-conserving diuretic may be all that is needed to deal with mild hyperkalemia.

Prevention of serious hyperkalemia by the administration, either orally or by retention enema, of cation exchange resins (eg, sodium polystyrene sulfonate [Kayexalate]) may be necessary in patients with renal impairment. Cation exchange resins cannot be used if the patient has a paralytic ileus, because intestinal perforation can occur. Kayexalate binds with other cations in the GI tract and contributes to the development of hypomagnesemia and hypocalcemia; it may also cause sodium retention and fluid overload, and should be used with caution in patients with heart failure.

Emergency Pharmacologic Therapy

If serum potassium levels are dangerously elevated, it may be necessary to administer IV calcium gluconate. Within minutes after administration, calcium antagonizes the action of hyperkalemia on the heart, but it does not reduce the serum potassium concentration. Calcium chloride and calcium gluconate are not interchangeable; calcium gluconate contains 4.5 mEq of calcium and calcium chloride contains 13.6 mEq of calcium. Therefore, caution is required.

Monitoring the blood pressure is essential to detect hypotension, which may result from the rapid IV administration of calcium gluconate. The ECG should be continuously monitored during administration; the appearance of bradycardia is an indication to stop the infusion. The myocardial protective effects of calcium last about 30 minutes. Extra caution is required if the patient has been "digitalized" (ie, has received accelerated dosages of a digitalis-based cardiac glycoside to reach a desired serum digitalis level rapidly); parenteral administration of calcium sensitizes the heart to digitalis and may precipitate digitalis toxicity.

IV administration of sodium bicarbonate may be necessary to alkalinize the plasma, cause a temporary shift of potassium into the cells, and furnish sodium to antagonize the cardiac effects of potassium (Vacca, 2008). Effects of this therapy begin within 30 to 60 minutes and may persist for hours; however, they are temporary.

IV administration of regular insulin and a hypertonic dextrose solution causes a temporary shift of potassium into the cells. Glucose and insulin therapy has an onset of action within 30 minutes and lasts for several hours. Loop diuretics, such as furosemide (Lasix), increase excretion of water by inhibiting sodium, potassium, and chloride reabsorption in the ascending loop of Henle and distal renal tubule.

Beta-2 agonists, such as albuterol (Proventil, Ventolin), are highly effective in decreasing potassium, but their use remains controversial, because they can cause tachycardia and chest discomfort (Porth & Matfin, 2009). Beta-2 agonists move potassium into the cells and may be used in the absence of ischemic cardiac disease. Their use is a stopgap measure that only temporarily protects the patient from hyperkalemia. If the hyperkalemic condition is not transient, actual removal of potassium from the body is required through cation exchange resins, peritoneal dialysis, hemodialysis, or other forms of renal replacement therapy.

Nursing Management

Patients at risk for potassium excess (eg, those with renal failure) need to be identified and closely monitored for signs of hyperkalemia. The nurse observes for signs of muscle weakness and dysrhythmias. The presence of paresthesias and GI symptoms such as nausea and intestinal colic are noted. Serum potassium levels, as well as BUN, creatinine, glucose, and arterial blood gas values, are monitored for patients at risk for developing hyperkalemia (Heitz & Horne, 2005).

Preventing Hyperkalemia

Measures are taken to prevent hyperkalemia in patients at risk, when possible, by encouraging the patient to adhere to the prescribed potassium restriction. Potassium-rich foods to be avoided include many fruits and vegetables, legumes, whole-grain breads, meat, milk, eggs, coffee, tea, and cocoa (Dudek, 2006). Conversely, foods with minimal potassium content include butter, margarine, cranberry juice or sauce, ginger ale, gumdrops or jellybeans, hard candy, root beer, sugar, and honey. Labels of cola beverages must be checked carefully because some are high in potassium and some are not.

Correcting Hyperkalemia

It is possible to exceed the tolerance for potassium if administered rapidly by the IV route. Therefore, care is taken to administer and monitor potassium solutions closely. Particular attention is paid to the solution's concentration and rate of administration. Potassium is not added to parenteral solutions on the nursing units but in the pharmacy. IV administration is via a volumetric infusion pump (Hayes, 2007b).

It is important to caution patients to use salt substitutes sparingly if they are taking other supplementary forms of potassium or potassium-conserving diuretics (O'Neill, 2007). Also, potassium-conserving diuretics, such as spironolactone (Aldactone), triamterene (Dyrenium), and amiloride (Midamor); potassium supplements; and salt substitutes should not be administered to patients with renal dysfunction. Most salt substitutes contain approximately 50 to 60 mEq of potassium per teaspoon.

Calcium Imbalances

More than 99% of the body's calcium is located in the skeletal system; it is a major component of bones and teeth. About 1% of skeletal calcium is rapidly exchangeable with blood calcium, and the rest is more stable and only slowly exchanged. The small amount of calcium located outside the bone circulates in the serum, partly bound to protein and partly ionized. Calcium plays a major role in transmitting nerve impulses and helps regulate muscle contraction

and relaxation, including cardiac muscle. Calcium is instrumental in activating enzymes that stimulate many essential chemical reactions in the body, and it also plays a role in blood coagulation. Because many factors affect calcium regulation, both hypocalcemia and hypercalcemia are relatively common disturbances.

The normal total serum calcium level is 8.6 to 10.2 mg/dL (2.2 to 2.6 mmol/L). Calcium exists in plasma in three forms: ionized, bound, and complexed. About 50% of the serum calcium exists in a physiologically active ionized form that is important for neuromuscular activity and blood coagulation; this is the only physiologically and clinically significant form. The normal ionized serum calcium level is 4.5 to 5.1 mg/dL (1.1 to 1.3 mmol/L). Less than half of the plasma calcium is bound to serum proteins, primarily albumin. The remainder is combined with nonprotein anions: phosphate, citrate, and carbonate.

Calcium is absorbed from foods in the presence of normal gastric acidity and vitamin D. It is excreted primarily in the feces, with the remainder excreted in the urine. The serum calcium level is controlled by PTH and calcitonin. As ionized serum calcium decreases, the parathyroid glands secrete PTH. This, in turn, increases calcium absorption from the GI tract, increases calcium reabsorption from the renal tubule, and releases calcium from the bone. The increase in calcium ion concentration suppresses PTH secretion. When calcium increases excessively, the thyroid gland secretes calcitonin, which inhibits calcium reabsorption from bone and decreases the serum calcium concentration.

CALCIUM DEFICIT (HYPOCALCEMIA)

Hypocalcemia (serum values lower than 8.6 mg/dL [2.15 mmol/L]) occurs in a variety of clinical situations. A patient may have a total body calcium deficit (as in osteoporosis) but a normal serum calcium level. Elderly people and those with disabilities, who spend an increased amount of time in bed, have an increased risk of hypocalcemia, because bed rest increases bone resorption.

Pathophysiology

Several factors can cause hypocalcemia, including primary hypoparathyroidism and surgical hypoparathyroidism. The latter is far more common. Not only is hypocalcemia associated with thyroid and parathyroid surgery, but it can also occur after radical neck dissection and is most likely in the first 24 to 48 hours after surgery. Transient hypocalcemia can occur with massive administration of citrated blood (ie, massive hemorrhage and shock), because citrate can combine with ionized calcium and temporarily remove it from the circulation.

Inflammation of the pancreas causes the breakdown of proteins and lipids. It is thought that calcium ions combine with the fatty acids released by lipolysis, forming soaps. As a result of this process, hypocalcemia occurs and is common in pancreatitis. Hypocalcemia may be related to excessive secretion of glucagon from the inflamed pancreas, which results in increased secretion of calcitonin.

Hypocalcemia is common in patients with renal failure, because these patients frequently have elevated serum phosphate levels. Hyperphosphatemia usually causes a reciprocal drop in the serum calcium level. Other causes of hypocalcemia include inadequate vitamin D consumption, magnesium deficiency, medullary thyroid carcinoma, low serum albumin levels, alkalosis, and alcohol abuse. Medications predisposing to hypocalcemia include aluminum-containing antacids, aminoglycosides, caffeine, cisplatin, corticosteroids, mithramycin, phosphates, isoniazid, and loop diuretics.

Clinical Manifestations

Tetany, the most characteristic manifestation of hypocalcemia and hypomagnesemia, refers to the entire symptom complex induced by increased neural excitability. These symptoms are caused by spontaneous discharges of both sensory and motor fibers in peripheral nerves. Sensations of tingling may occur in the tips of the fingers, around the mouth, and, less commonly, in the feet. Spasms of the muscles of the extremities and face may occur. Pain may develop as a result of these spasms. Hyperactive DTRs are another clinical sign associated with tetany.

Trousseau's sign (Fig. 14-6) can be elicited by inflating a blood pressure cuff on the upper arm to about 20 mm Hg above systolic pressure; within 2 to 5 minutes, carpal spasm (an adducted thumb, flexed wrist and metacarpophalangeal joints, extended interphalangeal joints with fingers together) will occur as ischemia of the ulnar nerve develops. Chvostek's sign consists of twitching of muscles enervated by the facial nerve when the region that is about 2 cm anterior to the earlobe, just below the zygomatic arch, is tapped.

Seizures may occur because hypocalcemia increases irritability of the central nervous system as well as of the peripheral nerves (Tocco, 2007). Other changes associated with hypocalcemia include mental changes such as depression, impaired memory, confusion, delirium, and hallucinations. A prolonged QT interval is seen on the ECG due to prolongation of the ST segment, and torsades de pointes, a type of ventricular tachycardia, may occur. Respiratory effects with decreasing calcium include dyspnea and laryngospasm. Signs and symptoms of chronic hypocalcemia include hyperactive bowel sounds, dry and brittle hair and nails, and abnormal clotting.

Figure 14-6 Trousseau's sign. Ischemia-induced carpal spasm can occur with hypocalcemia or hypomagnesemia. Occluding the brachial artery with a blood pressure cuff for 3 minutes can produce carpal spasm (contraction of the fingers and hand), which mimics the spasm that occurs with hypocalcemia or hypomagnesemia.

Osteoporosis is associated with prolonged low intake of calcium and represents a total body calcium deficit, even though serum calcium levels are usually normal. This disorder occurs in millions of Americans and is most common in postmenopausal women. It is characterized by loss of bone mass, which causes bones to become porous and brittle and therefore susceptible to fracture. See Chapter 68 for further discussion of osteoporosis.

Assessment and Diagnostic Findings

When evaluating serum calcium levels, the serum albumin level and the arterial pH must also be considered. Because abnormalities in serum albumin levels may affect interpretation of the serum calcium level, it may be necessary to calculate the corrected serum calcium if the serum albumin level is abnormal. For every decrease in serum albumin of 1 g/dL below 4 g/dL, the total serum calcium level is underestimated by approximately 0.8 mg/dL. The following is a quick method to calculate the corrected serum calcium level:

$$\text{Measured total serum } Ca^{++} \text{ level (mg/dL)} + 0.8 \times (4.0 - \text{Measured albumin level [g/dL]}) = \text{Corrected total calcium concentration (mg/dL)}$$

An example of the calculations needed to obtain the corrected total serum calcium level is as follows:

A patient's reported serum albumin level is 2.5 g/dL; the reported serum calcium level is 10.5 mg/dL. First, the decrease in serum albumin level from normal (ie, the difference from the normal albumin concentration of 4 g/dL) is calculated: 4 g/dL – 2.5 g/dL = 1.5 g/dL. Next, the following ratio is calculated:

$$0.8 \text{ mg/dL} : 1 \text{ g/dL} = X \text{ mg/dL} : 1.5 \text{ mg/dL}$$
$$X = 0.8 \times 1.5 \text{ mg/dL}$$
$$X = 1.2 \text{ mg/dL calcium}$$

Finally, 1.2 mg/dL is added to 10.5 mg/dL (the reported serum calcium level) to obtain the corrected total serum calcium level: 1.2 mg/dL + 10.5 mg/dL = 11.7 mg/dL.

Clinicians often discount a low serum calcium level in the presence of a similarly low serum albumin level. The ionized calcium level is usually normal in patients with reduced total serum calcium levels and concomitant hypoalbuminemia. When the arterial pH increases (alkalosis), more calcium becomes bound to protein. As a result, the ionized portion decreases. Symptoms of hypocalcemia may occur with alkalosis. Acidosis (low pH) has the opposite effect; that is, less calcium is bound to protein and therefore more exists in the ionized form. However, relatively small changes in serum calcium levels occur in these acid–base abnormalities.

Ideally, the ionized level of calcium should be measured in the laboratory. However, in many laboratories, only the total calcium level is reported; therefore, the concentration of the ionized fraction must be estimated by simultaneous measurement of the serum albumin level. PTH levels are decreased in hypoparathyroidism. Magnesium and phosphorus levels need to be assessed to identify possible causes of decreased calcium.

Medical Management

Acute symptomatic hypocalcemia is life-threatening and requires prompt treatment with IV administration of a calcium salt (Avent, 2007). Parenteral calcium salts include calcium gluconate, calcium chloride, and calcium gluceptate. Although calcium chloride produces a significantly higher ionized calcium level than calcium gluconate does, it is not used as often because it is more irritating and can cause sloughing of tissue if it infiltrates. Too-rapid IV administration of calcium can cause cardiac arrest, preceded by bradycardia. IV administration of calcium is particularly dangerous in patients receiving digitalis-derived medications, because calcium ions exert an effect similar to that of digitalis and can cause digitalis toxicity, with adverse cardiac effects. Therefore, calcium should be diluted in D_5W and administered as a slow IV bolus or a slow IV infusion using a volumetric infusion pump. The IV site must be observed often for any evidence of infiltration because of the risk of extravasation and resultant cellulitis or necrosis. A 0.9% sodium chloride solution should not be used with calcium because it increases renal calcium loss. Solutions containing phosphates or bicarbonate should not be used with calcium because they cause precipitation when calcium is added. The nurse must clarify with the physician and pharmacist which calcium salt to administer, because calcium gluconate yields 4.5 mEq of calcium and calcium chloride provides 13.6 mEq of calcium. Calcium replacement can cause postural hypotension; therefore, the patient is kept in bed during IV infusion, and blood pressure is monitored.

Vitamin D therapy may be instituted to increase calcium absorption from the GI tract; otherwise, the amount of calcium absorbed may not satisfy the body's calcium requirement. In addition, aluminum hydroxide, calcium acetate, or calcium carbonate antacids may be prescribed to decrease elevated phosphorus levels before treating hypocalcemia in the patient with chronic renal failure. Increasing the dietary intake of calcium to at least 1000 to 1500 mg/day in the adult is recommended. Calcium-containing foods include milk products; green, leafy vegetables; canned salmon; sardines; and fresh oysters. Hypomagnesemia can also cause tetany; if the tetany responds to IV calcium, then a low magnesium level is considered as a possible cause in chronic renal failure.

Nursing Management

It is important to observe for hypocalcemia in at-risk patients. Seizure precautions are initiated if hypocalcemia is severe. The status of the airway is closely monitored, because laryngeal stridor can occur. Safety precautions are taken, as indicated, if confusion is present.

It is important to teach the patient with hypocalcemia what foods are rich in calcium. The nurse must also advise the patient to consider calcium supplements if sufficient calcium is not consumed in the diet. Such supplements should be taken in divided doses with meals. Alcohol and caffeine in high doses inhibit calcium absorption, and moderate cigarette smoking increases urinary calcium excretion. The patient is also cautioned to avoid the overuse of laxatives and antacids that contain phosphorus, because their use decreases calcium absorption.

CALCIUM EXCESS (HYPERCALCEMIA)

Hypercalcemia (greater than 10.2 mg/dL [2.6 mmol/L]) is a dangerous imbalance when severe; in fact, hypercalcemic crisis has a mortality rate as high as 50% if not treated promptly.

Pathophysiology

The most common causes of hypercalcemia are malignancies and hyperparathyroidism. Malignant tumors can produce hypercalcemia by a variety of mechanisms (Stewart, 2005). The excessive PTH secretion associated with hyperparathyroidism causes increased release of calcium from the bones and increased intestinal and renal absorption of calcium. Calcifications of soft tissue occur when the calcium–phosphorus product (serum calcium × serum phosphorus) exceeds 70 mg/dL.

Bone mineral is lost during immobilization, and sometimes this causes elevation of total (and especially ionized) calcium in the bloodstream. However, symptomatic hypercalcemia from immobilization is rare; when it does occur, it is virtually limited to people with high calcium turnover rates (eg, adolescents during a growth spurt). Most cases of hypercalcemia secondary to immobility occur after severe or multiple fractures or spinal cord injury.

Thiazide diuretics can cause a slight elevation in serum calcium levels because they potentiate the action of PTH on the kidneys, reducing urinary calcium excretion. The milk-alkali syndrome has occurred in patients with peptic ulcer treated previously for a prolonged period with milk and alkaline antacids, particularly calcium carbonate. Vitamin A and D intoxication, as well as chronic lithium use and theophylline toxicity, can cause calcium excess. Calcium levels are inversely related to phosphorus levels.

Hypercalcemia reduces neuromuscular excitability because it suppresses activity at the myoneural junction. Decreased tone in smooth and striated muscle may cause symptoms such as muscle weakness, incoordination, anorexia, and constipation. Cardiac standstill can occur when the serum calcium level is about 18 mg/dL (4.5 mmol/L). Calcium enhances the inotropic effect of digitalis; therefore, hypercalcemia aggravates digitalis toxicity.

Clinical Manifestations

The symptoms of hypercalcemia are proportional to the degree of elevation of the serum calcium level. Anorexia, nausea, vomiting, and constipation are common symptoms of hypercalcemia. Dehydration occurs with nausea, vomiting, anorexia, and calcium reabsorption at the proximal renal tubule. Abdominal and bone pain may also be present. Abdominal distention and paralytic ileus may complicate severe hypercalcemic crisis. Excessive urination due to disturbed renal tubular function produced by hypercalcemia may occur. Severe thirst may occur with polyuria secondary to high solute (calcium) load. Patients with chronic hypercalcemia may develop symptoms similar to peptic ulcer disease because hypercalcemia increases the secretion of acid and pepsin in the stomach.

Confusion, impaired memory, slurred speech, lethargy, acute psychotic behavior, or coma may occur (Stewart, 2005). The more severe symptoms tend to appear when the serum calcium level is approximately 16 mg/dL (4 mmol/L) or higher. However, some patients become profoundly disturbed with serum calcium levels of only 12 mg/dL (3 mmol/L). These symptoms resolve as serum calcium levels return to normal after treatment.

Hypercalcemic crisis refers to an acute rise in the serum calcium level to 17 mg/dL (4.3 mmol/L) or higher. Severe thirst and polyuria are often present. Other findings may include muscle weakness, intractable nausea, abdominal cramps, severe constipation, diarrhea, peptic ulcer symptoms, and bone pain. Lethargy, confusion, and coma may also occur. This condition is dangerous and may result in cardiac arrest.

Assessment and Diagnostic Findings

The serum calcium level is greater than 10.2 mg/dL (2.6 mmol/L). Cardiovascular changes may include a variety of dysrhythmias (ie, heart blocks) and shortening of the QT interval and ST segment. The PR interval is sometimes prolonged. The double-antibody PTH test may be used to differentiate between primary hyperparathyroidism and malignancy as a cause of hypercalcemia: PTH levels are increased in primary or secondary hyperparathyroidism and suppressed in malignancy. X-rays may reveal bone changes if the patient has hypercalcemia secondary to a malignancy, bone cavitation, or urinary calculi. The Sulkowitch urine test analyzes the amount of calcium in the urine; in hypercalcemia, dense precipitation is observed due to hypercalciuria.

Medical Management

Therapeutic aims in hypercalcemia include decreasing the serum calcium level and reversing the process causing hypercalcemia. Treating the underlying cause (eg, chemotherapy for a malignancy, partial parathyroidectomy for hyperparathyroidism) is essential.

Pharmacologic Therapy

Measures include administering fluids to dilute serum calcium and promote its excretion by the kidneys, mobilizing the patient, and restricting dietary calcium intake. IV administration of 0.9% sodium chloride solution temporarily dilutes the serum calcium level and increases urinary calcium excretion by inhibiting tubular reabsorption of calcium. Administering IV phosphate can cause a reciprocal drop in serum calcium. Furosemide (Lasix) is often used in conjunction with administration of a saline solution; in addition to causing diuresis, furosemide increases calcium excretion. Although often overlooked, fluids and medications that contain calcium and dietary sources of calcium should be halted (Stewart, 2005).

Calcitonin can be used to lower the serum calcium level and is particularly useful for patients with heart disease or renal failure who cannot tolerate large sodium loads. Calcitonin reduces bone resorption, increases the deposition of calcium and phosphorus in the bones, and increases urinary excretion of calcium and phosphorus (Karch, 2008). Although several forms are available, calcitonin derived from salmon is commonly used. Skin testing for allergy to salmon calcitonin is necessary before the hormone is administered. Systemic allergic reactions are possible because this hormone is a protein; resistance to the medication may develop later because of antibody formation. Calcitonin is administered by intramuscular injection rather than subcutaneously, because patients with hypercalcemia have poor perfusion of subcutaneous tissue.

For patients with cancer, treatment is directed at controlling the condition by surgery, chemotherapy, or radiation therapy. Corticosteroids may be used to decrease bone turnover and tubular reabsorption for patients with sarcoidosis, myelomas, lymphomas, and leukemias; patients with solid tumors are less responsive. Some bisphosphonates (eg, etidronate disodium [Didronel], pamidronate disodium [Aredia], and ibandronate sodium [Boniva]) inhibit osteoclast activity. IV forms can cause fever, transient leukopenia, eye inflammation, nephrotic syndrome, and jaw osteonecrosis (Karch, 2008). Mithramycin, a cytotoxic antibiotic, inhibits bone resorption and thus lowers the serum calcium level. This agent must be used cautiously because it has significant side effects, including thrombocytopenia, nephrotoxicity, rebound hypercalcemia when discontinued, and hepatotoxicity. Inorganic phosphate salts can be administered orally or by nasogastric tube (in the form of Phospho-Soda or Neutra-Phos), rectally (as retention enemas), or intravenously. IV phosphate therapy is used with extreme caution in the treatment of hypercalcemia, because it can cause severe calcification in various tissues, hypotension, tetany, and acute renal failure.

Nursing Management

It is important to monitor for hypercalcemia in at-risk patients. Interventions such as increasing patient mobility and encouraging fluids can help prevent hypercalcemia, or at least minimize its severity. Hospitalized patients at risk for hypercalcemia should be encouraged to ambulate as soon as possible. Those who are outpatients and receive home care are instructed about the importance of frequent ambulation.

When encouraging oral fluids, the nurse considers the patient's likes and dislikes. Fluids containing sodium should be administered unless contraindicated, because sodium assists with calcium excretion. Patients are encouraged to drink 3 to 4 quarts of fluid daily. Adequate fiber in the diet is encouraged to offset the tendency for constipation. Safety precautions are implemented, as necessary, when mental symptoms of hypercalcemia are present. The patient and family are informed that these mental changes are reversible with treatment. Increased calcium increases the effects of digitalis; therefore, the patient is assessed for signs and symptoms of digitalis toxicity. Because ECG changes (premature ventricular contractions, paroxysmal atrial tachycardia, and heart block) can occur, the cardiac rate and rhythm are monitored for any abnormalities.

Magnesium Imbalances

Magnesium is the most abundant intracellular cation after potassium. It acts as an activator for many intracellular enzyme systems and plays a role in both carbohydrate and protein metabolism. The normal serum magnesium level is 1.3 to 2.3 mg/dL (0.62 to 0.95 mmol/L). Approximately one third of serum magnesium is bound to protein; the remaining two thirds exists as free cations—the active component (Mg^{++}). Magnesium balance is important in neuromuscular function. Because magnesium acts directly on the myoneural junction, variations in the serum level affect neuromuscular irritability and contractility. For example, an excess of magnesium diminishes the excitability of the muscle cells, whereas a deficit increases neuromuscular irritability and contractility. Magnesium produces its sedative effect at the neuromuscular junction, probably by inhibiting the release of the neurotransmitter acetylcholine. It also increases the stimulus threshold in nerve fibers.

Magnesium also affects the cardiovascular system, acting peripherally to produce vasodilation and decreased peripheral resistance. Magnesium is predominantly found in bone and soft tissues and eliminated by the kidneys.

MAGNESIUM DEFICIT (HYPOMAGNESEMIA)

Hypomagnesemia refers to a below-normal serum magnesium concentration (1.3 mg/dL [0.62 mmol/L]) and is frequently associated with hypokalemia and hypocalcemia. Magnesium is similar to calcium in two aspects: (1) it is the ionized fraction of magnesium that is primarily involved in neuromuscular activity and other physiologic processes, and (2) magnesium levels should be evaluated in combination with albumin levels. About 30% of magnesium is protein bound, principally to albumin. A decreased serum albumin level can, therefore, reduce the measured total magnesium concentration; however, it does not reduce the ionized plasma magnesium concentration.

Pathophysiology

An important route of magnesium loss is the GI tract. Loss of magnesium from the GI tract may occur with nasogastric suction, diarrhea, or fistulas. Because fluid from the lower GI tract has a higher concentration of magnesium (10 to 14 mEq/L) than fluid from the upper tract (1 to 2 mEq/L), losses from diarrhea and intestinal fistulas are more likely to induce magnesium deficit than are those from gastric suction. Although magnesium losses are relatively small in nasogastric suction, hypomagnesemia occurs if losses are prolonged and magnesium is not replaced through IV infusion. Because the distal small bowel is the major site of magnesium absorption, any disruption in small-bowel function (eg, intestinal resection or inflammatory bowel disease) can lead to hypomagnesemia. Hypomagnesemia is a common yet often overlooked imbalance in acutely and critically ill patients. It may occur with withdrawal from alcohol and administration of tube feedings or parenteral nutrition.

Alcoholism is currently the most common cause of symptomatic hypomagnesemia in the United States. The serum magnesium level should be measured at least every 2 or 3 days in patients undergoing withdrawal from alcohol. The serum magnesium level may be normal on admission but may decrease as a result of metabolic changes, such as the intracellular shift of magnesium associated with IV glucose administration.

During nutritional replacement, the major cellular electrolytes move from the serum to newly synthesized cells. Therefore, if the enteral or parenteral feeding formula is deficient in magnesium content, serious hypomagnesemia will occur. Because of this, serum magnesium levels should be measured at regular intervals in patients who are receiving parenteral or enteral feedings, especially those who have

undergone a period of starvation. Other causes of hypomagnesemia include the administration of aminoglycosides, cyclosporine, cisplatin, diuretics, digitalis, and amphotericin, as well as the rapid administration of citrated blood, especially to patients with renal or hepatic disease. Magnesium deficiency often occurs in diabetic ketoacidosis, secondary to increased renal excretion during osmotic diuresis and shifting of magnesium into the cells with insulin therapy. Other contributing causes are pregnancy, lactation, sepsis, burns, and hypothermia.

Clinical Manifestations

Clinical manifestations of hypomagnesemia are largely confined to the neuromuscular system. Some are due directly to the low serum magnesium level; others are due to secondary changes in potassium and calcium metabolism. Symptoms do not usually occur until the serum magnesium level has dropped to less than 1 mEq/L (0.5 mmol/L).

Among the neuromuscular changes are hyperexcitability with muscle weakness, tremors, and athetoid movements (slow, involuntary twisting and writhing). Others include tetany, nystagmus, vertigo, generalized tonic–clonic or focal seizures, laryngeal stridor, and positive Chvostek's and Trousseau's signs (see earlier discussion), which occur, in part, because of accompanying hypocalcemia.

Hypomagnesemia may be accompanied by marked alterations in mood. Apathy, depression, apprehension, and extreme agitation have been noted, as well as ataxia, dizziness, insomnia, and confusion. At times, delirium, auditory or visual hallucinations, and frank psychoses may occur.

Magnesium deficiency can disturb the ECG by prolonging the QRS, depressing the ST segment, and predisposing to cardiac dysrhythmias, such as premature ventricular contractions, supraventricular tachycardia, torsades de pointes (a form of ventricular tachycardia), and ventricular fibrillation. Increased susceptibility to digitalis toxicity is associated with low serum magnesium levels. This is important, because patients receiving digoxin are also likely to be receiving diuretic therapy, predisposing them to renal loss of magnesium. Hypercalcemia and hypokalemia may be refractory to correction until the magnesium level is corrected.

Assessment and Diagnostic Findings

On laboratory analysis, the serum magnesium level is less than 1.3 mg/dL (0.62 mmol/L). Urine magnesium may help identify the cause of magnesium depletion, and levels are measured after a loading dose of magnesium sulfate is administered. Two newer diagnostic techniques (nuclear magnetic resonance spectroscopy and the ion-selective electrode) are sensitive and direct means of measuring ionized serum magnesium levels.

Medical Management

Mild magnesium deficiency can be corrected by diet alone. Principal dietary sources of magnesium include green leafy vegetables, nuts, seeds, legumes, whole grains, seafood, peanut butter, and cocoa.

If necessary, magnesium salts can be administered orally in an oxide or gluconate form to replace continuous losses but can produce diarrhea. Patients receiving parenteral nutrition require magnesium in the IV solution to prevent hypomagnesemia. IV magnesium sulfate must be administered by an infusion pump and at a rate not to exceed 150 mg/min, or 67 mEq over 8 hours. Overt symptoms of hypomagnesemia are treated with parenteral administration of magnesium. A bolus dose of magnesium sulfate given too rapidly can produce alterations in cardiac conduction leading to heart block or asystole. Vital signs must be assessed frequently during magnesium administration to detect changes in cardiac rate or rhythm, hypotension, and respiratory distress. Monitoring urine output is essential before, during, and after magnesium administration; the physician is notified if urine volume decreases to less than 100 mL over 4 hours. Calcium gluconate must be readily available to treat hypocalcemic tetany or hypermagnesemia.

Nursing Management

The nurse should be aware of patients at risk for hypomagnesemia and observe them for its signs and symptoms. Patients receiving digitalis are monitored closely, because a deficit of magnesium can predispose them to digitalis toxicity. If hypomagnesemia is severe, seizure precautions are implemented (Tocco, 2007). Other safety precautions are instituted, as indicated, if confusion is observed. Because difficulty in swallowing (dysphagia) may occur in those with magnesium depletion, these patients should be screened for dysphagia.

Teaching plays a major role in treating magnesium deficit, particularly a deficit resulting from abuse of diuretic or laxative medications. In such cases, the nurse instructs the patient about the need to consume magnesium-rich foods. For patients experiencing hypomagnesemia from abuse of alcohol, the nurse provides teaching, counseling, support, and possible referral to alcohol abstinence programs or other professional help.

MAGNESIUM EXCESS (HYPERMAGNESEMIA)

Hypermagnesemia (serum levels over 2.3 mg/dL [0.95 mmol/L]) is a rare electrolyte abnormality, because the kidneys efficiently excrete magnesium. A serum magnesium level can appear falsely elevated if blood specimens are allowed to hemolyze or are drawn from an extremity with a tourniquet that was applied too tightly.

Pathophysiology

By far the most common cause of hypermagnesemia is renal failure. In fact, most patients with advanced renal failure have at least a slight elevation in serum magnesium levels. This condition is aggravated when such patients receive magnesium to control seizures.

Hypermagnesemia can occur in patients with untreated diabetic ketoacidosis when catabolism causes the release of cellular magnesium that cannot be excreted because of profound fluid volume depletion and resulting oliguria. A surplus of magnesium can also result from excessive magnesium administered to treat hypertension of pregnancy or to treat low hypomagnesemia. Increased serum magnesium levels can also occur in adrenocortical insufficiency, Addison's disease, or hypothermia. Excessive use of magnesium-

based antacids (eg, Maalox, Riopan, Mylanta) or laxatives (Milk of Magnesia) and medications that decrease GI motility, including opioids and anticholinergics, can also increase serum magnesium levels. Decreased elimination of magnesium or its increased absorption due to intestinal hypomotility from any cause can contribute to hypermagnesemia. Lithium intoxication can also cause an increase in serum magnesium levels. Extensive soft-tissue injury or necrosis as with trauma, shock, sepsis, cardiac arrest, or severe burns can also result in hypermagnesemia (Muller & Bell, 2008).

Clinical Manifestations

Acute elevation of the serum magnesium level depresses the central nervous system as well as the peripheral neuromuscular junction. At mildly increased levels, there is a tendency for lowered blood pressure because of peripheral vasodilation. Nausea, vomiting, weakness, soft-tissue calcifications, facial flushing, and sensations of warmth may also occur. At higher magnesium concentrations, lethargy, difficulty speaking (dysarthria), and drowsiness can occur. DTRs are lost, and muscle weakness and paralysis may develop. The respiratory center is depressed when serum magnesium levels exceed 10 mEq/L (5 mmol/L). Coma, atrioventricular heart block, and cardiac arrest can occur when the serum magnesium level is greatly elevated and not treated. High levels of magnesium also result in platelet clumping and delayed thrombin formation (Chernecky & Berger, 2007).

Assessment and Diagnostic Findings

On laboratory analysis, the serum magnesium level is greater than 2.3 mg/dL (0.95 mmol/L). Increased potassium and calcium are present concurrently. As creatinine clearance decreases to less than 3.0 mL/min, the serum magnesium levels increase. ECG findings may include a prolonged PR interval, tall T waves, a widened QRS, and a prolonged QT interval, as well as an atrioventricular block.

Medical Management

Hypermagnesemia can be prevented by avoiding the administration of magnesium to patients with renal failure and by carefully monitoring seriously ill patients who are receiving magnesium salts. In patients with severe hypermagnesemia, all parenteral and oral magnesium salts are discontinued. In emergencies, such as respiratory depression or defective cardiac conduction, ventilatory support and IV calcium gluconate are indicated. In addition, hemodialysis with a magnesium-free dialysate can reduce the serum magnesium to a safe level within hours. Administration of loop diuretics (Lasix) and sodium chloride or lactated Ringer's IV solution enhances magnesium excretion in patients with adequate renal function. IV calcium gluconate antagonizes the cardiovascular and neuromuscular effects of magnesium.

Nursing Management

Patients at risk for hypermagnesemia are identified and assessed. If hypermagnesemia is suspected, the nurse monitors the vital signs, noting hypotension and shallow respirations. The nurse also observes for decreased DTRs and changes in the level of consciousness. Medications that contain magnesium are not administered to patients with renal failure or compromised renal function, and patients with renal failure are cautioned to check with their health care providers before taking OTC medications. Caution is essential when preparing and administering magnesium-containing fluids parenterally, because available parenteral magnesium solutions (eg, 2-mL ampules, 50-mL vials) differ in concentration.

Phosphorus Imbalances

Phosphorus is a critical constituent of all the body's tissues. It is essential to the function of muscle and red blood cells; the formation of adenosine triphosphate (ATP) and of 2,3-diphosphoglycerate, which facilitates release of oxygen from hemoglobin; and the maintenance of acid–base balance, as well as the nervous system and the intermediary metabolism of carbohydrate, protein, and fat. It provides structural support to bones and teeth. Phosphorus is the primary anion of the ICF. About 85% of phosphorus is located in bones and teeth, 14% in soft tissue, and less than 1% in the ECF. The normal serum phosphorus level is 2.5 to 4.5 mg/dL (0.8 to 1.45 mmol/L) in adults.

PHOSPHORUS DEFICIT (HYPOPHOSPHATEMIA)

Hypophosphatemia is indicated by a value below 2.5 mg/dL (0.8 mmol/L). Although it often indicates phosphorus deficiency, hypophosphatemia may occur under a variety of circumstances in which total body phosphorus stores are normal. Conversely, phosphorus deficiency is an abnormally low content of phosphorus in lean tissues that may exist in the absence of hypophosphatemia. It can be caused by an intracellular shift of potassium from serum into cells, by increased urinary excretion of potassium, or by decreased intestinal absorption of potassium.

Pathophysiology

Hypophosphatemia may occur during the administration of calories to patients with severe protein–calorie malnutrition. It is most likely to result from overzealous intake or administration of simple carbohydrates. This syndrome can be induced in any person with severe protein–calorie malnutrition (eg, patients with anorexia nervosa or alcoholism, elderly debilitated patients who are unable to eat). As many as 50% of patients hospitalized because of chronic alcoholism have hypophosphatemia.

Marked hypophosphatemia may develop in malnourished patients who receive parenteral nutrition if the phosphorus loss is not corrected. Other causes of hypophosphatemia include heat stroke, prolonged intense hyperventilation, alcohol withdrawal, poor dietary intake, diabetic ketoacidosis, respiratory alkalosis, hepatic encephalopathy, and major thermal burns. Low magnesium levels, low potassium levels, and hyperparathyroidism related to increased urinary losses of phosphorus contribute to hypophosphatemia. Loss of phosphorus through the kidneys also occurs with acute volume expansion, osmotic diuresis, use of carbonic anhydrase inhibitors (acetazolamide

[Diamox]), and some malignancies. Respiratory alkalosis can cause a decrease in phosphorus because of an intracellular shift of phosphorus.

Excess phosphorus binding by antacids may decrease the phosphorus available from the diet to an amount lower than required to maintain serum phosphorus balance. The degree of hypophosphatemia depends on the amount of phosphorus in the diet compared to the dose of antacid. Phosphate can occur with chronic diarrhea or through severe potassium restriction. Vitamin D regulates intestinal ion absorption; therefore, a deficiency of vitamin D may cause decreased calcium and phosphorus levels, which may lead to osteomalacia (softened, brittle bones).

Clinical Manifestations

Most of the signs and symptoms of phosphorus deficiency appear to result from a deficiency of ATP, 2,3-diphosphoglycerate, or both. ATP deficiency impairs cellular energy resources; diphosphoglycerate deficiency impairs oxygen delivery to tissues, resulting in a wide range of neurologic manifestations, such as irritability, fatigue, apprehension, weakness, numbness, paresthesias, dysarthria, dysphagia, diplopia, confusion, seizures, and coma. Hypoxia leads to an increase in respiratory rate and respiratory alkalosis, causing phosphorus to move into the cells and potentiating hypophosphatemia. Hypophosphatemia may predispose a person to infection. In laboratory animals, hypophosphatemia is associated with depression of the chemotactic, phagocytic, and bacterial activity of granulocytes.

Muscle damage may develop as the ATP level in the muscle tissue declines. Clinical manifestations are muscle weakness, which may be subtle or profound and may affect any muscle group; muscle pain; and at times acute rhabdomyolysis (breakdown of skeletal muscle) (Spradling, 2007). Weakness of respiratory muscles may greatly impair ventilation. Hypophosphatemia also may predispose a person to insulin resistance and thus hyperglycemia. Chronic loss of phosphorus can cause bruising and bleeding from platelet dysfunction.

Assessment and Diagnostic Findings

On laboratory analysis, the serum phosphorus level is less than 2.5 mg/dL (0.80 mmol/L) in adults. When reviewing laboratory results, the nurse should keep in mind that glucose or insulin administration causes a slight decrease in the serum phosphorus level. PTH levels are increased in hyperparathyroidism. Serum magnesium may decrease due to increased urinary excretion of magnesium. Alkaline phosphatase is increased with osteoblastic activity. X-rays may show skeletal changes of osteomalacia or rickets.

Medical Management

Prevention of hypophosphatemia is the goal. In patients at risk for hypophosphatemia, serum phosphate levels should be closely monitored and correction initiated before deficits become severe. Adequate amounts of phosphorus should be added to parenteral solutions, and attention should be paid to the phosphorus levels in enteral feeding solutions.

Severe hypophosphatemia is dangerous and requires prompt attention. Aggressive IV phosphorus correction is usually limited to the patient whose serum phosphorus levels decrease to less than 1 mg/dL (0.3 mmol/L) and whose GI tract is not functioning. Possible dangers of IV administration of phosphorus include tetany from hypocalcemia and calcifications in tissues (blood vessels, heart, lung, kidney, eyes) from hyperphosphatemia. IV preparations of phosphorus are available as sodium or potassium phosphate. The rate of phosphorus administration should not exceed 10 mEq/h, and the site should be carefully monitored because tissue sloughing and necrosis can occur with infiltration. In less acute situations, oral phosphorus replacement is usually adequate.

Nursing Management

The nurse identifies patients who are at risk for hypophosphatemia and monitors them. Because malnourished patients receiving parenteral nutrition are at risk when calories are introduced too aggressively, preventive measures involve gradually introducing the solution to avoid rapid shifts of phosphorus into the cells.

For patients with documented hypophosphatemia, careful attention is given to preventing infection, because hypophosphatemia may alter the granulocytes. In patients requiring correction of phosphorus losses, the nurse frequently monitors serum phosphorus levels and documents and reports early signs of hypophosphatemia (apprehension, confusion, change in level of consciousness). If the patient experiences mild hypophosphatemia, foods such as milk and milk products, organ meats, nuts, fish, poultry, and whole grains should be encouraged. With moderate hypophosphatemia, supplements such as Neutra-Phos capsules (250 mg phosphorus/capsule; 7 mEq sodium and potassium), K-Phos (250 mg phosphorus/tablet; 14 mEq potassium), and Fleet's Phospho-Soda (815 mg phosphorus/5 mL) may be prescribed.

PHOSPHORUS EXCESS (HYPERPHOSPHATEMIA)

Hyperphosphatemia is a serum phosphorus level that exceeds 4.5 mg/dL (1.45 mmol/L) in adults.

Pathophysiology

Various conditions can lead to hyperphosphatemia, but the most common is renal failure. Other causes include increased intake, decreased output, or a shift from the intracellular to extracellular space. Conditions such as excessive vitamin D intake, administration of total parenteral nutrition, chemotherapy for neoplastic disease, hypoparathyroidism, metabolic or respiratory acidosis, diabetic ketoacidosis, acute hemolysis, high phosphate intake, profound muscle necrosis, and increased phosphorus absorption may also lead to this phosphorus imbalance. The primary complication of increased phosphorus is metastatic calcification (soft tissue, joints, and arteries), which occurs when the calcium–magnesium product (calcium × magnesium) exceeds 70 mg/dL.

Clinical Manifestations

An increased serum phosphorus level causes few symptoms. Symptoms that do occur usually result from decreased calcium levels and soft-tissue calcifications. The most important short-term consequence is tetany. Because of the recip-

rocal relationship between phosphorus and calcium, a high serum phosphorus level tends to cause a low serum calcium concentration. Tetany can result, causing tingling sensations in the fingertips and around the mouth. Anorexia, nausea, vomiting, bone and joint pain, muscle weakness, hyperreflexia, and tachycardia may occur.

The major long-term consequence is soft-tissue calcification, which occurs mainly in patients with a reduced glomerular filtration rate. High serum levels of inorganic phosphorus promote precipitation of calcium phosphate in nonosseous sites, decreasing urine output, impairing vision, and producing palpitations.

Assessment and Diagnostic Findings

On laboratory analysis, the serum phosphorus level exceeds 4.5 mg/dL (1.5 mmol/L) in adults. The serum calcium level is useful also for diagnosing the primary disorder and assessing the effects of treatments. X-rays may show skeletal changes with abnormal bone development. PTH levels are decreased in hypoparathyroidism. BUN and creatinine levels are used to assess renal function.

Medical Management

When possible, treatment is directed at the underlying disorder. For example, hyperphosphatemia may be related to volume depletion or respiratory or metabolic acidosis. In renal failure, elevated PTH production contributes to a high phosphorus level and bone disease. Measures to decrease the serum phosphate level and bind phosphorus in the GI tract of these patients include vitamin D preparations, such as calcitriol, which is available in both oral (Rocaltrol) and parenteral (Calcijex, paricalcitol [Zemplar]) forms. IV administration of calcitriol does not increase the serum calcium unless its dose is excessive, thus permitting more aggressive treatment of hyperphosphatemia with calcium-binding antacids (calcium carbonate or calcium citrate). Administration of Amphojel with meals is effective but can cause bone and central nervous system toxicity with long-term use. Restriction of dietary phosphate, forced diuresis with a loop diuretic, volume replacement with saline, and dialysis may also lower phosphorus. Surgery may be indicated for removal of large calcium and phosphorus deposits.

Nursing Management

The nurse monitors patients at risk for hyperphosphatemia. If a low-phosphorus diet is prescribed, the patient is instructed to avoid phosphorus-rich foods such as hard cheeses, cream, nuts, meats, whole-grain cereals, dried fruits, dried vegetables, kidneys, sardines, sweetbreads, and foods made with milk. When appropriate, the nurse instructs the patient to avoid phosphate-containing substances such as laxatives and enemas. The nurse also teaches the patient to recognize the signs of impending hypocalcemia and to monitor for changes in urine output.

Chloride Imbalances

Chloride, the major anion of the ECF, is found more in interstitial and lymph fluid compartments than in blood. Chloride is also contained in gastric and pancreatic juices, sweat, bile, and saliva. Sodium and chloride make up the largest electrolyte composition of the ECF and assist in determining osmotic pressure. Chloride is produced in the stomach, where it combines with hydrogen to form hydrochloric acid. Chloride control depends on the intake of chloride and the excretion and reabsorption of its ions in the kidneys. A small amount of chloride is lost in the feces.

The normal serum chloride level is 97 to 107 mEq/L (97 to 107 mmol/L). Inside the cell, the chloride level is 4 mEq/L. The serum level of chloride reflects a change in dilution or concentration of the ECF and does so in direct proportion to the sodium concentration. Serum osmolality parallels chloride levels as well. Aldosterone secretion increases sodium reabsorption, thereby increasing chloride reabsorption. The choroid plexus, which secretes cerebrospinal fluid in the brain, depends on sodium and chloride to attract water to form the fluid portion of the cerebrospinal fluid. Bicarbonate has an inverse relationship with chloride. As chloride moves from plasma into the red blood cells (called the chloride shift), bicarbonate moves back into the plasma. Hydrogen ions are formed, which then help release oxygen from hemoglobin. When the level of one of these three electrolytes (sodium, bicarbonate, or chloride) is disturbed, the other two are also affected. Chloride assists in maintaining acid–base balance and works as a buffer in the exchange of oxygen and carbon dioxide in red blood cells. Chloride is primarily obtained from the diet as table salt.

CHLORIDE DEFICIT (HYPOCHLOREMIA)

Hypochloremia is a serum chloride level below 97 mEq/L (97 mmol/L).

Pathophysiology

Hypochloremia can occur with GI tube drainage, gastric suctioning, gastric surgery, and severe vomiting and diarrhea. Administration of chloride-deficient IV solutions, low sodium intake, decreased serum sodium levels, metabolic alkalosis, massive blood transfusions, diuretic therapy, burns, and fever may cause hypochloremia. Administration of aldosterone, ACTH, corticosteroids, bicarbonate, or laxatives decreases serum chloride levels as well. As chloride decreases (usually because of volume depletion), sodium and bicarbonate ions are retained by the kidney to balance the loss. Bicarbonate accumulates in the ECF, which raises the pH and leads to hypochloremic metabolic alkalosis.

Clinical Manifestations

The signs and symptoms of hypochloremia are those of acid–base and electrolyte imbalances. The signs and symptoms of hyponatremia, hypokalemia, and metabolic alkalosis may also be present. Metabolic alkalosis is a disorder that results in a high pH and a high serum bicarbonate level as a result of excess alkali intake or loss of hydrogen ions. With compensation, the partial pressure of carbon dioxide in arterial blood ($PaCO_2$) increases to 50 mm Hg. Hyperexcitability of muscles, tetany, hyperactive DTRs, weakness, twitching, and muscle cramps may result. Hypokalemia can cause hypochloremia, resulting in cardiac dysrhythmias. In

addition, because low chloride levels parallel low sodium levels, a water excess may occur. Hyponatremia can cause seizures and coma.

Assessment and Diagnostic Findings

In addition to the chloride level, sodium and potassium levels are also evaluated, because these electrolytes are lost along with chloride. Arterial blood gas analysis identifies the acid–base imbalance, which is usually metabolic alkalosis. The urine chloride level, which is also measured, decreases in hypochloremia.

Medical Management

Treatment involves correcting the cause of hypochloremia and the contributing electrolyte and acid–base imbalances. Normal saline (0.9% sodium chloride) or half-strength saline (0.45% sodium chloride) solution is administered by IV to replace the chloride. If the patient is receiving a diuretic (loop, osmotic, or thiazide), it may be discontinued or another diuretic prescribed.

Ammonium chloride, an acidifying agent, may be prescribed to treat metabolic alkalosis; the dosage depends on the patient's weight and serum chloride level. This agent is metabolized by the liver, and its effects last for about 3 days. Its use should be avoided in patients with impaired liver or renal function.

Nursing Management

The nurse monitors the patient's I&O, arterial blood gas values, and serum electrolyte levels. Changes in the patient's level of consciousness and muscle strength and movement are reported to the physician promptly. Vital signs are monitored, and respiratory assessment is carried out frequently. The nurse provides and teaches the patient about foods with high chloride content. Foods high in chloride include tomato juice, bananas, dates, eggs, cheese, milk, salty broth, canned vegetables, and processed meats. A person who drinks free water (water without electrolytes) or bottled water and excretes large amounts of chloride needs instruction to avoid drinking this kind of water.

CHLORIDE EXCESS (HYPERCHLOREMIA)

Hyperchloremia exists when the serum level of chloride exceeds 107 mEq/L (107 mmol/L). Hypernatremia, bicarbonate loss, and metabolic acidosis can occur with high chloride levels.

Pathophysiology

High serum chloride levels are almost exclusively a result of iatrogenically induced hyperchloremic metabolic acidosis, stemming from excessive administration of chloride relative to sodium, most commonly as 0.9% normal saline solution, 0.45% normal saline solution, or lactated Ringer's solution (Muller & Bell, 2008). This condition can also be caused by the loss of bicarbonate ions via the kidney or the GI tract with a corresponding increase in chloride ions. Chloride ions in the form of acidifying salts accumulate, and acidosis occurs with a decrease in bicarbonate ions. Head trauma, increased perspiration, excess adrenocortical hormone production, and decreased glomerular filtration can lead to a high serum chloride level.

Clinical Manifestations

The signs and symptoms of hyperchloremia are the same as those of metabolic acidosis: hypervolemia and hypernatremia. Tachypnea; weakness; lethargy; deep, rapid respirations; diminished cognitive ability; and hypertension occur. If untreated, hyperchloremia can lead to a decrease in cardiac output, dysrhythmias, and coma. A high chloride level is accompanied by a high sodium level and fluid retention.

Assessment and Diagnostic Findings

The serum chloride level is 108 mEq/L (108 mmol/L) or greater, the serum sodium level is greater than 145 mEq/L (145 mmol/L), the serum pH is less than 7.35, and the serum bicarbonate level is less than 22 mEq/L (22 mmol/L). Urine chloride excretion increases.

Calculation of the serum anion gap is important in analyzing acid–base disorders. The sum of all negatively charged electrolytes (anions) equals the sum of all positively charged electrolytes (cations), with several anions that are not routinely measured leading to an anion gap. It is based primarily on three electrolytes: sodium, chloride, and bicarbonate or serum carbon dioxide (CO_2). A normal anion gap is 8 to 12 mEq/L (8 to 12 mmol/L). A low anion gap may be attributed to hypoproteinemia, whereas an elevated anion gap can be due to metabolic acidosis.

Medical Management

Correcting the underlying cause of hyperchloremia and restoring electrolyte, fluid, and acid–base balance are essential. Hypotonic IV solutions may be administered to restore balance. Lactated Ringer's solution may be prescribed to convert lactate to bicarbonate in the liver, which increases the bicarbonate level and corrects the acidosis. IV sodium bicarbonate may be administered to increase bicarbonate levels, which leads to the renal excretion of chloride ions because bicarbonate and chloride compete for combination with sodium. Diuretics may be administered to eliminate chloride as well. Sodium, chloride, and fluids are restricted.

Nursing Management

Monitoring vital signs, arterial blood gas values, and I&O is important to assess the patient's status and the effectiveness of treatment. Assessment findings related to respiratory, neurologic, and cardiac systems are documented, and changes are discussed with the physician. The nurse teaches the patient about the diet that should be followed to manage hyperchloremia and maintain adequate hydration.

ACID–BASE DISTURBANCES

Acid–base disturbances are commonly encountered in clinical practice. Identification of the specific acid–base imbalance is important in identifying the underlying cause of the disorder and determining appropriate treatment.

Plasma pH is an indicator of hydrogen ion (H^+) concentration. Homeostatic mechanisms keep pH within a normal range (7.35 to 7.45) (Ruholl, 2006; Swiderski & Byrum, 2007). These mechanisms consist of buffer systems, the kidneys, and the lungs. The H^+ concentration is extremely im-

portant: The greater the concentration, the more acidic the solution and the lower the pH. The lower the H^+ concentration, the more alkaline the solution and the higher the pH. The pH range compatible with life (6.8 to 7.8) represents a 10-fold difference in H^+ concentration in plasma.

Buffer systems prevent major changes in the pH of body fluids by removing or releasing H^+; they can act quickly to prevent excessive changes in H^+ concentration. Hydrogen ions are buffered by both intracellular and extracellular buffers. The body's major extracellular buffer system is the bicarbonate–carbonic acid buffer system, which is assessed when arterial blood gases are measured. Normally, there are 20 parts of bicarbonate (HCO_3^-) to one part of carbonic acid (H_2CO_3). If this ratio is altered, the pH will change. It is the ratio of HCO_3^- to H_2CO_3 that is important in maintaining pH, not absolute values. CO_2 is a potential acid; when dissolved in water, it becomes carbonic acid ($CO_2 + H_2O = H_2CO_3$). Therefore, when CO_2 is increased, the carbonic acid content is also increased, and vice versa. If either bicarbonate or carbonic acid is increased or decreased so that the 20:1 ratio is no longer maintained, acid–base imbalance results.

Less important buffer systems in the ECF include the inorganic phosphates and the plasma proteins. Intracellular buffers include proteins, organic and inorganic phosphates, and, in red blood cells, hemoglobin.

The kidneys regulate the bicarbonate level in the ECF; they can regenerate bicarbonate ions as well as reabsorb them from the renal tubular cells. In respiratory acidosis and most cases of metabolic acidosis, the kidneys excrete hydrogen ions and conserve bicarbonate ions to help restore balance. In respiratory and metabolic alkalosis, the kidneys retain hydrogen ions and excrete bicarbonate ions to help restore balance. The kidneys obviously cannot compensate for the metabolic acidosis created by renal failure. Renal compensation for imbalances is relatively slow (a matter of hours or days).

The lungs, under the control of the medulla, control the CO_2 and thus the carbonic acid content of the ECF. They do so by adjusting ventilation in response to the amount of CO_2 in the blood. A rise in the partial pressure of CO_2 in arterial blood ($PaCO_2$) is a powerful stimulant to respiration. Of course, the partial pressure of oxygen in arterial blood (PaO_2) also influences respiration. However, its effect is not as marked as that produced by the $PaCO_2$.

In metabolic acidosis, the respiratory rate increases, causing greater elimination of CO_2 (to reduce the acid load). In metabolic alkalosis, the respiratory rate decreases, causing CO_2 to be retained (to increase the acid load) (Swiderski & Byrum, 2007).

Acute and Chronic Metabolic Acidosis (Base Bicarbonate Deficit)

Metabolic acidosis is a common clinical disturbance characterized by a low pH (increased H^+ concentration) and a low plasma bicarbonate concentration. It can be produced by a gain of hydrogen ion or a loss of bicarbonate (Ruholl, 2006; Swiderski & Byrum, 2007). It can be divided clinically into two forms, according to the values of the serum anion gap: high anion gap acidosis and normal anion gap acidosis. The anion gap reflects normally unmeasured anions (phosphates, sulfates, and proteins) in plasma. Measuring the anion gap is essential in analyzing acid–base disorders correctly. The anion gap can be calculated by either one of the following equations:

$$\text{Anion gap} = Na^+ + K^+ - (Cl^- + HCO_3^-)$$
$$\text{Anion gap} = Na^+ - (Cl^- + HCO_3^-)$$

Potassium is often omitted from the equation because of its low level in the plasma; therefore, the second equation is used more often than the first.

The normal value for an anion gap is 8 to 12 mEq/L (8 to 12 mmol/L) without potassium in the equation. If potassium is included in the equation, the normal value for the anion gap is 12 to 16 mEq/L (12 to 16 mmol/L). The unmeasured anions in the serum normally account for less than 16 mEq/L of the anion production. An anion gap greater than 16 mEq (16 mmol/L) suggests excessive accumulation of unmeasured anions. An anion gap occurs because not all electrolytes are measured. More anions are left unmeasured than cations.

Pathophysiology

Normal anion gap acidosis results from the direct loss of bicarbonate, as in diarrhea, lower intestinal fistulas, ureterostomies, and use of diuretics; early renal insufficiency; excessive administration of chloride; and the administration of parenteral nutrition without bicarbonate or bicarbonate-producing solutes (eg, lactate). Normal anion gap acidosis is also referred to as hyperchloremic acidosis. A reduced or negative anion gap is primarily caused by hypoproteinemia. Disorders that cause a decreased or negative anion gap are rare compared to those related to an increased or high anion gap.

High anion gap acidosis results from excessive accumulation of fixed acid. If increased to 30 mEq/L (30 mmol/L) or more, then a high anion gap metabolic acidosis is present regardless of the values of pH and HCO_3^-. High ion gap occurs in ketoacidosis, lactic acidosis, the late phase of salicylate poisoning, uremia, methanol or ethylene glycol toxicity, and ketoacidosis with starvation. The hydrogen is buffered by HCO_3^-, causing the bicarbonate concentration to fall. In all of these instances, abnormally high levels of anions flood the system, increasing the anion gap above normal limits.

Clinical Manifestations

Signs and symptoms of metabolic acidosis vary with the severity of the acidosis but include headache, confusion, drowsiness, increased respiratory rate and depth, nausea, and vomiting. Peripheral vasodilation and decreased cardiac output occur when the pH drops to less than 7. Additional physical assessment findings include decreased blood pressure, cold and clammy skin, dysrhythmias, and shock. Chronic metabolic acidosis is usually seen with chronic renal failure.

Assessment and Diagnostic Findings

Arterial blood gas measurements are valuable in diagnosing metabolic acidosis. Expected blood gas changes include a low bicarbonate level (less than 22 mEq/L) and a low pH (less than 7.35) (Swiderski & Byrum, 2007). The cardinal

feature of metabolic acidosis is a decrease in the serum bicarbonate level. Hyperkalemia may accompany metabolic acidosis as a result of the shift of potassium out of the cells. Later, as the acidosis is corrected, potassium moves back into the cells and hypokalemia may occur. Hyperventilation decreases the CO_2 level as a compensatory action. Calculation of the anion gap is helpful in determining the cause of metabolic acidosis. An ECG detects dysrhythmias caused by the increased potassium.

Medical Management

Treatment is directed at correcting the metabolic imbalance (Ruholl, 2006). If the problem results from excessive intake of chloride, treatment is aimed at eliminating the source of the chloride. When necessary, bicarbonate is administered. Although hyperkalemia occurs with acidosis, hypokalemia may occur with reversal of the acidosis and subsequent movement of potassium back into the cells. Therefore, the serum potassium level is monitored closely, and hypokalemia is corrected as acidosis is reversed.

In chronic metabolic acidosis, low serum calcium levels are treated before the chronic metabolic acidosis is treated, to avoid tetany resulting from an increase in pH and a decrease in ionized calcium. Alkalizing agents may be administered. Treatment modalities may also include hemodialysis or peritoneal dialysis.

Acute and Chronic Metabolic Alkalosis (Base Bicarbonate Excess)

Metabolic alkalosis is a clinical disturbance characterized by a high pH (decreased H^+ concentration) and a high plasma bicarbonate concentration. It can be produced by a gain of bicarbonate or a loss of H^+ (Porth & Matfin, 2009).

Pathophysiology

Probably the most common cause of metabolic alkalosis is vomiting or gastric suction with loss of hydrogen and chloride ions. The disorder also occurs in pyloric stenosis, in which only gastric fluid is lost. Gastric fluid has an acid pH (usually 1 to 3), and loss of this highly acidic fluid increases the alkalinity of body fluids. Other situations predisposing to metabolic alkalosis include those associated with loss of potassium, such as diuretic therapy that promotes excretion of potassium (eg, thiazides, furosemide), and excessive adrenocorticoid hormones (as in hyperaldosteronism and Cushing's syndrome).

Hypokalemia produces alkalosis in two ways: (1) the kidneys conserve potassium, and therefore H^+ excretion increases; and (2) cellular potassium moves out of the cells into the ECF in an attempt to maintain near-normal serum levels (as potassium ions leave the cells, hydrogen ions must enter to maintain electroneutrality). Excessive alkali ingestion from antacids containing bicarbonate or from use of sodium bicarbonate during cardiopulmonary resuscitation can also cause metabolic alkalosis.

Chronic metabolic alkalosis can occur with long-term diuretic therapy (thiazides or furosemide), villous adenoma, external drainage of gastric fluids, significant potassium depletion, cystic fibrosis, and the chronic ingestion of milk and calcium carbonate.

Clinical Manifestations

Alkalosis is primarily manifested by symptoms related to decreased calcium ionization, such as tingling of the fingers and toes, dizziness, and hypertonic muscles. The ionized fraction of serum calcium decreases in alkalosis as more calcium combines with serum proteins. Because it is the ionized fraction of calcium that influences neuromuscular activity, symptoms of hypocalcemia are often the predominant symptoms of alkalosis. Respirations are depressed as a compensatory action by the lungs. Atrial tachycardia may occur. As the pH increases and hypokalemia develops, ventricular disturbances may occur. Decreased motility and paralytic ileus may also be evident.

Symptoms of chronic metabolic alkalosis are the same as for acute metabolic alkalosis, and as potassium decreases, frequent premature ventricular contractions or U waves are seen on the ECG.

Assessment and Diagnostic Findings

Evaluation of arterial blood gases reveals a pH greater than 7.45 and a serum bicarbonate concentration greater than 26 mEq/L. The $PaCO_2$ increases as the lungs attempt to compensate for the excess bicarbonate by retaining CO_2. This hypoventilation is more pronounced in semiconscious, unconscious, or debilitated patients than in alert patients. The former may develop marked hypoxemia as a result of hypoventilation. Hypokalemia may accompany metabolic alkalosis.

Urine chloride levels may help identify the cause of metabolic alkalosis if the patient's history provides inadequate information. Metabolic alkalosis is the setting in which urine chloride concentration may be a more accurate estimate of fluid volume than the urine sodium concentration. Urine chloride concentrations help to differentiate between vomiting, diuretic therapy, and excessive adrenocorticosteroid secretion as the cause of the metabolic alkalosis. In patients with vomiting or cystic fibrosis, those receiving nutritional repletion, and those receiving diuretic therapy, hypovolemia and hypochloremia produce urine chloride concentrations lower than 25 mEq/L. Signs of hypovolemia are not present, and the urine chloride concentration exceeds 40 mEq/L in patients with mineralocorticoid excess or alkali loading; these patients usually have expanded fluid volume. The urine chloride concentration should be less than 15 mEq/L when decreased chloride levels and hypovolemia occur.

Medical Management

Treatment of both acute and chronic metabolic alkalosis is aimed at correcting the underlying acid–base disorder (Gennari, 2005; Ruholl, 2006). Because of volume depletion from GI loss, the patient's fluid I&O must be monitored carefully.

Sufficient chloride must be supplied for the kidney to absorb sodium with chloride (allowing the excretion of excess bicarbonate). Treatment also includes restoring normal fluid volume by administering sodium chloride fluids (because continued volume depletion perpetuates the alkalosis). In patients with hypokalemia, potassium is adminis-

tered as KCl to replace both K^+ and Cl^- losses. H_2 receptor antagonists, such as cimetidine (Tagamet), reduce the production of gastric HCl, thereby decreasing the metabolic alkalosis associated with gastric suction. Carbonic anhydrase inhibitors are useful in treating metabolic alkalosis in patients who cannot tolerate rapid volume expansion (eg, patients with heart failure).

Acute and Chronic Respiratory Acidosis (Carbonic Acid Excess)

Respiratory acidosis is a clinical disorder in which the pH is less than 7.35 and the $PaCO_2$ is greater than 42 mm Hg. It may be either acute or chronic.

Pathophysiology

Respiratory acidosis is always due to inadequate excretion of CO_2 with inadequate ventilation, resulting in elevated plasma CO_2 concentrations and, consequently, increased levels of carbonic acid. In addition to an elevated $PaCO_2$, hypoventilation usually causes a decrease in PaO_2. Acute respiratory acidosis occurs in emergency situations, such as acute pulmonary edema, aspiration of a foreign object, atelectasis, pneumothorax, overdose of sedatives, sleep apnea, administration of oxygen to a patient with chronic hypercapnia (excessive CO_2 in the blood), severe pneumonia, and acute respiratory distress syndrome. Respiratory acidosis can also occur in diseases that impair respiratory muscles, such as muscular dystrophy, myasthenia gravis, and Guillain-Barré syndrome. Mechanical ventilation may be associated with hypercapnia if the rate of ventilation is inadequate and CO_2 retained.

Clinical Manifestations

Clinical signs in acute and chronic respiratory acidosis vary. Sudden hypercapnia (elevated $PaCO_2$) can cause increased pulse and respiratory rate, increased blood pressure, mental cloudiness, and a feeling of fullness in the head. An elevated $PaCO_2$, greater than 60 mm Hg, causes cerebrovascular vasodilation and increased cerebral blood flow. Ventricular fibrillation may be the first sign of respiratory acidosis in anesthetized patients.

If respiratory acidosis is severe, intracranial pressure may increase, resulting in papilledema and dilated conjunctival blood vessels. Hyperkalemia may result as the hydrogen concentration overwhelms the compensatory mechanisms and H^+ moves into cells, causing a shift of potassium out of the cell.

Chronic respiratory acidosis occurs with pulmonary diseases such as chronic emphysema and bronchitis, obstructive sleep apnea, and obesity. As long as the $PaCO_2$ does not exceed the body's ability to compensate, the patient will be asymptomatic. However, if the $PaCO_2$ increases rapidly, cerebral vasodilation will increase the intracranial pressure, and cyanosis and tachypnea will develop. Patients with chronic obstructive pulmonary disease (COPD) who gradually accumulate CO_2 over a prolonged period (days to months) may not develop symptoms of hypercapnia because compensatory renal changes have had time to occur.

NURSING ALERT

If the $PaCO_2$ is chronically higher than 50 mm Hg, the respiratory center becomes relatively insensitive to CO_2 as a respiratory stimulant, leaving hypoxemia as the major drive for respiration. Oxygen administration may remove the stimulus of hypoxemia, and the patient develops "carbon dioxide narcosis" unless the situation is quickly reversed. Therefore, oxygen is administered only with extreme caution.

Assessment and Diagnostic Findings

Arterial blood gas analysis reveals a pH lower than 7.35, a $PaCO_2$ greater than 42 mm Hg, and a variation in the bicarbonate level, depending on the duration of the acute respiratory acidosis. When compensation (renal retention of bicarbonate) has fully occurred, the arterial pH is within the lower limits of normal. Depending on the cause of respiratory acidosis, other diagnostic measures include monitoring of serum electrolyte levels, chest x-ray for determining any respiratory disease, and a drug screen if an overdose is suspected. An ECG to identify any cardiac involvement as a result of COPD may be indicated as well.

Medical Management

Treatment is directed at improving ventilation; exact measures vary with the cause of inadequate ventilation (Gennari, 2005). Pharmacologic agents are used as indicated. For example, bronchodilators help reduce bronchial spasm, antibiotics are used for respiratory infections, and thrombolytics or anticoagulants are used for pulmonary emboli (see Chapter 25).

Pulmonary hygiene measures are initiated, when necessary, to clear the respiratory tract of mucus and purulent drainage. Adequate hydration (2 to 3 L/day) is indicated to keep the mucous membranes moist and thereby facilitate the removal of secretions. Supplemental oxygen is administered as necessary.

Mechanical ventilation, used appropriately, may improve pulmonary ventilation. Inappropriate mechanical ventilation (eg, increased dead space, insufficient rate or volume settings, high fraction of inspired oxygen [FiO_2] with excessive CO_2 production) may cause such rapid excretion of CO_2 that the kidneys are unable to eliminate excess bicarbonate quickly enough to prevent alkalosis and seizures. For this reason, the elevated $PaCO_2$ must be decreased slowly. Placing the patient in a semi-Fowler's position facilitates expansion of the chest wall.

Treatment of chronic respiratory acidosis is the same as for acute respiratory acidosis.

Acute and Chronic Respiratory Alkalosis (Carbonic Acid Deficit)

Respiratory alkalosis is a clinical condition in which the arterial pH is greater than 7.45 and the $PaCO_2$ is less than 38 mm Hg. As with respiratory acidosis, acute and chronic conditions can occur.

Pathophysiology

Respiratory alkalosis is always caused by hyperventilation, which causes excessive "blowing off" of CO_2 and, hence, a decrease in the plasma carbonic acid concentration. Causes include extreme anxiety, hypoxemia, early phase of salicylate intoxication, gram-negative bacteremia, and inappropriate ventilator settings that do not match the patient's requirements.

Chronic respiratory alkalosis results from chronic hypocapnia, and decreased serum bicarbonate levels are the consequence. Chronic hepatic insufficiency and cerebral tumors are predisposing factors.

Clinical Manifestations

Clinical signs consist of lightheadedness due to vasoconstriction and decreased cerebral blood flow, inability to concentrate, numbness and tingling from decreased calcium ionization, tinnitus, and sometimes loss of consciousness. Cardiac effects of respiratory alkalosis include tachycardia and ventricular and atrial dysrhythmias (Heitz & Horne, 2005).

Assessment and Diagnostic Findings

Analysis of arterial blood gases assists in the diagnosis of respiratory alkalosis. In the acute state, the pH is elevated above normal as a result of a low $PaCO_2$ and a normal bicarbonate level. (The kidneys cannot alter the bicarbonate level quickly.) In the compensated state, the kidneys have had sufficient time to lower the bicarbonate level to a near-normal level. Evaluation of serum electrolytes is indicated to identify any decrease in potassium, as hydrogen is pulled out of the cells in exchange for potassium; decreased calcium, as severe alkalosis inhibits calcium ionization, resulting in carpopedal spasms and tetany; or decreased phosphate due to alkalosis, causing an increased uptake of phosphate by the cells. A toxicology screen should be performed to rule out salicylate intoxication.

Patients with chronic respiratory alkalosis are usually asymptomatic, and the diagnostic evaluation and plan of care are the same as for acute respiratory alkalosis.

Medical Management

Treatment depends on the underlying cause of respiratory alkalosis. If the cause is anxiety, the patient is instructed to breathe more slowly to allow CO_2 to accumulate or to breathe into a closed system (such as a paper bag). A sedative may be required to relieve hyperventilation in very anxious patients. Treatment of other causes of respiratory alkalosis is directed at correcting the underlying problem.

Mixed Acid–Base Disorders

Patients can simultaneously experience two or more independent acid–base disorders. A normal pH in the presence of changes in the $PaCO_2$ and plasma HCO_3^- concentration immediately suggests a mixed disorder. An example of a mixed disorder is the simultaneous occurrence of metabolic acidosis and respiratory acidosis during respiratory and cardiac arrest. The only mixed disorder that cannot occur is a mixed respiratory acidosis and alkalosis, because it is impossible to have alveolar hypoventilation and hyperventilation at the same time.

Compensation

Generally, the pulmonary and renal systems compensate for each other to return the pH to normal. In a single acid–base disorder, the system not causing the problem tries to compensate by returning the ratio of bicarbonate to carbonic acid to the normal 20:1. The lungs compensate for metabolic disturbances by changing CO_2 excretion. The kidneys compensate for respiratory disturbances by altering bicarbonate retention and H^+ secretion.

In respiratory acidosis, excess hydrogen is excreted in the urine in exchange for bicarbonate ions. In respiratory alkalosis, the renal excretion of bicarbonate increases, and hydrogen ions are retained. In metabolic acidosis, the compensatory mechanisms increase the ventilation rate and the renal retention of bicarbonate. In metabolic alkalosis, the respiratory system compensates by decreasing ventilation to conserve CO_2 and increase the $PaCO_2$. Because the lungs respond to acid–base disorders within minutes, compensation for metabolic imbalances occurs faster than compensation for respiratory imbalances. Table 14-5 summarizes compensation effects.

Blood Gas Analysis

Blood gas analysis is often used to identify the specific acid–base disturbance and the degree of compensation that has occurred. The analysis is usually based on an arterial blood sample, but if an arterial sample cannot be obtained, a mixed venous sample may be used. Results of arterial blood gas analysis provide information about alveolar ventilation, oxygenation, and acid–base balance. It is necessary to evaluate the concentrations of serum electrolytes (sodium, potassium, and chloride) and carbon dioxide along with arterial blood gas data, because they are often the first sign of an acid–base disorder. The health history, physical examination, previous blood gas results, and serum electrolytes should always be part of the assessment used to determine the cause of the acid–base disorder (Porth & Matfin, 2009). Responding to isolated sets of blood gas results without these data can lead to serious errors in interpretation. Treatment

Table 14-5 ACID–BASE DISORDERS AND COMPENSATION

Disorder	Initial Event	Compensation
Respiratory acidosis	↓ pH, ↑ or normal HCO_3^-, ↑ $PaCO_2$	↑ Renal acid excretion and ↑ serum HCO_3^-
Respiratory alkalosis	↑ pH, ↓ or normal HCO_3^-, ↓ $PaCO_2$	↓ Renal acid excretion and ↓ serum HCO_3^-
Metabolic acidosis	↓ pH, ↓ HCO_3^-, ↓ or normal $PaCO_2$	Hyperventilation with resulting ↓ PaCO (conserves HCO_3^-)
Metabolic alkalosis	↑ pH, ↑ HCO_3^-, ↑ or normal $PaCO_2$	Hypoventilation with resulting ↑ $PaCO_2$

Table 14-6 NORMAL VALUES FOR ARTERIAL AND MIXED VENOUS BLOOD

Parameter	Arterial Blood	Mixed Venous Blood
pH	7.35–7.45	7.32–7.42
$PaCO_2$	35–45 mm Hg	38–52 mm Hg
PaO_2*	70–100 mm Hg	24–48 mm Hg
HCO_3^-	19–25 mEq/L	19–25 mEq/L
Base excess/deficit	±5 mEq/L	±5 mEq/L
Oxygen saturation	>90–95%	40–70%

*At altitudes of 3000 feet and higher, the values for oxygen are decreased.

of the underlying condition usually corrects most acid–base disorders. Table 14-6 compares normal ranges of venous and arterial blood gas values. See also Chart 14-2.

Parenteral Fluid Therapy

When no other route of administration is available, fluids are administered by IV in hospitals, outpatient diagnostic and surgical settings, clinics, and homes to replace fluids, administer medications, and provide nutrients.

Purpose

The choice of an IV solution depends on the purpose of its administration. Generally, IV fluids are administered to achieve one or more of the following goals:

- To provide water, electrolytes, and nutrients to meet daily requirements
- To replace water and correct electrolyte deficits
- To administer medications and blood products

IV solutions contain dextrose or electrolytes mixed in various proportions with water. Pure, electrolyte-free water can never be administered by IV because it rapidly enters red blood cells and causes them to rupture.

Types of Intravenous Solutions

Solutions are often categorized as **isotonic, hypotonic,** or **hypertonic,** according to whether their total osmolality is the same as, less than, or greater than that of blood, respec-

CHART 14-2 *Assessing for Arterial Blood Gases*

The following steps are recommended to evaluate arterial blood gas values. They are based on the assumption that the average values are:

$pH = 7.4$
$PaCO_2 = 40$ mm Hg
$HCO_3^- = 24$ mEq/L

1. *First, note the pH.* It can be high, low, or normal, as follows:
   pH >7.4 (alkalosis)
   pH <7.4 (acidosis)
   pH = 7.4 (normal)
   A normal pH may indicate perfectly normal blood gases, *or* it may be an indication of a *compensated* imbalance. A compensated imbalance is one in which the body has been able to correct the pH by either respiratory or metabolic changes (depending on the primary problem). For example, a patient with primary metabolic acidosis starts out with a low bicarbonate level but a normal CO_2 level. Soon afterward, the lungs try to compensate for the imbalance by exhaling large amounts of CO_2 (hyperventilation). As another example, a patient with primary respiratory acidosis starts out with a high CO_2 level; soon afterward, the kidneys attempt to compensate by retaining bicarbonate. If the compensatory mechanism is able to restore the bicarbonate–to–carbonic acid ratio back to 20:1, full compensation (and thus normal pH) will be achieved.
2. The next step is to determine the primary cause of the disturbance. This is done by evaluating the $PaCO_2$ and HCO_3^- in relation to the pH.
   Example: pH >7.4 (alkalosis)
   a. If the $PaCO_2$ is <40 mm Hg, the primary disturbance is respiratory alkalosis. (This situation occurs when a patient hyperventilates and "blows off" too much CO_2. Recall that CO_2 dissolved in water becomes carbonic acid, the acid side of the "carbonic acid–bicarbonate buffer system.")
   b. If the HCO_3^- is >24 mEq/L, the primary disturbance is metabolic alkalosis. (This situation occurs when the body gains too much bicarbonate, an alkaline substance. Bicarbonate is the basic or alkaline side of the "carbonic acid–bicarbonate buffer system.")
   Example: pH <7.4 (acidosis)
   a. If the $PaCO_2$ is >40 mm Hg, the primary disturbance is respiratory acidosis. (This situation occurs when a patient hypoventilates and thus retains too much CO_2, an acidic substance.)
   b. If the HCO_3^- is <24 mEq/L, the primary disturbance is metabolic acidosis. (This situation occurs when the body's bicarbonate level drops, either because of direct bicarbonate loss or because of gains of acids such as lactic acid or ketones.)
3. The next step involves determining if compensation has begun. This is done by looking at the value other than the primary disorder. If it is moving in the same direction as the primary value, compensation is under way. Consider the following gases:

	pH	$PaCO_2$	HCO_3^-
(1)	7.2	60 mm Hg	24 mEq/L
(2)	7.4	60 mm Hg	37 mEq/L

   The first set (1) indicates acute respiratory acidosis without compensation (the $PaCO_2$ is high, the HCO_3^- is normal). The second set (2) indicates chronic respiratory acidosis. Note that compensation has taken place; that is, the HCO_3^- has elevated to an appropriate level to balance the high $PaCO_2$ and produce a normal pH.
4. Two distinct acid–base disturbances may occur simultaneously. These can be identified when the pH does not explain one of the changes.
   Example: Metabolic and respiratory acidosis

a. pH	7.2	decreased acid
b. $PaCO_2$	52	increased acid
c. HCO_3	13	decreased acid

5. Evaluate the patient to determine if the clinical signs and symptoms are compatible with acid–base analysis.

tively (see earlier discussion of osmolality). Electrolyte solutions are considered isotonic if the total electrolyte content (anions + cations) is approximately 310 mEq/L, hypotonic if the total electrolyte content is less than 250 mEq/L, and hypertonic if the total electrolyte content is greater than 375 mEq/L. The nurse must also consider a solution's osmolality, keeping in mind that the osmolality of plasma is approximately 300 mOsm/L (300 mmol/L). For example, a 10% dextrose solution has an osmolality of approximately 505 mOsm/L.

Isotonic Fluids

Fluids that are classified as isotonic have a total osmolality close to that of the ECF and do not cause red blood cells to shrink or swell. The composition of these fluids may or may not approximate that of the ECF. Isotonic fluids expand the ECF volume. One liter of isotonic fluid expands the ECF by 1 L; however, it expands the plasma by only 0.25 L because it is a crystalloid fluid and diffuses quickly into the ECF compartment. For the same reason, 3 L of isotonic fluid is needed to replace 1 L of blood loss. Because these fluids expand the intravascular space, patients with hypertension and heart failure should be carefully monitored for signs of fluid overload.

D_5W

A solution of D_5W has a serum osmolality of 252 mOsm/L. Once administered, the glucose is rapidly metabolized, and this initially isotonic solution then disperses as a hypotonic fluid, one-third extracellular and two-thirds intracellular. It is essential to consider this action of D_5W, especially if the patient is at risk for increased intracranial pressure. During fluid resuscitation, this solution should not be used, because hyperglycemia can result. Therefore, D_5W is used mainly to supply water and to correct an increased serum osmolality. About 1 L of D_5W provides fewer than 200 kcal and is a minor source of the body's daily caloric requirements.

Normal Saline Solution

Normal saline (0.9% sodium chloride) solution has a total osmolality of 308 mOsm/L. Because the osmolality is entirely contributed by electrolytes, the solution remains within the ECF. For this reason, normal saline solution is often used to correct an extracellular volume deficit. Although referred to as "normal," it contains only sodium and chloride and is not identical to ECF. It is used with administration of blood transfusions and to replace large sodium losses, such as in burn injuries. It is not used for heart failure, pulmonary edema, renal impairment, or sodium retention. Normal saline does not supply calories.

Other Isotonic Solutions

Several other solutions contain ions in addition to sodium and chloride and are somewhat similar to the ECF in composition. Lactated Ringer's solution contains potassium and calcium in addition to sodium chloride. It is used to correct dehydration and sodium depletion and replace GI losses. Lactated Ringer's solution contains bicarbonate precursors as well. These solutions are marketed, with slight variations, under various trade names.

Hypotonic Fluids

One purpose of hypotonic solutions is to replace cellular fluid, because it is hypotonic compared with plasma. Another is to provide free water for excretion of body wastes. At times, hypotonic sodium solutions are used to treat hypernatremia and other hyperosmolar conditions. Half-strength saline (0.45% sodium chloride) solution, with an osmolality of 154 mOsm/L, is frequently used. Multiple-electrolyte solutions are also available. Excessive infusions of hypotonic solutions can lead to intravascular fluid depletion, decreased blood pressure, cellular edema, and cell damage. These solutions exert less osmotic pressure than the ECF.

Hypertonic Fluids

When normal saline solution or lactated Ringer's solution contains 5% dextrose, the total osmolality exceeds that of the ECF. However, the dextrose is quickly metabolized, and only the isotonic solution remains. Therefore, any effect on the intracellular compartment is temporary. Similarly, with hypotonic multiple-electrolyte solutions containing 5% dextrose, once the dextrose is metabolized, these solutions disperse as hypotonic fluids. Higher concentrations of dextrose, such as 50% dextrose in water, are strongly hypertonic and must be administered into central veins so that they can be diluted by rapid blood flow.

Saline solutions are also available in osmolar concentrations greater than that of the ECF. These solutions draw water from the ICF to the ECF and cause cells to shrink. If administered rapidly or in large quantity, they may cause an extracellular volume excess and precipitate circulatory overload and dehydration. As a result, these solutions must be administered cautiously and usually only when the serum osmolality has decreased to dangerously low levels. Hypertonic solutions exert an osmotic pressure greater than that of the ECF.

Other Intravenous Substances

When the patient is unable to tolerate food, nutritional requirements are often met using the IV route. Solutions may include high concentrations of glucose (such as 50% dextrose in water), protein, or fat to meet nutritional requirements (see Chapter 36). The IV route may also be used to administer colloids, plasma expanders, and blood products. Examples of blood products include whole blood, packed red blood cells, albumin, and cryoprecipitate; these are discussed in more detail in Chapter 33.

Many medications are also delivered by the IV route, either by continuous infusion or intermittent bolus directly into the vein. Because IV medications enter the circulation rapidly, administration by this route is potentially very hazardous. All medications can produce adverse reactions; however, medications administered by the IV route can cause these reactions within seconds to minutes after administration, because the medications are delivered directly into the bloodstream. Administration rates and recommended dilutions for individual medications are available in specialized texts pertaining to IV medications and in manufacturers' package inserts; these should be consulted to ensure safe IV administration of medications.

NURSING ALERT

The nurse must assess the patient for a history of allergic reactions to medications. Although this is important when any medication is to be administered, it is even more important with IV administration, because the medication is delivered directly into the bloodstream.

Nursing Management of the Patient Receiving Intravenous Therapy

The ability to perform venipuncture to gain access to the venous system for administering fluids and medication is an expected nursing skill in many settings. This responsibility includes selecting the appropriate venipuncture site and type of cannula and being proficient in the technique of vein entry. The nurse should demonstrate competency in and knowledge of catheter placement according to the Nurse Practice Act applicable in his or her state and should follow the rules and regulations, organizational policies and procedures, and practice guidelines of that state's board of nursing (Alexander, 2006).

Infusion therapy is initiated by a health care provider who prescribes the type and amount of solution, additives (if any), and rate of flow. When administering parenteral fluids, the nurse monitors the patient's response to the fluids, considering the fluid volume, the fluid content, and the patient's clinical status.

Preparing to Administer Intravenous Therapy

Before performing venipuncture, the nurse carries out hand hygiene, applies gloves, and informs the patient about the procedure. The nurse selects the most appropriate insertion site and type of cannula for a particular patient.

Choosing an Intravenous Site

Many sites can be used for IV therapy, but ease of access and potential hazards vary. Veins of the extremities are designated as peripheral locations and are ordinarily the only sites used by nurses. Because they are relatively safe and easy to enter, arm veins are most commonly used (Fig. 14-7). The metacarpal, cephalic, basilic, and median veins and their branches are recommended sites because of their size and ease of access. Leg veins should rarely, if ever, be used because of the high risk of thromboembolism. Additional sites to avoid include veins distal to a previous IV infiltration or phlebitic area, sclerosed or thrombosed veins, an arm with an arteriovenous shunt or fistula, and an arm affected by edema, infection, blood clot, deformity, severe scarring, or skin breakdown. The arm on the side of a mastectomy is avoided because of impaired lymphatic flow.

Central veins commonly used by physicians include the subclavian and internal jugular veins. It is possible to gain access to (or cannulate) these larger vessels even when peripheral sites have collapsed, and they allow for the administration of hyperosmolar solutions. However, the potential hazards are much greater and include inadvertent entry into an artery or the pleural space.

Ideally, both arms and hands are carefully inspected before a specific venipuncture site that does not interfere with mobility is chosen. For this reason, the antecubital fossa is avoided, except as a last resort. The most distal site of the arm or hand is generally used first, so that subsequent IV access sites can be moved progressively upward. The following factors should be considered when selecting a site for venipuncture:

- Condition of the vein
- Type of fluid or medication to be infused
- Duration of therapy
- Patient's age and size
- Whether the patient is right- or left-handed
- Patient's medical history and current health status
- Skill of the person performing the venipuncture

After applying a tourniquet, the nurse palpates and inspects the vein. The vein should feel firm, elastic, engorged, and round—not hard, flat, or bumpy. Because arteries lie close to veins in the antecubital fossa, the vessel should be palpated for arterial pulsation (even with a tourniquet on), and cannulation of pulsating vessels should be avoided. General guidelines for selecting a cannula include the following:

- Length: 0.75 to 1.25 inches long
- Diameter: narrow diameter of the cannula to occupy minimal space within the vein
- Gauge:
  - 20 to 22 gauge for most IV fluids; a larger gauge for caustic or viscous solutions
  - 14 to 18 gauge for blood administration and for trauma patients and those undergoing surgery
  - 22 to 24 gauge for elderly patients

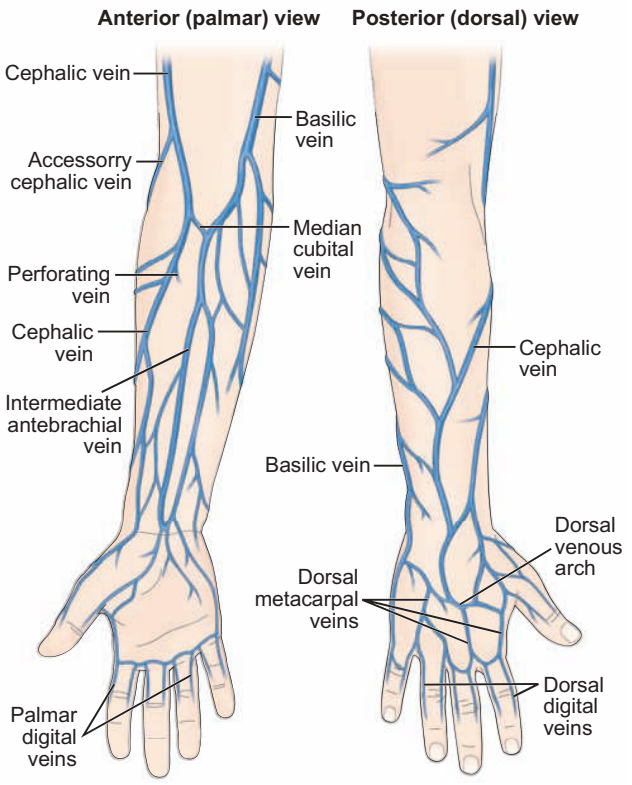

Figure 14-7 Site selection for peripheral cannulation of veins: anterior (palmar) veins at left, posterior (dorsal) veins at right. Adapted from Agur, A. M. R., Lee, M. J. & Boileau Grant, M. J. (1999). *Grant's atlas of anatomy* (10th ed.). Philadelphia: Lippincott Williams & Wilkins.

Hand veins are easiest to cannulate. Cannula tips should not rest in a flexion area (eg, the antecubital fossa), because this could inhibit the IV flow (Hadaway & Millam, 2007).

Selecting Venipuncture Devices

Equipment used to gain access to the vasculature includes cannulas, needleless IV delivery systems, and peripherally inserted central catheter (PICC) or midline catheter vascular access devices.

Cannulas

Most peripheral access devices are cannulas. They have an obturator inside a tube that is later removed. *Catheter* and *cannula* are terms that are used interchangeably. The main types of cannula devices available are those referred to as winged infusion sets (butterfly) with a steel needle or as over-the-needle catheters with wings; indwelling plastic cannulas that are inserted over a steel needle; and indwelling plastic cannulas that are inserted through a steel needle. Scalp vein or butterfly needles are short steel needles with plastic wing handles. These are easy to insert, but because they are small and nonpliable, infiltration occurs easily. The use of these needles should be limited to obtaining blood specimens or administering bolus injections or infusions lasting only a few hours, because they increase the risk of vein injury and infiltration. Insertion of an over-the-needle catheter requires the additional step of advancing the catheter into the vein after venipuncture. Because these devices are less likely to cause infiltration, they are frequently preferred over winged infusion sets.

Plastic cannulas inserted through a hollow needle are usually called intracatheters. They are available in long lengths and are well suited for placement in central locations. Because insertion requires threading the cannula through the vein for a relatively long distance, these can be difficult to insert. The most commonly used infusion device is the over-the-needle catheter. A hollow metal stylet is preinserted into the catheter and extends through the distal tip of the catheter to allow puncture of the vessel, in an effort to guide the catheter as the venipuncture is performed. The vein is punctured and a flashback of blood appears in the closed chamber behind the catheter hub. The catheter is threaded through the stylet into the vein and the stylet is then removed.

To select the ideal product for use, consideration should be given to which product provides the greatest patient satisfaction and offers quality, cost-effective infusion care. All devices should be radiopaque to determine catheter location by x-ray, if necessary. All catheters increase the risk of thrombus formation to varying degrees. Biocompatibility, another characteristic of a catheter, ensures that inflammation and irritation do not occur. Silicone catheters are the most bioinert catheters available today.

Needleless Intravenous Delivery Systems. In an effort to decrease needlestick injuries and exposure to bloodborne pathogens, the federal government has legislated and agencies have implemented needleless IV delivery systems. These systems have built-in protection against needlestick injuries and provide a safe means of using and disposing of an IV administration set (which consists of tubing, an area for inserting the tubing into the container of IV fluid, and an adapter for connecting the tubing to the needle). Numerous companies produce needleless components. IV line connectors allow the simultaneous infusion of IV medications and other intermittent medications (known as a piggyback delivery) without the use of needles; this method is being used more frequently, moving away from use of the traditional stylet. An example is a self-sheathing stylet that is recessed into a rigid chamber at the hub of the catheter when its insertion is complete. Other designs have placed the stylet at the end of a flexible wire to avoid needlesticks.

Many types of these devices are on the market. Each institution must evaluate products to determine its own needs based on Occupational Safety and Health Administration (OSHA) guidelines and the institution's policies and procedures.

Peripherally Inserted Central Catheter or Midline Catheter Access Lines. Patients who need moderate- to long-term parenteral therapy often receive a PICC or a midline catheter. These catheters are also used for patients with limited peripheral access (eg, obese or emaciated patients, IV/injection drug users) who require IV antibiotics, blood, and parenteral nutrition. For these devices to be used, the veins must be pliable (not sclerosed or hardened) and not subject to repeated puncture. If these veins are damaged, then central venous access via the subclavian or internal jugular vein, or surgical placement of an implanted port or a vascular access device, must be considered as an alternative (Anderson, 2005; Ludeman, 2007). Table 14-7 compares PICC and midline catheters. Both PICC and midline catheters have the advantages of reducing cost, avoiding repeated venipunctures, and decreasing the incidence of catheter-related infections when compared with centrally placed catheters.

The principles for inserting these lines are much the same as those for inserting peripheral catheters; however, their insertion should be undertaken only by practitioners who are experienced and specially skilled in inserting IV lines.

The physician prescribes the line and the solution to be infused. Insertion of either catheter requires sterile technique. The size of the catheter lumen chosen is based on the type of solution, the patient's body size, and the vein to be used. The patient's consent is obtained before use of these catheters. Use of the dominant arm is recommended as the site for inserting the cannula into the superior vena cava to ensure adequate arm movement, which encourages blood flow and reduces the risk of dependent edema.

Teaching the Patient

Except in emergency situations, the patient should be prepared in advance for an IV infusion. The venipuncture, the expected length of infusion, and activity restrictions are explained. If the patient requires alternative formats (eg, interpreter, large-print written materials) to understand the procedure, these should be provided. Then the patient should have an opportunity to ask questions and express concerns. For example, some patients believe that they will die if small bubbles in the tubing enter their veins.

Table 14-7 COMPARISON OF PERIPHERALLY INSERTED CENTRAL AND MIDLINE CATHETERS

	Peripherally Inserted Central Catheter (PICC)	Midline Catheter
Indications	Parenteral nutrition; IV fluid replacement; administration of chemotherapy agents, analgesics, and antibiotics; removal of blood specimens; administration of blood products	Parenteral nutrition; IV fluid replacement; administration of analgesics and antibiotics (no solution or medications with a pH <5 or >9 or osmolarity >500 mOsm/L); removal of blood specimens
Features	Single-, double-, and triple-lumen catheters available (16–24 gauge) 50–70-cm in length; sizes range from 2–7 Fr	Single- and double-lumen catheters available (16–24 gauge) 7.5–20-cm in length; catheter can increase two gauges in size as it softens
Material	Radiopaque, polymer (polyurethane), silastic materials; flexible	Silicone, polyurethane, and their derivatives; available impregnated with heparin to ↓ thrombogenicity (radiopaque or clear, with radiopaque strip)
Insertion sites	Venipuncture performed in the antecubital fossa, above or below it into the basilic, cephalic, or axillary veins of the dominant arm. Median basilic is ideal insertion site.	Venipuncture performed 112 inches above or below antecubital fossa through brachial cephalic, basilic, or median cubital vein.
Catheter placement	Tip of catheter lies in lower third of superior vena cava. Catheter is placed via median basilic, median cubital, or median cephalic vein at antecubital fossa.	Catheter lies between antecubital area and head of clavicle (tip in axilla region). Tip terminates in the proximal portion of extremity below axilla and proximal to central veins and is advanced 3–10 inches.
Insertion method	Sedation and NPO not required. Through-the-needle technique, with or without a guidewire, breakaway needle with introducer or cannula with introducer (peel-away sheath). (A peripherally inserted central catheter can also be used as midline catheter.) Insertion can be accomplished at bedside using sterile technique. Right arm placement is a more direct route to vena cava. Arm to be used should be positioned in abduction to 90-degree angle. Consent is required. Ultrasound-guided placement can allow access to difficult veins at bedside or in x-ray department with fluoroscopy. Catheter may stay in place for up to 12 months.	No separate guidewire or introducer needed. Stiff catheter is passed using catheter advancement tab. Insertion can be accomplished at bedside using sterile technique. Arm to be used should be positioned in abduction to 45-degree angle. Consent is required. The catheter should never be reused. Catheter may stay in place for 2–4 weeks.
Potential complications	Malposition, pneumothorax, hemothorax, hydrothorax, dysrhythmias, nerve or tendon damage, respiratory distress, catheter embolism, thrombophlebitis, or catheter occlusion. Compared with centrally placed catheters, venipuncture in antecubital space reduces risk of insertion complications.	Thrombosis, phlebitis, air embolism, infection, vascular perforation, bleeding, catheter transection, occlusion
Contraindications	Dermatitis, cellulitis, lymphedema, compromised anatomy, burns, high fluid volume infusions, rapid bolus injections, hemodialysis, and venous thrombosis. No clamping of this catheter or splinting of arm permitted. No blood pressure or tourniquets to be used on extremity where peripherally inserted central catheter is inserted.	Dermatitis, cellulitis, burns, high fluid volume infusions, rapid bolus injection, hemodialysis, and venous thrombosis. No blood pressure or tourniquet to be used on extremity where catheter is placed. Patient should avoid heavy lifting with arm that has catheter.
Catheter maintenance	Sterile dressing types and changes are according to agency protocol, training, and competency requirements. Catheter is secured with stabilization device.	Sterile dressing types and changes are according to agency protocol, training, and competency requirements. Catheter must be anchored securely to prevent its dislodgment and can be secured with stabilization device.
Postplacement	Chest x-ray needed to confirm placement of catheter tip	Chest x-ray to assess placement may be obtained if unable to flush catheter, if no free flow blood return, if difficulty with catheter advancement, if guidewire is difficult to remove or bent on removal, or catheter migration is suspected.
Assessment	Daily measurement of arm circumference (4 inches above insertion site) and length of exposed catheter	Daily measurement of arm circumference (4 inches above insertion site) and length of exposed catheter
Removal	Catheter should be removed when no longer indicated for use, if contaminated, or if complications occur. Arm is abducted during removal. Patient should be in dorsal recumbent position with head of bed flat and should perform Valsalva maneuver while catheter is withdrawn. Pressure is applied on removal with sterile dressing and antiseptic ointment to site. Dressing is changed every 24–48 h until epithelialization occurs.	Catheter should be removed when no longer indicated for use, if contaminated, or if complications occur. Arm is abducted during removal. Pull gently from insertion site no more than 1/4–1/2 inch at a time to prevent vasospasm. Pressure is applied on removal with a sterile dressing and antiseptic ointment to site. Dressing is changed every 24–48 h until epithelialization occurs.

After acknowledging this fear, the nurse can explain that usually only relatively large volumes of air administered rapidly are dangerous.

Preparing the Intravenous Site

Before preparing the skin, the nurse should ask the patient if he or she is allergic to latex or iodine, products commonly used in preparing the skin for IV therapy. Excessive hair at the selected site may be removed by clipping to increase the visibility of the veins and to facilitate insertion of the cannula and adherence of dressings to the IV insertion site. Because infection can be a major complication of IV therapy, the IV device, the fluid, the container, and the tubing must be sterile. The nurse must perform hand hygiene and put on gloves. Gloves (nonsterile, disposable) must be worn during the venipuncture procedure because of the likelihood of coming into contact with bloodborne pathogens. The insertion site is prepared according to institutional policy (Rosenthal, 2007; Todd, 2006).

Performing Venipuncture

Guidelines and a suggested sequence for venipuncture are presented in Chart 14-3. For veins that are very small or particularly fragile, modifications in the technique may be necessary. Alternative methods can be found in journal articles or in specialized textbooks on IV therapy. Institutional policies and procedures determine whether all nurses must be certified to perform venipuncture. A nurse certified in IV therapy or an IV team can be consulted to assist with initiating IV therapy. To avoid multiple unsuccessful attempts, causing unnecessary trauma to the patient and limiting future vascular access, no more than two attempts at cannulation by any one nurse should be made (Alexander, 2006).

Maintaining Therapy

Maintaining an existing IV infusion is a nursing responsibility that demands knowledge of the solutions being administered and the principles of flow. In addition, patients must be assessed carefully for both local and systemic complications.

Factors Affecting Flow

The flow of an IV infusion is governed by the same principles that govern fluid movement in general:

- Flow is directly proportional to the height of the liquid column. Raising the height of the infusion container may improve a sluggish flow.
- Flow is directly proportional to the diameter of the tubing. The clamp on IV tubing regulates the flow by changing the tubing diameter. In addition, the flow is faster through large-gauge rather than small-gauge cannulas.
- Flow is inversely proportional to the length of the tubing. Adding extension tubing to an IV line decreases the flow.
- Flow is inversely proportional to the viscosity of a fluid. Viscous IV solutions, such as blood, require a larger cannula than do water or saline solutions.

Monitoring Flow

Because so many factors influence an IV set to gravity flow, a solution does not necessarily continue to run at the speed originally set. Therefore, the nurse monitors IV infusions frequently to make sure that the fluid is flowing at the intended rate. The IV container should be marked to indicate at a glance whether the correct amount has infused. The flow rate is calculated when the solution is originally started and then monitored at least hourly. To calculate the flow rate, the nurse determines the number of drops delivered per milliliter; this varies with equipment and is usually printed on the administration set packaging. A formula that can be used to calculate the drop rate is

$$\text{gtt/mL of infusion set/60 (min in 1 hr)} \times \text{total hourly volume} = \text{gtt/min}$$

Flushing of a vascular device is performed to ensure patency and to prevent the mixing of incompatible medications or solutions. This procedure should be carried out at established intervals, according to hospital policy and procedure, especially for intermittently used catheters. Most manufacturers and researchers suggest the use of preservative-free 0.9% sodium chloride for flushing (Alexander, 2006). The volume of the flush solution should be at least twice the volume capacity of the catheter. The catheter should be clamped before the syringe is completely empty and withdrawn to prevent reflux of blood into the lumen, which could cause catheter clotting.

A variety of electronic infusion devices are available to assist in IV fluid delivery. These devices allow more accurate administration of fluids and medications than is possible with routine gravity-flow setups. A pump is a positive-pressure device that uses pressure to infuse fluid at a pressure of 10 psi; newer models use a pressure of 5 psi. The pressure exerted by the pump overrides vascular resistance (increased tubing length, low height of the IV container).

Volumetric pumps calculate the volume delivered by measuring the volume in a reservoir that is part of the set and is calibrated in milliliters per hour (mL/h). A controller is an infusion assist device that relies on gravity for infusion; the volume is calibrated in drops (gtt) per minute. A controller uses a drop sensor to monitor the flow. Factors essential for the safe use of pumps include alarms to signify the presence of air in the IV line or an occlusion. The standard for the accurate delivery of fluid or medication via an electronic IV infusion pump is plus or minus 5%. The manufacturer's directions must be read carefully before use of any infusion pump or controller, because there are many variations in available models. Use of these devices does not eliminate the need for the nurse to monitor the infusion and the patient frequently. The nurse must be knowledgeable about flow control devices and competent regarding their use.

Discontinuing an Infusion

IV therapy should be discontinued as prescribed by an appropriate health care provider or on assessment by the nurse that contamination, phlebitis, or infiltration has occurred. The removal of an IV catheter is associated with two possible dangers: bleeding and catheter embolism. To prevent excessive bleeding, a dry, sterile pressure dressing should be held over the site as the catheter is removed. Firm pressure is applied until bleeding stops.

If a plastic IV catheter is severed, the loose fragment can travel to the right ventricle and block blood flow. To detect

CHART 14-3

Guidelines for Starting an Intravenous Infusion

Equipment

- Tourniquet
- Tape
- IV solution, tubing and catheter
- Chlorhexidine gluconate, povidone–iodine, or alcohol swabs
- Nonlatex gloves
- Transparent dressing, bandage, or sterile gauze
- Padded, appropriate-length arm board

Implementation

Nursing Action	Rationale
1. Verify prescription for IV therapy, check solution label, and identify patient. Check for allergies (ie, latex, iodine).	1. Serious errors can be avoided by careful checking. Checking for allergies reduces risk of allergic reaction.
2. Explain procedure to patient.	2. Knowledge increases patient comfort and cooperation.
3. Perform hand hygiene and put on disposable nonlatex gloves.	3. Asepsis is essential to prevent infection. Use of nonlatex gloves prevents exposure of nurse to patient's blood and of patient and nurse to latex.
4. Apply a tourniquet 4 to 6 inches above the site and identify a suitable vein.	4. This will distend the veins and allow them to be visualized.
5. Choose site. Use distal veins of hands and arms first.	5. Careful site selection will increase likelihood of successful venipuncture and preservation of vein. Using distal sites first preserves sites proximal to the previously cannulated site for subsequent venipunctures. Veins of feet and lower extremity should be avoided due to risk of thrombophlebitis. (In consultation with the physician, the saphenous vein of the ankle or dorsum of the foot may occasionally be used.)
6. Choose IV cannula or catheter.	6. Length and gauge of cannula should be appropriate for both site and purpose of infusion. The shortest gauge and length needed to deliver prescribed therapy should be used. Inspect the needle or cannula to make sure there are no imperfections.
7. Prepare equipment by connecting infusion bag and tubing, run solution through tubing to displace air, and cover end of tubing.	7. This prevents delay; equipment must be ready to connect immediately after successful venipuncture to prevent clotting.
8. Raise bed to comfortable working height and position for patient; adjust lighting. Position patient's arm below heart level to encourage capillary filling. Place protective pad on bed under patient's arm.	8. Proper positioning will increase likelihood of success and provide comfort for patient.
9. Depending on agency policy and procedure, lidocaine 1% (without epinephrine) 0.1–0.2 mL may be injected locally to the IV site or a transdermal analgesic cream (EMLA) may be applied to the site prior to IV placement or blood withdrawal. Alternatively, topical application of lidocaine (Numby Stuff) or an intradermal injection of bacteriostatic 0.9% sodium chloride may be used to produce a local anesthetic effect.	9. This reduces pain locally from procedure and decreases anxiety about pain.
10. Palpate for a pulse distal to the tourniquet. Ask patient to open and close fist several times or position patient's arm in a dependent position to distend a vein.	10. The tourniquet should never be tight enough to occlude arterial flow. If a radial pulse cannot be palpated distal to the tourniquet, it is too tight. A new tourniquet should be used for each patient to prevent the transmission of microorganisms. A blood pressure cuff may be used for elderly patients to avoid rupture of the veins. A clenched fist encourages the vein to become round and turgid. Positioning the arm below the level of the patient's heart promotes capillary filling. Warm packs applied for 10 to 20 minutes prior to venipuncture can promote vasodilation. Bedside ultrasound-guided visualization of vein location and assessment of venous pathway and flow using ultrasonic waves may also be used.

Continued

CHART 14-3

Guidelines for Starting an Intravenous Infusion (Continued)

Nursing Action	Rationale
11. Prepare site by scrubbing with chlorhexidine gluconate or povidone–iodine swabs for 2 to 3 minutes in circular motion, moving outward from injection site. Allow to dry. a. If the site selected is excessively hairy, clip hair. (Check agency's policy and procedure about this practice.) b. Isopropyl alcohol 70% is an alternative solution that may be used.	11. Strict asepsis and careful site preparation are essential to prevent infection. a. Hair removal should be performed with scissors or electric clippers. Shaving should not be done with a razor because of the potential for microabrasions that increase the risk of infection. Depilatories should not be used due to the potential for dermal allergic reactions and/or irritation.
12. With hand not holding the venous access device, steady patient's arm and use finger or thumb to pull skin taut over vessel.	12. Applying traction to the vein helps to stabilize it.
13. Holding needle bevel up and at 5- to 25-degree angle, depending on the depth of the vein, pierce skin to reach but not penetrate vein.	13. Bevel-down technique is necessary for small veins to prevent extravasation. One-step method of catheter insertion directly into vein with immediate thrust through the skin is excellent for large veins but may cause a hematoma if used in small veins.
14. Decrease angle of needle further until nearly parallel with skin, then enter vein either directly above or from the side in one quick motion.	14. Two-stage procedure decreases chance of thrusting needle through posterior wall of vein as skin is entered. No attempt should be made to reinsert the stylet because of risk of severing or puncturing the catheter.
15. If backflow of blood is visible, straighten angle and advance needle. Additional steps for catheter inserted over needle: a. Advance needle 0.6 cm (1/4 to 1/2 inch) after successful venipuncture. b. Hold needle hub, and slide catheter over the needle into the vein. Never reinsert needle into a plastic catheter or pull the catheter back into the needle. c. Remove needle while pressing lightly on the skin over the catheter tip; hold catheter hub in place. d. Never reinsert a stylet back into a catheter. e. Never reuse the same catheter.	15. Backflow may not occur if vein is small; this position decreases chance of puncturing posterior wall of vein. a. Advancing the needle slightly makes certain the plastic catheter has entered the vein. b. Reinsertion of the needle or pulling the catheter back can sever the catheter, causing catheter embolism. c. Slight pressure prevents bleeding before tubing is attached. d. The stylet can shear off a piece of the plastic if reinserted. e. Reusing the same catheter can cause infection.
16. Release tourniquet and attach infusion tubing; open clamp enough to allow drip.	16. Releasing the tourniquet restores blood flow and avoids potential ischemic damage to the area distal to the IV insertion site.
17. Cover the insertion site with a transparent dressing, bandage, or sterile gauze according to hospital policy and procedure. Tape in place with nonallergenic tape but do not encircle extremity. Tape a small loop of IV tubing onto dressing.	17. Transparent dressings allow assessment of the insertion site for phlebitis, infiltration, and infection without removing the dressing. Tape applied around extremity can act as a tourniquet and impede blood flow and infusion of fluid. The loop decreases the chance of inadvertent cannula removal if the tubing is pulled.
18. Label with type and length of cannula, date, time, and initials.	18. Labeling facilitates assessment and safe discontinuation.
19. A padded, appropriate-length arm board may be applied to an area of flexion (neurovascular checks should be performed frequently).	19. This secures cannula placement and allows correct flow rate (neurovascular checks assess nerve, muscle, and vascular function to be sure function is not affected by immobilization).
20. Calculate infusion rate and regulate flow of infusion. For hourly IV rate use the following formula: gtt/mL of infusion set/60 (min in h) × total hourly vol = gtt/min	20. Infusion must be regulated carefully to prevent overinfusion or underinfusion. Calculation of the IV rate is essential for the safe delivery of fluids. Safe administration requires knowledge of the volume of fluid to be infused, total infusion time, and calibration of the administration set (found on the IV tubing package; 10, 12, 15, or 60 drops to deliver 1 mL of fluid).
21. Document date and time therapy initiated; type and amount of solution; additives and dosages; flow rate; gauge, length, and type of vascular access device; catheter insertion site; type of dressing applied; patient response to procedure; patient teaching and name and title of the health care provider who inserted the catheter.	21. Documentation is essential to promote continuity of care.
22. Discard needles, stylets, or guidewires into a puncture-resistant needle container that meets OSHA guidelines. Remove gloves and perform hand hygiene.	22. Proper disposal of sharps decreases risk of needlesticks.

this complication when the catheter is removed, the nurse compares the expected length of the catheter with its actual length. Plastic catheters should be withdrawn carefully and their length measured to detect a fragment that has broken off in the vein. Both of these actions must be documented in the patient's medical record.

Great care must be exercised when using scissors around the dressing site. If the catheter clearly has been severed, the nurse can attempt to occlude the vein above the site by applying a tourniquet to prevent the catheter from entering the central circulation (until surgical removal is possible). The physician must be notified immediately. It is better to prevent a potentially fatal problem than to deal with it after it has occurred. Catheter embolism can be prevented easily by following simple rules:

- Avoid using scissors near the catheter.
- Avoid withdrawing the catheter through the insertion needle.
- Follow the manufacturer's guidelines carefully (eg, cover the needle point with the bevel shield to prevent severing the catheter).

Managing Systemic Complications

IV therapy predisposes the patient to numerous hazards, including both local and systemic complications. Systemic complications occur less frequently but are usually more serious than local complications. They include circulatory overload, air embolism, febrile reaction, and infection.

Fluid Overload

Overloading the circulatory system with excessive IV fluids causes increased blood pressure and central venous pressure. Signs and symptoms of fluid overload include moist crackles on auscultation of the lungs, edema, weight gain, dyspnea, and rapid, shallow respirations. Possible causes include rapid infusion of an IV solution or hepatic, cardiac, or renal disease. The risk of fluid overload and subsequent pulmonary edema is especially increased in elderly patients with cardiac disease; this is referred to as circulatory overload. Its treatment includes decreasing the IV rate, monitoring vital signs frequently, assessing breath sounds, and placing the patient in a high Fowler's position. The physician is contacted immediately. This complication can be avoided by using an infusion pump and by carefully monitoring all infusions. Complications of circulatory overload include heart failure and pulmonary edema.

Air Embolism

The risk of air embolism is rare but ever-present. It is most often associated with cannulation of central veins. Manifestations of air embolism include palpitations, dyspnea, and cyanosis; hypotension; weak, rapid pulse; loss of consciousness; and chest, shoulder, and low back pain. Treatment calls for immediately clamping the cannula and replacing a leaking or open infusion system, placing the patient on the left side in the Trendelenburg position, assessing vital signs and breath sounds, and administering oxygen. Air embolism can be prevented by using locking adapters on all lines, filling all tubing completely with solution, and using an air detection alarm on an IV infusion pump. Complications of air embolism include shock and death. The amount of air necessary to induce death in humans is not known; however, the rate of entry is probably as important as the actual volume of air.

Infection

Pyrogenic substances in either the infusion solution or the IV administration set can cause bloodstream infections (Todd, 2006). Signs and symptoms include an abrupt temperature elevation shortly after the infusion is started, backache, headache, increased pulse and respiratory rate, nausea and vomiting, diarrhea, chills and shaking, and general malaise. In severe sepsis, vascular collapse and septic shock may occur. See Chapter 15 for a discussion of septic shock.

Infection ranges in severity from local involvement of the insertion site to systemic dissemination of organisms through the bloodstream, as in sepsis. Measures to prevent infection are essential at the time the IV line is inserted and throughout the entire infusion. Prevention includes the following:

- Careful hand hygiene before every contact with any part of the infusion system or the patient
- Examining the IV containers for cracks, leaks, or cloudiness, which may indicate a contaminated solution
- Using strict aseptic technique
- Firmly anchoring the IV cannula to prevent to-and-fro motion (eg, a catheter stabilization device will help)
- Inspecting the IV site daily and replacing a soiled or wet dressing with a dry sterile dressing (antimicrobial agents that should be used for site care include 2% tincture of iodine, 10% povidone–iodine, alcohol, or chlorhexidine gluconate, used alone or in combination)
- Disinfecting injection/access ports with antimicrobial solution before and after each use
- Removing the IV cannula at the first sign of local inflammation, contamination, or complication
- Replacing the peripheral IV cannula every 72 to 96 hours, or as indicated (Chart 14-4)
- Replacing the IV cannula inserted during emergency conditions (with questionable asepsis) as soon as possible
- Using a 0.2-μm air-eliminating and bacteria/particulate retentive filter with non–lipid-containing solutions that require filtration. The filter can be added to the proximal or distal end of the administration set. If added to the proximal end between the fluid container and the tubing spike, the filter ensures sterility and particulate removal from the infusate container and prevents inadvertent infusion of air. If added to the distal end of the administration set, it filters air particles and contaminants introduced from add-on devices, secondary administration sets, or interruptions to the primary system. Filters should be located as close to the catheter insertion site as possible (Alexander, 2006)
- Replacing the solution bag and administration set in accordance with agency policy and procedure
- Infusing or discarding medication or solution within 24 hours of its addition to an administration set
- Changing primary and secondary continuous administration sets every 72 hours, intermittent administra-

CHART 14-4 NURSING RESEARCH PROFILE

When Should a Peripheral IV Catheter Be Changed?

Gallant, P. & Schultz, A. (2006). Evaluation of a visual infusion phlebitis scale for determining appropriate discontinuation of peripheral intravenous catheters. *Journal of Infusion Nursing, 29*(6), 333–345.

Purpose

Phlebitis is a common occurrence at peripheral IV sites. It is known that phlebitis is related to certain types of medication, infusates the patient is receiving, and length of time the catheter remains in the vein. Therefore, it has been recommended that peripheral IV sites be rotated at prescribed intervals (eg, 48 to 72 hours) to reduce the rate of phlebitis. This study evaluated the use of a visual infusion phlebitis scale for determining appropriate discontinuation of peripheral IV catheters.

Design

A descriptive correlation design was used to compare phlebitis rates between peripheral IV sites that had been indwelling for up to 96 hours with those that had been indwelling for longer than 96 hours. Researchers monitored 851 IV sites in 513 patients in a cardiac surgery critical care unit and in a cardiothoracic stepdown unit. Skin preparation, IV insertion, and dressing site care were according to hospital policy, with insertions initiated using 18-gauge or smaller needles in the antecubital area, forearm, hand, or wrist. Researchers used the Visual Infusion Phlebitis (VIP) scale to rate phlebitis using a range of scores from 0 (no symptoms) to 5 (purulent drainage, redness, and a palpable cord greater than 3 inches). All scores of 2 (pain, redness, warmth, and/or edema extending from 1 to 2 inches above the site) resulted in a change in catheters. The researchers evaluated each IV site daily and documented reasons for removal of catheters, time of catheter removal, and any medications administered through the IV route.

Findings

Chi-square analysis demonstrated no statistically significant difference between the two groups in age, gender, or the type of surgery for the patients who had peripheral IV catheters indwelling for less than 96 hours compared with those who had catheters for more than 96 hours. There was a significant increase in the rate of phlebitis in patients who needed IV restarts (13.4%) as compared with a phlebitis rate of 2.7% in patients who had only one IV inserted (no restarts). There was a significant increase in phlebitis rates when certain medications (antibiotics, diltiazem, potassium chloride, and amiodarone) were infused compared with those in which none of these medications was infused. The VIP scale was found to be a reliable and valid method for determining when a peripheral catheter should be removed.

Nursing Implications

In the sample studied, the peripheral IV catheters in place for longer than 96 hours did not result in significantly increased rates of phlebitis or greater risk of bacteremia compared to IV lines removed at 96 hours. Routine restarting of IV lines and administration of certain medications increased the risk of phlebitis. The VIP scale was useful for determining when catheters should be removed. Clinical assessment remains essential for safe patient care.

tion sets every 24 hours, or immediately if contamination is suspected (Alexander, 2006)
- Using administration sets with a twist-lock design (Rosenthal, 2007)

Managing Local Complications

Local complications of IV therapy include infiltration and extravasation, phlebitis, thrombophlebitis, hematoma, and clotting of the needle.

Infiltration and Extravasation

Infiltration is the unintentional administration of a nonvesicant solution or medication into surrounding tissue. This can occur when the IV cannula dislodges or perforates the wall of the vein. Infiltration is characterized by edema around the insertion site, leakage of IV fluid from the insertion site, discomfort and coolness in the area of infiltration, and a significant decrease in the flow rate. When the solution is particularly irritating, sloughing of tissue may result. Close monitoring of the insertion site is necessary to detect infiltration before it becomes severe.

Infiltration is usually easily recognized if the insertion area is larger than the same site of the opposite extremity; however, it is not always so obvious. A common misconception is that a backflow of blood into the tubing proves that the catheter is properly placed within the vein. However, if the catheter tip has pierced the wall of the vessel, IV fluid will seep into tissues as well as flow into the vein. Although blood return occurs, infiltration may have occurred as well. A more reliable means of confirming infiltration is to apply a tourniquet above (or proximal to) the infusion site and tighten it enough to restrict venous flow. If the infusion continues to drip despite the venous obstruction, infiltration is present.

As soon as the nurse detects infiltration, the infusion should be stopped, the IV catheter discontinued, and a sterile dressing applied to the site after careful inspection to determine the extent of infiltration. The infiltration of any amount of blood product, irritant, or vesicant is considered the most severe.

The IV infusion should be started in a new site or proximal to the infiltration if the same extremity must be used again. A warm compress may be applied to the site if small volumes of noncaustic solutions have infiltrated over a long period, or if the solution was isotonic with a normal pH; the affected extremity should be elevated to promote the absorption of fluid. If the infiltration is recent and the solution was hypertonic or had an increased pH, a cold compress may be applied to the area. Infiltration can be detected and treated early by inspecting the site every hour for redness, pain, edema, blood return, coolness at the site, and IV fluid leaking from the IV site. Using the appropriate size and type of cannula for the vein prevents this complication. The Infusion Nursing Standards of Practice state that a standard-

CHART 14-5 *Assessing for Infiltration*

Grade	Clinical Criteria
0	No clinical symptoms
1	Skin blanched, edema less than 1 inch in any direction, cool to touch, with or without pain
2	Skin blanched, edema 1 to 6 inches in any direction, cool to touch, with or without pain
3	Skin blanched, translucent, gross edema greater than 6 inches in any direction, cool to touch, mild to moderate pain, possible numbness
4	Skin blanched, translucent, skin tight, leaking, skin discolored, bruised, swollen, gross edema greater than 6 inches in any direction, deep pitting tissue edema, circulatory impairment, moderate to severe pain, infiltration of any amount of blood products, irritant, or vesicant

Adapted from Alexander, M. (2006). Infusion nursing standards of practice. *Journal of Infusion Nursing, 29*(IS), S1–S92.

ized infiltration scale should be used to document the infiltration (Alexander, 2006) (Chart 14-5).

Extravasation is similar to infiltration, with an inadvertent administration of vesicant or irritant solution or medication into the surrounding tissue. Medications such as dopamine, calcium preparations, and chemotherapeutic agents can cause pain, burning, and redness at the site. Blistering, inflammation, and necrosis of tissues can occur. The extent of tissue damage is determined by the concentration of the medication, the quantity that extravasated, the location of the infusion site, the tissue response, and the duration of the process of extravasation.

The infusion must be stopped and the physician notified promptly. The agency's protocol to treat extravasation is initiated; the protocol may specify specific treatments, including antidotes specific to the medication that extravasated, and may indicate whether the IV line should remain in place or be removed before treatment. The protocol often specifies infiltration of the infusion site with an antidote prescribed after assessment by the physician, removal of the cannula, and application of warm compresses to sites of extravasation from vinca alkaloids or cold compresses to sites of extravasation from alkylating and antibiotic vesicants. The affected extremity should not be used for further cannula placement. Thorough neurovascular assessments of the affected extremity must be performed frequently (Hadaway, 2007).

Reviewing the institution's IV policy and procedures and incompatibility charts and checking with the pharmacist before administering any IV medication, whether peripherally or centrally, are recommended to determine incompatibilities and vesicant potential to prevent extravasation. Careful, frequent monitoring of the IV site, avoiding insertion of IV devices in areas of flexion, securing the IV line, and using the smallest catheter possible that accommodates the vein help minimize the incidence and severity of this complication. In addition, when vesicant medication is administered by IV push, it should be given through a side port of an infusing IV solution to dilute the medication and decrease the severity of tissue damage if extravasation occurs. Extravasation is rated as grade 4 on the infiltration scale.

Phlebitis

Phlebitis is defined as inflammation of a vein, which can be categorized as chemical, mechanical, or bacterial; however, two or more of these types of irritation often occur simultaneously. Chemical phlebitis can be caused by an irritating medication or solution (increased pH or high osmolality of a solution), rapid infusion rates, and medication incompatibilities. Mechanical phlebitis results from long periods of cannulation, catheters in flexed areas, catheter gauges larger than the vein lumen, and poorly secured catheters. Bacterial phlebitis can develop from poor hand hygiene, lack of aseptic technique, failure to check all equipment before use, and failure to recognize early signs and symptoms of phlebitis. Other factors include poor venipuncture technique, catheter in place for a prolonged period, and failure to adequately secure the catheter. Phlebitis is characterized by a reddened, warm area around the insertion site or along the path of the vein, pain or tenderness at the site or along the vein, and swelling. The incidence of phlebitis increases with the length of time the IV line is in place (see Chart 14-4), the composition of the fluid or medication infused (especially its pH and tonicity), the size and site of the cannula inserted, ineffective filtration, inadequate anchoring of the line, and the introduction of microorganisms at the time of insertion. The Infusion Nursing Society has identified specific standards for assessing phlebitis (Alexander, 2006); these appear in Chart 14-6. Phlebitis is graded according to the most severe presenting indication.

CHART 14-6 *Assessing for Phlebitis*

Grade	Clinical Criteria
0	No clinical symptoms
1	Erythema at access site with or without pain
2	Pain at access site Erythema, edema, or both
3	Pain at access site Erythema, edema, or both Streak formation Palpable venous cord (1 inch or shorter)
4	Pain at access site with erythema Streak formation Palpable venous cord (longer than 1 inch) Purulent drainage

Note: If this scale is not being used in an institution, then the description associated with the number can be used to describe the assessment.

Adapted from Alexander, M. (2006). Infusion nursing standards of practice. *Journal of Infusion Nursing, 29*(1S), S1–S92.

Treatment consists of discontinuing the IV line and restarting it in another site, and applying a warm, moist compress to the affected site. Phlebitis can be prevented by using aseptic technique during insertion, using the appropriate-size cannula or needle for the vein, considering the composition of fluids and medications when selecting a site, observing the site hourly for any complications, anchoring the cannula or needle well, and changing the IV site according to agency policy and procedures.

Thrombophlebitis

Thrombophlebitis refers to the presence of a clot plus inflammation in the vein. It is evidenced by localized pain, redness, warmth, and swelling around the insertion site or along the path of the vein, immobility of the extremity because of discomfort and swelling, sluggish flow rate, fever, malaise, and leukocytosis.

Treatment includes discontinuing the IV infusion; applying a cold compress first, to decrease the flow of blood and increase platelet aggregation, followed by a warm compress; elevating the extremity; and restarting the line in the opposite extremity (see Chart 14-4). If the patient has signs and symptoms of thrombophlebitis, the IV line should not be flushed (although flushing may be indicated in the absence of phlebitis to ensure cannula patency and to prevent mixing of incompatible medications and solutions). The catheter should be cultured after the skin around the catheter is cleaned with alcohol. If purulent drainage exists, the site is cultured before the skin is cleaned.

Thrombophlebitis can be prevented by avoiding trauma to the vein at the time the IV line is inserted, observing the site every hour, and checking medication additives for compatibility.

Hematoma

Hematoma results when blood leaks into tissues surrounding the IV insertion site. Leakage can result if the opposite vein wall is perforated during venipuncture, the needle slips out of the vein, or insufficient pressure is applied to the site after removal of the needle or cannula. The signs of a hematoma include ecchymosis, immediate swelling at the site, and leakage of blood at the insertion site.

Treatment includes removing the needle or cannula and applying light pressure with a sterile, dry dressing; applying ice for 24 hours to the site to avoid extension of the hematoma; elevating the extremity; assessing the extremity for any circulatory, neurologic, or motor dysfunction; and restarting the line in the other extremity if indicated (see Chart 14-4). A hematoma can be prevented by carefully inserting the needle and by using diligent care with patients who have a bleeding disorder, are taking anticoagulant medication, or have advanced liver disease.

Clotting and Obstruction

Blood clots may form in the IV line as a result of kinked IV tubing, a very slow infusion rate, an empty IV bag, or failure to flush the IV line after intermittent medication or solution administrations. The signs are decreased flow rate and blood backflow into the IV tubing.

If blood clots in the IV line, the infusion must be discontinued and restarted in another site with a new cannula and administration set. The tubing should not be irrigated or milked. Neither the infusion rate nor the solution container should be raised, and the clot should not be aspirated from the tubing. Clotting of the needle or cannula may be prevented by not allowing the IV solution bag to run dry, taping the tubing to prevent kinking and maintain patency, maintaining an adequate flow rate, and flushing the line after intermittent medication or other solution administration. In some cases, a specially trained nurse or physician may inject a thrombolytic agent into the catheter to clear an occlusion resulting from fibrin or clotted blood.

Promoting Home and Community-Based Care

Teaching Patients Self-Care

At times, IV therapy must be administered in the home setting, in which case much of the daily management rests with the patient and family. Teaching becomes essential to ensure that the patient and family can manage the IV fluid and infusion correctly and avoid complications. Written instructions as well as demonstration and return demonstration help reinforce the key points for all these functions.

Continuing Care

Home infusion therapies cover a wide range of treatments, including antibiotic, analgesic, and antineoplastic medications; blood or blood component therapy; and parenteral nutrition. When direct nursing care is necessary, arrangements are made to have an infusion nurse visit the home and administer the IV therapy as prescribed. In addition to implementing and monitoring the IV therapy, the nurse carries out a comprehensive assessment of the patient's condition and continues to teach the patient and family about the skills involved in overseeing the IV therapy setup. Any dietary changes that may be necessary because of fluid or electrolyte imbalances are explained or reinforced during such sessions.

Periodic laboratory testing may be necessary to assess the effects of IV therapy and the patient's progress. Blood specimens may be obtained by a laboratory near the patient's home, or a home visit may be arranged to obtain blood specimens for analysis.

The nurse collaborates with the case manager in assessing the patient, family, and home environment; developing a plan of care in accordance with the patient's treatment plan and level of ability; and arranging for appropriate referral and follow-up if necessary. Any necessary equipment may be provided by the agency or purchased by the patient, depending on the terms of the home care arrangements. Appropriate documentation is necessary to assist in obtaining third-party payment for the service provided.

CRITICAL THINKING EXERCISES

1 A 38-year-old woman is admitted with a chief complaint of shortness of breath and polyuria for the past 6 weeks. She is hypotensive. Her pulse rate is 110 bpm, and her lungs are clear to auscultation. Her laboratory test results are as follows: pH 7.32; sodium 131 mEq/L; glucose 600 mg/dL; $PaCO_2$ 28 mm Hg; potassium 4.5 mEq/L; cre-

atinine 1.4 mg/dL; HCO_3^- 14 mEq/L; chloride 95 mEq/L; BUN 30 mg/dL. What fluid and electrolyte or acid–base disorder is the patient experiencing? What IV fluids would you anticipate being prescribed? Give the rationale for their use. What treatments would address the patient's fluid and electrolyte or acid–base disorders?

2 A 54-year-old obese man who has smoked one pack per day for the past 25 years has had a productive cough for the last 3 months and shortness of breath with little exertion. His wife complains of his loud snoring. His blood pressure is 130/90 mm Hg and pulse rate 126 bpm. His arterial blood gas results are as follows: pH 7.29; $PaCO_2$ 72 mm Hg; HCO_3^- 34 mEq/L; PaO_2 50 mm Hg. How do you interpret the patient's blood gas values? What treatment would you anticipate?

3 An 85-year-old woman is brought to the hospital with a decreased fluid intake for the past 4 days and weakness. She is not in respiratory distress. Her laboratory test results are as follows: sodium 145 mEq/L; potassium 1.9 mEq/L; chloride 86 mEq/L; pH 7.58; $PaCO_2$ 49 mm Hg; HCO_3^- 44 mEq/L. What fluid and electrolyte or acid–base disorders is the patient experiencing? Outline the nursing plan of care to address the patient's fluid and electrolyte or acid–base disorders. Give the rationale for the nursing interventions for this patient.

4 A 58-year-old man on the surgical unit is scheduled for an appendectomy and needs an IV for hydration and administration of preoperative medications. What aspects of the patient history must be assessed prior to administration of IV fluids and mediations? Describe the site selection process and the factors that affect the choice of an IV site. What factors need to be considered in preparing to administer IV therapy to this patient?

EBP **5** A 35-year-old obese woman has been receiving IV therapy for the past 72 hours. The nurse plans to change the IV site today. A nurse on the IV team has suggested the use of clinical assessment criteria to assess the need for the IV change. What is the evidence for use of clinical criteria in this case? What criteria would you use to assess the strength of the evidence for the use of clinical criteria? Which criteria would you use in this patient's case?

The Smeltzer suite offers these additional resources to enhance learning and facilitate understanding of this chapter:

- thePoint online resource, thepoint.lww.com/Smeltzer12E
- Student CD-ROM included with the book
- *Study Guide to Accompany Brunner & Suddarth's Textbook of Medical-Surgical Nursing*
- *Handbook for Brunner & Suddarth's Textbook of Medical-Surgical Nursing*

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*Asterisk indicates nursing reseach.
**Double asterisk indicates classic reference.

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chapter 15

Shock and Multiple Organ Dysfunction Syndrome

LEARNING OBJECTIVES

On completion of this chapter, the learner will be able to:

1. Describe shock and its underlying pathophysiology.
2. Compare clinical findings of the compensatory, progressive, and irreversible stages of shock.
3. Describe organ dysfunction that may occur with shock.
4. Describe similarities and differences in shock due to hypovolemic, cardiogenic, neurogenic, anaphylactic, and septic shock states.
5. Identify medical and nursing management priorities in treating patients in shock.
6. Identify vasoactive medications used in treating shock, and describe nursing implications associated with their use.
7. Discuss the importance of nutritional support in all forms of shock.
8. Discuss the role of nurses in psychosocial support of patients experiencing shock and their families.
9. Discuss multiple organ dysfunction syndrome.

GLOSSARY

anaphylactic shock: circulatory shock state resulting from a severe allergic reaction producing an overwhelming systemic vasodilation and relative hypovolemia

biochemical mediators: messenger substances that may be released by a cell to create an action at that site or be carried by the bloodstream to a distant site before being activated; also called cytokines

cardiogenic shock: shock state resulting from impairment or failure of the myocardium

circulatory shock: shock state resulting from displacement of blood volume creating a relative hypovolemia and inadequate delivery of oxygen to the cells; also called distributive shock

colloids: intravenous solutions that contain molecules that are too large to pass through capillary membranes

crystalloids: intravenous electrolyte solutions that move freely between the intravascular compartment and interstitial spaces

hypovolemic shock: shock state resulting from decreased intravascular volume due to fluid loss

multiple organ dysfunction syndrome: presence of altered function of two or more organs in an acutely ill patient such that interventions are necessary to support continued organ function

neurogenic shock: shock state resulting from loss of sympathetic tone causing relative hypovolemia

septic shock: circulatory shock state resulting from overwhelming infection causing relative hypovolemia

shock: physiologic state in which there is inadequate blood flow to tissues and cells of the body

systemic inflammatory response syndrome: overwhelming inflammatory response in the absence of infection causing relative hypovolemia and decreased tissue perfusion

Shock is a life-threatening condition with a variety of underlying causes. It is characterized by inadequate perfusion that, if untreated, results in cell death. The progression of shock is neither linear nor predictable, and shock states, especially septic shock, comprise a current area of aggressive clinical research. Nurses caring for patients with shock and for those at risk for shock must understand the underlying mechanisms of shock and recognize its subtle as well as more obvious signs. Rapid assessment with early recognition and response to shock states is essential to the patient's recovery.

Overview of Shock

Shock can best be defined as a condition in which widespread perfusion to the cells is inadequate to deliver oxygen and nutrients to support vital organs and cellular function (VonRueden, Bolton & Vary, 2008). Adequate blood flow to the tissues and cells requires an adequate cardiac pump, effective vasculature or circulatory system, and sufficient blood volume. If one of these components is impaired, perfusion to the tissues is threatened or compromised. Without treatment, inadequate blood flow to the cells results in poor delivery of oxygen and nutrients, cellular hypoxia, and cell death that progresses to organ dysfunction and eventually death.

Shock affects all body systems. It may develop rapidly or slowly, depending on the underlying cause. During shock, the body struggles to survive, calling on all its homeostatic mechanisms to restore blood flow. Any insult to the body can create a cascade of events resulting in poor tissue perfusion. Therefore, almost any patient with any disease state may be at risk for developing shock. Conventionally, the primary underlying pathophysiologic process and underlying disorder are used to classify the shock state (eg, hypovolemic shock, cardiogenic shock, and circulatory shock [all discussed later in the chapter]).

Regardless of the initial cause of shock, certain physiologic responses are common to all types of shock. These physiologic responses include hypoperfusion of tissues, hypermetabolism, and activation of the inflammatory response. The body responds to shock states by activating the sympathetic nervous system and mounting a hypermetabolic and inflammatory response. Once shock develops, the patient's survival may have more to do with the body's ability to effectively respond to it than with the initial cause of shock. Failure of compensatory mechanisms to effectively restore physiologic balance is the final pathway of all shock states and results in end-organ dysfunction and death (Cocchi, Kimlin, Walsh, et al., 2007; Dellinger, Levy, Carlet, et al., 2008; King, 2007; VonRueden, et al., 2008).

Nursing care of patients with shock requires ongoing systematic assessment. Many of the interventions required in caring for patients with shock call for close collaboration with other members of the health care team and rapid implementation of prescribed therapies. Nurses must anticipate these therapies because they need to be implemented with speed and accuracy.

Normal Cellular Function

Energy metabolism occurs within the cell, where nutrients are chemically broken down and stored in the form of adenosine triphosphate (ATP). Cells use this stored energy to perform necessary functions, such as active transport, muscle contraction, and biochemical synthesis, as well as specialized cellular functions, such as the conduction of electrical impulses. ATP can be synthesized aerobically (in the presence of oxygen) or anaerobically (in the absence of oxygen). Aerobic metabolism yields far greater amounts of ATP per mole of glucose than does anaerobic metabolism; therefore, it is a more efficient and effective means of producing energy. In addition, anaerobic metabolism results in the accumulation of the toxic end product, lactic acid, which must be removed from the cell and transported to the liver for conversion into glucose and glycogen.

Pathophysiology

Cellular Changes

In shock, the cells lack an adequate blood supply and are deprived of oxygen and nutrients; therefore, they must produce energy through anaerobic metabolism. This results in low energy yields from nutrients and an acidotic intracellular environment. Because of these changes, normal cell function ceases (Fig. 15-1). The cell swells and the cell membrane becomes more permeable, allowing electrolytes and fluids to seep out of and into the cell. The sodium–potassium pump becomes impaired; cell structures, primarily the mitochondria, are damaged; and death of the cell results.

Glucose is the primary substrate required for the production of cellular energy in the form of ATP. In stress states, catecholamines, cortisol, glucagons, and inflammatory cytokines and mediators are released, causing hyperglycemia and insulin resistance to mobilize glucose for cellular metabolism. Activation of these substances promotes gluconeogenesis, which is the formation of glucose from noncarbohydrate sources such as proteins and fats. Glycogen that has been stored in the liver is converted to glucose through glycogenolysis to meet metabolic needs, increasing the blood glucose concentration (ie, hyperglycemia).

Continued activation of the stress response by shock states causes a depletion of glycogen stores, resulting in increased proteolysis and eventual organ failure (Vincent, 2007). The inability of the body to have enough nutrients and oxygen for normal cellular metabolism causes a buildup of metabolic end products in the cells and interstitial spaces. Cellular metabolism is impaired, and a negative feedback loop is initiated.

Vascular Responses

Local regulatory mechanisms, referred to as autoregulation, stimulate vasodilation or vasoconstriction in response to **biochemical mediators** (ie, cytokines) released by the cell, communicating the need for oxygen and nutrients (King, 2007). A biochemical mediator is a substance released by a cell or immune cells such as macrophages; the substance triggers an action at a cell site or travels in the bloodstream to a distant site, where it triggers action. Researchers are learning more every day about the physiologic actions of more than 100 known cytokines (VonRueden, et al., 2008).

Blood Pressure Regulation

Three major components of the circulatory system—blood volume, the cardiac pump, and the vasculature—must respond effectively to complex neural, chemical, and

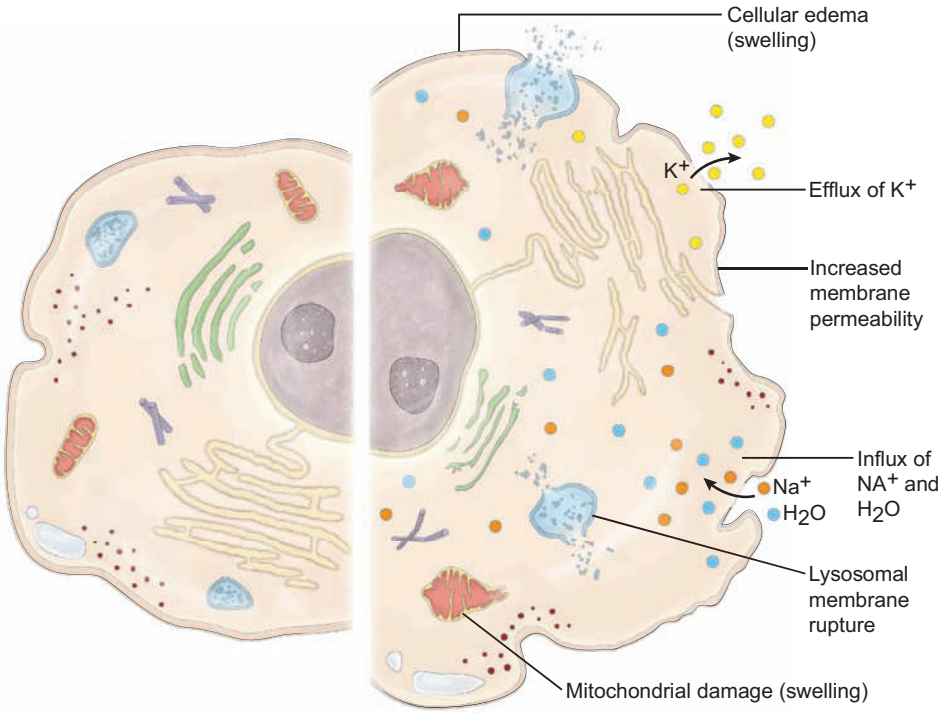

Figure 15-1 Cellular effects of shock. The cell swells and the cell membrane becomes more permeable; fluids and electrolytes seep from and into the cell. Mitochondria and lysosomes are damaged, and the cell dies.

hormonal feedback systems to maintain an adequate blood pressure (BP) and perfuse body tissues. BP is regulated through a complex interaction of neural, chemical, and hormonal feedback systems affecting both cardiac output and peripheral resistance. This relationship is expressed in the following equation:

Mean arterial BP = Cardiac output × Peripheral resistance

Cardiac output is a product of the stroke volume (the amount of blood ejected from the left ventricle during systole) and heart rate. Peripheral resistance is determined by the diameter of the arterioles.

Tissue perfusion and organ perfusion depend on mean arterial pressure (MAP), or the average pressure at which blood moves through the vasculature. MAP must exceed 65 mm Hg for cells to receive the oxygen and nutrients needed to metabolize energy in amounts sufficient to sustain life (Dellinger, et al., 2008). True MAP can be calculated only by complex methods. Frequently, MAP is calculated by automatic blood pressure machines, however, the nurse must ensure accurate blood pressure measurement is obtained before interpreting data from automated vital sign equipment.

BP is regulated by baroreceptors (pressure receptors) located in the carotid sinus and aortic arch. These pressure receptors are responsible for monitoring the circulatory volume and regulating neural and endocrine activities (see Chapter 14 for further description). When BP drops, catecholamines (epinephrine and norepinephrine) are released from the adrenal medulla. These increase heart rate and cause vasoconstriction, thus restoring BP. Chemoreceptors, also located in the aortic arch and carotid arteries, regulate BP and respiratory rate using much the same mechanism in response to changes in oxygen and carbon dioxide concentrations in the blood. These primary regulatory mechanisms can respond to changes in BP on a moment-to-moment basis.

The kidneys regulate BP by releasing renin, an enzyme needed for the conversion of angiotensin I to angiotensin II, a potent vasoconstrictor. This stimulation of the renin–angiotensin mechanism and the resulting vasoconstriction indirectly lead to the release of aldosterone from the adrenal cortex, which promotes the retention of sodium and water. The increased concentration of sodium in the blood stimulates the release of antidiuretic hormone (ADH) by the pituitary gland. ADH causes the kidneys to retain water further in an effort to raise blood volume and BP. These secondary regulatory mechanisms may take hours or days to respond to changes in BP. The relationship between the initiation of shock and the responsiveness of primary and secondary regulatory mechanisms that compensate for deficits in blood volume, the pumping effectiveness of the heart, or vascular tone, which may result because of the shock state, is noted in Figure 15-2.

Stages of Shock

Shock is believed to progress along a continuum of stages. Shock can be identified as early or late, depending on the signs and symptoms and the overall severity of organ dysfunction. A convenient way to understand the physiologic responses and subsequent clinical signs and symptoms of shock is to divide the continuum into separate stages: compensatory (stage 1), progressive (stage 2), and irreversible (stage 3). The earlier that medical and nursing

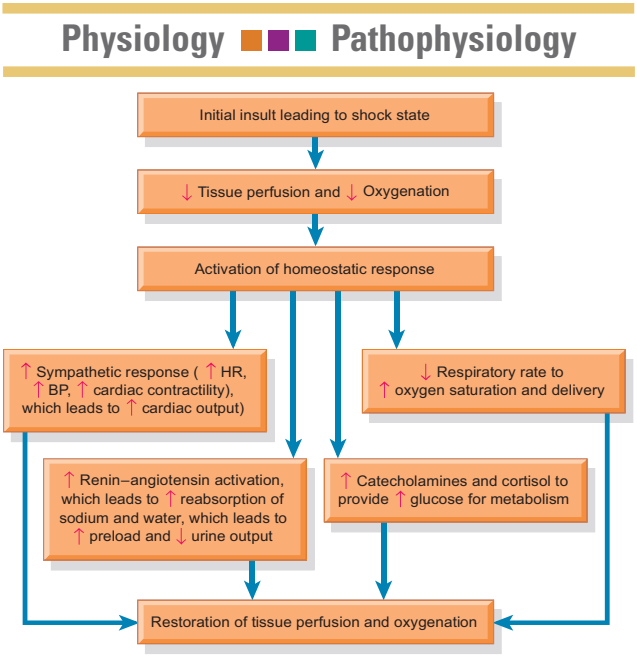

Figure 15-2 Compensatory mechanisms in shock.

interventions are initiated along this continuum, the greater the patient's chance of survival. Current research is focusing on assessing patients at greatest risk for shock and implementing early and aggressive interventions to reverse tissue hypoxia (King, 2007; Otero, Nguygen, Huang, et al., 2006). Studies suggest that the window of opportunity that increases the likelihood of patient survival occurs when aggressive therapy begins within 6 hours of identifying a shock state, especially septic shock (Otero, et al., 2006; Rivers, McIntyre, Morro, et al., 2005).

COMPENSATORY STAGE

In the compensatory stage of shock, the BP remains within normal limits. Vasoconstriction, increased heart rate, and increased contractility of the heart contribute to maintaining adequate cardiac output. This results from stimulation of the sympathetic nervous system and subsequent release of catecholamines (epinephrine and norepinephrine). Patients display the often-described "fight or flight" response. The body shunts blood from organs such as the skin, kidneys, and gastrointestinal tract to the brain, heart, and lungs to ensure adequate blood supply to these vital organs. As a result, the skin is cool and clammy, bowel sounds are hypoactive, and urine output decreases in response to the release of aldosterone and ADH.

Clinical Manifestations

Despite a normal BP, the patient shows numerous clinical signs indicating inadequate organ perfusion (Table 15-1). The result of inadequate perfusion is anaerobic metabolism and a buildup of lactic acid, producing metabolic acidosis. The respiratory rate increases in response to metabolic acidosis. This rapid respiratory rate facilitates removal of excess carbon dioxide but raises the blood pH and often causes a compensatory respiratory alkalosis. The alkalotic state causes mental status changes, such as confusion or combativeness, as well as arteriolar dilation. If treatment begins in this stage of shock, the prognosis for the patient is better than in later stages.

Medical Management

Medical treatment is directed toward identifying the cause of the shock, correcting the underlying disorder so that shock does not progress, and supporting those physiologic processes that thus far have responded successfully to the threat. Because compensation cannot be maintained indefinitely, measures such as fluid replacement and medication therapy must be initiated to maintain an adequate BP and reestablish and maintain adequate tissue perfusion (Otero, et al., 2006).

Nursing Management

As stated earlier, intervention as soon as possible along the continuum of shock is the key to improving the patient's prognosis. The nurse must systematically assess the patient at risk for shock to recognize the subtle clinical signs of the compensatory stage before the patient's BP drops. Special considerations related to recognizing early signs of shock in the elderly patient are given in Chart 15-1.

Table 15-1 CLINICAL FINDINGS IN STAGES OF SHOCK

	Stage		
Finding	**Compensatory**	**Progressive**	**Irreversible**
Blood pressure	Normal	Systolic <80–90 mm Hg Requires fluids resuscitation to support blood pressure	Requires mechanical or pharmacologic support
Heart rate	>100 bpm	>150 bpm	Erratic or asystole
Respiratory status	>20 breaths/min $PaCO_2$ <32 mm Hg	Rapid, shallow respirations; crackles PaO_2 <80 mm Hg $PaCO_2$ >45 mm Hg	Requires intubation and mechanical ventilation and oxygenation
Skin	Cold, clammy	Mottled, petechiae	Jaundice
Urinary output	Decreased	0.5 mL/kg/h	Anuric, requires dialysis
Mentation	Confusion	Lethargy	Unconscious
Acid–base balance	Respiratory alkalosis	Metabolic acidosis	Profound acidosis

CHART 15-1

Recognizing Shock in Older Patients

The physiologic changes associated with aging, coupled with pathologic and chronic disease states, place older people at increased risk for developing a state of shock and possibly multiple organ dysfunction syndrome (MODS). Elderly people can recover from shock if it is detected and treated early with aggressive and supportive therapies. Nurses play an essential role in assessing and interpreting subtle changes in older patients' responses to illness.

- Medications such as beta-blocking agents (metoprolol [Lopressor]) used to treat hypertension may mask tachycardia, a primary compensatory mechanism to increase cardiac output, during hypovolemic states.
- The aging immune system may not mount a truly febrile response (temperature more than 38°C [100.4°F]), but an increasing trend in body temperature should be addressed. The patient may also report increased fatigue and malaise in the absence of a febrile response.
- The heart does not function well in hypoxemic states, and the aging heart may respond to decreased myocardial oxygenation with dysrhythmias that may be misinterpreted as a normal part of the aging process.
- There is a progressive decline in respiratory muscle strength, maximal ventilation, and response to hypoxia. Older patients have a decreased respiratory reserve and decompensate more quickly.
- Changes in mentation may be inappropriately misinterpreted as dementia. Older people with a sudden change in mentation should be aggressively treated for the presence of infection and organ hypoperfusion.

Monitoring Tissue Perfusion

In assessing tissue perfusion, the nurse observes for changes in level of consciousness, vital signs (including pulse pressure), urinary output, skin, and laboratory values (eg, base deficit and lactic acid levels). In the compensatory stage of shock, serum sodium and blood glucose levels are elevated in response to the release of aldosterone and catecholamines.

The nurse should monitor the patient's hemodynamic status and promptly report deviations to the physician, assist in identifying and treating the underlying disorder by continuous in-depth assessment of the patient, administer prescribed fluids and medications, and promote patient safety. Vital signs are key indicators of hemodynamic status and BP is an indirect measure of tissue hypoxia. The nurse should report a systolic BP lower than 90 mm Hg or a drop in systolic BP of 40 mm Hg from baseline.

Pulse pressure correlates well with stroke volume. Pulse pressure is calculated by subtracting the diastolic measurement from the systolic measurement; the difference is the pulse pressure (Cottingham, 2006). Normally, the pulse pressure is 30 to 40 mm Hg. Narrowing or decreased pulse pressure is an earlier indicator of shock than a drop in systolic BP. Decreased or narrowing pulse pressure, an early indication of decreased stroke volume, is illustrated in the following example:

$$\text{Systolic BP} - \text{Diastolic BP} = \text{Pulse pressure}$$

Normal pulse pressure:

$$120 \text{ mg Hg} - 80 \text{ mm Hg} = 40 \text{ mm Hg}$$

Narrowing of pulse pressure:

$$90 \text{ mm Hg} - 70 \text{ mm Hg} = 20 \text{ mm Hg}$$

Elevation of the diastolic BP with release of catecholamines and attempts to increase venous return through vasoconstriction is an early compensatory mechanism in response to decreased stroke volume, BP, and overall cardiac output.

NURSING ALERT

By the time BP drops, damage has already been occurring at the cellular and tissue levels. Therefore, the patient at risk for shock must be assessed and monitored closely before the BP falls.

Continuous central venous oximetry ($Sc\bar{v}O_2$) monitoring may be used to evaluate mixed venous blood oxygen saturation and the severity of tissue hypoperfusion states. A central catheter is introduced into the superior vena cava (SVC), and a sensor on the catheter measures the oxygen saturation of the blood in the SVC as blood returns to the heart and pulmonary system for reoxygenation. A normal $Sc\bar{v}O_2$ value is 70% (Goodrich, 2006; Rivers, et al., 2005). Body tissues use approximately 25% of the oxygen delivered to them during normal metabolism. During states of stress, such as shock, more oxygen is consumed and the $Sc\bar{v}O_2$ saturation is lower, indicating that the tissues are consuming more oxygen.

Interventions focus on decreasing tissue oxygen requirements and increasing perfusion to deliver more oxygen to the tissues. For instance, sedating agents may be administered to lower metabolic demands, the patient's pain may be treated with intravenous (IV) opioid agents, or measures to prevent shivering, decrease metabolic demands for oxygen. Supplemental oxygen and mechanical ventilation may be required to increase the delivery of oxygen in the blood. Administration of IV fluids and medications supports blood pressure and cardiac output, and the transfusion of packed red blood cells enhances oxygen transport. Monitoring tissue oxygen consumption with $Sc\bar{v}O_2$ is a minimally invasive measure to more accurately assess tissue oxygenation in the compensatory stage of shock before changes in vital signs detect altered tissue perfusion (Dellinger, et al., 2008; Goodrich, 2006; Otero, et al., 2006).

New technologies allow clinicians to detect changes in tissue perfusion before changes in classic signs (BP, heart rate, and urine output) indicative of hypoperfusion occur.

Two of these technologies include sublingual capnometry and near-infrared spectroscopy. Sublingual capnometry, a noninvasive technology, provides information about the degree of hypoperfusion based on the sublingual partial pressure of carbon dioxide (PCO_2) (Goodrich, 2006). A probe is placed under the patient's tongue, and PCO_2 levels are derived from the blood flow found in the mucosal bed. During shock, an elevated PCO_2 indicates poor tissue perfusion. Near-infrared spectroscopy (NIRS), a continuous noninvasive technology, uses light transmission to measure skeletal muscle oxygenation as an indicator of shock. The NIRS probe is applied to the thenar muscle that is located on the palm of the hand near the thumb, and it measures the oxygen saturation of tissue by determining the amount of infrared light absorption. Low values of tissue oxygenation (eg, less than 80%) indicate severity of shock; the lower the value, the more severe the tissue hypoxia.

Although treatments are prescribed and initiated by the physician, the nurse usually implements them, operates and troubleshoots equipment used in treatment, monitors the patient's status during treatment, and evaluates the immediate effects of treatment. In addition, the nurse assesses the response of the patient and family to the crisis and its treatment.

Reducing Anxiety

Patients and their families often become anxious and apprehensive when they face a major threat to health and well-being and are the focus of attention of many health care providers. Providing brief explanations about the diagnostic and treatment procedures, supporting the patient during these procedures, and providing information about their outcomes are usually effective in reducing stress and anxiety and thus promoting the patient's physical and mental well-being. Speaking in a calm reassuring voice and using gentle touch also help ease the patient's concerns. These actions may provide comfort for critically ill, frightened patients (Benner, 2004; Duran, Oman, Abel, et al., 2007). Research has repeatedly shown that family members have certain needs during a health-related crisis, including needing honest, consistent, and thorough communication with health care providers; needing physical and emotional closeness to the patient; sensing that health care providers care about their patients; seeing the patient frequently; and knowing exactly what has been done for the patient (Duran, et al., 2007).

The nurse should advocate that family members be present during procedures and while patient care is provided. The presence of family provides a necessary connection and support for the patient during a time of crisis.

Promoting Safety

The nurse must be vigilant for potential threats to the patient's safety, because a high anxiety level and altered mental status impair judgment. In this stage of shock, patients who were previously cooperative and followed instructions may now disrupt IV lines and catheters and complicate their condition. Therefore, close monitoring and frequent reorientation interventions are essential.

PROGRESSIVE STAGE

In the second stage of shock, the mechanisms that regulate BP can no longer compensate, and the MAP falls below normal limits. Patients are clinically hypotensive; this is defined as a systolic BP of less than 90 mm Hg or a decrease in systolic BP of 40 mm Hg from baseline (Dellinger, et al., 2008; VonRueden, et al., 2008).

Pathophysiology

Although all organ systems suffer from hypoperfusion at this stage, several events perpetuate the shock syndrome. First, the overworked heart becomes dysfunctional, the body's inability to meet increased oxygen requirements produces ischemia, and biochemical mediators cause myocardial depression (Dellinger, et al., 2008; Otero, et al., 2006; VonRueden, et al., 2008). This leads to failure of the cardiac pump, even if the underlying cause of the shock is not of cardiac origin. Second, the autoregulatory function of the microcirculation fails in response to the numerous biochemical mediators released by the cells, resulting in increased capillary permeability, with areas of arteriolar and venous constriction further compromising cellular perfusion (King, 2007; VonRueden, et al., 2008). At this stage, the prognosis worsens. The relaxation of precapillary sphincters causes fluid to leak from the capillaries, creating interstitial edema and return of less fluid to the heart. In addition, the inflammatory response to injury is activated, and proinflammatory and anti-inflammatory mediators are released, which activate the coagulation system in an effort to reestablish homeostasis (King, 2007). The body mobilizes energy stores and increases oxygen consumption to meet the increased metabolic needs of the underperfused tissues and cells.

Even if the underlying cause of the shock is reversed, the sequence of compensatory responses to the decrease in tissue perfusion perpetuates the shock state, and a vicious circle ensues. The cellular reactions that occur during the progressive stage of shock are an active area of clinical research. It is believed that the body's response to shock or lack of response in this stage of shock may be the primary factor determining the patient's survival.

Clinical Manifestations

Chances of survival depend on the patient's general health before the shock state as well as the amount of time it takes to restore tissue perfusion. As shock progresses, organ systems decompensate.

Respiratory Effects

The lungs, which become compromised early in shock, are affected at this stage. Subsequent decompensation of the lungs increases the likelihood that mechanical ventilation will be needed. Respirations are rapid and shallow. Crackles are heard over the lung fields. Decreased pulmonary blood flow causes arterial oxygen levels to decrease and carbon dioxide levels to increase. Hypoxemia and biochemical mediators cause an intense inflammatory response and pulmonary vasoconstriction, perpetuating pulmonary capillary hypoperfusion and hypoxemia. The hypoperfused alveoli

stop producing surfactant and subsequently collapse. Pulmonary capillaries begin to leak, causing pulmonary edema, diffusion abnormalities (shunting), and additional alveolar collapse. This condition is called acute lung injury (ALI); as ALI continues, interstitial inflammation and fibrosis are common consequences, leading to acute respiratory distress syndrome (ARDS) (Cocci, et al., 2007; Girard, Kess, Fuchs, et al., 2008; Villar, Perez-Mendez, Lopez, et al., 2007). Further explanation of ALI and ARDS, as well as their nursing management, can be found in Chapter 23.

Cardiovascular Effects

A lack of adequate blood supply leads to dysrhythmias and ischemia. The heart rate is rapid, sometimes exceeding 150 bpm. The patient may complain of chest pain and even suffer a myocardial infarction. Levels of cardiac enzymes (eg, myocardial creatine kinase [CK-MB] and cardiac troponin I [cTn-I]) increase. In addition, myocardial depression and ventricular dilation may further impair the heart's ability to pump enough blood to the tissues to meet oxygen requirements.

New laboratory markers can be used to assess the function of the heart. B-type natriuretic peptide (BNP) is one of these markers. BNP is increased when the ventricle is overdistended; therefore, elevations in BNP can be used to assess ventricular function of patients in shock states (Wilson Tang & Francis, 2005).

Neurologic Effects

As blood flow to the brain becomes impaired, mental status deteriorates. Changes in mental status occur with decreased cerebral perfusion and hypoxia. Initially, the patient may exhibit subtle changes in behavior or agitation and confusion. Subsequently, lethargy increases, and the patient begins to lose consciousness.

Renal Effects

When the MAP falls below 70 mm Hg (Cottingham, 2006; Dellinger, et al., 2008; King, 2007; Pinsky, 2007), the glomerular filtration rate of the kidneys cannot be maintained, and drastic changes in renal function occur. Acute renal failure (ARF) may develop. ARF is characterized by an increase in blood urea nitrogen (BUN) and serum creatinine levels, fluid and electrolyte shifts, acid–base imbalances, and a loss of the renal-hormonal regulation of BP. Urinary output usually decreases to less than 0.5 mL/kg/h (or less than 30 mL/h) but may vary depending on the phase of ARF. For further information about ARF, see Chapter 44.

Hepatic Effects

Decreased blood flow to the liver impairs the ability of liver cells to perform metabolic and phagocytic functions. Consequently, the patient is less able to metabolize medications and metabolic waste products, such as ammonia and lactic acid. Metabolic activities of the liver, including gluconeogenesis and glycogenolysis, are impaired. The patient becomes more susceptible to infection as the liver fails to filter bacteria from the blood. Liver enzymes (aspartate aminotransferase [AST], alanine aminotransferase [ALT], lactate dehydrogenase [LDH]) and bilirubin levels are elevated, and the patient appears jaundiced.

Gastrointestinal Effects

Gastrointestinal (GI) ischemia can cause stress ulcers in the stomach, putting the patient at risk for GI bleeding. In the small intestine, the mucosa can become necrotic and slough off, causing bloody diarrhea. Beyond the local effects of impaired perfusion, GI ischemia leads to bacterial toxin translocation, in which bacterial toxins enter the bloodstream through the lymphatic system. In addition to causing infection, bacterial toxins can cause cardiac depression, vasodilation, increased capillary permeability, and an intense inflammatory response with activation of additional biochemical mediators. The net result is interference with healthy cellular functioning and their ability to metabolize nutrients (Stapleton, Jones & Heyland, 2007).

Hematologic Effects

The combination of hypotension, sluggish blood flow, metabolic acidosis, coagulation system imbalance, and generalized hypoxemia can interfere with normal hemostatic mechanisms. In shock states, the inflammatory cytokines activate the clotting cascade, causing deposition of microthrombi in multiple areas of the body and consumption of clotting factors. The alterations of the hematologic system, including imbalance of the clotting cascade, are linked to the overactivation of the inflammatory response of injury (Remick, 2007a; VonRueden, et al., 2008). Disseminated intravascular coagulation (DIC) may occur either as a cause or as a complication of shock. In this condition, widespread clotting and bleeding occur simultaneously. Bruises (ecchymoses) and bleeding (petechiae) may appear in the skin. Coagulation times (eg, prothrombin time [PT], activated partial thromboplastin time [aPTT]) are prolonged. Clotting factors and platelets are consumed and require replacement therapy to achieve hemostasis. Further discussion of DIC appears in Chapter 33.

Medical Management

Specific medical management in the progressive stage of shock depends on the type of shock and its underlying cause. It is also based on the degree of decompensation in the organ systems. Medical management specific to each type of shock is discussed later in this chapter. Although there are several differences in medical management by type of shock, some medical interventions are common to all types. These include the use of appropriate IV fluids and medications to restore tissue perfusion by the following methods:

- Supporting the respiratory system
- Optimizing intravascular volume
- Supporting the pumping action of the heart
- Improving the competence of the vascular system

Other aspects of management may include early enteral nutritional support, aggressive hyperglycemic control with IV insulin (Hafidh, Reuter, Chassels, et al., 2007; Vanhorebeek, Langouche & Van den Berghe, 2007), and use of antacids, histamine-2 (H_2) blockers, or antipeptic agents to reduce the risk of GI ulceration and bleeding.

NURSING ALERT

Tight glycemic control (blood glucose, 80 to 110 mg/dL) has been shown to reduce morbidity and mortality of acutely ill patients.

Nursing Management

Nursing care of patients in the progressive stage of shock requires expertise in assessing and understanding shock and the significance of changes in assessment data. Early interventions are essential to the survival of patients; therefore, suspecting that a patient may be in shock and reporting subtle changes in assessment are imperative. Patients in the progressive stage of shock are cared for in the intensive care setting to facilitate close monitoring (hemodynamic monitoring, electrocardiographic [ECG] monitoring, arterial blood gases, serum electrolyte levels, physical and mental status changes); rapid and frequent administration of various prescribed medications and fluids; and possibly interventions with supportive technologies, such as mechanical ventilation, dialysis, and intra-aortic balloon pump.

Working closely with other members of the health care team, the nurse carefully documents treatments, medications, and fluids that are administered, recording the time, dosage or volume, and patient response. In addition, the nurse coordinates both the scheduling of diagnostic procedures that may be carried out at the bedside and the flow of health care personnel involved in the care of patients.

Preventing Complications

The nurse helps reduce the risk of related complications and monitors the patient for early signs of complications. Monitoring includes evaluating blood levels of medications, observing invasive vascular lines for signs of infection, and checking neurovascular status if arterial lines are inserted, especially in the lower extremities. Simultaneously, the nurse promotes the patient's safety and comfort by ensuring that all procedures, including invasive procedures and arterial and venous punctures, are carried out using correct aseptic techniques and that venous and arterial puncture and infusion sites are maintained with the goal of preventing infection. Nursing interventions that reduce the incidence of ventilator-associated pneumonias must also be implemented. These include frequent oral care, aseptic suction technique, turning, and elevating the head of the bed at least 30 degrees to prevent aspiration (Carson, Tyner, Sanders, et al., 2007; Dellinger, et al., 2008). Positioning and repositioning of the patient to promote comfort and maintain skin integrity are essential.

Promoting Rest and Comfort

Efforts are made to minimize the cardiac workload by reducing the patient's physical activity and treating pain and anxiety. Promoting patient rest and comfort is a priority. To ensure that the patient obtains as much uninterrupted rest as possible, the nurse performs only essential nursing activities. To conserve the patient's energy, the nurse should protect the patient from temperature extremes (eg, excessive warmth or cold, and shivering), which can increase the metabolic rate and oxygen consumption and thus the cardiac workload. The patient should not be warmed too quickly, and warming blankets should not be applied, because they can cause vasodilation and a subsequent drop in BP.

Supporting Family Members

Because patients in shock receive intense attention by the health care team, families may be overwhelmed and frightened. Family members may be reluctant to ask questions or seek information for fear that they will be in the way or will interfere with the attention given to the patient. The nurse should make sure that the family is comfortably situated and kept informed about the patient's status. Often, families need advice from the health care team to get some rest; family members are more likely to take this advice if they feel that the patient is being well cared for and that they will be notified of any significant changes in the patient's status. A visit from the hospital chaplain may be comforting and provides some attention to the family while the nurse concentrates on the patient.

IRREVERSIBLE STAGE

The irreversible (or refractory) stage of shock represents the point along the shock continuum at which organ damage is so severe that the patient does not respond to treatment and cannot survive. Despite treatment, BP remains low. Renal and liver failure, compounded by the release of necrotic tissue toxins, creates an overwhelming metabolic acidosis. Anaerobic metabolism contributes to a worsening lactic acidosis. Reserves of ATP are almost totally depleted, and mechanisms for storing new supplies of energy have been destroyed. Respiratory system failure prevents adequate oxygenation and ventilation despite mechanical ventilatory support, and the cardiovascular system is ineffective in maintaining an adequate MAP for perfusion. Multiple organ dysfunction progressing to complete organ failure has occurred, and death is imminent. Multiple organ dysfunction can occur as a progression along the shock continuum or as a syndrome unto itself and is described in more detail later in this chapter.

Medical Management

Medical management during the irreversible stage of shock is usually the same as for the progressive stage. Although the patient may have progressed to the irreversible stage, the judgment that the shock is irreversible can be made only retrospectively on the basis of the patient's failure to respond to treatment. Strategies that may be experimental (eg, investigational medications, such as antibiotic agents and immunomodulation therapy) may be tried to reduce or reverse the severity of shock.

Nursing Management

As in the progressive stage of shock, the nurse focuses on carrying out prescribed treatments, monitoring the patient, preventing complications, protecting the patient from injury, and providing comfort. Offering brief explanations to the patient

about what is happening is essential even if there is no certainty that the patient hears or understands what is being said. Simple comfort measures, including reassuring touches, should continue to be provided despite the patient's nonresponsiveness to verbal stimuli (Benner, 2004, Duran, et al., 2007).

As it becomes obvious that the patient is unlikely to survive, the family must be informed about the prognosis and likely outcome. Opportunities should be provided throughout the patient's care for the family to see, touch, and talk to the patient. Close family friends or spiritual advisors may be of comfort to the family members in dealing with the inevitable death of their loved one. Whenever possible and appropriate, the patient's family should be approached regarding any living wills, advance directives, or other written or verbal wishes the patient may have shared in the event that he or she became unable to participate in end-of-life decisions. In some cases, ethics committees may assist families and health care teams in making difficult decisions.

During this stage of shock, the family may misinterpret the actions of the health care team. They have been told that nothing has been effective in reversing the shock and that the patient's survival is very unlikely, yet they find physicians and nurses continuing to work feverishly on the patient. Distraught, grieving families may interpret this as a chance for recovery when none exists, and family members may become angry when the patient dies. Conferences with all members of the health care team and the family promote better understanding by the family of the patient's prognosis and the purpose for management measures. During these conferences, it is essential to explain that the equipment and treatments being provided are intended for patient comfort and do not suggest that the patient will recover. Family members should be encouraged to express their wishes concerning the use of life-support measures.

General Management Strategies in Shock

As described previously and in the discussion of types of shock to follow, management in all types and all phases of shock includes the following:

- Support of the respiratory system with supplemental oxygen and/or mechanical ventilation to provide optimal oxygenation (see Chapter 25)
- Fluid replacement to restore intravascular volume
- Vasoactive medications to restore vasomotor tone and improve cardiac function
- Nutritional support to address the metabolic requirements that are often dramatically increased in shock

Therapies described in this section require collaboration among all members of the health care team to ensure that the manifestations of shock are quickly identified and that adequate and timely treatment is instituted to achieve the best outcome possible.

Fluid Replacement

Fluid replacement, also referred to as fluid resuscitation, is administered in all types of shock. The type of fluids administered and the speed of delivery vary, but fluids are administered to improve cardiac and tissue oxygenation, which in part depends on flow. The fluids administered may include **crystalloids** (electrolyte solutions that move freely between intravascular and interstitial spaces), **colloids** (large-molecule IV solutions), and blood components (packed red blood cells, fresh frozen plasma, and platelets).

Crystalloid and Colloid Solutions

The best fluid to treat shock remains controversial. In emergencies, the "best" fluid is often the fluid that is readily available. Fluid resuscitation should be initiated early in shock to maximize intravascular volume. There is no consensus regarding whether crystalloids or colloids should be used; however, with crystalloids, more fluid is necessary to restore intravascular volume (Roberts, Alderson, Bunn, et al., 2007).

Crystalloids are electrolyte solutions that move freely between the intravascular compartment and the interstitial spaces. Isotonic crystalloid solutions are often selected because they contain the same concentration of electrolytes as the extracellular fluid and therefore can be given without altering the concentrations of electrolytes in the plasma. IV crystalloids commonly used for resuscitation in hypovolemic shock include 0.9% sodium chloride solution (normal saline) and lactated Ringer's solution (Boswell & Scalea, 2008; Cottingham, 2006). Ringer's lactate is an electrolyte solution containing the lactate ion, which should not be confused with lactic acid. The lactate ion is converted to bicarbonate, which helps buffer the overall acidosis that occurs in shock. A disadvantage of using isotonic crystalloid solutions is that some of the volume administered is lost to the interstitial compartment and some remains in the intravascular compartment. This occurs as a consequence of cellular permeability that occurs during shock. Diffusion of crystalloids into the interstitial space means that more fluid must be administered than the amount lost (Cottingham, 2006; Roberts, et al., 2007).

Care must be taken when rapidly administering isotonic crystalloids to avoid both underresuscitating and overresuscitating the patient in shock. Insufficient fluid replacement is associated with a higher incidence of morbidity and mortality from lack of tissue perfusion, whereas excessive fluid administration can cause systemic and pulmonary edema that progresses to ARDS, abdominal compartment syndrome, and multiple organ dysfunction syndrome (MODS).

Depending on the cause of the hypovolemia, a hypertonic crystalloid solution, such as 3% sodium chloride, is sometimes administered in hypovolemic shock. These solutions exert a large osmotic force that pulls fluid from the intracellular space to the extracellular space to achieve a fluid balance (Cottingham, 2006). This osmotic effect results in fewer fluids being administered to restore intravascular volume. Complications associated with use of hypertonic solutions include excessive serum osmolality, which can cause rapid fluid shifts overwhelming the heart, and hypernatremia.

Generally, IV colloidal solutions are similar to plasma proteins, in that they contain molecules that are too large to pass through capillary membranes. Colloids expand intravascular volume by exerting oncotic pressure, thereby pulling fluid into the intravascular space. Colloidal solutions

have the same effect as hypertonic solutions in increasing intravascular volume, but less volume of fluid is required than with crystalloids. In addition, colloids have a longer duration of action than crystalloids, because the molecules remain within the intravascular compartment longer.

Typically, if colloids are used to treat tissue hypoperfusion, albumin is the agent prescribed. Albumin is a plasma protein; an albumin solution is prepared from human plasma and is heated during production to reduce its potential to transmit disease. The disadvantage of albumin is its high cost compared to crystalloid solutions. Synthetic colloid preparations, such as hetastarch and dextran solution, may also be used for colloid infusions; however, dextran may interfere with platelet aggregation and, therefore, is not indicated if hemorrhage is the cause of the hypovolemic shock or if the patient has a coagulation disorder.

NURSING ALERT

With all colloidal solutions, side effects include the rare occurrence of anaphylactic reactions. Nurses must monitor patients closely.

Complications of Fluid Administration

Close monitoring of the patient during fluid replacement is necessary to identify side effects and complications. The most common and serious side effects of fluid replacement are cardiovascular overload and pulmonary edema. The patient receiving fluid replacement must be monitored frequently for adequate urinary output, changes in mental status, skin perfusion, and changes in vital signs. Lung sounds are auscultated frequently to detect signs of fluid accumulation. Adventitious lung sounds, such as crackles, may indicate pulmonary edema.

Abdominal compartment syndrome (ACS) is a serious complication that may occur when large volumes of fluid are administered. It may also occur after trauma, abdominal surgery, severe pancreatitis, or sepsis (Brush, 2007). In ACS, fluid leaks into the intra-abdominal cavity, increasing pressure that is displaced onto surrounding vessels and organs. Venous return, preload, and cardiac output are compromised. The pressure also elevates the diaphragm, making it difficult to breathe effectively. The renal system and GI systems also begin to show signs of dysfunction (eg, decreased urine output, absent bowel sounds, intolerance of tube feeding). Abdominal compartment pressure can be measured. Normally, it is 0 to 5 mm Hg, and a pressure of 12 mm Hg is considered to be indicative of intra-abdominal hypertension (Brush, 2007). If ACS is present, interventions that usually include surgical decompression are necessary to relieve the pressure.

NURSING ALERT

When administering large volumes of crystalloid solutions, monitor the lungs for adventitious sounds and signs and symptoms of interstitial edema (eg, abdominal compartment syndrome).

Often a right atrial pressure line (also known as a central venous pressure [CVP] line) is inserted. In addition to physical assessment, the right atrial pressure value helps in monitoring the patient's response to fluid replacement. A normal right atrial pressure value or CVP is 4 to 12 mm Hg or cm H_2O. Several readings are obtained to determine a range, and fluid replacement is continued to achieve a CVP of at least 8 mm Hg (Dellinger, et al., 2008). With newer technologies, right atrial catheters can be placed that allow the monitoring of intravascular pressures and venous oxygen levels. Assessment of venous oxygenation (venous oxygen saturation ([SvO_2], or $Sc\bar{v}O_2$ with a CVP line) is helpful in evaluating the adequacy of intravascular volume (Goodrich, 2006; Rivers, et al., 2005). Hemodynamic monitoring with arterial and pulmonary artery lines may be implemented to allow close monitoring of the patient's perfusion and cardiac status as well as response to therapy. For additional information about hemodynamic monitoring, see Chapter 26.

Vasoactive Medication Therapy

Vasoactive medications are administered in all forms of shock to improve the patient's hemodynamic stability when fluid therapy alone cannot maintain adequate MAP. Specific medications are selected to correct the particular hemodynamic alteration that is impeding cardiac output. These medications help increase the strength of myocardial contractility, regulate the heart rate, reduce myocardial resistance, and initiate vasoconstriction.

Vasoactive medications are selected for their action on receptors of the sympathetic nervous system. These receptors are known as alpha-adrenergic and beta-adrenergic receptors. Beta-adrenergic receptors are further classified as beta-1 and beta-2 adrenergic receptors. When alpha-adrenergic receptors are stimulated, blood vessels constrict in the cardiorespiratory and GI systems, skin, and kidneys. When beta-1 adrenergic receptors are stimulated, heart rate and myocardial contraction increase. When beta-2 adrenergic receptors are stimulated, vasodilation occurs in the heart and skeletal muscles, and the bronchioles relax. The medications used in treating shock consist of various combinations of vasoactive medications to maximize tissue perfusion by stimulating or blocking the alpha- and beta-adrenergic receptors.

When vasoactive medications are administered, vital signs must be monitored frequently (at least every 15 minutes until stable, or more often if indicated). Vasoactive medications should be administered through a central venous line, because infiltration and extravasation of some vasoactive medications can cause tissue necrosis and sloughing. An IV pump or controller should be used to ensure that the medications are delivered safely and accurately.

Individual medication dosages are usually titrated by the nurse, who adjusts drip rates based on the prescribed dose and the patient's response. Dosages are changed to maintain the MAP at a physiologic level that ensures adequate tissue perfusion (usually greater than 65 mm Hg).

NURSING ALERT

Vasoactive medications should never be stopped abruptly, because this could cause severe hemodynamic instability, perpetuating the shock state.

Dosages of vasoactive medications should be tapered, and the patient should be weaned from medication with frequent monitoring of BP (every 15 minutes). Table 15-2 presents some of the commonly prescribed vasoactive medications used in the treatment of shock. Occasionally, the patient does not respond as expected to vasoactive medications. A current topic of active research is evaluation of patients' adrenal function. Recent studies suggest that critically ill patients should be evaluated for corticosteroid insufficiency, and if this condition is present, corticosteroid replacement (eg, hydrocortisone) should be initiated (Dellinger, et al., 2008).

Nutritional Support

Nutritional support is an important aspect of care for patients with shock. Increased metabolic rates during shock increase energy requirements and therefore caloric requirements. Patients in shock may require more than 3000 calories daily. The release of catecholamines early in the shock continuum causes depletion of glycogen stores in about 8 to 10 hours. Nutritional energy requirements are then met by breaking down lean body mass. In this catabolic process, skeletal muscle mass is broken down even when the patient has large stores of fat or adipose tissue. Loss of skeletal muscle greatly prolongs the patient's recovery time.

Parenteral or enteral nutritional support should be initiated as soon as possible. Enteral nutrition is preferred, promoting GI function through direct exposure to nutrients and limiting infectious complications associated with parenteral feeding (Vincent, 2007). In addition, glutamine (an essential amino acid during stress), which may be administered in enteral formulas, is important in the immunologic function of the GI tract, providing a fuel source for lymphocytes and macrophages (Stapleton, et al., 2007).

Stress ulcers occur frequently in acutely ill patients because of the compromised blood supply to the GI tract. Therefore, antacids, H_2 blockers (eg, famotidine [Pepcid], ranitidine [Zantac]), and proton pump inhibitors (eg, lansoprazole [Prevacid]) are prescribed to prevent ulcer formation by inhibiting gastric acid secretion or increasing gastric pH.

Hypovolemic Shock

Nurses who care for patients in the different stages of shock must tailor interventions to the type of shock, whether hypovolemic, cardiogenic, or circulatory shock. **Hypovolemic shock**, the most common type of shock, is characterized by decreased intravascular volume. Body fluid is contained in the intracellular and extracellular compartments. Intracellular fluid accounts for about two thirds of the total body water. The extracellular body fluid is found in one of two compartments: intravascular (inside blood vessels) or interstitial (surrounding tissues). The volume of interstitial fluid is about three to four times that of intravascular fluid. Hypovolemic shock occurs when there is a reduction in intravascular volume by 15% to 30%, which represents a loss of 750 to 1500 mL of blood in a 70-kg (154-lb) person (American College of Surgeons, 2006).

Pathophysiology

Hypovolemic shock can be caused by external fluid losses, as in traumatic blood loss, or by internal fluid shifts, as in severe dehydration, severe edema, or ascites (Chart 15-2). Intravascular volume can be reduced both by fluid loss and by fluid shifting between the intravascular and interstitial compartments.

The sequence of events in hypovolemic shock begins with a decrease in the intravascular volume. This results in decreased venous return of blood to the heart and subsequent decreased ventricular filling. Decreased ventricular filling results in decreased stroke volume (amount of blood ejected from the heart) and decreased cardiac output. When cardiac output drops, BP drops and tissues cannot be adequately perfused (Fig. 15-3).

Table 15-2 VASOACTIVE AGENTS USED IN TREATING SHOCK

Medication	Desired Action in Shock	Disadvantages
Inotropic Agents Dobutamine (Dobutrex) Dopamine (Intropin) Epinephrine (Adrenalin) Milrinone (Primacor)	Improve contractility, increase stroke volume, increase cardiac output	Increase oxygen demand of the heart
Vasodilators Nitroglycerin (Tridil) Nitroprusside (Nipride)	Reduce preload and afterload, reduce oxygen demand of heart	Cause hypotension
Vasopressor Agents Norepinephrine (Levophed) Dopamine (Intropin) Phenylephrine (Neo-Synephrine) Vasopressin (Pitressin)	Increase blood pressure by vasoconstriction	Increase afterload, thereby increasing cardiac workload; compromise perfusion to skin, kidneys, lungs, gastrointestinal tract

CHART 15-2 *Risk Factors for Hypovolemic Shock*

External: Fluid Losses	Internal: Fluid Shifts
• Trauma	• Hemorrhage
• Surgery	• Burns
• Vomiting	• Ascites
• Diarrhea	• Peritonitis
• Diuresis	• Dehydration
• Diabetes insipidus	

Medical Management

Major goals in the treatment of hypovolemic shock are to restore intravascular volume to reverse the sequence of events leading to inadequate tissue perfusion, to redistribute fluid volume, and to correct the underlying cause of the fluid loss as quickly as possible. Depending on the severity of shock and the patient's condition, it is likely that efforts will be made to address all three goals simultaneously.

Treatment of the Underlying Cause

If the patient is hemorrhaging, efforts are made to stop the bleeding. This may involve applying pressure to the bleeding site or surgical interventions to stop internal bleeding. If the cause of the hypovolemia is diarrhea or vomiting, medications to treat diarrhea and vomiting are administered while efforts are made to identify and treat the cause.

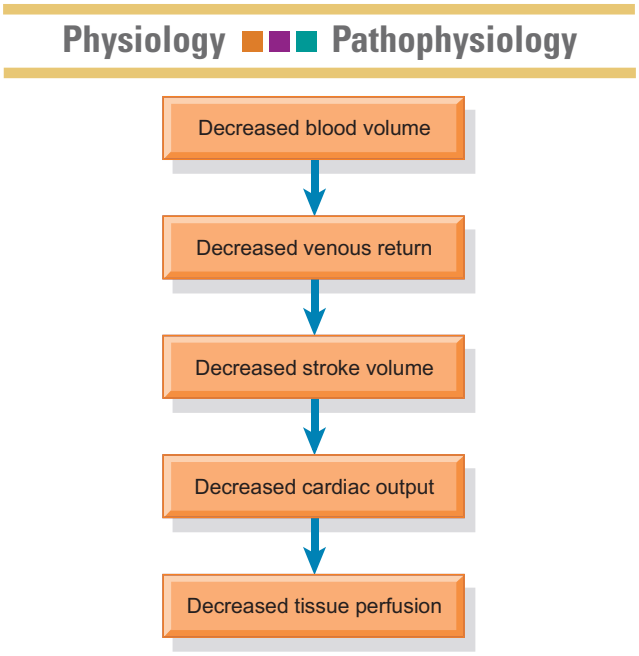

Figure 15-3 Pathophysiologic sequence of events in hypovolemic shock.

In elderly patients, dehydration may be the cause of hypovolemic shock.

Fluid and Blood Replacement

Beyond reversing the primary cause of the decreased intravascular volume, fluid replacement is of primary concern. At least two large-gauge IV lines are inserted to establish access for fluid administration. Two IV lines allow simultaneous administration of fluid, medications, and blood component therapy if required. Because the goal of the fluid replacement is to restore intravascular volume, it is necessary to administer fluids that will remain in the intravascular compartment to avoid fluid shifts from the intravascular compartment into the intracellular compartment. Table 15-3 summarizes the fluids commonly used in the treatment of shock.

As discussed earlier, crystalloid solutions such as lactated Ringer's solution or 0.9% sodium chloride solution are commonly used to treat hypovolemic shock as large amounts of fluid must be administered to restore intravascular volume. If hypovolemia is primarily due to blood loss, the American College of Surgeons recommends administration of 3 mL of crystalloid solution for each milliliter of estimated blood loss. This is referred to as the 3:1 rule (American College of Surgeons, 2006). Colloid solutions (eg, albumin, hetastarch) may also be used. Dextran is not indicated if the cause of the hypovolemic shock is hemorrhage, because it interferes with platelet aggregation.

Blood products, which are also colloids, may need to be administered, particularly if the cause of the hypovolemic shock is hemorrhage. The decision to give blood is based on the patient's lack of response to only crystalloid resuscitation, the volume of blood lost, the need for hemoglobin to assist with oxygen transport, and the necessity to correct the patient's coagulopathy. It should be noted that research indicates that patients who receive massive blood transfusions to achieve near-normal hemoglobin levels tend to have poorer outcomes than those with low hemoglobin levels (eg, less than 7.0 g/dL) (Dellinger, et al., 2008; Holcomb & Hess, 2006). Packed red blood cells are administered to replenish the patient's oxygen-carrying capacity in conjunction with other fluids that will expand volume. Currently, the need for transfusions is based on the patient's oxygenation needs, which are determined by vital signs, blood gas values, and clinical appearance rather than an arbitrary laboratory value. An area of active research is the development of synthetic forms of blood (ie, compounds capable of carrying oxygen in the same way that blood does) as potential alternatives to blood component therapy.

Redistribution of Fluid

In addition to administering fluids to restore intravascular volume, positioning the patient properly assists fluid redistribution. A modified Trendelenburg position (Fig. 15-4) is recommended in hypovolemic shock. Elevation of the legs promotes the return of venous blood. A full Trendelenburg position makes breathing difficult and does not increase BP or cardiac output (Bridges & Jarquin-Valdivia, 2005).

Table 15-3 FLUID REPLACEMENT IN SHOCK

Deliver a minimum of 20 mL/kg of crystalloid (or colloid equivalent).

Fluids	Advantages	Disadvantages
Crystalloids		
0.9% sodium chloride (normal saline solution)	Widely available, inexpensive	Requires large volume of infusion; can cause hypernatremia, pulmonary edema, abdominal compartment syndrome
Lactated Ringer's	Lactate ion helps buffer metabolic acidosis	Requires large volume of infusion; can cause metabolic acidosis, pulmonary edema, abdominal compartment syndrome
Hypertonic saline (3%)	Small volume needed to restore intravascular volume	Danger of hypernatremia and cardiovascular compromise from rapid fluid shifts
Colloids		
Albumin (5%, 25%)	Rapidly expands plasma volume	Expensive; requires human donors; limited supply; can cause heart failure
Dextran	Synthetic plasma expander	Interferes with platelet aggregation; not recommended for hemorrhagic shock
Hetastarch	Synthetic plasma expander	Prolongs bleeding and clotting times

Pharmacologic Therapy

If fluid administration fails to reverse hypovolemic shock, then vasoactive medications that prevent cardiac failure are given. Medications are also administered to reverse the cause of the dehydration. For example, insulin is administered if dehydration is secondary to hyperglycemia, desmopressin (DDAVP) is administered for diabetes insipidus, antidiarrheal agents for diarrhea, and antiemetic medications for vomiting.

Nursing Management

Primary prevention of shock is an essential focus of nursing care. Hypovolemic shock can be prevented in some instances by closely monitoring patients who are at risk for fluid deficits and assisting with fluid replacement before intravascular volume is depleted. In other circumstances, nursing care focuses on assisting with treatment targeted at the cause of the shock and restoring intravascular volume.

General nursing measures include ensuring safe administration of prescribed fluids and medications and documenting their administration and effects. Another important nursing role is monitoring for complications and side effects of treatment and reporting them promptly.

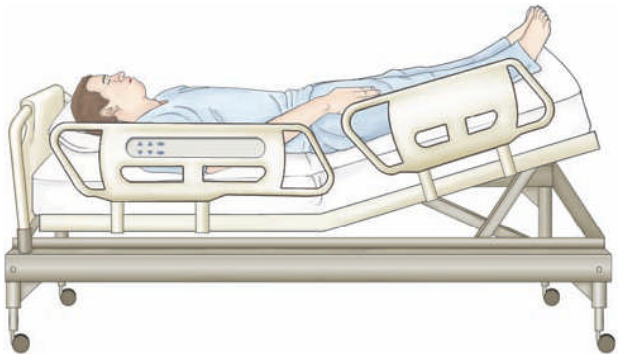

Figure 15-4 Proper positioning (modified Trendelenburg) for the patient who shows signs of shock. The lower extremities are elevated to an angle of about 20 degrees; the knees are straight, the trunk is horizontal, and the head is slightly elevated.

Administering Blood and Fluids Safely

Administering blood transfusions safely is a vital nursing role. In emergency situations, it is important to acquire blood specimens quickly, to obtain a baseline complete blood count, and to type and cross-match the blood in anticipation of blood transfusions. A patient who receives a transfusion of blood products must be monitored closely for adverse effects (see Chapter 33).

Fluid replacement complications can occur, often when large volumes are administered rapidly. Therefore, the nurse monitors the patient closely for cardiovascular overload, signs of difficulty breathing, and pulmonary edema. The risk of these complications is increased in the elderly and in patients with preexisting cardiac disease. Hemodynamic pressures, vital signs, arterial blood gases, serum lactate levels, hemoglobin and hematocrit levels, and fluid intake and output (I&O) are among the parameters monitored. Temperature should also be monitored closely to ensure that rapid fluid resuscitation does not cause hypothermia. IV fluids may need to be warmed during the administration of large volumes. Physical assessment focuses on observing the jugular veins for distention and monitoring jugular venous pressure. Jugular venous pressure is low in hypovolemic shock; it increases with effective treatment and is significantly increased with fluid overload and heart failure. The nurse must monitor cardiac and respiratory status closely and report changes in BP, pulse pressure, CVP, heart rate and rhythm, and lung sounds to the physician.

Implementing Other Measures

Oxygen is administered to increase the amount of oxygen carried by available hemoglobin in the blood. A patient who is confused may feel apprehensive with an oxygen mask or cannula in place, and frequent explanations about the need for the mask may reduce some of the patient's fear and anxiety. Simultaneously, the nurse must direct efforts to the safety and comfort of the patient.

Cardiogenic Shock

Cardiogenic shock occurs when the heart's ability to contract and to pump blood is impaired and the supply of oxygen is inadequate for the heart and tissues. The causes of cardiogenic shock are known as either coronary or noncoronary. Coronary cardiogenic shock is more common than noncoronary cardiogenic shock and is seen most often in patients with acute myocardial infarction (MI) resulting in damage to a significant portion of the left ventricular myocardium (Aymong, Ramanathan & Buller, 2007). Patients who experience an anterior wall MI are at greatest risk for cardiogenic shock because of the potentially extensive damage to the left ventricle caused by occlusion of the left anterior descending coronary artery. Noncoronary causes of cardiogenic shock are related to conditions that stress the myocardium (eg, severe hypoxemia, acidosis, hypoglycemia, hypocalcemia, and tension pneumothorax) as well as conditions that result in ineffective myocardial function (eg, cardiomyopathies, valvular damage, cardiac tamponade, dysrhythmias).

Pathophysiology

In cardiogenic shock, cardiac output, which is a function of both stroke volume and heart rate, is compromised. When stroke volume and heart rate decrease or become erratic, BP falls and tissue perfusion is reduced. Blood supply for tissues and organs and for the heart muscle itself is inadequate, resulting in impaired tissue perfusion. Because impaired tissue perfusion weakens the heart and impairs its ability to pump, the ventricle does not fully eject its volume of blood at systole. As a result, fluid accumulates in the lungs. This sequence of events can occur rapidly or over a period of days (Fig. 15-5).

Clinical Manifestations

Patients in cardiogenic shock may experience the pain of angina, develop dysrhythmias, complain of fatigue, express feelings of doom, and show signs of hemodynamic instability.

Medical Management

The goals of medical management in cardiogenic shock are to limit further myocardial damage and preserve the healthy myocardium and to improve the cardiac function by increasing cardiac contractility, decreasing ventricular afterload, or both (Aymong, et al., 2007; Iakobishvili & Hasdai, 2007; Mann & Nolan, 2006). In general, these goals are achieved by increasing oxygen supply to the heart muscle while reducing oxygen demands.

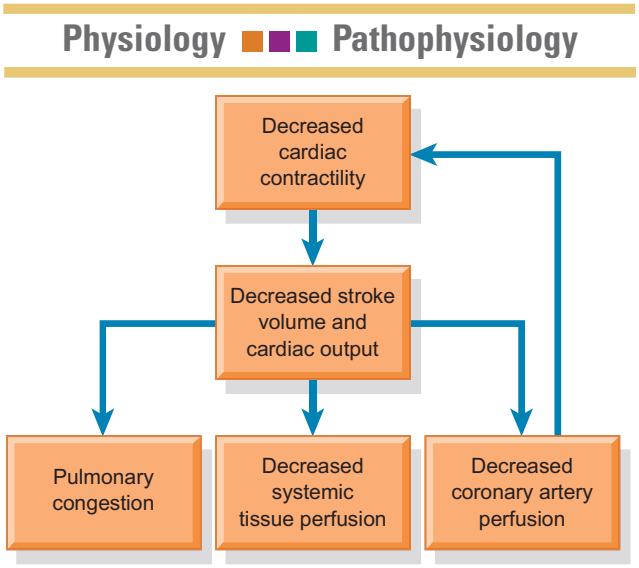

Figure 15-5 Pathophysiologic sequence of events in cardiogenic shock.

Correction of Underlying Causes

As with all forms of shock, the underlying cause of cardiogenic shock must be corrected. It is necessary first to treat the oxygenation needs of the heart muscle to ensure its continued ability to pump blood to other organs. In the case of coronary cardiogenic shock, the patient may require thrombolytic therapy, a percutaneous coronary intervention (PCI), coronary artery bypass graft (CABG) surgery, intraaortic balloon pump therapy, or some combination of these treatments. In the case of noncoronary cardiogenic shock, interventions focus on correcting the underlying cause, such as replacement of a faulty cardiac valve, correction of a dysrhythmia, correction of acidosis and electrolyte disturbances, or treatment of the tension pneumothorax.

Initiation of First-Line Treatment

Oxygenation

In the early stages of shock, supplemental oxygen is administered by nasal cannula at a rate of 2 to 6 L/min to achieve an oxygen saturation exceeding 90%. Monitoring of arterial blood gas values and pulse oximetry values helps determine whether the patient requires a more aggressive method of oxygen delivery.

Pain Control

If a patient experiences chest pain, IV morphine is administered for pain relief. In addition to relieving pain, morphine dilates the blood vessels. This reduces the workload of the heart by both decreasing the cardiac filling pressure (preload) and reducing the pressure against which the heart muscle has to eject blood (afterload). Morphine also decreases the patient's anxiety.

Hemodynamic Monitoring

Hemodynamic monitoring is initiated to assess the patient's response to treatment. In many institutions, this is performed in the intensive care unit (ICU), where an arterial line can be inserted. The arterial line enables accurate and

continuous monitoring of BP and provides a port from which to obtain frequent arterial blood samples without having to perform repeated arterial punctures. A multilumen pulmonary artery catheter is inserted to allow measurement of the pulmonary artery pressures, myocardial filling pressures, cardiac output, and pulmonary and systemic resistance. For more information, see Chapter 30.

Laboratory Marker Monitoring

Laboratory markers for ventricular dysfunction (eg, BNP) and cardiac enzyme levels (CK-MB and cTn-I) are measured, and serial 12-lead ECGs are obtained to assess the degree of myocardial damage. Continuous ECG and ST-segment monitoring is also used to closely monitor the patient for ischemic changes.

Fluid Therapy

Appropriate fluid administration is also necessary in the treatment of cardiogenic shock. Administration of fluids must be monitored closely to detect signs of fluid overload. Incremental IV fluid boluses are cautiously administered to determine optimal filling pressures for improving cardiac output.

NURSING ALERT

A fluid bolus should never be given rapidly, because rapid fluid administration in patients with cardiac failure may result in acute pulmonary edema.

Pharmacologic Therapy

Vasoactive medication therapy consists of multiple pharmacologic strategies to restore and maintain adequate cardiac output. In coronary cardiogenic shock, the aims of vasoactive medication therapy are improved cardiac contractility, decreased preload and afterload, and stabilized heart rate and rhythm.

Because improving contractility and decreasing cardiac workload are opposing pharmacologic actions, two types of medications may be administered in combination: inotropic agents and vasodilators. Inotropic medications increase cardiac output by mimicking the action of the sympathetic nervous system, activating myocardial receptors to increase myocardial contractility (inotropic action) or increasing the heart rate (chronotropic action). These agents may also enhance vascular tone, increasing preload. Vasodilators are used primarily to decrease afterload, reducing the workload of the heart and the oxygen demand. Vasodilators also decrease preload. Medications commonly combined to treat cardiogenic shock include dobutamine, nitroglycerin, and dopamine (see Table 15-2).

Dobutamine. Dobutamine produces inotropic effects by stimulating myocardial beta-receptors, increasing the strength of myocardial activity and improving cardiac output. Myocardial alpha-adrenergic receptors are also stimulated, resulting in decreased pulmonary and systemic vascular resistance (decreased afterload). Dobutamine enhances the strength of cardiac contraction, improving stroke volume ejection and overall cardiac output (Iakobishvili & Hasdai, 2007; Mann & Nolan, 2006).

Nitroglycerin. IV nitroglycerin in low doses acts as a venous vasodilator and therefore reduces preload. At higher doses, nitroglycerin causes arterial vasodilation and therefore reduces afterload as well. These actions, in combination with dobutamine, increase cardiac output while minimizing cardiac workload. In addition, vasodilation enhances blood flow to the myocardium, improving oxygen delivery to the weakened heart muscle (Iakobishvili & Hasdai, 2007).

Dopamine. Dopamine is a sympathomimetic agent that has varying vasoactive effects depending on the dosage. It may be used with dobutamine and nitroglycerin to improve tissue perfusion. Doses of 2 to 8 μg/kg/min improve contractility (inotropic action), slightly increase the heart rate (chronotropic action), and may increase cardiac output. Doses that are higher than 8 μg/kg/min predominantly cause vasoconstriction, which increases afterload and thus increases cardiac workload. Because this effect is undesirable in patients with cardiogenic shock, dopamine doses must be carefully titrated.

Low-dose dopamine (ie, 0.5 to 3.0 μg/kg/min) neither improves renal flow, changes the need for renal support, nor reduces mortality (Freidrich, Adhikari, Herridge, et al., 2005; Iakobishvili & Hasdai, 2007). Thus, low-dose dopamine is no longer recommended. However, some patients respond to lower dosages of dopamine for its inotropic effects (Friedrich, et al., 2005).

In severe metabolic acidosis, which occurs in the later stages of shock, the effectiveness of dopamine is diminished. To maximize the effectiveness of any vasoactive agent, metabolic acidosis must first be corrected (Dellinger, et al., 2008).

Other Vasoactive Medications. Additional vasoactive agents that may be used in managing cardiogenic shock include norepinephrine, epinephrine, milrinone, vasopressin, and phenylephrine. Each of these medications stimulates different receptors of the sympathetic nervous system. A combination of these medications may be prescribed, depending on the patient's response to treatment. All vasoactive medications have adverse effects, making specific medications more useful than others at different stages of shock. Diuretics such as furosemide may be administered to reduce the workload of the heart by reducing fluid accumulation (see Table 15-2).

Antiarrhythmic Medications. Multiple factors, such as hypoxemia, electrolyte imbalances, and acid–base imbalances, contribute to serious cardiac dysrhythmias in all patients with shock. In addition, as a compensatory response to decreased cardiac output and BP, the heart rate increases beyond normal limits. This impedes cardiac output further by shortening diastole and thereby decreasing the time for ventricular filling. Consequently, antiarrhythmic medications are required to stabilize the heart rate. For a full discussion of cardiac dysrhythmias as well as commonly prescribed medications, see Chapter 27. General principles regarding the administration of vasoactive medications are discussed later in this chapter.

Mechanical Assistive Devices

If cardiac output does not improve despite supplemental oxygen, vasoactive medications, and fluid boluses, mechanical assistive devices are used temporarily to improve the heart's ability to pump. Intra-aortic balloon counterpulsation is one means of providing temporary circulatory

assistance (see Chapter 30). Other means of mechanical assistance include left and right ventricular assist devices (VADs) and total temporary artificial hearts (see Chapters 29 and 30). VADs are utilized frequently as bridge therapy to either recovery or heart transplantation. Another short-term means of providing cardiac or pulmonary support to the patient in cardiogenic shock is through an extracorporeal device similar to the cardiopulmonary bypass (CPB) system used in open-heart surgery (see Chapter 28). CPB is used only in emergency situations until definitive treatment, such as heart transplantation, can be initiated.

Nursing Management

Preventing Cardiogenic Shock

Identifying at-risk patients early, promoting adequate oxygenation of the heart muscle, and decreasing cardiac workload can prevent cardiogenic shock. This can be accomplished by conserving the patient's energy, promptly relieving angina, and administering supplemental oxygen. Often, however, cardiogenic shock cannot be prevented. In such instances, nursing management includes working with other members of the health care team to prevent shock from progressing and to restore adequate cardiac function and tissue perfusion.

Monitoring Hemodynamic Status

A major role of the nurse is monitoring the patient's hemodynamic and cardiac status. Arterial lines and ECG monitoring equipment must be well maintained and functioning properly. The nurse anticipates the medications, IV fluids, and equipment that might be used and is ready to assist in implementing these measures. Changes in hemodynamic, cardiac, and pulmonary status and laboratory values are documented and reported promptly. In addition, adventitious breath sounds, changes in cardiac rhythm, and other abnormal physical assessment findings are reported immediately.

Administering Medications and Intravenous Fluids

The nurse plays a critical role in the safe and accurate administration of IV fluids and medications. Fluid overload and pulmonary edema are risks because of ineffective cardiac function and accumulation of blood and fluid in the pulmonary tissues. The nurse documents and records medications and treatments that are administered as well as the patient's response to treatment.

The nurse must be knowledgeable about the desired effects as well as the side effects of medications. For example, it is important to monitor the patient for decreased BP after administering morphine or nitroglycerin. Patients receiving thrombolytic therapy must be monitored for bleeding. Arterial and venous puncture sites must be observed for bleeding, and pressure must be applied at the sites if bleeding occurs. Neurologic assessment is essential after the administration of thrombolytic therapy to assess for the potential complication of cerebral hemorrhage associated with this therapy. IV infusions must be observed closely because tissue necrosis and sloughing may occur if vasopressor medications infiltrate the tissues. It is necessary to monitor urine output, BUN, and serum creatinine levels to detect decreased renal function secondary to the effects of cardiogenic shock or its treatment.

Maintaining Intra-Aortic Balloon Counterpulsation

The nurse plays a critical role in caring for the patient receiving intra-aortic balloon counterpulsation (see Chapter 30). The nurse makes ongoing timing adjustments of the balloon pump to maximize its effectiveness by synchronizing it with the cardiac cycle. The patient is at risk for circulatory compromise to the leg on the side where the catheter for the balloon has been inserted; therefore, the nurse must check the neurovascular status of the lower extremities frequently.

Enhancing Safety and Comfort

Throughout care, the nurse must take an active role in safeguarding the patient, enhancing comfort, and reducing anxiety. This includes administering medication to relieve chest pain, preventing infection at the multiple arterial and venous line insertion sites, protecting the skin, and monitoring respiratory and renal function. Proper positioning of the patient promotes effective breathing without decreasing BP and may also increase patient comfort while reducing anxiety.

Brief explanations about procedures that are being performed and the use of comforting touch often provide reassurance to the patient and family. The family is usually anxious and benefits from opportunities to see and talk to the patient. Explanations of treatments and the patient's responses are often comforting to family members.

Circulatory Shock

Circulatory shock occurs when blood volume pools in peripheral blood vessels. This abnormal displacement of blood volume causes a relative hypovolemia because not enough blood returns to the heart, which leads to inadequate tissue perfusion. The ability of the blood vessels to constrict helps return the blood to the heart. The vascular tone is determined both by central regulatory mechanisms, as in BP regulation, and by local regulatory mechanisms, as in tissue demands for oxygen and nutrients. Therefore, circulatory shock can be caused either by a loss of sympathetic tone or by release of biochemical mediators from cells.

The varied mechanisms leading to the initial vasodilation in circulatory shock provide the basis for the further subclassification of shock into three types: septic shock, neurogenic shock, and anaphylactic shock. These types of circulatory shock cause variations in the pathophysiologic chain of events and are explained here separately. In all types of circulatory shock, massive arterial and venous dilation promotes peripheral pooling of blood. Arterial dilation reduces systemic vascular resistance. Initially, cardiac output can be high, both from the reduction in afterload (systemic vascular resistance) and from the heart muscle's increased effort to maintain perfusion despite the incompetent vasculature. Pooling of blood in the periphery results in decreased venous return. Decreased venous return results in decreased stroke volume and decreased cardiac output. Decreased cardiac output, in turn, causes decreased BP and ultimately decreased tissue perfusion. Figure 15-6 presents the pathophysiologic sequence of events in circulatory shock.

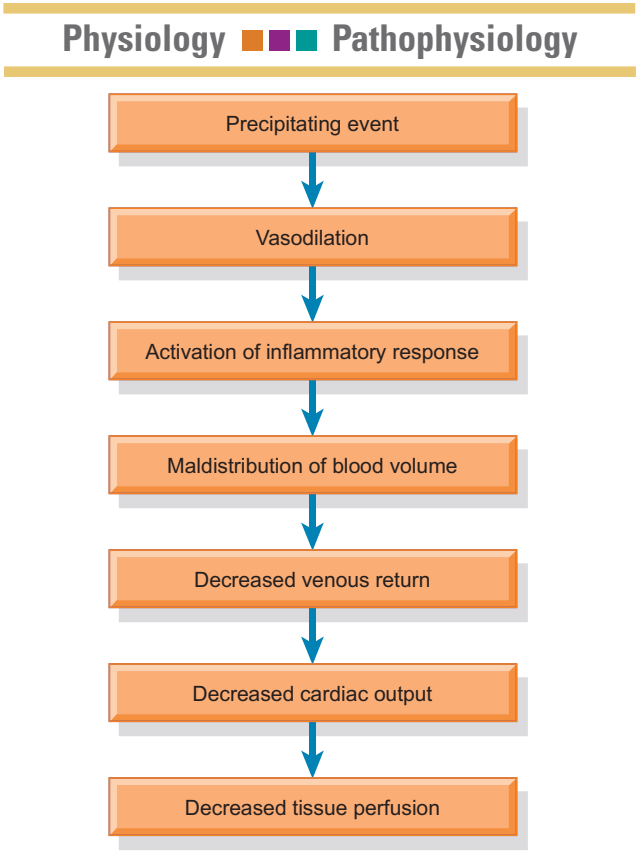

Figure 15-6 Pathophysiologic sequence of events in circulatory shock.

SEPTIC SHOCK

Septic shock, the most common type of circulatory shock, is caused by widespread infection (Chart 15-3). Despite the increased sophistication of antibiotic therapy, the incidence of septic shock has continued to rise during the past 60 years. It is the most common cause of death in noncoronary ICUs in the United States. Each year, severe sepsis affects an estimated 750,000 people in the United States. As the population ages, the rate is expected to rise to 1 million cases a year by 2010 (Surviving Sepsis Campaign, 2007). Finding and aggressively treating the source of infection and quickly restoring tissue perfusion are important interventions that may positively influence the clinical outcome.

Health care–associated infections (infections not incubating at the time of admission to the health care setting) in critically ill patients that may progress to septic shock most frequently originate (in decreasing order of frequency) in the bloodstream (bacteremia), lungs, and urinary tract (urosepsis) (Arugon & Sole, 2006). Other infections include intraabdominal infections and wound infections. Of increasing concern are bacteremias associated with intravascular catheters and indwelling urinary catheters (Aragon & Sole, 2006; Institute for Healthcare Improvement [IHI], 2007).

Additional risk factors that contribute to the growing incidence of septic shock are the increased use of invasive procedures and indwelling medical devices; the increased number of antibiotic-resistant microorganisms; and the increasingly older population (Aragon & Sole, 2006; King, 2007). Elderly patients are at particular risk for sepsis because of decreased physiologic reserves and an aging immune system (Marik, 2006). Other patients at risk are those undergoing surgical and other invasive procedures; those with malnutrition or immunosuppression; and those with chronic illness such as diabetes mellitus, hepatitis, chronic renal failure, and immunodeficiency disorders (Aragon & Sole, 2006; King, 2007).

The incidence of septic shock can be reduced by using strict infection control practices, beginning with thorough hand-hygiene techniques (Aragon & Sole, 2006). Other interventions include implementing programs to prevent central line infection; early débriding of wounds to remove necrotic tissue; carrying out standard precautions and adhering to infection control practices, including the use of meticulous aseptic technique; and properly cleaning and maintaining equipment.

A significant body of research has been conducted in the past decade in an effort aimed at reducing the morbidity and mortality caused by septic shock and at clarifying the understanding of sepsis and related disorders (Chart 15-4). In 1991, 2003, and again in early 2008, critical care experts and infectious disease experts systematically reevaluated the body of research and provided evidence-based recommendations for the acute management of patients with sepsis and septic shock (Dellinger, et al., 2008; Vincent & Abraham, 2006).

CHART 15-3 *Risk Factors for Circulatory Shock*

Septic Shock
- Immunosuppression
- Extremes of age (<1 yr and >65 yr)
- Malnourishment
- Chronic illness
- Invasive procedures

Anaphylactic Shock
- Penicillin sensitivity
- Transfusion reaction
- Bee sting allergy
- Latex sensitivity
- Severe allergy to some foods or medications

Neurogenic Shock
- Spinal cord injury
- Spinal anesthesia
- Depressant action of medications
- Glucose deficiency

Pathophysiology

Gram-negative bacteria traditionally have been the most commonly implicated microorganisms in septic shock. However, there is also an increased incidence of gram-positive bacterial infections, and gram-positive bacteria currently account for 50% of cases of septic shock (Smith & McInnis, 2007). Other infectious agents, such as viruses and fungi, also can cause septic shock. However, it is estimated that 20% to 30% of patients with severe sepsis may never have an identifiable site of infection (King, 2007).

When microorganisms invade body tissues, patients exhibit an immune response. This immune response provokes the activation of biochemical cytokines and mediators associated with an inflammatory response and produces a

Chart 15-4• *Definitions to Promote Recognition and Earlier Treatment of Patients With Sepsis*

Bacteremia: the presence of bacteria in the blood

Infection: the presence of microorganisms that trigger an inflammatory response

Hypotension: a systolic blood pressure <90 mm Hg or a drop in systolic blood pressure of ≥40 mm Hg from the patient's baseline blood pressure

Systemic inflammatory response syndrome (SIRS): a syndrome resulting from a *severe clinical insult* that initiates an overwhelming inflammatory response by the body; clinical signs and symptoms may include
- Temperature >38°C or <36°C (>100.4°F or <96.8°F)
- Heart rate >90 bpm
- Respiratory rate >20 breaths/min or $PaCO_2$ <32 mm Hg
- WBC count >12,000 cells/mm^3, <4000 cells/mm^3, or >10% immature WBC (bands)

Sepsis: a systemic response to *infection;* manifested by two or more of the SIRS criteria as a consequence of documented or presumed infection

Severe sepsis: the presence of signs and symptoms of sepsis associated with organ dysfunction, hypotension, or hypoperfusion; clinical signs and symptoms include those of sepsis as well as
- Lactic acidosis
- Oliguria
- Altered level of consciousness
- Thrombocytopenia and coagulation disorders
- Altered hepatic function

Septic shock: shock associated with sepsis; characterized by symptoms of sepsis plus hypotension and hypoperfusion despite adequate fluid volume replacement

Multiple organ dysfunction syndrome (MODS): the presence of altered function of one or more organs in an acutely ill patient requiring intervention and support of the organs to achieve physiologic functioning required for homeostasis; clinical signs and symptoms may be
- Cardiovascular: hypotension and hypoperfusion
- Respiratory: hypoxemia, hypercarbia, adventitious breath sounds
- Renal: increased creatinine, decreased urine output
- Hematologic: thrombocytopenia, coagulation abnormalities
- Metabolic: lactic acidemia, metabolic acidosis
- Neurologic: altered level of consciousness
- Hepatic: elevated liver function tests, hyperbilirubinemia

From Levy, M. M., Fink, M. P., Marshall, J. C., et al. (2003). 2001 SCCM/ESICM/ACCP/ATS/SIS International Sepsis Definitions Conference. *Critical Care Medicine, 31*(4), 1250–1256; and Dellinger, R. P., Levy, M. M., Carlet, J. M., et al. (2008). Surviving sepsis campaign: International guidelines for management of severe sepsis and septic shock: 2008. *Critical Care Medicine, 36*(1), 296–327.

complex cascade of physiologic events that leads to poor tissue perfusion. Increased capillary permeability, which leads to fluid seeping from the capillaries, and vasodilation are two such effects that interrupt the ability of the body to provide adequate perfusion, oxygen, and nutrients to the tissues and cells. In addition, proinflammatory and anti-inflammatory cytokines released during the inflammatory response activate the coagulation system, which begins to form clots whether or not bleeding is present. The imbalance of the inflammatory response and the clotting and fibrinolysis cascades are considered critical elements of the devastating physiologic progression that occurs in patients with severe sepsis.

Sepsis is an evolving process, with neither clearly definable clinical signs and symptoms nor predictable progression. Initial physiologic changes are subtle. In the early stage of septic shock, BP may remain within normal limits, or the patient may be hypotensive but responsive to fluids. The heart rate increases, progressing to tachycardia. Hyperthermia and fever, with warm, flushed skin and bounding pulses, is evident. The respiratory rate is elevated. Urinary output may remain at normal levels or decrease. GI status may be compromised, as evidenced by nausea, vomiting, diarrhea, or decreased bowel sounds. Signs of hypermetabolism include increased serum glucose and insulin resistance. Subtle changes in mental status, such as confusion or agitation, may be present. The lactate level is elevated because of the maldistribution of blood. Inflammatory markers such as white blood cell counts and C-reactive protein are also elevated (King, 2007).

As sepsis progresses, tissues become less perfused and acidotic, compensation begins to fail, and the patient begins to show signs of organ dysfunction. The cardiovascular system also begins to fail, the BP does not respond to fluid resuscitation and vasoactive agents, and signs of end-organ damage are evident (eg, renal failure, pulmonary failure, hepatic failure). As sepsis progresses to septic shock, the BP drops, and the skin becomes cool, pale, and mottled. Temperature may be normal or below normal. Heart and respiratory rates remain rapid. Urine production ceases, and multiple organ dysfunction progressing to death occurs.

Systemic inflammatory response syndrome (SIRS) presents clinically like sepsis and is part of the initial continuum of sepsis. The physiologic presentation of SIRS is similar to sepsis, except there is no identifiable source of infection (Dellinger, et al., 2008; King, 2007). SIRS stimulates an overwhelming inflammatory immunologic and hormonal response similar to that seen in septic patients. Any overwhelming insult stimulates SIRS and may progress to sepsis. Therefore, despite an absence of infection, antibiotic agents may still be administered because of the possibility of unrecognized infection. Additional therapies directed to support patients with SIRS are similar to those for sepsis. If the inflammatory process progresses, septic shock may develop.

Medical Management

Current treatment of sepsis and septic shock involves identification and elimination of the cause of infection. Current goals are to identify and treat patients in early sepsis within 6 hours to optimize patient outcome (Otero, et al., 2006; Rivers, et al, 2005). Several screening tools can be used to help identify patients with severe sepsis. Chart 15-5 provides key elements that may help identify patients with sepsis and guide interventions in the treatment of severe sepsis and

Chart 15-5 • *Early Identification and Treatment of Patients with Sepsis and Severe Sepsis*

Questions to ask:
Does the patient meet criteria for systemic inflammatory response syndrome (SIRS) (see Chart 15-4)?
Does the patient have signs or symptoms of infection?
- Positive blood cultures
- Currently receiving antibiotic or antifungal therapy
- Examination or chest x-ray suggestive of pneumonia
- Suspected infected wound, abdomen, urine, or other source of infection

Does the patient have signs of acute organ dysfunction?
- Cardiovascular: systolic BP <90 mm Hg or mean arterial pressure (MAP) <65 mm Hg, or drop in systolic BP >40 mm Hg from baseline BP
  - Is hypotension responsive to fluid resuscitation, or is vasopressor support needed?
  - Is the serum lactate >4 mmol/L?
- Respiratory: respiratory rate >20 breaths/min or $PaCO_2$ <32 mm Hg
  - Is increasing oxygen or mechanical ventilator support needed?
- Renal: urine output <0.5 mL/kg/h
- Hematologic: laboratory analysis and signs and symptoms of coagulopathies
- Metabolic: insulin resistance, metabolic acidosis, or serum lactate >4 mmol/L
- Hepatic: elevated liver function tests or hyperbilirubinemia
- Central nervous system: changes in level of consciousness ranging from agitation to coma

Early interventions:
- Aggressive fluid resuscitation with 20 mL/kg/h of crystalloid (or colloid equivalent)
  - Give fluids to achieve a target central venous pressure of 8 to 12 mm Hg, MAP >65 mm Hg, urine output >0.5 mL/kg/h, and an $ScvO_2$ >70%
  - Vasopressor agents are used if fluid resuscitation does not restore an effective blood pressure and cardiac output
- Obtain blood, sputum, urine, and wound cultures and administer broad-spectrum antibiotics
- Support the respiratory system with mechanical ventilation
- Transfuse with packed red blood cells when hemoglobin is <7 g/dL
- Provide adequate IV sedation; avoid the use of neuromuscular blockade agents when possible
- Control serum glucose <150 mg/dL with IV insulin therapy
- Implement interventions and medications to prevent deep vein thrombosis and stress ulcer prophylaxis
- Consider IV steroid therapy if the patient is not responding to fluid resuscitation and vasopressor therapy
- Consider administration of recombinant human activated protein C (drotrecogin alfa) in adult patients with sepsis-induced organ dysfunction with clinical assessment of high risk of death

From Dellinger, R. P., Levy, M. M., Carlet, J. M., et al. (2008). Surviving sepsis campaign: International guidelines for management of severe sepsis and septic shock: 2008. *Critical Care Medicine, 36*(1), 296–327; and Rivers, E. P., McIntyre, L., Morro, D. C., et al. (2005). Early and innovative interventions for severe sepsis and septic shock: Taking advantage of a window of opportunity. *Canadian Medical Association Journal, 173*(9), 1054–1065.

septic shock (Dellinger, et al., 2008; King, 2007; Otero, et al., 2006; Surviving Sepsis Campaign, 2007).

Rapid identification of the infectious source is also a critical element in management. Specimens of blood, sputum, urine, wound drainage, and tips of invasive catheters are collected for culture using aseptic technique. Any potential routes of infection must be identified and treated. IV lines are removed and reinserted at alternate sites. Antibiotic-coated IV central lines may be inserted to decrease the risk of invasive line-related bacteremia in high-risk patients (King, 2007). If possible, urinary catheters are removed. Any abscesses are drained, and necrotic areas are débrided.

Research efforts are focusing on better identification and early aggressive treatment of patients with sepsis, rapid and effective restoration of tissue perfusion, evaluation and treatment of the patient's immune response, and treatment of dysregulation of the coagulation system that seems to occur with severe sepsis (Remick, 2007a).

Fluid Replacement Therapy

Fluid replacement must be instituted to correct the tissue hypoperfusion that results from the incompetent vasculature and the inflammatory response. Reestablishing tissue perfusion through aggressive fluid resuscitation is the key to management of severe sepsis and septic shock (Dellinger, et al., 2008; Otero, et al., 2006). See Chart 15-5 for a list of the treatment endpoints of fluid resuscitation.

Pharmacologic Therapy

If the identity of the infecting organism is unknown, broad-spectrum antibiotic agents are started until culture and sensitivity reports are received (Dellinger, et al., 2008; Smith & McInnis, 2007), at which time the antibiotic agents may be changed to agents that are more specific to the infecting organism and less toxic to the patient.

Treatment of the dysregulation of the coagulation system that occurs in patients with severe sepsis and septic shock remains controversial. Nonetheless, current guidelines recommend the administration of recombinant human activated protein C (rhAPC; drotrecogin alfa [Xigris]) to patients with end-organ dysfunction and high risk of death (Dellinger, et al., 2008). In sepsis, an imbalance in proinflammatory mediators activates the coagulation cascade and deposits microthrombi that alter tissue perfusion. Drotrecogin alfa (Xigris) acts as an antithrombotic, anti-inflammatory, and profibrinolytic agent. Drotrecogin alfa acts as an anti-inflammatory cytokine, it stimulates fibrinolysis, restoring balance in the coagulation–anticoagulation homeostatic process of the body's inflammatory response to injury and infection.

Drotrecogin alfa has provided a significant breakthrough in the successful pharmacologic treatment of patients with sepsis. The medication should be administered as early as possible in the sequence of pathophysiologic events of sepsis. It is not without side effects, bleeding being the most common serious effect. Stopping the medication reduces

the risk of bleeding. The patient should be evaluated with regard to the relative risk of bleeding versus the potential benefit from the medication. Drotrecogin alfa is contraindicated in patients with active internal bleeding, recent hemorrhagic stroke, intracranial surgery, or head injury.

Nutritional Therapy

Aggressive nutritional supplementation is critical in the management of septic shock, because malnutrition further impairs the patient's resistance to infection. Nutritional supplementation should be initiated within the first 24 hours after ICU admission (Stapleton, et al., 2007), and continuous infusions of insulin are used to control hyperglycemia (Dellinger, et al., 2008; Vanhorebeek, et al., 2007). Enteral feedings are preferred to the parenteral route because of the increased risk of iatrogenic infection associated with IV catheters; however, enteral feedings may not be possible if decreased perfusion to the GI tract reduces peristalsis and impairs absorption.

Nursing Management

Nurses caring for patients in any setting must keep in mind the risks of sepsis and the high mortality rate associated with sepsis, severe sepsis, and septic shock. All invasive procedures must be carried out with aseptic technique after careful hand hygiene. In addition, IV lines, arterial and venous puncture sites, surgical incisions, traumatic wounds, urinary catheters, and pressure ulcers must be monitored for signs of infection. Nurses need to identify patients who are at particular risk for sepsis and septic shock (ie, elderly and immunosuppressed patients and those with extensive trauma, burns, or diabetes), keeping in mind that these high-risk patients may not develop typical or classic signs of infection and sepsis. For example, confusion may be the first sign of infection and sepsis in elderly patients.

When caring for a patient with septic shock, the nurse collaborates with other members of the health care team to identify the site and source of sepsis and the specific organisms involved. The nurse often obtains appropriate specimens for culture and sensitivity.

Elevated body temperature (hyperthermia) is common with sepsis and raises the patient's metabolic rate and oxygen consumption. Fever is one of the body's natural mechanisms for fighting infections. Therefore, elevated temperatures may not be treated unless they reach dangerous levels (more than 40°C [104°F]) or unless the patient is uncomfortable. Efforts may be made to reduce the temperature by administering acetaminophen or applying a hypothermia blanket. During these therapies, the nurse monitors the patient closely for shivering, which increases oxygen consumption. Efforts to increase comfort are important if the patient experiences fever, chills, or shivering.

The nurse administers prescribed IV fluids and medications, including antibiotic agents and vasoactive medications, to restore vascular volume. Because of decreased perfusion, serum concentrations of antibiotic agents that are normally cleared by the kidneys and liver may increase and produce toxic effects. Therefore, the nurse monitors blood levels (antibiotic agents, BUN, creatinine, white blood cell count, hemoglobin, hematocrit, platelet levels, coagulation studies) and reports changes to the physician. As with other types of shock, the nurse monitors the patient's hemodynamic status, fluid intake and output, and nutritional status. Daily weights and close monitoring of serum albumin and prealbumin levels help determine the patient's protein requirements.

NEUROGENIC SHOCK

In **neurogenic shock**, vasodilation occurs as a result of a loss of balance between parasympathetic and sympathetic stimulation. Sympathetic stimulation causes vascular smooth muscle to constrict, and parasympathetic stimulation causes vascular smooth muscle to relax or dilate. The patient experiences a predominant parasympathetic stimulation that causes vasodilation lasting for an extended period, leading to a relative hypovolemic state. However, blood volume is adequate, because the vasculature is dilated; the blood volume is displaced, producing a hypotensive (low BP) state. The overriding parasympathetic stimulation that occurs with neurogenic shock causes a drastic decrease in the patient's systemic vascular resistance and bradycardia. Inadequate BP results in the insufficient perfusion of tissues and cells that is common to all shock states.

Neurogenic shock can be caused by spinal cord injury, spinal anesthesia, or other nervous system damage (see Chart 15-3). It may also result from the depressant action of medications or from lack of glucose (eg, insulin reaction or shock). Neurogenic shock may have a prolonged course (spinal cord injury) or a short one (syncope or fainting). Normally, during states of stress, the sympathetic stimulation causes the BP and heart rate to increase. In neurogenic shock, the sympathetic system is not able to respond to body stressors. Therefore, the clinical characteristics of neurogenic shock are signs of parasympathetic stimulation. It is characterized by dry, warm skin rather than the cool, moist skin seen in hypovolemic shock. Another characteristic is hypotension with bradycardia, rather than the tachycardia that characterizes other forms of shock.

Medical Management

Treatment of neurogenic shock involves restoring sympathetic tone, either through the stabilization of a spinal cord injury or, in the instance of spinal anesthesia, by positioning the patient properly. Specific treatment depends on the cause of the shock. Further discussion of management of patients with a spinal cord injury is presented in Chapter 63. If hypoglycemia (insulin shock) is the cause, glucose is rapidly administered (see Chapter 41).

Nursing Management

It is important to elevate and maintain the head of the bed at least 30 degrees to prevent neurogenic shock when a patient receives spinal or epidural anesthesia. Elevation of the head helps prevent the spread of the anesthetic agent up the spinal cord. In suspected spinal cord injury, neurogenic shock may be prevented by carefully immobilizing the patient to prevent further damage to the spinal cord.

Nursing interventions are directed toward supporting cardiovascular and neurologic function until the usually transient episode of neurogenic shock resolves. Applying

anti-embolism stockings and elevating the foot of the bed may minimize pooling of blood in the legs. Pooled blood increases the risk of thrombus formation. Therefore, the nurse must check the patient daily for any lower extremity pain, redness, tenderness, and warmth. If the patient complains of pain and objective assessment of the calf is suspicious, the patient should be evaluated for deep vein thrombosis. Administration of heparin or low-molecular-weight heparin (Lovenox) as prescribed, application of anti-embolism stockings, or use of pneumatic compression of the legs may prevent thrombus formation. Passive range of motion of the immobile extremities helps promote circulation.

A patient who has experienced a spinal cord injury may not report pain caused by internal injuries. Therefore, in the immediate postinjury period, the nurse must monitor the patient closely for signs of internal bleeding that could lead to hypovolemic shock.

ANAPHYLACTIC SHOCK

Anaphylactic shock occurs rapidly and is life-threatening. Because anaphylactic shock occurs in patients already exposed to an antigen and who have developed antibodies to it, it can often be prevented. Patients with known allergies should understand the consequences of subsequent exposure to the antigen and should wear medical identification that lists their sensitivities. This could prevent inadvertent administration of a medication that would lead to anaphylactic shock. In addition, patients and families need instruction about emergency use of medications for treatment of anaphylaxis.

Anaphylactic shock is caused by a severe allergic reaction when patients who have already produced antibodies to a foreign substance (antigen) develop a systemic antigen–antibody reaction (see Chart 15-3). This process requires that the patient has previously been exposed to the substance. An antigen–antibody reaction provokes mast cells to release potent vasoactive substances, such as histamine or bradykinin, causing widespread vasodilation and capillary permeability. Characteristics of severe anaphylaxis usually include rapid onset of hypotension, neurologic compromise, respiratory distress, and cardiac arrest (Brown, 2007).

Medical Management

Treatment of anaphylactic shock requires removing the causative antigen (eg, discontinuing an antibiotic agent), administering medications that restore vascular tone, and providing emergency support of basic life functions. Epinephrine is given for its vasoconstrictive action. Diphenhydramine (Benadryl) is administered to reverse the effects of histamine, thereby reducing capillary permeability. These medications are given intravenously. Nebulized medications, such as albuterol (Proventil), may be given to reverse histamine-induced bronchospasm.

If cardiac arrest and respiratory arrest are imminent or have occurred, cardiopulmonary resuscitation is performed. Endotracheal intubation or tracheotomy may be necessary to establish an airway. IV lines are inserted to provide access for administering fluids and medications. Anaphylaxis and specific chemical mediators are discussed further in Chapter 53.

Nursing Management

The nurse has an important role in preventing anaphylactic shock. The nurse must assess all patients for allergies or previous reactions to antigens (eg, medications, blood products, foods, contrast agents, latex) and communicate the existence of these allergies or reactions to others. In addition, the nurse assesses the patient's understanding of previous reactions and steps taken by the patient and family to prevent further exposure to antigens. When new allergies are identified, the nurse advises the patient to wear or carry identification that names the specific allergen or antigen.

When administering any new medication, the nurse observes all patients for allergic reactions. This is especially important with IV medications, including antibiotics. Previous adverse drug reactions increase the risk that the patient will develop an undesirable reaction to a new medication. If the patient reports an allergy to a medication, the nurse must be aware of the risks involved in the administration of similar medications.

At hospital and outpatient diagnostic testing sites, the nurse must identify patients who are at risk for anaphylactic reactions to contrast agents (radiopaque, dyelike substances that may contain iodine) used for diagnostic tests. Patients with a known allergy to iodine or fish and those who have had previous allergic reactions to contrast agents are at high risk. This information must be communicated to the staff at the diagnostic testing site, including x-ray personnel. The nurse must be knowledgeable about the clinical signs of anaphylaxis, must take immediate action if signs and symptoms occur, and must be prepared to begin cardiopulmonary resuscitation if cardiorespiratory arrest occurs.

Community health and home care nurses who administer medications, including antibiotic agents, in the patient's home or other settings must be prepared to administer epinephrine subcutaneously or intramuscularly in the event of an anaphylactic reaction.

After recovery from anaphylaxis, the patient and family require an explanation of the event. Furthermore, the nurse provides instruction about avoiding future exposure to antigens and administering emergency medications to treat anaphylaxis (see Chapter 53).

Multiple Organ Dysfunction Syndrome

Multiple organ dysfunction syndrome (MODS) is altered organ function in acutely ill patients that requires medical intervention to support continued organ function. It is another phase in the progression of shock states. The actual incidence of MODS is difficult to determine, because it develops with acute illnesses that compromise tissue perfusion. Dysfunction of one organ system is associated with 20% mortality, and if more than four organs fail, the mortality may reach 70% (VonRueden, et al., 2008).

Pathophysiology

MODS may be a complication of any form of shock caused by inadequate tissue perfusion. The precise mechanism by which MODS occurs remains unknown. However, MODS

frequently occurs toward the end of the continuum of septic shock when tissue perfusion cannot be effectively restored. It is not possible to predict which patients who experience shock will develop MODS, partly because much of the organ damage occurs at the cellular level and therefore cannot be directly observed or measured. However, a pattern of progressive organ dysfunction and failure typically occurs; organ failure usually begins in the lungs, and cardiovascular instability as well as failure of the hepatic, GI, renal, immunologic, and central nervous systems follow (Abraham & Singer, 2007; VonRueden, et al., 2008). Advanced age, malnutrition, and coexisting disease appear to increase the risk of MODS in acutely ill patients.

Clinical Manifestations

The clinical presentation of MODS is insidious; tissues become hypoperfused at both a microcellular and macrocellular level, eventually causing organ dysfunction that requires intervention to support organ function.

In MODS, the sequence of organ dysfunction varies depending on the patient's primary illness and comorbidities prior to experiencing shock. For simplicity of presentation, the classic pattern is described. Typically, the lungs are the first organs to show signs of dysfunction. The patient experiences progressive dyspnea and respiratory failure requiring intubation and mechanical ventilation (see Chapters 23 and 25). The patient usually remains hemodynamically stable but may require increasing amounts of IV fluids and vasoactive agents to support the BP and cardiac output. Signs of a hypermetabolic state, characterized by hyperglycemia (elevated blood glucose level), hyperlactic acidemia (excess lactic acid in the blood), and increased BUN, are present. The metabolic rate may be 1.5 to 2 times the basal metabolic rate. At this time, there is a severe loss of skeletal muscle mass (autocatabolism) to meet the high energy demands of the body.

After approximately 7 to 10 days, signs of hepatic dysfunction (eg, elevated bilirubin and liver function tests) and renal dysfunction (eg, elevated creatinine and anuria) are evident. As the lack of tissue perfusion continues, the hematologic system becomes dysfunctional, with worsening immunocompromise and increasing risk of bleeding. The cardiovascular system becomes unstable and unresponsive to vasoactive agents, and the patient's neurologic response progresses to a state of unresponsiveness or coma.

The goal of all shock states is to reverse the tissue hypoperfusion and hypoxia. If effective tissue perfusion is restored before organs become dysfunctional, the patient's condition stabilizes. Along the septic shock continuum, the onset of organ dysfunction is an ominous prognostic sign; the more organs that fail, the worse the outcome.

Medical Management

Prevention remains the top priority in managing MODS. Elderly patients are at increased risk for MODS because of the lack of physiologic reserve and the natural degenerative process, especially immune compromise (Marik, 2006). Early detection and documentation of initial signs of infection are essential in managing MODS in elderly patients. Subtle changes in mentation and a gradual rise in temperature are early warning signs. Other patients at risk for MODS are those with chronic illness, malnutrition, immunosuppression, or surgical or traumatic wounds.

If preventive measures fail, treatment measures to reverse MODS are aimed at (1) controlling the initiating event, (2) promoting adequate organ perfusion, and (3) providing nutritional support.

Nursing Management

The general plan of nursing care for patients with MODS is the same as that for patients with septic shock. Primary nursing interventions are aimed at supporting the patient and monitoring organ perfusion until primary organ insults are halted. Providing information and support to family members is a critical role of the nurse. It is important that the health care team address end-of-life decisions to ensure that supportive therapies are congruent with the patient's wishes (see Chapter 17).

Promoting Communication

Nurses should encourage frequent and open communication about treatment modalities and options to ensure that the patient's wishes regarding medical management are met. For patients who survive MODS, it is essential that they be informed about the goals of rehabilitation and expectations for progress toward these goals, because massive loss of skeletal muscle mass makes rehabilitation a long, slow process. A strong nurse–patient relationship built on effective communication provides needed encouragement during this phase of recovery.

Promoting Home and Community-Based Care

Teaching Patients Self-Care

Patients who experience and survive shock may have been unable to get out of bed for an extended period of time and are likely to have a slow, prolonged recovery. The patient and family are instructed about strategies to prevent further episodes of shock by identifying the factors implicated in the initial episode. In addition, the patient and family require instruction about assessments needed to identify the complications that may occur after the patient is discharged from the hospital. Depending on the type of shock and its management, the patient or family may require instruction about treatment modalities such as emergency administration of medications, IV therapy, parenteral or enteral nutrition, skin care, exercise, and ambulation. The patient and family are also instructed about the need for gradual increases in ambulation and other activity. The need for adequate nutrition is another crucial aspect of teaching.

Continuing Care

Because of the physical toll associated with recovery from shock, patients may be cared for in a long-term care facility or rehabilitation setting after hospital discharge. Alternatively, a referral may be made for home care. The home care nurse assesses the patient's physical status and monitors

recovery. The nurse also assesses the adequacy of treatments that are continued at home and the ability of the patient and family to cope with these treatments. The patient is likely to require close medical supervision until complete recovery occurs. The home care nurse reinforces the importance of continuing medical care and helps the patient and family identify and mobilize community resources.

CRITICAL THINKING EXERCISES

1 A patient with a history of severe osteoarthritis is prescribed glucosamine and chondroitin supplements. The patient's chart states that he has no known drug allergies, but he does have food allergies that include shell fish and avocados. Fifteen minutes after the first dose of the medication is administered, the patient complains of anxiety, shortness of breath, and chest discomfort. He is flushed and visibly uncomfortable. What are your nursing priorities in providing care to this patient? What assessment data do you need to obtain to determine if this patient is experiencing cardiogenic or anaphylactic shock? What nursing interventions and medical treatments would you anticipate for cardiogenic shock? What risks did the patient have that may have increased his likelihood of experiencing anaphylactic shock? In terms of anaphylactic shock, what nursing interventions and medical treatments would you anticipate?

EBP **2** An elderly man with a 16-year history of Parkinson's disease is admitted with sudden, increasing confusion and combative behavior. You know that changes in mental status may be an early sign of sepsis in the elderly. How would you assess this patient for the possibility of sepsis? What risk factors place an older patient at higher risk for sepsis? How would you ensure the accuracy of vital signs and interpretation of vital signs in the older patient experiencing sepsis? What is the evidence base for these risk factors? How would the management of the elderly patient differ from that of a younger patient?

3 A 32-year-old man is admitted with severe pancreatitis. He has a long history of addiction to alcohol and was recently on a "drinking binge." The patient is agitated and exhibiting nervous behavior. His BP is 106/88 mm Hg, heart rate is 126 bpm, respiratory rate is 32 breaths/min, and he has not voided for the past 3 hours. Is the patient most likely experiencing withdrawal from alcohol or a type of shock? Describe the type of shock that poses the greatest risk for this patient. What interventions should you anticipate to prevent the progression of shock or development of MODS? Given the patient's history, what organ(s) is least likely to tolerate prolonged tissue hypoperfusion? What assessment data would you look at to monitor organ dysfunction in this patient?

4 A 23-year-old patient underwent surgical repair of her shoulder. She had spinal anesthesia for the surgery and currently has a patent epidural catheter for pain management. What types of shock are possible in this patient? What therapy directed at prevention or treatment of shock would you anticipate? Describe the rationale for the therapies that you have identified. How would you use the patient's history and symptom presentation to help you identify shock states? Describe likely symptoms and the underlying pathophysiology of the shock state.

The Smeltzer suite offers these additional resources to enhance learning and facilitate understanding of this chapter:

- thePoint online resource, thepoint.lww.com/Smeltzer12E
- Student CD-ROM included with the book
- *Study Guide to Accompany Brunner & Suddarth's Textbook of Medical-Surgical Nursing*
- *Handbook for Brunner & Suddarth's Textbook of Medical-Surgical Nursing*

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**Asterisk indicate nursing research.*

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chapter 16

Oncology: Nursing Management in Cancer Care

LEARNING OBJECTIVES

On completion of this chapter, the learner will be able to:

1. Compare the structure and function of the normal cell and the cancer cell.
2. Differentiate between benign and malignant tumors.
3. Identify agents and factors that have been found to be carcinogenic.
4. Describe the significance of health education and preventive care in decreasing the incidence of cancer.
5. Differentiate among the purposes of surgical procedures used in cancer treatment, diagnosis, prophylaxis, palliation, and reconstruction.
6. Describe the roles of surgery, radiation therapy, chemotherapy, targeted therapy, hematopoietic stem cell transplantation, and other therapies in treating cancer.
7. Describe the special nursing needs of patients receiving chemotherapy.
8. Describe nursing care related to common nursing diagnoses associated with cancer: impaired skin integrity, alopecia, nutritional problems, and altered body image.
9. Identify potential complications for the patient with cancer and discuss associated nursing care.
10. Describe the concept of hospice in providing care for patients with advanced cancer.
11. Identify assessment parameters and nursing management of patients with oncologic emergencies.

GLOSSARY

alopecia: hair loss
anaplasia: cells that lack normal cellular characteristics and differ in shape and organization with respect to their cells of origin; usually, anaplastic cells are malignant
apoptosis: programmed cell death
benign: not cancerous; benign tumors may grow but are unable to spread to other areas
biologic response modifier (BRM) therapy: use of agents or treatment methods that can alter the immunologic relationship between the tumor and the host to provide a therapeutic benefit
biopsy: a diagnostic procedure to remove a small sample of tissue to be examined microscopically to detect malignant cells
brachytherapy: delivery of radiation therapy through internal implants
cancer: a disease process whereby cells proliferate abnormally, ignoring growth-regulating signals in the environment surrounding the cells
carcinogenesis: process of transforming normal cells into malignant cells
chemotherapy: use of medications to kill tumor cells by interfering with cellular functions and reproduction
control: containment of the growth of cancer cells

GLOSSARY *(Continued)*

cure: prolonged survival and disappearance of all evidence of disease so that the patient has the same life expectancy as anyone else in his or her age group
cytokines: substances produced by cells of the immune system to enhance production and functioning of components of the immune system
dysplasia: bizarre cell growth resulting in cells that differ in size, shape, or arrangement from other cells of the same type of tissue
extravasation: leakage of medication from the veins into the subcutaneous tissues
grading: identification of the type of tissue from which the tumor originated and the degree to which the tumor cells retain the functional and structural characteristics of the tissue of origin
graft-versus-host disease (GVHD): an immune response initiated by T lymphocytes of donor tissue against the recipient's tissues (skin, gastrointestinal tract, liver); an undesirable response
graft-versus-tumor effect: the donor cell response against the malignancy; a desirable response
hyperplasia: increase in the number of cells of a tissue; most often associated with periods of rapid body growth
malignant: having cells or processes that are characteristic of cancer
metaplasia: conversion of one type of mature cell into another type of cell
metastasis: spread of cancer cells from the primary tumor to distant sites
myelosuppression: suppression of the blood cell–producing function of the bone marrow
nadir: lowest point of white blood cell depression after therapy that has toxic effects on the bone marrow
neoplasia: uncontrolled cell growth that follows no physiologic demand
neutropenia: abnormally low absolute neutrophil count
oncology: field or study of cancer
palliation: relief of symptoms and promotion of comfort and quality of life
radiation therapy: use of ionizing radiation to interrupt the growth of malignant cells
staging: process of determining the extent of disease, including tumor size and spread or metastasis to distant sites
stomatitis: inflammation of the oral tissues, often associated with some chemotherapeutic agents and radiation therapy to the head and neck region
targeted therapies: cancer treatments that seek to minimize the negative effects on healthy tissues by disrupting specific cancer cell functions, such as malignant transformation, communication pathways, processes for growth and metastasis, and genetic coding
thrombocytopenia: decrease in the number of circulating platelets; associated with the potential for bleeding
tumor-specific antigen (TSA): protein on the membrane of cancer cells that distinguishes the malignant cell from a benign cell of the same tissue type
vesicant: substance that can cause tissue necrosis and damage, particularly when extravasated
xerostomia: dry oral cavity resulting from decreased function of salivary glands

Cancer is not a single disease with a single cause; rather, it is a group of distinct diseases with different causes, manifestations, treatments, and prognoses. Cancer nursing practice covers all age groups and nursing specialties and is carried out in a variety of health care settings, including the home, community, acute care institutions, outpatient centers, rehabilitation, and long-term care facilities. The scope, responsibilities, and goals of cancer nursing, also called **oncology** nursing, are as diverse and complex as those of any nursing specialty. Because many people associate cancer with pain and death, nurses need to identify their own reactions to cancer and set realistic goals to meet the challenges inherent in caring for patients with cancer. In addition, cancer nurses must be prepared to support patients and families through a wide range of physical, emotional, social, cultural, and spiritual crises.

Epidemiology of Cancer

Although cancer affects people of all ages, most cancers occur in people older than 65 years of age. Overall, the incidence of cancer is higher in men than in women and higher in industrialized sectors and nations.

More than 1.4 million Americans are diagnosed each year with cancer, affecting one of various body sites (Fig. 16-1). Cancer is second only to cardiovascular disease as a leading cause of death in the United States. Although the numbers of cancer deaths have decreased slightly, more than 560,000 Americans were expected to die from a malignant process in 2008. The leading causes of cancer death in the United States, in order of frequency, are lung, prostate, and colorectal cancer in men and lung, breast, and colorectal cancer in women (Jemal, Siegel, Ward, et al., 2007).

For all cancer sites combined, African American men have a 15% higher incidence rate and a 38% higher death rate than Caucasian men. African American women have a 9% lower incidence rate, but an 18% higher death rate than Caucasian women for all cancer sites combined. Factors contributing to disparities in cancer morbidity and mortality in this group vary by site and are related to exposure, economics, education, access to health care, and other issues that are not clearly understood (Jemal, et al., 2007).

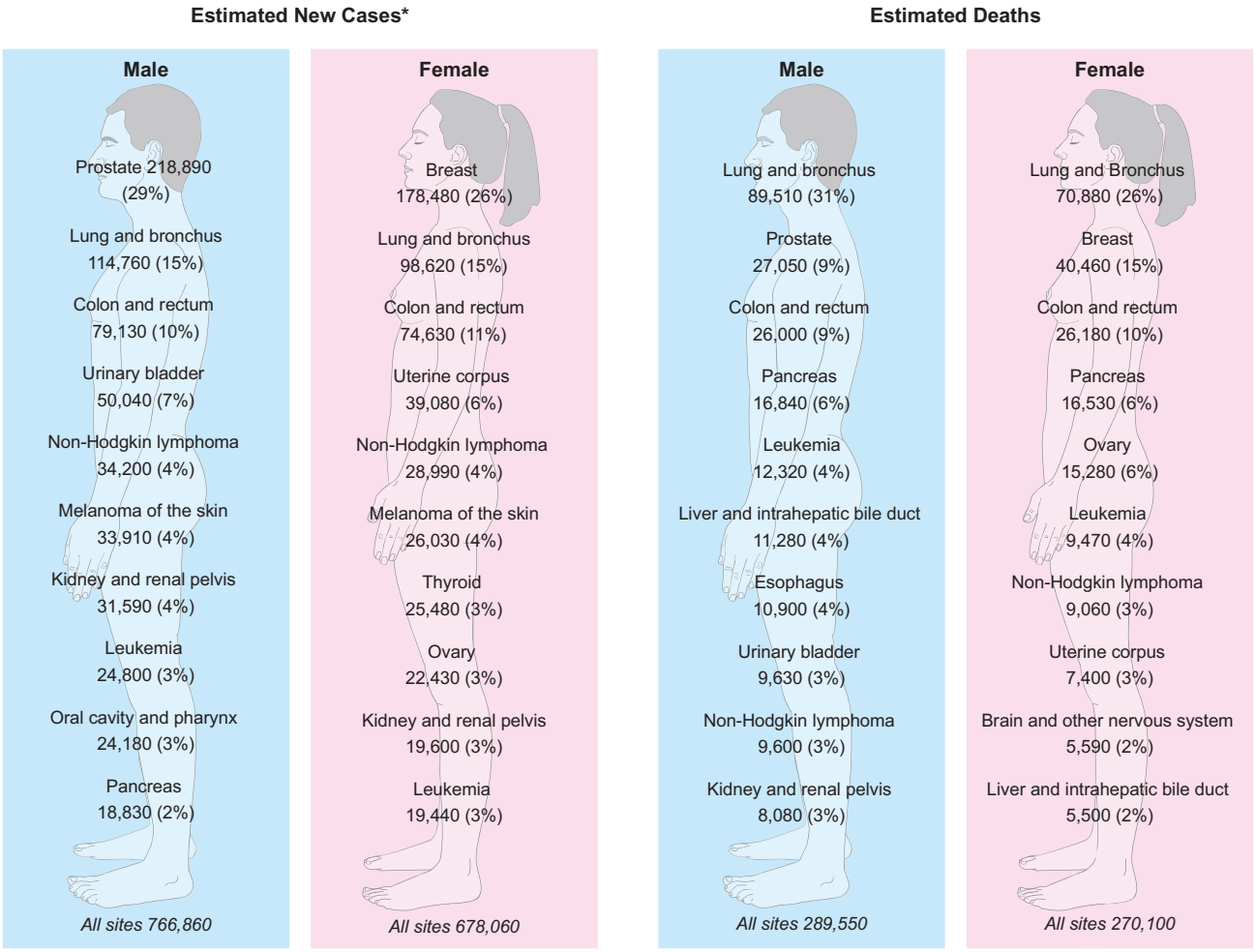

Figure 16-1 Ten leading types of cancer by gender determined on the basis of estimated new cancer cases and deaths in the United States in 2007.
*Excludes basal and squamous cell skin cancers and in situ cancers except urinary bladder. Note: Percentages may not total 100% because of rounding. Redrawn from Jemal, A., Siegel, R., Ward, E., et al. (2007). Cancer statistics. *CA Cancer Journal for Clinicians, 57*(1), 43–66.

Pathophysiology of the Malignant Process

Cancer is a disease process that begins when an abnormal cell is transformed by the genetic mutation of the cellular DNA. This abnormal cell forms a clone and begins to proliferate abnormally, ignoring growth-regulating signals in the environment surrounding the cell. The cells acquire invasive characteristics, and changes occur in surrounding tissues. The cells infiltrate these tissues and gain access to lymph and blood vessels, which carry the cells to other areas of the body.

Proliferative Patterns

During the lifespan, various body tissues normally undergo periods of rapid or proliferative growth that must be distinguished from malignant growth activity. Several patterns of cell growth exist: **hyperplasia, metaplasia, dysplasia, anaplasia,** and **neoplasia.** Cancerous cells are described as **malignant** neoplasms. They demonstrate uncontrolled cell growth that follows no physiologic demand (neoplasia). **Benign** (noncancerous) and malignant growths are classified and named by tissue of origin (eg, benign tumors of the meninges are called meningioma and malignant tumors of the meninges are called meningeal sarcoma).

Benign and malignant cells differ in many cellular growth characteristics, including the method and rate of growth, ability to metastasize or spread, general effects, destruction of tissue, and ability to cause death. These differences are summarized in Table 16-1. The degree of anaplasia (cells that lack normal cellular characteristics and differ in shape and organization with respect to their cells of origin) ultimately determines the malignant potential.

Characteristics of Malignant Cells

Despite their individual differences, all cancer cells share some common cellular characteristics related to the cell membrane, special proteins, the nuclei, chromosomal abnormalities, and the rate of mitosis and growth. The cell membranes are altered in cancer cells, which affect fluid movement in and out of the cell. The cell membrane of malignant cells also contains proteins called **tumor-specific antigens** (eg, carcinoembryonic antigen [CEA] and prostate-specific antigen [PSA]), which develop over time as the cells become less differentiated (mature). These proteins distinguish malignant cells from benign cells of the same tissue type. They may be useful in measuring the extent of disease in a person and in tracking the course of illness during treatment or relapse. Malignant cellular membranes also contain less fibronectin, a cellular cement. They are therefore less cohesive and do not adhere to adjacent cells readily.

Typically, nuclei of cancer cells are large and irregularly shaped (pleomorphism). Nucleoli, structures within the nucleus that house ribonucleic acid (RNA), are larger and more numerous in malignant cells, perhaps because of increased RNA synthesis. Chromosomal abnormalities (translocations, deletions, additions) and fragility of chromosomes are commonly found when cancer cells are analyzed.

Mitosis (cell division) occurs more frequently in malignant cells than in normal cells. As the cells grow and divide, more glucose and oxygen are needed. If glucose and oxygen are unavailable, malignant cells use anaerobic metabolic channels to produce energy, which makes the cells less dependent on the availability of a constant oxygen supply.

Invasion and Metastasis

Malignant disease processes have the ability to allow the spread or transfer of cancerous cells from one organ or body part to another by invasion and metastasis.

Table 16-1 CHARACTERISTICS OF BENIGN AND MALIGNANT NEOPLASMS

Characteristics	Benign	Malignant
Cell characteristics	Well-differentiated cells that resemble normal cells of the tissue from which the tumor originated	Cells are undifferentiated and often bear little resemblance to the normal cells of the tissue from which they arose
Mode of growth	Tumor grows by expansion and does not infiltrate the surrounding tissues; usually encapsulated	Grows at the periphery and sends out processes that infiltrate and destroy the surrounding tissues
Rate of growth	Rate of growth is usually slow	Rate of growth is variable and depends on level of differentiation; the more anaplastic the tumor, the faster its growth
Metastasis	Does not spread by metastasis	Gains access to the blood and lymphatic channels and metastasizes to other areas of the body
General effects	Is usually a localized phenomenon that does not cause generalized effects unless its location interferes with vital functions	Often causes generalized effects, such as anemia, weakness, and weight loss
Tissue destruction	Does not usually cause tissue damage unless its location interferes with blood flow	Often causes extensive tissue damage as the tumor outgrows its blood supply or encroaches on blood flow to the area; may also produce substances that cause cell damage
Ability to cause death	Does not usually cause death unless its location interferes with vital functions	Usually causes death unless growth can be controlled

Invasion, which refers to the growth of the primary tumor into the surrounding host tissues, occurs in several ways. Mechanical pressure exerted by rapidly proliferating neoplasms may force fingerlike projections of tumor cells into surrounding tissue and interstitial spaces. Malignant cells are less adherent and may break off from the primary tumor and invade adjacent structures. Malignant cells are thought to possess or produce specific destructive enzymes (proteinases), such as collagenases (specific to collagen), plasminogen activators (specific to plasma), and lysosomal hydrolyses. These enzymes are thought to destroy surrounding tissue, including the structural tissues of the vascular basement membrane, facilitating invasion of malignant cells. The mechanical pressure of a rapidly growing tumor may enhance this process.

Metastasis is the dissemination or spread of malignant cells from the primary tumor to distant sites by direct spread of tumor cells to body cavities or through lymphatic and blood circulation. Tumors growing in or penetrating body cavities may shed cells or emboli that travel within the body cavity and seed the surfaces of other organs. This can occur in ovarian cancer when malignant cells enter the peritoneal cavity and seed the peritoneal surfaces of such abdominal organs as the liver or pancreas. Patterns of metastasis can be partially explained by circulatory patterns and by specific affinity for certain malignant cells to bind to molecules in specific body tissue.

Lymphatic and Hematogenous Spread

Lymph and blood are key mechanisms by which cancer cells spread. Lymphatic spread (the transport of tumor cells through the lymphatic circulation) is the most common mechanism of metastasis. Tumor emboli enter the lymph channels by way of the interstitial fluid, which communicates with lymphatic fluid. Malignant cells also may penetrate lymphatic vessels by invasion. After entering the lymphatic circulation, malignant cells either lodge in the lymph nodes or pass between the lymphatic and venous circulations. Tumors arising in areas of the body with rapid and extensive lymphatic circulation are at high risk for metastasis through lymphatic channels. Breast tumors frequently metastasize in this manner through axillary, clavicular, and thoracic lymph channels.

Hematogenous spread is the dissemination of malignant cells via the bloodstream and is directly related to the vascularity of the tumor. Few malignant cells can survive the turbulence of arterial circulation, insufficient oxygenation, or destruction by the body's immune system. In addition, the structure of most arteries and arterioles is far too secure to permit malignant invasion. Those malignant cells that do survive are able to attach to endothelium and attract fibrin, platelets, and clotting factors to seal themselves from immune system surveillance. The endothelium retracts, allowing the malignant cells to enter the basement membrane and secrete lysosomal enzymes. These enzymes destroy surrounding body tissues, allowing implantation.

Angiogenesis

Angiogenesis is the growth of new capillaries from the host tissue by the release of growth factors and enzymes such as vascular endothelial growth factor (VEGF). These proteins rapidly stimulate formation of new blood vessels, which helps malignant cells obtain the necessary nutrients and oxygen. It is also through this vascular network that tumor emboli can enter the systemic circulation and travel to distant sites. Large tumor emboli that become trapped in the microcirculation of distant sites may further metastasize to other sites. Therapies that target VEGF or its receptors are being used to treat many cancers effectively (see Targeted Therapies).

Carcinogenesis

Molecular Process

Malignant transformation, or **carcinogenesis,** is thought to be at least a three-step cellular process, involving initiation, promotion, and progression. During *initiation,* initiators (carcinogens), such as chemicals, physical factors, and biologic agents, escape normal enzymatic mechanisms and alter the genetic structure of the cellular DNA. Normally, these alterations are reversed by DNA repair mechanisms or the changes initiate programmed cellular death. Occasionally, cells escape these protective mechanisms, and permanent cellular mutations occur. These mutations usually are not significant to cells until the second step of carcinogenesis.

During *promotion,* repeated exposure to promoting agents (cocarcinogens) causes the expression of abnormal or mutant genetics information even after long latency periods. Latency periods for the promotion of cellular mutations vary with the type of agent and the dosage of the promoter as well as the innate characteristics of the target cell.

Cellular oncogenes are responsible for the vital cellular functions of growth and differentiation. Cellular proto-oncogenes act as an "on switch" for cellular growth. Proto-oncogenes are influenced by multiple growth factors that stimulate cell proliferation, such as epidermal growth factor (EGF) and transforming growth factor alpha. Another proto-oncogene that plays an important role in cancer development is the *k-ras (KRAS2)* oncogene located on chromosome 12.

Just as proto-oncogenes "turn on" cellular growth, cancer suppressor genes "turn off," or regulate, unneeded cellular proliferation. When suppressor genes mutate or lose their regulatory capabilities, malignant cells are allowed to reproduce. The *p53 (TP53)* gene is a tumor suppressor gene that is frequently implicated in many human cancers. This gene determines whether cells will live or die after their DNA is damaged. **Apoptosis** is the innate cellular process of programmed cell death. Alterations in *TP53* may decrease apoptotic signals, thus giving rise to a survival advantage for mutant cell populations. Mutant *TP53* is associated with a poor prognosis and may be associated with determining response to treatment. Once this genetic expression occurs in cells, the cells begin to produce mutant cell populations that are different from their original cellular ancestors.

During *progression,* the altered cells exhibit increased malignant behavior. These cells have a propensity to invade adjacent tissues and to metastasize. Agents that initiate or promote cellular transformation are referred to as carcinogens.

Etiology

Categories of agents or factors implicated in carcinogenesis include viruses and bacteria, physical agents, chemical agents, genetic or familial factors, dietary factors, and hormonal agents.

Viruses and Bacteria

Viruses are difficult to evaluate as a cause of human cancers because they are difficult to isolate. However, infectious causes are considered or suspected when specific cancers appear in clusters. Viruses are thought to incorporate themselves in the genetic structure of cells, thus altering future generations of that cell population, perhaps leading to cancer. For example, the Epstein-Barr virus is highly suspect as a cause in Burkitt lymphoma, nasopharyngeal cancers, and some types of non-Hodgkin and Hodgkin lymphoma.

Bacteria have been evaluated as a cause of cancer over the years but with little evidence to support the link of bacteria to cancer. Chronic inflammatory reactions to bacteria and the production of carcinogenic metabolites are possible mechanisms under investigation. In the early 1990s, the International Agency for Research on Cancer (IARC) identified *Helicobacter pylori* (*H. pylori*) as the first bacterium to be termed a definite cause of cancer in humans. *H. pylori* has been associated with an increased incidence of gastric malignancy related to chronic superficial gastritis, with resultant atrophic and metaplastic changes to the gastric mucosa (Schottenfeld & Bebbe-Dimmer, 2006).

Physical Agents

Physical factors associated with carcinogenesis include exposure to sunlight or radiation, chronic irritation or inflammation, and tobacco use.

Excessive exposure to the ultraviolet rays of the sun, especially in fair-skinned, blue- or green-eyed people, increases the risk of skin cancers. Factors such as clothing styles (sleeveless shirts or shorts); use of sunscreens; occupation; recreational habits; and environmental variables, including humidity, altitude, and latitude, all play a role in the amount of exposure to ultraviolet light.

Exposure to ionizing radiation can occur with repeated diagnostic x-ray procedures or with radiation therapy used to treat disease. Fortunately, improved x-ray equipment minimizes the risk of extensive radiation exposure. Radiation therapy used in disease treatment and exposure to radioactive materials at nuclear weapon manufacturing sites or nuclear power plants are associated with a higher incidence of leukemias, multiple myeloma, and cancers of the lung, bone, breast, thyroid, and other tissues. Background radiation from the natural decay processes that produce radon has also been associated with lung cancer. Homes with high levels of trapped radon should be ventilated to allow the gas to disperse into the atmosphere.

Chemical Agents

About 75% of all cancers are thought to be related to the environment. Most hazardous chemicals produce their toxic effects by altering DNA structure in body sites distant from chemical exposure. The liver, lungs, and kidneys are the organ systems most often affected, presumably because of their roles in detoxifying chemicals.

Tobacco smoke, thought to be the single most lethal chemical carcinogen, accounts for at least 30% of cancer deaths. Smoking is strongly associated with cancers of the lung, head and neck, esophagus, stomach, pancreas, cervix, kidney, and bladder and with acute myeloblastic leukemia. More than 4,000 individual chemicals have been identified in tobacco and tobacco smoke, including more than 60 chemicals that are known carcinogens. Tobacco may also act synergistically with other substances, such as alcohol, asbestos, uranium, and viruses, to promote cancer development. Chewing tobacco is associated with cancers of the oral cavity, which primarily occurs in men younger than 40 years of age. Considerable research has also substantiated the effect of secondhand cigarette smoke as an environmental risk factor for both smokers and nonsmokers (American Cancer Society [ACS], 2008c;2008d).

Many chemical substances found in the workplace have proved to be carcinogens or cocarcinogens. In the United States, carcinogens are classified by two federal agencies: the National Toxicology Program of the Department of Health and Human Services and the Environmental Protection Agency's Integrated Risk Information System (IRIS). The Centers for Disease Control and Prevention established the National Institute for Occupational Safety and Health to provide occupational exposure limits and guidelines for protection of the workforce as regulated by the Occupational Safety and Health Act of 1970. The extensive list of suspected chemical substances continues to grow and includes aromatic amines and aniline dyes; pesticides and formaldehydes; arsenic, soot, and tars; asbestos; benzene; betel nut and lime; cadmium; chromium compounds; nickel and zinc ores; wood dust; beryllium compounds; and polyvinyl chloride.

Genetics and Familial Factors

Almost every cancer type has been shown to run in families. This may be due to genetics, shared environments, cultural or lifestyle factors, or chance alone. Genetic factors play a role in cancer cell development. Abnormal chromosomal patterns and cancer have been associated with extra chromosomes, too few chromosomes, or translocated chromosomes. Specific cancers with underlying genetic abnormalities include Burkitt lymphoma, chronic myelogenous leukemia, meningiomas, acute leukemias, retinoblastomas, Wilms tumor, and skin cancers, including malignant melanoma. Additionally, there are syndromes that represent a cluster of cancers that are identified by a specific genetic alteration that is inherited across generations of a family. In these families, the associated genetic mutation is found in all cells and represents an inherited susceptibility to cancer for all family members who carry the mutation.

Approximately 5% of cancers in adults display a pattern of cancers suggestive of a familial predisposition. The hallmarks of families with a hereditary cancer syndrome include cancer in two or more first-degree or second-degree relatives, early onset of cancer in family members younger than 50 years of age, the same type of cancer in several family members, individual family members with more than one type of cancer, and a rare cancer in one or more family members. There is also evidence of an autosomal dominant inheritance pattern of cancers affecting several generations of the family.

Since the early 1990s, there have been considerable advances in the recognition of inherited cancer susceptibility syndromes and in the ability to isolate and identify the inherited genetic mutation responsible for the cancer patterns. Discoveries of mutations in genes related to critical

CHART 16-1

GENETICS IN NURSING PRACTICE
Concepts and Challenges in Management of the Patient with Cancer

Cancer is a genetic disease. Every phase of carcinogenesis is affected by multiple gene mutations. Some of these mutations are inherited (present in germ-line cells), but most (90%) are somatic mutations that are acquired mutations in specific cells. Examples of cancers influenced by genetics include:

- Cowden syndrome
- Familial adenomatous polyposis
- Familial melanoma syndrome
- Hereditary breast and ovarian cancer
- Hereditary nonpolyposis colon cancer
- Neurofibromatosis type 1
- Retinoblastoma

Nursing Assessments

Family History Assessment

- Obtain information about both maternal and paternal sides of family.
- Obtain cancer history of at least three generations.
- Look for clustering of cancers that occur at young ages, multiple primary cancers in one individual, cancer in paired organs, and two or more close relatives with the same type of cancer suggestive of hereditary cancer syndromes

Patient Assessment

- Physical findings that may predispose the patient to cancer, such as multiple colon polyps, suggestive of a polyposis syndrome
- Skin findings, such as atypical moles, that may be related to familial melanoma syndrome
- Multiple *café au lait* spots, axillary freckling, and two or more neurofibromas associated with neurofibromatosis type 1
- Facial trichilemmomas, mucosal papillomatosis, multinodular thyroid goiter or thyroid adenomas, macrocephaly, fibrocystic breasts and other fibromas or lipomas related to Cowden syndrome

Management Issues Specific to Genetics

- Assess patient's understanding of genetics factors related to his or her cancer
- Refer for cancer risk assessment when a hereditary cancer syndrome is suspected so that patient and family can discuss inheritance risk with other family members and availability of genetic testing
- Offer appropriate genetics information and resources
- Assess patient's understanding of genetics information
- Provide support to patients and families with known genetic test results for hereditary cancer syndromes
- Participate in the management and coordination of risk-reduction measures for those with known gene mutations

Genetics Resources

American Cancer Society—offers general information about cancer and support resources for families, www.cancer.org

Gene Clinics—a listing of common genetic disorders with up-to-date clinical summaries, genetic counseling, and testing information, www.geneclinics.org

Genetic Alliance—a directory of support groups for patients and families with genetic conditions, www.geneticalliance.org

National Cancer Institute—a listing of cancers with clinical summaries and treatment reviews, information on genetic risks for cancer, listing of cancer centers providing genetic cancer risk assessment services, www.cancernet.nci.nih.gov

National Organization of Rare Disorders—a directory of support groups and information for patients and families with rare genetic disorders, www.rarediseases.org

OMIM: Online Mendelian Inheritance in Man—a complete listing of known inherited genetic conditions, www.ncbi.nlm.nih.gov/Omim/mimstats.html

cell control functions, such as tumor suppression, DNA repair mechanisms, and oncogenes, have enabled the appropriate identification of families at risk for these syndromes. Examples of these syndromes include hereditary breast and ovarian cancer syndrome (*BRCA1* and *BRCA2*) and multiple endocrine neoplasia syndrome (*MEN1* and *MEN2*) (Chart 16-1). Cancers associated with familial inheritance syndromes include nephroblastomas, pheochromocytomas, and breast, ovarian, colorectal, stomach, thyroid, renal, prostate, and lung cancers (Nussbaum, McInnes & Willard, 2007).

Dietary Factors

Dietary factors are also linked to environmental cancers. Dietary substances can be proactive (protective), carcinogenic, or cocarcinogenic. The risk of cancer increases with long-term ingestion of carcinogens or cocarcinogens or chronic absence of protective substances in the diet.

Dietary substances that appear to increase the risk of cancer include fats, alcohol, salt-cured or smoked meats, nitrate-containing and nitrite-containing foods, and red and processed meats. Alcohol increases the risk of cancers of the mouth, pharynx, larynx, esophagus, liver, colorectum, and breast. Alcohol intake should be limited to no more than two drinks per day for men and one drink per day for women. Greater consumption of vegetables and fruits is associated with a decreased risk of lung, esophageal, stomach, and colorectal cancers (Kushi, Byers, Doyle, et al., 2006).

A high caloric dietary intake is also associated with an increased cancer risk. Obesity is clearly associated with endometrial cancer, postmenopausal breast cancers, and colon, esophagus, and kidney cancers. There is evidence that obesity also increases the risk for cancers of the pancreas, gallbladder, thyroid, ovary, cervix, prostate, and for multiple myeloma and Hodgkin lymphoma (Kushi, et al., 2006).

Hormonal Agents

Tumor growth may be promoted by disturbances in hormonal balance, either by the body's own (endogenous) hormone production or by administration of exogenous hormones. Cancers of the breast, prostate, and uterus are thought to depend on endogenous hormonal levels for growth. Diethylstilbestrol (DES) has long been recognized as a cause of vaginal carcinomas. Oral contraceptives and prolonged estrogen therapy are associated with an increased

incidence of hepatocellular, endometrial, and breast cancers, but they decrease the risk of ovarian cancer. The combination of estrogen and progesterone appears safer than estrogen alone in decreasing the risk of endometrial cancers; however, studies support discontinuing hormonal therapy containing both estrogen and progestin because of the increased risk of breast cancer, coronary heart disease, stroke, and blood clots (Chlebowski, Anderson, Pettinger, et al., 2008).

Hormonal changes related to the female reproductive cycle are also associated with cancer incidence. Early onset of menses under age 12 and delayed onset of menopause after age 55, nulliparity (never giving birth), and delayed childbirth after age 30 are all associated with an increased risk of breast cancer. Increased numbers of pregnancies are associated with a decreased incidence of breast, endometrial, and ovarian cancers.

Role of the Immune System

In humans, malignant cells are capable of developing on a regular basis. However, some evidence indicates that the immune system can detect the development of malignant cells and destroy them before cell growth becomes uncontrolled. When the immune system fails to identify and stop the growth of malignant cells, clinical cancer develops.

Patients who are immunocompromised have an increased incidence of cancer. Organ transplant recipients who receive immunosuppressive therapy to prevent rejection of the transplanted organ have an increased incidence of lymphoma, Kaposi's sarcoma, squamous cell cancer of the skin, and cervical and anogenital cancers (Herman, Rogers & Ratner, 2007). Patients with immunodeficiency diseases, such as acquired immunodeficiency syndrome (AIDS), have an increased incidence of Kaposi's sarcoma, lymphoma, rectal cancer, and head and neck cancers (Grulich, Vajdic & Cozen, 2007). Some patients who have received alkylating chemotherapeutic agents to treat cancer have an increased incidence of secondary malignancies (Tward, Glenn, Pulsipher, et al., 2007). Autoimmune diseases, such as rheumatoid arthritis and Sjögren syndrome, are associated with increased cancer development (Wolf & Michaud, 2007). Finally, age-related changes, such as declining organ function, increased incidence of chronic diseases, and diminished immunocompetence, may contribute to an increased incidence of cancer in older people.

Normal Immune Responses

Normally, an intact immune system has the ability to combat cancer cells in several ways. Usually, the immune system recognizes as foreign certain antigens on the cell membranes of many cancer cells. These antigens, known as tumor-associated antigens (also called tumor cell antigens), are capable of stimulating both cellular and humoral immune responses.

Along with the macrophages, T lymphocytes, the soldiers of the cellular immune response, are responsible for recognizing tumor-associated antigens. When T lymphocytes recognize tumor antigens, other T lymphocytes that are toxic to the tumor cells are stimulated. These lymphocytes proliferate and are released into the circulation. In addition to possessing cytotoxic (cell-killing) properties, T lymphocytes can stimulate other components of the immune system to rid the body of malignant cells.

Certain lymphokines, which are substances produced by lymphocytes, are capable of killing or damaging various types of malignant cells. Other lymphokines can mobilize other immune system cells, such as macrophages, that disrupt cancer cells. Interferon, a substance produced by the body in response to viral infection, also possesses some antitumor properties. Antibodies produced by B lymphocytes, associated with the humoral immune response, also defend the body against malignant cells. These antibodies act either alone or in combination with the complement system or the cellular immune system.

Natural killer (NK) cells are a major component of the body's defense against cancer. NK cells are a subpopulation of lymphocytes that act by directly destroying cancer cells or by producing lymphokines and enzymes that assist in cell destruction.

Immune System Failure

Several theories explain how malignant cells can survive and proliferate despite the elaborate immune system defense mechanisms. If the body fails to recognize the malignant cell as different from "self" (ie, as nonself or foreign), the immune response may not be stimulated. When tumors do not possess tumor-associated antigens that label them as foreign, the immune response is not alerted. This allows the tumor to grow too large to be managed by normal immune mechanisms.

Tumor antigens may combine with the antibodies produced by the immune system and hide or disguise themselves from normal immune defense mechanisms. These tumor antigen–antibody complexes can suppress further production of antibodies. Tumors can also alter their appearance or produce substances that impair usual immune responses. These substances promote tumor growth and increase the patient's susceptibility to infection. After prolonged contact with a tumor antigen, the body may be depleted of the specific lymphocytes and no longer be able to mount an appropriate immune response.

Abnormal concentrations of host suppressor T lymphocytes may play a role in cancer development. Suppressor T lymphocytes normally assist in regulating antibody production and diminishing immune responses when they are no longer required. Low levels of antibodies and high levels of suppressor cells have been found in patients with multiple myeloma, a cancer associated with hypogammaglobulinemia (low amounts of serum antibodies). Carcinogens, such as viruses and certain chemicals, including chemotherapeutic agents, may weaken the immune system and ultimately enhance tumor growth.

Detection and Prevention of Cancer

Nurses and physicians have traditionally been involved with tertiary prevention, the care, and rehabilitation of patients after cancer diagnosis and treatment. However, the American Cancer Society, the National Cancer Institute, clinicians, and researchers also place emphasis on primary

CHART 16-2

HEALTH PROMOTION

American Cancer Society (ACS) Guidelines on Nutrition and Physical Activity for Cancer Prevention

ACS Recommendations for Individual Choices

- Maintain a healthy weight throughout life
  - Balance caloric intake with physical activity
  - Avoid excessive weight gain throughout the life cycle
  - Achieve and maintain a healthy weight if currently overweight or obese
- Adopt a physically active lifestyle
  - Adults: engage in at least 30 minutes of moderate to vigorous physical activity, above usual activities, on 5 or more days of the week; 45 to 60 minutes of intentional physical activity are preferable
  - Children and adolescents: engage in at least 60 minutes per day of moderate to vigorous physical activity at least 5 days per week
- Consume a healthy diet, with an emphasis on plant sources
  - Choose foods and beverages in amounts that help achieve and maintain a healthy weight
  - Eat five or more servings of a variety of vegetables and fruits each day
  - Choose whole grains in preference to processed (refined) grains
  - Limit consumption of processed and red meats

If you drink alcoholic beverages, limit consumption. Drink no more than one drink per day for women or two per day for men.

ACS Recommendations for Community Action

- Public, private, and community organizations should work to create social and physical environments that support the adoption and maintenance of healthful nutrition and physical activity behaviors
- Increase access to healthful foods in schools, worksites, and communities
- Provide safe, enjoyable, and accessible environments for physical activity in schools, and for transportation and recreation in communities

From Kushi, L. H., Byers, T., Doyle, C., et al. (2006). American Cancer Society guidelines on nutrition and physical activity for cancer prevention: Reducing the risk of cancer with healthy food choices and physical activity. *CA Cancer Journal for Clinicians, 56*(5), 254–281, with permission.

and secondary prevention of cancer. Nurses must be aware of factors such as race, cultural influences, access to care, patient–physician and patient–nurse relationships, level of education, income, and age that influence the knowledge, attitudes, and beliefs individuals have about cancer. These factors also may affect the health-promoting behaviors that people practice.

Primary Prevention

Primary prevention is concerned with reducing the risks of disease through health promotion strategies. It is estimated that almost one third of all cancers worldwide could be prevented through primary prevention efforts (Williams-Brown & Singh, 2005). By acquiring the knowledge and skills necessary to educate the community about cancer risk, nurses in all settings play a key role in cancer prevention. One way to reduce the risk of cancer is to help patients avoid known carcinogens. Another strategy involves encouraging patients to make dietary and lifestyle changes (smoking cessation, decreased caloric intake, increased physical activity) that studies show influence the risk for cancer. Nurses use their teaching and counseling skills to provide patient education and support public education campaigns through organizations, such as the ACS, that guide patients and families in taking steps to reduce cancer risks through health promotion behaviors (Chart 16-2).

Several clinical trials have been conducted to identify medications or supplements that may help reduce the incidence of certain types of cancer. For example, large-scale breast cancer prevention studies supported by the National Cancer Institute (NCI) indicated that chemoprevention with the medication tamoxifen (Nolvadex) can reduce the incidence of breast cancer by 50% in women at high risk for breast cancer (Fisher, Constantino, Wickerham, et al., 2005). Currently, the NCI (2008) lists 110 ongoing clinical trials exploring chemoprevention strategies.

Secondary Prevention

Secondary prevention programs promote screening and early detection activities such as breast and testicular self-examination and Papanicolaou (Pap) tests. Many organizations conduct cancer screening events that focus on cancers with the highest incidence rates or those that have improved survival rates if diagnosed early, such as breast or prostate cancer. These events offer education and examinations such as mammograms, digital rectal examinations, and PSA blood tests for minimal or no cost. These programs often target people who lack access to health care insurance or who cannot afford to participate on their own.

The evolving understanding of the role of genetics in cancer cell development has contributed to prevention and screening efforts. Many centers across the country are offering innovative cancer risk evaluation programs that provide in-depth screening and follow-up screening for people who are found to be at high risk for cancer. Nurses in all settings can develop programs that identify risks for patients and families and that incorporate teaching and counseling into all educational efforts, particularly for patients and families with a high incidence of cancer. Nurses and physicians can encourage people to comply with detection efforts as suggested by the ACS (Table 16-2).

Table 16-2 AMERICAN CANCER SOCIETY (ACS) RECOMMENDATIONS FOR THE EARLY DETECTION OF CANCER IN AVERAGE-RISK ASYMPTOMATIC PEOPLE

Cancer Site	Population	Test or Procedure	Frequency
Breast	Women, aged ≥20 years	Breast self-examination (BSE)	Beginning in their early 20s, women should be told about the benefits and limitations of BSE. The importance of prompt reporting of any new breast symptoms to a health professional should be emphasized. Women who choose to do BSE should receive instruction and have their technique reviewed on the occasion of a periodic health examination. It is acceptable for women to choose not to do BSE or to do BSE irregularly.
		Clinical breast examination (CBE)	For women in their 20s and 30s, it is recommended that CBE be part of a periodic health examination, preferably at least every 3 years. Asymptomatic women aged ≥40 years should continue to receive a clinical breast examination as part of a periodic health examination, preferably annually.
		Mammography	Begin annual mammography at age 40 years.*
Colorectal	Men and women, aged ≥50 years	Fecal occult blood test (FOBT)† *or* fecal immunochemical test (FIT), *or* Flexible sigmoidoscopy, *or* Fecal occult blood test (FOBT)† and flexible sigmoidoscopy,‡ *or* contrast barium enema (DCBE), *or*	Annual, starting at age 50 years Every 5 years, starting at age 50 years Annual FOBT (or FIT) and flexible sigmoidoscopy every 5 years, starting at age 50 years DCBE every 5 years, starting at age 50 years Colonoscopy every 10 years, starting at age 50 years
Prostate	Men, aged ≥50 years	Digital rectal examination (DRE) and prostate-specific antigen (PSA) test	The PSA test and the DRE should be offered annually, starting at age 50 years, for men who have a life expectancy of at least 10 years§
Cervix	Women, aged ≥18 years	Pap test	Cervical cancer screening should begin approximately 3 years after a woman begins having vaginal intercourse, but no later than age 21 years. Screening should be done every year with conventional Pap tests or every 2 years using liquid-based Pap tests. At or after age 30 years, women who have had normal test results in a row may get screened every 2 to 3 years with cervical cytology (either conventional or liquid-based Pap test) alone, or every 3 years with a human papillomavirus DNA test, plus cervical cytology. Women aged ≥70 years who have had or more normal Pap tests and no abnormal Pap tests in the 10 years and women who have had a total hysterectomy may choose to stop cervical cancer screening.
Endometrial	Women, at menopause	At the time of menopause, women at average risk should be informed about risks and symptoms of endometrial cancer and strongly encouraged to report any unexpected bleeding or spotting to their physicians.	
Cancer-related checkup	Men and women, aged ≥20 years	On the occasion of a periodic health examination, the cancer-related check-up should include examination for cancers of the thyroid, testicles, ovaries, lymph nodes, oral cavity, and skin, as well as health counseling about tobacco, sun exposure, diet and nutrition, risk factors, sexual practices, and environmental and occupational exposures.	

*Beginning at age 40 years, annual clinical breast examination should be performed prior to mammography.

†FOBT, as it is sometimes done in physicians' offices with the single stool sample collected on a fingertip during a DRE, is not an adequate substitute for the recommended at-home procedure of collecting two samples from three consecutive specimens, and is not recommended. Toilet bowl FOBT tests also are not recommended. In comparison with guaiac-based tests for the detection of occult blood, immunochemical tests are more patient-friendly and are likely to be equal or better in sensitivity and specificity. There is no justification for repeating FOBT in response to an initial positive finding. Patients with a positive screening FOBT should undergo colonoscopy.

‡Flexible sigmoidoscopy together with FOBT is preferred compared with FOBT or flexible sigmoidoscopy alone.

§Information should be provided to men about the benefits and limitations of testing so that an informed decision about testing can be made with the clinician's assistance.

From Smith, R. A., Cokkinides, V. & Eyre, H. J. (2007). Cancer screening in the United States, 2007: A review of current guidelines, practices and prospects. *CA Cancer Journal for Clinicians*, 57(2), 90–104, with permission.

Diagnosis of Cancer

A cancer diagnosis is based on assessment of physiologic and functional changes and results of the diagnostic evaluation. Patients with suspected cancer undergo extensive testing to (1) determine the presence and extent of tumor, (2) identify possible spread (metastasis) of disease or invasion of other body tissues, (3) evaluate the function of involved and uninvolved body systems and organs, and (4) obtain tissue and cells for analysis, including evaluation of tumor stage and grade. The diagnostic evaluation includes a review of systems, physical examination, imaging studies, laboratory tests of blood, urine and other body fluids, and surgical and pathology reports. Knowledge of suspicious symptoms and of the behavior of particular types of cancer assists in determining relevant diagnostic tests (Table 16-3).

Patients undergoing extensive testing may be fearful of the procedures and anxious about the possible test results. Nurses help relieve the patient's fear and anxiety by explaining the tests to be performed, the sensations likely to be experienced, and the patient's role in the test procedures. The nurse encourages the patient and family to voice their fears about the test results, supports the patient and family throughout the test period, and reinforces and clarifies information conveyed by the physician. The nurse also encourages the patient and family to communicate and share their concerns and to discuss their questions and concerns with one another.

Tumor Staging and Grading

A complete diagnostic evaluation includes identifying the stage and grade of the tumor. This is accomplished prior to treatment to provide baseline data for evaluating outcomes of therapy and to maintain a systematic and consistent approach to ongoing diagnosis and treatment. Treatment options and prognosis are based on staging and grading.

Staging determines the size of the tumor and the existence of local invasion and distant metastasis. Several systems exist for classifying the anatomic extent of disease. The tumor, nodes, and metastasis (TNM) system is frequently used (American Joint Committee on Cancer, 2006) (Chart 16-3). A variety of other staging systems are also used to describe the extent of cancers, such as central nervous system (CNS) cancers, hematologic cancers, and malignant melanoma, which are not well described by the TNM system. Staging systems also provide a convenient shorthand notation that condenses lengthy descriptions into manageable terms for comparisons of treatments and prognoses.

Table 16-3 DIAGNOSTIC AIDS USED TO DETECT CANCER

Test	Description	Examples of Diagnostic Uses
Tumor marker identification	Analysis of substances found in body—tissues, blood, or other body fluids that are made by the tumor or by the body in response to the tumor	Breast, colon, lung, ovarian, testicular, prostate cancers
Genetic profiling	Analysis for the presence of mutations (alterations) in genes found in tumors or body tissues. Assists in diagnosis, selection of treatment, prediction of response to therapy, and risk of progression or recurrence	Breast, lung, kidney, ovarian, brain cancers, leukemia, and lymphoma (many uses of genetic profiling are considered investigational)
Mammography	Use of x-ray images of the breast	Breast cancer
Magnetic resonance imaging (MRI)	Use of magnetic fields and radiofrequency signals to create sectioned images of various body structures	Neurologic, pelvic, abdominal, thoracic, breast cancers
Computed tomography (CT)	Use of narrow-beam x-ray to scan successive layers of tissue for a cross-sectional view	Neurologic, pelvic, skeletal, abdominal, thoracic cancers
Fluoroscopy	Use of x-rays that identify contrasts in body tissue densities; may involve the use of contrast agents	Skeletal, lung, gastrointestinal cancers
Ultrasonography (ultrasound)	High-frequency sound waves echoing off body tissues are converted electronically into images; used to assess tissues deep within the body	Abdominal and pelvic cancers
Endoscopy	Direct visualization of a body cavity or passageway by insertion of an endoscope into a body cavity or opening; allows tissue biopsy, fluid aspiration, and excision of small tumors. Used for diagnostic and therapeutic purposes	Bronchial, gastrointestinal cancers
Nuclear medicine imaging	Uses intravenous injection or ingestion of radioisotope substances followed by imaging of tissues that have concentrated the radioisotopes	Bone, liver, kidney, spleen, brain, thyroid cancers
Positron emission tomography (PET)	Through the use of a tracer, provides black and white or color-coded images of the biologic activity of a particular area, rather than its structure. Used in detection of cancer or its response to treatment	Lung, colon, liver, pancreatic, head and neck cancers; Hodgkin and non-Hodgkin lymphoma and melanoma
PET fusion	Use of a PET scanner and a CT scanner in one machine to provide an image combining anatomic detail, spatial resolution, and functional metabolic abnormalities	See PET
Radioimmunoconjugates	Monoclonal antibodies are labeled with a radioisotope and injected intravenously into the patient; the antibodies that aggregate at the tumor site are visualized with scanners	Colorectal, breast, ovarian, head and neck cancers; lymphoma and melanoma

Chart 16-3 • *TNM Classification System*

T The extent of the primary tumor
N The absence or presence and extent of regional lymph node metastasis
M The absence or presence of distant metastasis

The use of numerical subsets of the TNM components indicates the progressive extent of the malignant disease.

Primary Tumor (T)

Tx Primary tumor cannot be assessed
T0 No evidence of primary tumor
Tis Carcinoma in situ
T1, T2, T3, T4 Increasing size and/or local extent of the primary tumor

Regional Lymph Nodes (N)

Nx Regional lymph nodes cannot be assessed
N0 No regional lymph node metastasis
N1, N2, N3 Increasing involvement of regional lymph nodes

Distant Metastasis (M)

Mx Distant metastasis cannot be assessed
M0 No distant metastasis
M1 Distant metastasis

From American Joint Committee on Cancer. (2006). *AJCC cancer staging atlas.* Chicago: Springer Science and Business Media, Inc.

Grading refers to the classification of the tumor cells. Grading systems seek to define the type of tissue from which the tumor originated and the degree to which the tumor cells retain the functional and histologic characteristics of the tissue of origin (differentiation). Samples of cells used to establish the grade of a tumor may be obtained from tissue scrapings, body fluids, secretions, or washings, biopsy, or surgical excision. This information helps the health care team predict the behavior and prognosis of various tumors. The tumor is assigned a numeric value ranging from I to IV. Grade I tumors, also known as well-differentiated tumors, closely resemble the tissue of origin in structure and function. Tumors that do not clearly resemble the tissue of origin in structure or function are described as poorly differentiated or undifferentiated and are assigned grade IV. These tumors tend to be more aggressive and less responsive to treatment than well-differentiated tumors.

Management of Cancer

Treatment options offered to cancer patients should be based on treatment goals for each specific type of cancer. The range of possible treatment goals may include complete eradication of malignant disease (**cure**), prolonged survival and containment of cancer cell growth (**control**), or relief of symptoms associated with the disease (**palliation**).

The health care team, the patient, and the patient's family must have a clear understanding of the treatment options and goals. Open communication and support are vital as the patient and family periodically reassess treatment plans and goals when complications of therapy develop or disease progresses.

Multiple modalities are commonly used in cancer treatment. A variety of approaches, including surgery, radiation therapy, chemotherapy, and targeted therapies, may be used at various times throughout treatment. Understanding the principles of each and how they interrelate is important in understanding the rationale and goals of treatment.

Surgery

Surgical removal of the entire cancer remains the ideal and most frequently used treatment method. However, the specific surgical approach may vary for several reasons. Diagnostic surgery is the definitive method of identifying the cellular characteristics that influence all treatment decisions. Surgery may be the primary method of treatment, or it may be prophylactic, palliative, or reconstructive.

Diagnostic Surgery

Diagnostic surgery, such as a **biopsy,** is usually performed to obtain a tissue sample for analysis of cells suspected to be malignant. In most instances, the biopsy is taken from the actual tumor, but in some situations, it is necessary to biopsy lymph nodes near the suspicious tumor. Many cancers can metastasize from the primary site to other areas of the body through the lymphatic circulation. Knowing whether adjacent lymph nodes contain tumor cells helps physicians plan for systemic therapies instead of or in addition to surgery or radiation, to combat tumor cells that have gone beyond the primary tumor site. The use of injectable dyes and nuclear medicine imaging can help the surgeon identify the sentinel lymph node or the initial lymph node to which the primary tumor and surrounding tissue drains. Sentinel lymph node biopsy (SLNB), also known as sentinel lymph node mapping, is a minimally invasive surgical approach that in some instances has replaced more invasive lymph node dissections (lymphadenectomy) and their associated complications such as lymphedema and delayed healing. SLNB has been widely adopted for regional lymph node staging in selected cases of melanoma and breast cancer (Chen, Iddings, Scheri, et al., 2006).

Biopsy Types

The three most common biopsy methods are the excisional, incisional, and needle methods (Szopa, 2005). The choice of biopsy is determined by the size and location of the tumor, the type of treatment anticipated if the cancer diagnosis is confirmed, and the need for surgery and general anesthesia. The biopsy method that allows for the least invasive approach while permitting the most representative tissue sample is chosen. Occasionally diagnostic imaging techniques are used to assist in locating the suspicious lesion and to facilitate accurate tissue sampling. The patient and family are given the opportunity and time to discuss the options before definitive plans are made. The nurse serves as the patient's advocate and liaison between the patient and physician to facilitate this process.

Excisional biopsy is most frequently used for easily accessible tumors of the skin, breast, and upper or lower gastrointestinal and upper respiratory tracts. In many cases, the surgeon can remove the entire tumor as well as the surrounding marginal tissues. The removal of normal tissue beyond the tumor area decreases the possibility that residual microscopic disease cells may lead to a recurrence of the tumor. This approach not only provides the pathologist, who

stages and grades the cells, with the entire tissue specimen but also decreases the chance of seeding the tumor (disseminating cancer cells throughout surrounding tissues).

Incisional biopsy is performed if the tumor mass is too large to be removed. In this case, a wedge of tissue from the tumor is removed for analysis. The cells of the tissue wedge must be representative of the tumor mass so that the pathologist can provide an accurate diagnosis. If the specimen does not contain representative tissue and cells, negative biopsy results do not guarantee the absence of cancer.

Excisional and incisional approaches are often performed through endoscopy. However, surgical incision may be required to determine the anatomic extent or stage of the tumor. For example, a diagnostic or staging laparotomy (the surgical opening of the abdomen to assess malignant abdominal disease) may be necessary to assess malignancies such as gastric cancer.

Needle biopsies are performed to sample suspicious masses that are easily accessible, such as some growths in the breasts, thyroid, lung, liver, and kidney. Needle biopsies are most often performed on an outpatient basis. They are fast, relatively inexpensive, easy to perform, and usually require only local anesthesia. In general, the patient experiences slight and temporary physical discomfort. In addition, the surrounding tissues are disturbed only minimally, thus decreasing the likelihood of seeding cancer cells. Needle aspiration biopsy involves aspirating tissue fragments through a needle guided into an area suspected of bearing disease. Occasionally, x-ray, computed tomography (CT) scanning, ultrasonography, or magnetic resonance imaging (MRI) is used to help locate the suspicious area and guide the placement of the needle. In some instances, the aspiration biopsy does not yield enough tissue to permit accurate diagnosis. A needle core biopsy uses a specially designed needle to obtain a small core of tissue. Most often, this specimen is sufficient to permit accurate diagnosis.

Surgery as Primary Treatment

When surgery is the primary approach in treating cancer, the goal is to remove the entire tumor or as much as is feasible (a procedure sometimes called debulking) and any involved surrounding tissue, including regional lymph nodes.

Two common surgical approaches used for treating primary tumors are local and wide excisions. Local excision, often performed on an outpatient basis, is warranted when the mass is small. It includes removal of the mass and a small margin of normal tissue that is easily accessible. Wide or radical excisions (en bloc dissections) include removal of the primary tumor, lymph nodes, adjacent involved structures, and surrounding tissues that may be at high risk for tumor spread (Szopa, 2005). This surgical method can result in disfigurement and altered functioning, necessitating rehabilitation or reconstructive procedures. However, wide excisions are considered if the tumor can be removed completely and the chances of cure or control are good.

Video-assisted endoscopic surgery is increasingly replacing surgery associated with long incisions and extended recovery periods to minimize surgical trauma and shorten patient recovery time without compromising surgical outcomes (Swanson, Herndon, D'Amico, et al., 2007). In this minimally invasive procedure, an endoscope with intense lighting and an attached multichip mini-camera is inserted into the body through a small incision. The surgical instruments are inserted into the surgical field through one or two additional small incisions, each about 3 cm in length. The camera transmits the image of the involved area to a monitor so the surgeon can manipulate the instruments to perform the necessary procedure. Such surgery is now being used for many thoracic and abdominal surgeries.

Salvage surgery is an additional treatment option that uses an extensive surgical approach to treat the local recurrence of a cancer after the use of a less extensive primary approach. A mastectomy to treat recurrent breast cancer after primary lumpectomy and radiation is an example of salvage surgery.

In addition to surgery that uses surgical blades or scalpels to excise the mass and surrounding tissues, several other types of surgical techniques are available. Table 16-4 identifies these techniques and provides examples of their use in the patient with cancer. A multidisciplinary approach to patient care is essential for the patient undergoing cancer-related surgery. The effects of surgery on the patient's body image, self-esteem, and functional abilities are addressed. If necessary, a plan for postoperative rehabilitation is made before the surgery is performed.

The growth and dissemination of cancer cells may have produced distant micrometastases by the time the patient

Table 16-4 SELECTED TECHNIQUES USED TO REMOVE OR DESTROY TUMORS

Type of Procedure	Description	Examples of Use
Electrosurgery	Use of an electric current to destroy tumor cells	Basal and squamous cell skin cancers
Cryosurgery	Use of liquid nitrogen or a very cold probe to freeze tissue and cause cell destruction	Cervical and prostate cancers
Chemosurgery	Use of chemicals or chemotherapy applied directly to tissue to cause destruction	Intraperitoneal chemotherapy for ovarian cancer involving the abdomen and peritoneum
Laser surgery	Use of light and energy aimed at an exact tissue location and depth to vaporize cancer cells (also referred to as photocoagulation or photoablation)	Dyspnea associated with endobronchial obstructions
Photodynamic therapy	Intravenous administration of a light-sensitizing agent (hematoporphyrin derivative [HPD]) that is taken up by cancer cells, followed by exposure to laser light within 24–48 hours; causes cancer cell death	Palliative treatment of dysphagia associated with esophageal and dyspnea associated with endobronchial obstructions
Radiofrequency ablation (RFA)	Uses localized application of thermal energy that destroys cancer cells through heat: temperatures exceed 50°C (122°F)	Nonresectable liver tumors, pain control with bone metastasis

Table 16-5 INDICATIONS FOR PALLIATIVE SURGERY

Procedure	Indications
Pleural drainage tube placement	Pleural effusion
Peritoneal drainage tube placement (Tenckhoff catheter)	Ascites
Abdominal shunt placement (Levine shunt)	Ascites
Pericardial drainage tube placement	Pericardial effusion
Colostomy or ileostomy	Bowel obstruction
Gastrostomy, jejunostomy tube placement	Upper gastrointestinal tract obstruction
Biliary stent placement	Biliary obstruction
Bone stabilization	Displaced bone fracture related to metastatic disease
Excision of solitary metastatic lesion	Metastatic lung, liver or brain lesion
Ureteral stent placement	Ureteral obstruction
Nerve block	Pain
Cordotomy	Pain
Venous access device placement (for administering parenteral analgesics)	Pain
Epidural catheter placement (for administering epidural analgesics)	Pain
Hormone manipulation (removal of ovaries, testes, adrenals, pituitary)	Tumors that depend on hormones for growth

seeks treatment. Therefore, attempting to remove wide margins of tissue in the hope of "getting all the cancer" may not be feasible. This reality substantiates the need for a coordinated multidisciplinary approach to cancer therapy.

Once the surgery has been completed, one or more additional (or adjuvant) modalities may be chosen to increase the likelihood of destroying the remaining cancer cells. However, some cancers that are treated surgically in the very early stages (eg, skin and testicular cancers) are considered to be curable without additional therapies.

Prophylactic Surgery

Prophylactic surgery involves removing nonvital tissues or organs that are at increased risk to develop cancer. The following factors are considered when physicians, nurses, patients, and families discuss possible prophylactic surgery:

- Family history and genetic predisposition
- Presence or absence of symptoms
- Potential risks and benefits
- Ability to detect cancer at an early stage
- The patient's acceptance of the postoperative outcome

Colectomy, mastectomy, and oophorectomy are examples of prophylactic surgeries. Identification of genetic markers indicative of a predisposition to develop some types of cancer plays a role in decisions concerning prophylactic surgeries. However, what is adequate justification for prophylactic surgery remains controversial. For example, several factors are considered when deciding to proceed with a prophylactic mastectomy, including a strong family history of breast cancer; positive *BRCA1* or *BRCA2* findings; an abnormal physical finding on breast examination, such as progressive nodularity and cystic disease; a proven history of breast cancer in the opposite breast; abnormal mammography findings; and abnormal biopsy results (Calhoun & Anderson, 2006). Prophylactic surgery is offered selectively to patients and discussed thoroughly with patients and families. Preoperative teaching and counseling, as well as long-term follow-up, are provided.

Palliative Surgery

When cure is not possible, the goals of treatment are to make the patient as comfortable as possible and to promote quality of life as defined by the patient and his or her family. Palliative surgery is performed in an attempt to relieve complications of cancer, such as ulceration, obstruction, hemorrhage, pain, and malignant effusion (Table 16-5). Honest and informative communication with the patient and family about the goal of surgery is essential to avoid false hope and disappointment.

Reconstructive Surgery

Reconstructive surgery may follow curative or radical surgery in an attempt to improve function or obtain a more desirable cosmetic effect. It may be performed in one operation or in stages. The surgeon who will perform the surgery discusses possible reconstructive surgical options with the patient before the primary surgery is performed. Reconstructive surgery may be indicated for breast, head and neck, and skin cancers.

The nurse recognizes the patient's needs and the impact that altered functioning and body image may have on quality of life. The nurse provides the patient and family with opportunities to discuss these issues. The individual needs of the patient undergoing reconstructive surgery must be accurately assessed and addressed.

Nursing Management in Cancer Surgery

Patients undergoing surgery for cancer require general perioperative nursing care, as described in Unit 4 of this text, along with specific care related to age, organ impairment, nutritional deficits, disorders of coagulation, and altered immunity that may increase the risk of postoperative complications. Combining other treatment methods, such as radiation and chemotherapy, with surgery also contributes to postoperative complications, such as infection, impaired wound healing, altered pulmonary or renal function, and the development of deep vein thrombosis. In these situations, the nurse completes a thorough preoperative assessment for

factors that may affect the patient undergoing the surgical procedure.

Patients who are undergoing surgery for the diagnosis or treatment of cancer may be anxious about the surgical procedure, possible findings, postoperative limitations, changes in normal body functions, and prognosis. The patient and family require time and assistance to deal with the possible changes and outcomes resulting from the surgery.

The nurse provides education and emotional support by assessing the needs of the patient and family and by discussing their fears and coping mechanisms. The nurse encourages the patient and family to take an active role in decision making when possible. If the patient or family asks about the results of diagnostic testing and surgical procedures, the nurse's response is guided by the information the physician has previously conveyed to the patient and family. The patient and family may ask the nurse to explain and clarify information that the physician initially provided but that they did not grasp because they were anxious and overwhelmed at the time. It is important that the nurse communicate frequently with the physician and other members of the health care team to be certain that the information provided is consistent.

Postoperatively, the nurse assesses the patient's responses to the surgery and monitors the patient for possible complications, such as infection, bleeding, thrombophlebitis, wound dehiscence, fluid and electrolyte imbalance, and organ dysfunction. The nurse also provides for the patient's comfort. Postoperative teaching addresses wound care, activity, nutrition, and medication information.

Plans for discharge, follow-up, home care, and treatment are initiated as early as possible to ensure continuity of care from hospital to home or from a cancer referral center to the patient's local hospital and health care provider. Patients and families are encouraged to use community resources such as the American Cancer Society for support and information.

Radiation Therapy

More than half of patients with cancer receive a form of **radiation therapy** at some point during treatment. Radiation may be used to cure cancer, as in thyroid carcinomas, localized cancers of the head and neck, and cancers of the uterine cervix. Radiation therapy may also be used to control malignant disease when a tumor cannot be removed surgically or when local nodal metastasis is present, or it can be used neoadjuvantly (prior to local definitive treatment) with or without chemotherapy to reduce the size of a tumor to enable surgical resection. Radiation therapy may be used prophylactically to prevent the spread of a primary cancer to a distant area (eg, irradiating the brain to prevent leukemic infiltration or metastatic lung cancer). Palliative radiation therapy is used to relieve the symptoms of metastatic disease, especially when the cancer has spread to the brain, bone, or soft tissue, or to treat oncologic emergencies, such as superior vena cava syndrome, bronchial airway obstruction, or spinal cord compression.

Two types of ionizing radiation—electromagnetic radiation (x-rays and gamma rays) and particulate radiation (electrons, beta particles, protons, neutrons, and alpha particles)—can lead to tissue disruption. The most harmful tissue disruption is the direct alteration of the DNA molecule within the cells of the tissue. Ionizing radiation breaks the strands of the DNA helix, leading to cell death. It can also lead to the formation of free radicals and irreversibly damage DNA. If the DNA is incapable of repair, the cell may die immediately, or it may initiate cellular suicide, a genetically programmed cell death (Bruner, Haas & Gosselin-Acomb, 2006; Yarbro, Hansen-Frogge & Goodman, 2005).

Cells are most vulnerable to the disruptive effects of radiation during DNA synthesis and mitosis (early S, G_2, and M phases of the cell cycle). Therefore, those body tissues that undergo frequent cell division are most sensitive to radiation therapy. These tissues include bone marrow, lymphatic tissue, epithelium of the gastrointestinal tract, hair cells, and gonads. Slower-growing tissues and tissues at rest (eg, muscle, cartilage, and connective tissues) are relatively radioresistant (less sensitive to the effects of radiation). However, it is important to remember that radiation therapy is localized treatment, and only the tissues that are within the treatment field will be affected by the radiation therapy.

A radiosensitive tumor is one that can be destroyed by a dose of radiation that still allows for cell regeneration in the normal tissue. Tumors that are well oxygenated also appear to be more sensitive to radiation. In theory, therefore, radiation therapy may be enhanced if more oxygen can be delivered to tumors. In addition, if the radiation is delivered when most tumor cells are cycling through the cell cycle, the number of cancer cells destroyed (cell kill) is maximal. Radiation sensitivity is also enhanced in tumors that are smaller in size and that contain cells that are rapidly dividing (highly proliferative) and poorly differentiated (no longer resembling the tissue of origin) (Bruner, et al., 2006).

Certain chemicals, including chemotherapy agents, act as radiosensitizers and sensitize hypoxic (oxygen-poor) tumors to the effects of radiation therapy. Combinations of chemotherapy and radiation therapy are typically used to take advantage of the radiosensitizing effects of chemotherapy and achieve an improved survival benefit while minimizing side effects of such therapy.

Radiation Dosage

The radiation dosage depends on the sensitivity of the target tissues to radiation, the size of the tumor, tissue tolerance of the surrounding normal tissues, and critical structures adjacent to the tumor target. The lethal tumor dose is defined as that dose that will eradicate 95% of the tumor yet preserve normal tissue. In external beam radiation, the total radiation dose is delivered over several weeks in daily doses called fractions. This allows healthy tissue to repair and to achieve greater cell kill by exposing more cells to the radiation as they begin active cell division. Repeated radiation treatments over time (fractionated doses) also allow for the periphery of the tumor to be reoxygenated repeatedly, because tumors shrink from the outside inward. This increases the radiosensitivity of the tumor, thereby increasing tumor cell death (Bruner, et al., 2006; Yarbro, et al., 2005).

Administration of Radiation

Radiation therapy can be administered in a variety of ways depending on the source of radiation used, the location of the tumor, and the type of cancer targeted. The primary

applications include teletherapy (external beam radiation), **brachytherapy** (internal radiation), systemic (radioisotopes), and contact or surface molds.

External Radiation

External beam radiation therapy (EBRT) is the most commonly used form of radiation therapy. The energy utilized in EBRT is either generated from a linear accelerator or from a unit that generates energy directly from a core source of radioactive material such as a GammaKnife™ unit. Through computerized software programs, both approaches are able to shape an invisible beam of highly charged electrons to penetrate the body and target a tumor with pinpoint accuracy. Depending on the size, shape, and location of the tumor, different energy levels are generated to produce a carefully shaped beam that will destroy the targeted tumor, yet spare the surrounding healthy tissue and vital organs in an effort to reduce the treatment toxicities for the patient. With advances in computer technology, these beams can be shaped to a two-dimensional or three-dimensional shape to conform to the exact shape of the tumor as measured by imaging studies such as positron emission tomography (PET), CT, or MRI scans. Recent treatment enhancements in EBRT include the ability to direct different energy levels at different angles directed at the tumor, called intensity modulated radiation therapy (IMRT), which enables higher doses to be delivered to the tumor while sparing the important healthy structures surrounding the tumor. IMRT can be administered as standard daily fractions or as "hyperfractionated" twice daily fractions, which shortens the duration of the patient's treatment schedule. Image-guided radiation therapy (IGRT) uses continuous monitoring of the tumor with ultrasound or CT scans during the treatment to allow for automatic adjustment of the target as the tumor changes shape or position, again in an effort to spare the healthy surrounding tissue and reduce side effects (Sharpe, Craig & Moseley, 2007). The most recent treatment enhancements now include respiratory–gating, where the treatment delivery is actually synchronized with the patient's respiratory cycle, enabling the beam to be adjusted as the tumor moves (Dawson & Jaffray, 2007).

Gamma rays generated from the spontaneous decay of naturally occurring solid source of radioactivity, such as cobalt-60, is one of the oldest forms of EBRT. With the advent of modern linear accelerators, the use of solid radioactive elements are confined primarily to the GammaKnife™ stereotactic radiosurgery unit, which is used as a one-time, high-dose delivery of EBRT for treatment of benign and malignant intracranial lesions.

Stereotactic body radiotherapy (SBRT) is another form of EBRT using higher doses of radiation to penetrate very deeply into the body to control deep-seated tumors that cannot be treated by other approaches such as surgery. SBRT is delivered with considerably higher treatment fraction doses over a short span of time, usually 1 to 5 treatment days, in contrast to 6 to 8 weeks for conventional EBRT (Timmerman, Kavanagh, Cho, et al., 2007). Specialized linear accelerators with the capability of robotically moving around the patient are used to deliver SBRT, such as the CyberKnife™, Trilogy™, and TomoTherapy™ delivery systems, which are being utilized more commonly in community hospital settings.

Proton therapy is another very different approach to EBRT. Proton therapy utilizes high-linear energy transfer (LET) in the form of charged protons generated by a large magnetic unit called a cyclotron. The advantage of proton therapy is that it is capable of delivering its high-energy dose to a deep-seated tumor, with no energy exiting through the patient's healthy tissue behind the tumor, allowing for treatment of deep tumors in close proximity to critical structures such as the heart or major blood vessels (Thornton, Fitzek, Klein, et al., 2007). Due to the limited number of proton units in the United States, most treatment has been investigational in the area of localized prostate cancer, inoperable early stage lung cancer, uveal melanoma, and head and neck tumors. With recent expansion in the number of proton therapy centers, investigation of treatment advantages utilizing proton therapy will be a research priority in the future (Schulz-Ertner & Tsujii, 2007).

Internal Radiation

Internal radiation implantation, or brachytherapy, delivers a high dose of radiation to a localized area. The specific radioisotope for implantation is selected on the basis of its half-life, which is the time it takes for half of its radioactivity to decay. Internal radiation can be implanted by means of needles, seeds, beads, or catheters into body cavities (vagina, abdomen, pleura) or interstitial compartments (breast, prostate). Patients may have many fears or concerns about internal radiation and the nurse must be prepared to explain the various approaches and safety precautions that will be used to protect both the patient and the staff.

Brachytherapy may be delivered as a temporary or a permanent implant. Temporary applications may be delivered as high-dose radiation (HDR) for short periods of time or low-dose radiation (LDR) for a more extended period of time. The primary advantage of HDR sources of brachytherapy is that treatment time is shorter, there is reduced exposure to personnel, and the procedure can typically be performed as an outpatient procedure over several days. HDR brachytherapy can be used for intraluminal, interstitial, intracavitary, and surface lesions.

Intraluminal brachytherapy involves the insertion of catheters or hollow tubes into the lumens of organs so that the radioisotope can be delivered as close to the tumor bed as possible. Obstructive lesions in the bronchus, esophagus, or bile duct can be treated with this approach. Contact or surface application is used for treatment of tumors of the eye such as retinoblastoma in children or ocular melanoma in adults.

Intracavitary radioisotopes are frequently used to treat gynecologic cancers. In these malignancies, the radioisotopes are inserted into specially positioned applicators after their placement is verified by x-ray. Treatment can be achieved with either HDR or LDR brachytherapy sources depending on the extent of disease. LDR therapy requires hospitalization as the patient is treated over several days. Nursing care of the hospitalized LDR patient is essential to maximize effective safe delivery of the therapy and prevention of complications. The patient is maintained on bed rest in a specially prepared private room typically for 72 hours and log-rolled to prevent displacement of the intracavitary

delivery device. An indwelling urinary catheter is inserted to ensure that the bladder remains empty. Low-residue diets and antidiarrheal agents are provided to prevent bowel movements during therapy, which would displace the radioisotopes. Visitors and personnel must limit their time and proximity to the patient due to the risk of radiation exposure. HDR intracavitary brachytherapy is typically delivered as an outpatient procedure in the radiation therapy department over several days.

Interstitial implants, used in treating such malignancies as prostate, pancreatic, or breast cancer, may be temporary or permanent, depending on the radioisotopes used. These implants usually consist of seeds, needles, wires, or small catheters positioned to provide a local radiation source and are infrequently dislodged. With internal radiation therapy, the farther the tissue is from the radiation source, the lower the dosage delivered to the tissue. This spares the noncancerous tissue from the radiation dose. Prostate seed therapy is probably the most frequently used type of interstitial brachytherapy, where small radioactive seeds are placed directly into the prostate gland under ultrasound guidance. These seeds are permanently placed and appropriate safety precautions must be employed for several days due to the risk of radiation exposure to others. Recently, partial breast irradiation utilizing a technique for interstitial isotope employing the MammoSite™ device has shown benefit in certain localized breast cancers. MammoSite™ involves the placement of an inflatable balloon within the cavity created after surgical resection of the breast tumor. HDR brachytherapy fractions are delivered via a radioactive seed inserted into the balloon over the course of 5 days. Studies have shown comparable 5-year outcomes for selected patients, with minimal toxicities and excellent cosmesis, when compared with outcomes with whole breast EBRT for postlumpectomy patients. The advantages for patients are reduced treatment time (5 days versus 6 to 8 weeks), less radiation exposure to healthy tissues and adjacent organs (heart and lungs), less skin reaction, and improved cosmesis of the breast. Nursing care for these patients must include instruction in rigorous catheter care and wound management, as the patient is treated as an outpatient with a double-lumen catheter projecting from the breast (Benitez, Keisch, Vicini, et al., 2007).

Systemic brachytherapy involves the IV administration of a therapeutic radioactive isotope targeted to a specific tumor. Radioactive iodine (I^{131}) is a widely used form of systemic brachytherapy and is the primary treatment for thyroid cancer. Strontium 89 is utilized for bone metastases, samarium 153 is used for metastatic bone lesions, and phosphorus 32 is used for treatment of malignant ascites associated with ovarian cancer. Radioisotopes are now also being used as radioimmunotherapy for the treatment of refractory non-Hodgkin lymphoma (NHL). Radioimmunotherapy involves the administration of a radionuclide chemically conjugated (bound) to a monoclonal antibody (discussed later in this chapter) that specifically targets NHL tumor cells, delivering the radionuclide directly to the tumor and sparing the surrounding healthy tissue. There are currently two radioimmunotherapy agents available for treatment of NHL: ibritumomab tiuxetan (Zevalin), which utilizes yttrium 90 as the radioactive beta emitting nucleotide, and iodine 131 tositumomab (Bexxar), which utilizes I^{131} as the beta and gamma emitting radionuclide.

Toxicity

Toxicity of radiation therapy is localized to the region being irradiated. Toxicity may be increased if concomitant chemotherapy is administered. Acute local reactions occur when normal cells in the treatment area are also destroyed and cellular death exceeds cellular regeneration. Body tissues most affected are those that normally proliferate rapidly, such as the skin; the epithelial lining of the gastrointestinal tract, including the oral cavity; and the bone marrow. Altered skin integrity is a common effect and can include **alopecia** (hair loss). Skin reactions are identified and graded by severity along a continuum ranging from erythema and dry desquamation (flaking of skin), to moist desquamation (dermis exposed, skin oozing serous fluid), and potentially, ulceration. Re-epithelialization occurs after treatments have been completed (McQuestion, 2006).

Alterations in oral mucosa secondary to radiation therapy include **stomatitis** (inflammation of the oral tissues), **xerostomia** (dryness of the mouth), change and loss of taste, and decreased salivation. The entire gastrointestinal mucosa may be involved, and esophageal irritation with chest pain and dysphagia may result. Anorexia, nausea, vomiting, and diarrhea may occur if the stomach or colon is in the irradiated field. Symptoms subside and gastrointestinal re-epithelialization occurs after treatments have been completed.

Bone marrow cells proliferate rapidly, and if sites containing bone marrow (eg, the iliac crest, sternum) are included in the radiation field, anemia, leukopenia (decreased white blood cells [WBCs]), and **thrombocytopenia** (a decrease in platelets) may result. The patient is then at increased risk for infection and bleeding until blood cell counts return to normal. Chronic anemia may occur (Bruner, et al., 2006; Yarbro, et al., 2005).

Research to develop cytoprotective agents that can protect normal tissue from radiation damage continues. The most commonly used cytoprotectant is amifostine (Ethyol), which is utilized in head and neck cancer patients to reduce acute and chronic xerostomia while preserving antitumor efficacy (Bruner, et al., 2006; Hogle, 2007).

Certain systemic side effects are also commonly experienced by patients receiving radiation therapy. These side effects include fatigue, malaise, and anorexia and may be secondary to substances released when tumor cells break down. The effects are temporary and most often subside with the cessation of treatment.

Late effects (months to years after treatment) of radiation therapy may also occur in various body tissues. These effects are chronic, usually produce fibrotic changes secondary to a decreased vascular supply, and are irreversible. Severe late effects may affect the lungs, heart, central nervous system, and bladder. Toxicities may intensify when radiation is combined with other treatment modalities.

Nursing Management in Radiation Therapy

The nurse assesses the patient's skin and oropharyngeal mucosa regularly when radiation therapy is directed to these areas. In addition, nutritional status and general feeling of

well-being are assessed throughout the course of treatment. Evidence-based treatment protocols for nursing management of the toxicities associated with radiation therapy are the focus of nursing research. Assessment and management of these problems are discussed in more detail in the Plan of Nursing Care: The Patient With Cancer (Chart 16-7).

If systemic symptoms, such as weakness and fatigue, occur, the nurse explains that these symptoms are a result of the treatment and do not represent deterioration or progression of the disease. The assessment and nursing management of fatigue is discussed in more detail in the Nursing Care of Patients With Cancer: Fatigue (p. 380).

Protecting Caregivers

When the patient has a radioactive implant in place, the nurse and other health care providers need to protect themselves as well as the patient from the effects of radiation. Patients receiving internal radiation emit radiation while the implant is in place; therefore, contact with the health care team is guided by principles of time, distance, and shielding to minimize exposure of personnel to radiation. Specific instructions are usually provided by the radiation safety officer from the x-ray department and specify the maximum time that can be spent safely in the patient's room, the shielding equipment to be used, and special precautions and actions to be taken if the implant is dislodged. Safety precautions used in caring for a patient receiving brachytherapy include assigning the patient to a private room, posting appropriate notices about radiation safety precautions, having staff members wear dosimeter badges, making sure that pregnant staff members are not assigned to the patient's care, prohibiting visits by children or pregnant visitors, limiting visits from others to 30 minutes daily, and seeing that visitors maintain a 6-foot distance from the radiation source.

Patients with seed implants typically are able to return home; radiation exposure to others is minimal. Information about any precautions, if needed, is provided to the patient and family members to ensure safety. Depending on the dose and energy emitted by a systemic radionuclide, patients may or may not require special precautions or hospitalization (Bruner, et al., 2006). The nurse should explain the rationale for these precautions to keep the patient from feeling unduly isolated.

Chemotherapy

In **chemotherapy,** antineoplastic agents are used in an attempt to destroy tumor cells by interfering with cellular functions, including replication. Chemotherapy is used primarily to treat systemic disease rather than localized lesions that are amenable to surgery or radiation. Chemotherapy may be combined with surgery, radiation therapy, or both to reduce tumor size preoperatively (neoadjuvant), to destroy any remaining tumor cells postoperatively (adjuvant), or to treat some forms of leukemia or lymphoma (primary). The goals of chemotherapy (cure, control, palliation) must be realistic because they will determine the medications that are used and the aggressiveness of the treatment plan.

Cell Kill and the Cell Cycle

Each time a tumor is exposed to a chemotherapy agent, a percentage of the tumor cells (20% to 99%, depending on dosage) is destroyed. Repeated doses of chemotherapy are necessary over a prolonged period to achieve regression of the tumor. Eradication of 100% of the tumor is almost impossible. Instead, the goal of treatment is eradication of enough of the tumor so that the remaining tumor cells can be destroyed by the body's immune system.

Actively proliferating cells within a tumor are the most sensitive to chemotherapeutic agents (the ratio of dividing cells to resting cells is referred to as the growth fraction). Nondividing cells capable of future proliferation are the least sensitive to antineoplastic medications and consequently are potentially dangerous. However, the nondividing cells must be destroyed to eradicate a cancer. Repeated cycles of chemotherapy or sequencing of multiple chemotherapeutic agents are used to kill more tumor cells by destroying these nondividing cells as they begin active cell division.

Reproduction of both healthy and malignant cells follows the cell cycle pattern (Fig. 16-2). The cell cycle time is the time required for one tissue cell to divide and reproduce two identical daughter cells. The cell cycle of any cell has four distinct phases, each with a vital underlying function:

1. G_1 phase—RNA and protein synthesis occur
2. S phase—DNA synthesis occurs

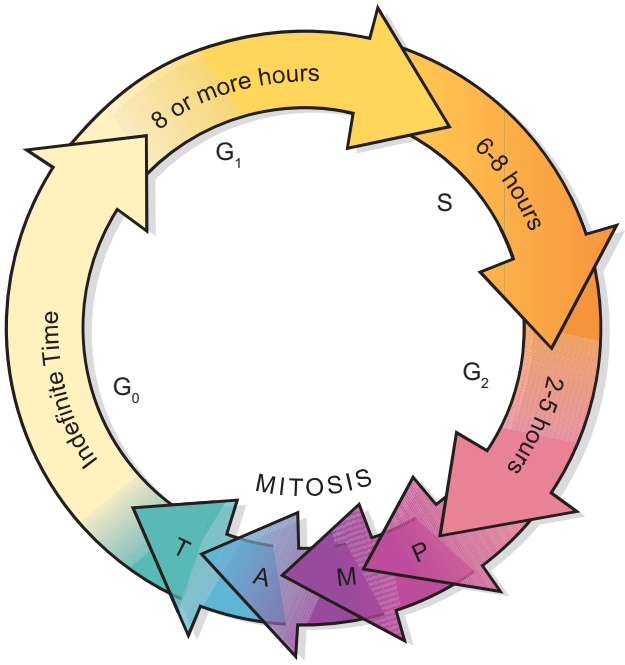

Figure 16-2 Phases of the cell cycle extend over the interval between the midpoint of mitosis and the subsequent end point in mitosis in a daughter cell. G_1 is the postmitotic phase during which ribonucleic acid (RNA) and protein syntheses are increased and cell growth occurs. G_0 is the resting, or dormant, phase of the cell cycle. In the S phase, nucleic acids are synthesized and chromosomes are replicated in preparation for cell mitosis. During G_2, RNA and protein synthesis occur as in G_1. P, prophase; M, metaphase; A, anaphase; T, telophase. Redrawn from Porth, C. M. & Matfin, G. (2009). *Pathophysiology: Concepts of altered health states* (8th ed.). Philadelphia: Lippincott Williams & Wilkins.

3. G_2 phase—premitotic phase; DNA synthesis is complete, mitotic spindle forms
4. Mitosis—cell division occurs

The G_0 phase, the resting or dormant phase of cells, can occur after mitosis and during the G_1 phase. Within the G_0 phase are those dangerous cells that are not actively dividing but have the potential for replicating. The administration of certain chemotherapeutic agents (as well as some other forms of therapy) is coordinated with the cell cycle.

Classification of Chemotherapeutic Agents

Chemotherapeutic agents may be classified by their relationship to the cell cycle. Certain chemotherapeutic agents that are specific to certain phases of the cell cycle are termed cell cycle–specific agents. These agents destroy cells that are actively reproducing by means of the cell cycle; most affect cells in the S phase by interfering with DNA and RNA synthesis. Other agents, such as the vinca or plant alkaloids, are specific to the M phase, where they halt mitotic spindle formation. Chemotherapeutic agents that act independently of the cell cycle phases are termed cell cycle–nonspecific agents. These agents usually have a prolonged effect on cells, leading to cellular damage or death. Many treatment plans combine cell cycle–specific and cell cycle–nonspecific agents to increase the number of vulnerable tumor cells killed during a treatment period (Polovich, White & Kelleher, 2005).

Chemotherapeutic agents are also classified by chemical group, each with a different mechanism of action. These include the alkylating agents, nitrosoureas, antimetabolites, antitumor antibiotics, plant alkaloids, hormonal agents, and miscellaneous agents. The classification, mechanism of action, common drugs, cell cycle specificity, and common side effects of selected antineoplastic agents are listed in Table 16-6.

Chemotherapeutic agents from every category may be used to enhance tumor cell kill during therapy by creating multiple cellular lesions. Combined medication therapy relies on agents of differing toxicities and with synergistic actions. Use of combination therapy also prevents the development of drug-resistant mechanisms.

Combining older medications with other agents, such as levamisole (Ergamisole*), leucovorin (Wellcovorin), hormones, or interferons, has shown some benefit in combating resistance of cells to chemotherapeutic agents. Newer investigational agents are being studied for effectiveness in resistant tumor lines.

Administration of Chemotherapeutic Agents

Chemotherapeutic agents may be administered in the hospital, outpatient center, or home setting by topical, oral, intravenous, intramuscular, subcutaneous, arterial, intracavitary, and intrathecal routes. The route of administration depends on the type of agent; the required dose; and the type, location, and extent of tumor being treated. Guidelines for the safe administration of chemotherapy have been developed by the Oncology Nursing Society (Polovich, et al., 2005). Patient education is essential to maximize safety if chemotherapy is administered in the home (Chart 16-4).

*Ergamisol (without an "e" at end) is not available in U.S.

Dosage

Dosage of antineoplastic agents is based primarily on the patient's total body surface area, previous response to chemotherapy or radiation therapy, and function of major organ systems. Dosages are determined to maximize cell kill while minimizing impact on healthy tissues and subsequent toxicities. The therapeutic effect may be compromised if inadequate dosing is required due to toxicities. Modification of dosage is often required if critical laboratory values or the patient's symptoms indicate unacceptable or dangerous toxicities. Various laboratory tests are performed prior to, during, and after chemotherapy to determine optimal treatment options, evaluate the patient's response, and monitor toxicity. Laboratory and physical assessments of the hematologic, hepatic, renal, cardiovascular, and pulmonary systems are critical in evaluating the response to chemotherapy (Duong & Loh, 2006; Nirenburg, Bush, Davis, et al., 2006).

Chemotherapy treatment regimens include standard dosage therapy, dose-dense regimens, and myeloablative regimens with bone marrow or peripheral stem cell transplant. For certain chemotherapeutic agents, there is a maximum lifetime dose limit that must be adhered to because of the danger of long-term irreversible organ complications (eg, because of the risk of cardiomyopathy, doxorubicin [Adriamycin] has a cumulative lifetime dose limit of 550 mg/m^2).

Extravasation

Antineoplastic chemotherapeutic agents are additionally classified by their potential to damage soft tissue if they inadvertently leak from a vein (**extravasation**). The consequences of extravasation range from mild discomfort to severe tissue destruction, depending on whether the agent is classified as a nonvesicant, irritant, or vesicant. Irritant agents induce inflammatory reactions but usually cause no permanent tissue damage. **Vesicants** are those agents that, if deposited into the subcutaneous tissue (extravasation), cause tissue necrosis and damage to underlying tendons, nerves, and blood vessels. Although the complete mechanism of tissue destruction is unclear, it is known that the pH of many antineoplastic agents is responsible for the severe inflammatory reaction as well as the ability of these agents to bind to tissue DNA. Sloughing and ulceration of the tissue progresses to tissue necrosis and may be so severe that skin grafting may be necessary. The full extent of tissue damage may take several weeks to become apparent. Medications classified as vesicants include many of the commonly used agents: cisplatin (Platinol-AQ), dactinomycin (Cosmegen), daunorubicin (DaunoXome), doxorubicin, nitrogen mustard (Mustargen), mitomycin (Mutamycin), paclitaxel (Taxol), vinblastine (Velban), vincristine (Oncovin), vindesine (Eldisine), and vinorelbine (Navelbine) (Sauerland, Engelking, Wickham, et al., 2006).

Only specially trained physicians and nurses should administer vesicants. Careful selection of peripheral veins, skilled venipuncture, and careful administration of medications are essential. Indications of extravasation during administration of vesicant agents include the following:

- Absence of blood return from the intravenous (IV) catheter
- Resistance to flow of IV fluid
- Burning or pain, swelling or redness at the site

Table 16-6 ANTINEOPLASTIC AGENTS

Drug Class and Examples	Mechanism of Action	Cell Cycle Specificity	Common Side Effects
Alkylating Agents			
Busulfan (Busulfex, Myleran), carboplatin (Paraplatin), chlorambucil (Leukeran), cisplatin (Platinol-AQ), cyclophosphamide (Cytoxan), dacarbazine (DTIC-Dome), hexamethylene amine or altretamine (Hexalen), ifosfamide (Ifex), melphalan (Alkeran), nitrogen mustard (Mustargen), oxaliplatin (Eloxatin), thiotepa (Thioplex)	Alter DNA structure by misreading DNA code, initiating breaks in the DNA molecule, cross-linking DNA strands	Cell cycle–nonspecific	Bone marrow suppression, nausea, vomiting, cystitis (cyclophosphamide, ifosfamide), stomatitis, alopecia, gonadal suppression, renal toxicity (cisplatin)
Nitrosoureas Carmustine (BCNU [BiCNU, Gliadel]), lomustine or CCNU (CeeNU), semustine (methyl CCNU [MeCCNU]), streptozocin (Zanosar)	Similar to the alkylating agents; cross the blood–brain barrier	Cell cycle–nonspecific	Delayed and cumulative myelosuppression, especially thrombocytopenia; nausea, vomiting
Topoisomerase I Inhibitors Irinotecan (Camptosar) Topotecan (Hycamtin)	Induce breaks in the DNA strand by binding to enzyme topoisomerase I, preventing cells from dividing	Cell cycle–specific (S phase)	Bone marrow suppression, diarrhea, nausea, vomiting, hepatotoxicity
Antimetabolites 5-Azacytadine, capecitabine (Xeloda), cytarabine (DepoCyt, Tarabine) edatrexate fludarabine (Fludara), 5-fluorouracil (5-FU), gemcitabine (Gemzar), hydroxyurea (Droxia, Hydrea), cladribine (Leustatin), 6-mercaptopurine (Purinethol), methotrexate (Trexall, Rheumatrex), pentostatin (Nipent), 6-thioguanine (Tabloid)	Interferes with the biosynthesis of metabolites or nucleic acids necessary for RNA and DNA synthesis	Cell cycle–specific (S phase)	Nausea, vomiting, diarrhea, bone marrow suppression, proctitis, stomatitis, renal toxicity (methotrexate), hepatotoxicity
Antitumor Antibiotics Bleomycin (BLM, Blenoxane), dactinomycin (Cosmegen), daunorubicin (DaunoXome), doxorubicin (Adriamycin), idarubicin (Idamycin), mitomycin (Mutamycin), mitoxantrone (Novantrone), plicamycin (Mithracin)	Interfere with DNA synthesis by binding DNA; prevent RNA synthesis	Cell cycle–nonspecific	Bone marrow suppression, nausea, vomiting, alopecia, anorexia, cardiac toxicity (daunorubicin, doxorubicin)
Mitotic Spindle Poisons *Plant alkaloids:* etoposide (Toposar), teniposide (Vumon) vinblastine (Velban), vincristine (VCR [Oncovin]), vindesine (Eldisine), vinorelbine (Navelbine)	Arrest metaphase by inhibiting mitotic tubular formation (spindle); inhibit DNA and protein synthesis	Cell cycle–specific (M phase)	Bone marrow suppression (mild with VCR), neuropathies (VCR), stomatitis
Taxanes: paclitaxel (Taxol), docetaxel (Taxotere)	Arrest metaphase by inhibiting tubulin depolymerization	Cell cycle–specific (M phase)	Bradycardia, hypersensitivity reactions, bone marrow suppression, alopecia, neuropathies
Hormonal Agents Androgens and antiandrogens, estrogens and antiestrogens, progestins and antiprogestins, aromatase inhibitors, luteinizing hormone–releasing hormone analogues, steroids	Bind to hormone receptor sites that alter cellular growth; block binding of estrogens to receptor sites (antiestrogens); inhibit RNA synthesis; suppress aromatase of P450 system, which decreases level	Cell cycle–nonspecific	Hypercalcemia, jaundice, increased appetite, masculinization, feminization, sodium and fluid retention, nausea, vomiting, hot flashes, vaginal estrogen dryness
Miscellaneous Agents Asparaginase (Elspar), procarbazine (Matulane)	Unknown or too complex to categorize	Varies	Anorexia, nausea, vomiting, bone marrow suppression, hepatotoxicity, anaphylaxis, hypotension, altered glucose metabolism

CHART 16-4 HOME CARE CHECKLIST

Chemotherapy Administration

At the completion of the home care instruction, the patient or caregiver will be able to:	PATIENT	CAREGIVER
• Demonstrate how to administer the chemotherapy agent in the home.	✔	✔
• Demonstrate safe disposal of needles, syringes, IV supplies, or unused chemotherapy medications.	✔	✔
• List possible side effects of chemotherapeutic agents.	✔	✔
• List complications of medications necessitating a call to the nurse or physician.	✔	✔
• List complications of medications necessitating a visit to the emergency department.	✔	✔
• List names and telephone numbers of resource personnel involved in care (ie, home care nurse, infusion services, IV vendor, equipment company).	✔	✔
• Explain treatment plan (protocol) and importance of upcoming visits to physician.	✔	✔

NURSING ALERT

If extravasation is suspected, the medication administration is stopped immediately, and dependent on the drug, an attempt is made to aspirate any remaining drug from the extravasation site through the existing needle.

Institutional nursing policies should be available to identify nursing intervention and an extravasation kit should be readily available with all of the emergency equipment and antidote medication, as well as a quick reference for how to properly manage an extravasation of the specific vesicant agent used. Application of heat or cold is very dependent on the drug administered, and nurses should refer to their hospital policy. In general, cold compresses are indicated for doxorubicin extravasation but are of no benefit for taxane or oxaliplatin (Eloxatin) extravasation. Warm compresses are recommended for vinca alkaloid extravasation. Depending on the guidelines for specific agents, extravasation management may include aspiration of any infiltrated medication from the tissues and injection of a neutralizing solution into the area to reduce tissue damage. Selection of the neutralizing solution depends on the extravasated agent. Recent research has suggested that dexrazoxane (Totect) IV infusion for 3 days has benefit in treatment of anthracycline (ie, doxorubicin) extravasation with prevention of tissue necrosis (Schulmeister, 2007). Application of topical ointments, such as dexamethasone (Decadron) ointment, has been reported with variable levels of effectiveness. Recommendations and guidelines for managing vesicant extravasation have been issued by individual medication manufacturers, pharmacies, and the Oncology Nursing Society, and they differ from one medication to the next (Gullatte, 2007; Sauerland, et al., 2006).

Prevention of extravasation is essential and relies on vigilant nursing care. Vesicant chemotherapy should never be administered in peripheral veins involving the hand or wrist. Peripheral administration is permitted for short duration infusions only, and placement of the venipuncture site should be on the forearm area using a soft, plastic catheter. For any frequent, or prolonged administration of antineoplastic vesicants, right atrial silastic catheters, implanted venous access devices, or peripherally inserted central catheters (PICC) should be inserted to promote safety during medication administration and reduce problems with access to the circulatory system (Figs. 16-3 and 16-4). Indwelling or subcutaneous catheters require vigilant nursing care. Complications associated with their use include infection and thrombosis (Arch, 2007).

Hypersensitivity Reactions

Most of the available chemotherapeutic agents have the potential to cause hypersensitivity reactions; however, the overall incidence of hypersensitivity reactions to these agents is only about 5%. Understanding and managing hypersensitivity reactions is critical when caring for patients receiving chemotherapy because these reactions are potentially

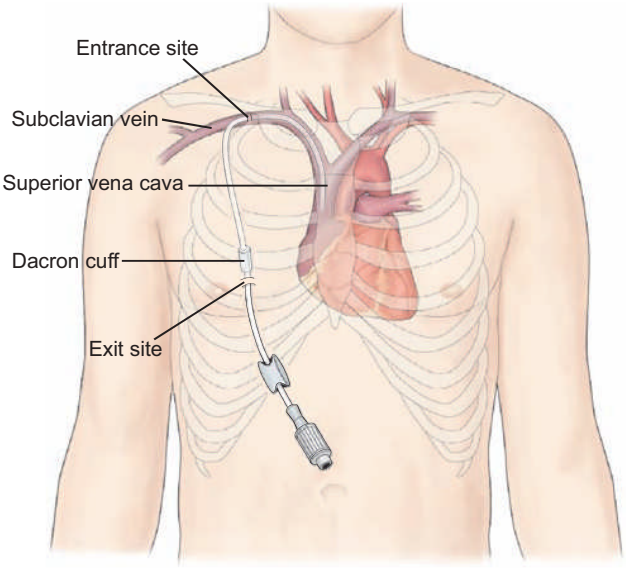

Figure 16-3 Right atrial catheter. The right atrial catheter is inserted into the subclavian vein and advanced until its tip lies in the superior vena cava just above the right atrium. The proximal end is then tunneled from the entry site through the subcutaneous tissue of the chest wall and brought out through an exit site on the chest. The Dacron cuff anchors the catheter in place and serves as a barrier to infection.

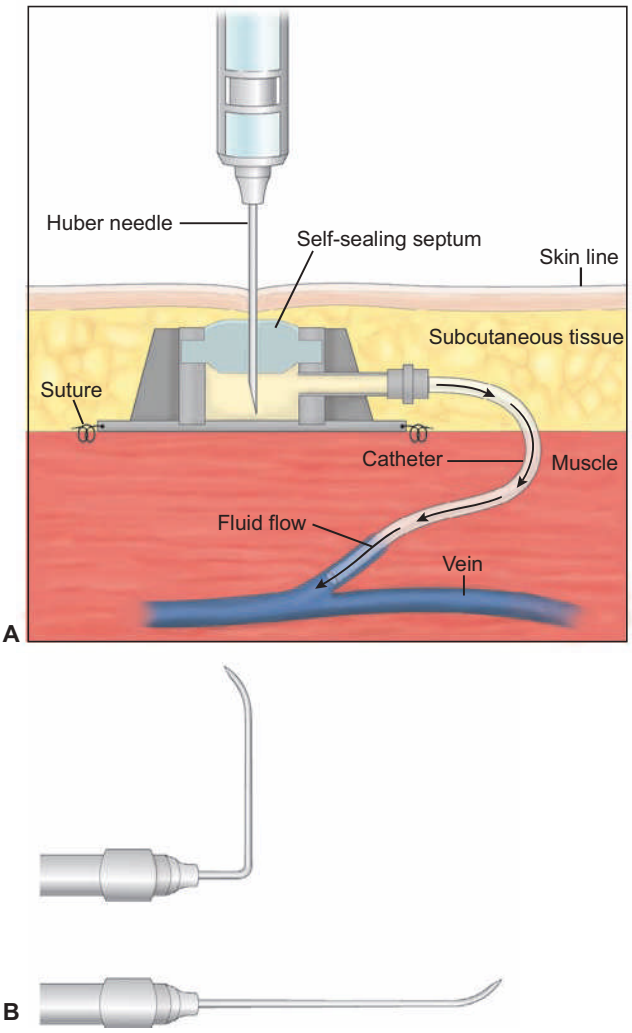

Figure 16-4 Implanted vascular access device. (**A**) A schematic diagram of an implanted vascular access device used for administration of medications, fluids, blood products, and nutrition. The self-sealing septum permits repeated puncture by Huber needles without damage or leakage. (**B**) Two Huber needles used to enter the implanted vascular port. The 90-degree needle is used for top-entry ports for continuous infusions.

life-threatening. Prevention is the first line of defense, and nurses need to have a clear understanding of which agents have the potential for precipitating hypersensitivity reactions, determining the patient's responses to certain agents via skin testing, and providing appropriate premedication before administering agents with a high potential for causing hypersensitivity reactions. Education of patients should emphasize the importance of adhering to prescribed self-administered premedication before presenting to the infusion center and recognizing and reporting the signs and symptoms to the nurse once their infusion has started. Early intervention can prevent progression of a reaction to systemic anaphylaxis. Most reactions coincide with chemotherapy agent administration, but some reactions can be delayed or occur after several uneventful courses of therapy. Although patients may react to the first infusion of a chemotherapy agent, repeated exposure increases the likelihood of a reaction along with other predisposing risk factors such as preexisting allergic reactions to food, blood products, and other medications. Emergency medication and resuscitation equipment should be easily accessible.

The usual chemotherapy hypersensitivity reaction is categorized as a type I immediate, immunoglobulin E mediated reaction. Type I hypersensitivity reactions may present as a local reaction and then rapidly progress to systemic anaphylaxis, or the initial presentation may be an acute life-threatening anaphylaxis. Symptoms include generalized itching with localized or generalized urticaria; flushing of the face, hands, or feet; chest tightness; agitation; nausea and vomiting; dyspnea and bronchospasm; difficulty speaking; feeling of impending doom; and hypotension (Gobel, 2005; Wilkes & Barton-Burke, 2007). The medication should be discontinued immediately and emergency procedures initiated. Many institutions have developed specific protocols for responding to hypersensitivity reactions including standing orders for administration of emergency medications (de Lemos, 2006). Chapter 53 presents further discussion of allergic reaction.

For some chemotherapeutic agents, especially if they are essential in the treatment plan, desensitization procedures may be possible, and the patient is retreated with the agent at reduced dosages or slower infusion rates. Premedication regimens including corticosteroids, histamine-1 and histamine-2 antagonists, and antipyretics are routinely preadministered for certain chemotherapy agents to prevent or minimize potential reactions.

Doxorubicin or daunorubicin can create localized allergic reactions referred to as flare reaction. Patients typically experience a hot, flushed sensation with urticaria and pruritis. The nurse must confirm that the reaction is indeed a flare and not an extravasation. The infusion can be temporarily discontinued and restarted at a slower infusion rate after consultation with the physician and IV administration of hydrocortisone (Solu-Cortef Hydrocortone).

Toxicity

Toxicity associated with chemotherapy can be acute or chronic. Cells with rapid growth rates (eg, epithelium, bone marrow, hair follicles, sperm) are very susceptible to damage, and various body systems may be affected as well.

Gastrointestinal System. Nausea and vomiting are the most common side effects of chemotherapy and may persist for as long as 24 to 48 hours after its administration. Delayed nausea and vomiting may persist for as long as 1 week after chemotherapy.

A number of mechanisms are responsible for the occurrence of nausea and vomiting, including activation of receptors found in the chemoreceptor trigger zone (CTZ) of the medulla, stimulation of the peripheral autonomic and vestibular pathways, cognitive stimulation, or a combination of factors. Medications that can decrease nausea and vomiting include serotonin blockers, such as ondansetron (Zofran), granisetron (Kytril), dolasetron (Anzemet), and palonosetron (Aloxi), which block serotonin receptors of the gastrointestinal tract and CTZ, and dopaminergic blockers, such as metoclopramide (Reglan), which block dopamine receptors of the CTZ. Newer agents include

neurokinin 1 receptor antagonists (eg, aprepitant [Emend]), which block the activity of substance P, a potent neurotransmitter involved in stimulating nausea and vomiting (Jordan, Sippel & Schmoll, 2007).

Nausea and vomiting involve multiple pathways; therefore, corticosteroids, phenothiazines, sedatives, and histamines are helpful, especially when used in combination with serotonin blockers to provide improved antiemetic protection. Delayed nausea and vomiting that occur longer than 48 to 72 hours after chemotherapy are troublesome for some patients. To minimize discomfort, some antiemetic medications are necessary for the first week at home after chemotherapy. Nonpharmacologic approaches such as relaxation techniques, imagery, and acupressure (Dribble, Luce, Cooper, et al., 2007) can also help decrease stimuli contributing to symptoms. Small frequent meals, bland foods, and comfort foods may reduce the frequency or severity of these symptoms.

The epithelium that lines the oral cavity is susceptible to the effects of chemotherapy; as a result, stomatitis is common. The entire gastrointestinal tract is susceptible to mucositis (inflammation of the mucosal lining), and diarrhea is a common result. Antimetabolites and antitumor antibiotics are the major culprits in mucositis and other gastrointestinal symptoms, including diarrhea, which can be severe in some patients.

Hematopoietic System. Most chemotherapeutic agents cause **myelosuppression** (depression of bone marrow function), resulting in decreased production of WBCs (leukopenia), granulocytes (neutropenia), red blood cells (RBCs) (anemia), and platelets (thrombocytopenia) and increased risk of infection and bleeding. Depression of these cells is the usual reason for limiting the dose of the chemotherapeutic agents. Myelosuppression is predictable, and patients usually reach their nadir counts (point at which blood counts are lowest) 7 to 14 days after chemotherapy has been administered. At this time nurses anticipate associated toxicities, especially febrile neutropenia (fever associated with neutrophil count less than 1500 cells/mm^3). Frequent monitoring of blood cell counts is essential and strategies are implemented to protect patients from infection and injury, particularly while blood cell counts are depressed (Duong & Loh, 2006; Nirenberg, et al., 2006).

Other agents, called colony-stimulating factors (granulocyte colony-stimulating factor [G-CSF] and granulocyte-macrophage colony-stimulating factor [GM-CSF]), can be administered after chemotherapy to stimulate the bone marrow to produce WBCs, especially neutrophils, at an accelerated rate, thus decreasing the duration of neutropenia. G-CSF and GM-CSF decrease the episodes of infection and the need for antibiotics and allow for more timely cycling of chemotherapy with less need to reduce the dosage. Erythropoietin (EPO) stimulates RBC production, thus decreasing the symptoms of chronic anemia and reducing the need for blood transfusions. Interleukin 11 (IL-11) stimulates the production of platelets and can be used to prevent and treat thrombocytopenia (platelet count less than 100,000) but has had limited use because of toxicities such as fatigue, edema, dysrhythmias, and syncope (Burcat & McAdams, 2007; Hurter & Bush, 2007; Nirenberg, et al., 2006).

Renal System. Chemotherapeutic agents can damage the kidneys because of their direct effects during excretion and the accumulation of end products after cell lysis. Cisplatin, methotrexate (Trexall, Rheumatrex), and mitomycin are particularly toxic to the kidneys. Rapid tumor cell lysis after chemotherapy results in increased urinary excretion of uric acid, which can cause renal damage. In addition, intracellular contents are released into the circulation, resulting in hyperkalemia, hyperphosphatemia, and hypocalcemia. (See later discussion of tumor lysis syndrome.)

Monitoring blood urea nitrogen (BUN), serum creatinine, creatinine clearance, and serum electrolyte levels is essential. Adequate hydration, diuresis, alkalinization of the urine to prevent formation of uric acid crystals, and allopurinol may be used to prevent these side effects (Duong & Loh, 2006; Gullatte, 2006). Amifostine has demonstrated an ability to minimize renal toxicities associated with cisplatin, cyclophosphamide (Cytoxan), and ifosfamide (Ifex) therapy (Hogle, 2007).

Hemorrhagic cystitis is a bladder toxicity resulting from cyclophosphamide and ifosfamide therapy. Hematuria can range from microscopic to frank bleeding with symptoms ranging from transient irritative urination, dysuria, suprapubic pain, to life-threatening hemorrhage. Protection of the bladder focuses on aggressive IV hydration, frequent voiding, and diuresis. Mesna (Mesnex) is a cytoprotectant agent that binds with the toxic metabolites of cyclophosphamide or ifosfamide in the kidneys to prevent hemorrhagic cystitis (Hogle, 2007; Wilkes & Barton-Burke, 2007).

Cardiopulmonary System. Anthracyclines (daunorubicin and doxorubicin) are known to cause irreversible cumulative cardiac toxicities, especially when total dosage reaches 600 mg/m^2 and 550 mg/m^2, respectively. If these agents are administered in the presence of thoracic radiation therapy or other agents with cardiotoxicity potential, their cumulative dose limit is reduced to 450 mg/m^2. Dexrazoxane (Zinecard) has been utilized as a cardioprotectant when doxorubicin is needed in individuals who have already received a cumulative dose of 300 mg/m^2 and continuation of therapy is deemed beneficial (Wilkes & Barton-Burke, 2007; Hogle, 2007). Cardiac ejection fraction (volume of blood ejected from the heart with each beat) and signs of heart failure must be monitored closely.

Bleomycin (Blenoxane), carmustine (BCNU), and busulfan (Busulfex, Myleran) have cumulative toxic effects on lung function, resulting in pulmonary fibrosis. Therefore, patients are monitored closely for changes in pulmonary function, including pulmonary function test results. Total cumulative doses of bleomycin should not exceed 400 U, and carmustine should not exceed 1400 mg.

Capillary leak syndrome with resultant pulmonary edema is a toxic effect of cytarabine (DepoCyt, Tarabine) (AraC), mitomycin C, cyclophosphamide, and BCNU. Subtle onset of dyspnea and cough may progress rapidly to acute respiratory distress and subsequent respiratory failure (Wilkes & Barton-Burke, 2007).

Reproductive System. Testicular and ovarian function can be affected by chemotherapeutic agents, resulting in possible sterility. Normal ovulation, early menopause, or permanent sterility may occur. In men, temporary or permanent

azoospermia (absence of spermatozoa) may develop. Because treatment may damage reproductive cells, banking of sperm is often recommended for men before treatment is initiated. Patients and their partners need to be informed about potential changes in reproductive function resulting from chemotherapy. They are advised to use reliable methods of birth control while receiving chemotherapy and not to assume that sterility has resulted.

Neurologic System. Chemotherapy-induced neurotoxicity can affect the CNS, peripheral nervous system (PNS), the cranial nerves or a combination; it is a dose-limiting toxicity. The blood–brain barrier can protect the CNS and PNS from the toxic effects of most water soluble chemotherapy agents, but neurotoxicity characterized by metabolic encephalopathy can occur with ifosfamide, high-dose methotrexate, and cytarabine. With repeated doses, the taxanes and plant alkaloids, especially vincristine, can cause peripheral neurologic damage with sensory alterations in the feet and hands. These sensations can be described as tingling, pricking, or numbness of the extremities, burning or freezing pain, sharp, stabbing, or electric shock–like pain and extreme sensitivity to touch. If unreported by patients or undetected, progressive motor axon damage can lead to loss of deep tendon reflexes, with muscle weakness, loss of balance and coordination, and paralytic ileus. Although usually reversible, these side effects may take many months to resolve. Along with the usual paresthesias of the hands and feet, oxaliplatin has a unique and frightening neurotoxicity presentation that is often precipitated by exposure to cold and is characterized by pharyngolaryngeal dysesthesia consisting of lip paresthesia, discomfort or tightness in the back of the throat, inability to breathe, and jaw pain. Patients receiving oxaliplatin must be instructed to avoid drinking cold fluids or going outside with hands and feet exposed to cold temperatures to avoid exacerbation of these symptoms. Cisplatin may cause peripheral neuropathies and hearing loss due to damage to the acoustic nerve (Wilkes & Barton-Burke, 2007). The ability of cytoprotectant agents to prevent these significant neurotoxicities, including amifostine, is being studied (Hogle, 2006; Wilkes & Barton-Burke, 2007).

Fatigue. Fatigue, a distressing side effect for most patients that greatly affects quality of life, can last for months after treatment. Assessment and nursing management of fatigue are discussed in the Nursing Care of Patients With Cancer section of this chapter. In 2006, the Oncology Nursing Society conducted an exhaustive review of evidence-based interventions for fatigue management to provide guidelines for nurses to effectively intervene and assist their patients (Mitchell, Beck, Hood, et al., 2007).

Nursing Management in Chemotherapy

Nurses play an important role in assessing and managing many of the problems experienced by patients undergoing chemotherapy. Chemotherapeutic agents affect both normal and malignant cells, meaning that these problems are often widespread, affecting many body systems.

Assessing Fluid and Electrolyte Status

Anorexia, nausea, vomiting, altered taste, mucositis, and diarrhea put patients at risk for nutritional and fluid and electrolyte disturbances. Therefore, it is important for the nurse to assess the patient's nutritional and fluid and electrolyte status frequently and to use creative ways to encourage an adequate fluid and dietary intake.

Modifying Risks for Infection and Bleeding

Suppression of the bone marrow and immune system is expected and frequently serves as a guide in determining appropriate chemotherapy dosage but increases the risk of anemia, infection, and bleeding disorders. Nursing assessment and care address factors that would further increase the patient's risk. The nurse's role in decreasing the risk for infection and bleeding is discussed further in the Nursing Care of Patients With Cancer (see p. 366).

Administering Chemotherapy

The local effects of the chemotherapeutic agent are also of concern. The patient is observed closely during its administration because of the risk and consequences of extravasation, particularly of vesicant agent. Local difficulties or problems with administration of chemotherapeutic agents are brought to the attention of the physician promptly so that corrective measures can be taken immediately to minimize local tissue damage. (See previous discussion of extravasation.)

Protecting Caregivers

Nurses involved in handling chemotherapeutic agents may be exposed to low doses of the agents by direct contact, inhalation, or ingestion. Urinalyses of personnel repeatedly exposed to cytotoxic agents have demonstrated mutagenic activity. Although long-term studies of nurses who handle chemotherapeutic agents have not been conducted, it is known that chemotherapeutic agents are associated with secondary formation of cancers and chromosome abnormalities. In addition, nausea, vomiting, dizziness, alopecia, and nasal mucosal ulcerations have occurred in health care personnel who have handled chemotherapeutic agents. The Occupational Safety and Health Administration, Oncology Nursing Society, hospitals, and other health care agencies have developed specific precautions for health care providers involved in the preparation and administration of chemotherapy (Chart 16-5) (Polovich, et al., 2005; Wilkes & Barton-Burke, 2007). Nurses must be familiar with their institutional policies regarding personal protective equipment, handling and disposal of chemotherapeutic agents and supplies, and management of accidental spills or exposures. Emergency spill kits should be readily available in any treatment area where chemotherapy is prepared or administered. Precautions must also be taken when handling any bodily fluids or excreta from the patient, as many agents are excreted unaltered in urine and feces. Nurses have a responsibility to educate patients, caregivers, assistive personnel, and housekeepers concerning these precautions.

Bone Marrow Transplantation

Although surgery, radiation therapy, and chemotherapy have improved survival rates for patients with cancer, many cancers that initially respond to therapy recur. This is true of hematologic cancers that affect the bone marrow and solid tumor cancers treated with lower doses of antineoplastics to

Chart 16-5 • *Safety in Administering Chemotherapy*

Safety recommendations from the Occupational Safety and Health Administration (OSHA), Oncology Nursing Society (ONS), hospitals, and other health care agencies for the preparation and handling of antineoplastic agents follow:

- Use a biologic safety cabinet for the preparation of all chemotherapy agents.
- Wear surgical gloves when handling antineoplastic agents and the excretions of patients who received chemotherapy.
- Wear disposable, long-sleeved gowns when preparing and administering chemotherapy agents.
- Use Luer-Lok fittings on all intravenous tubing used to deliver chemotherapy.
- Dispose of all equipment used in chemotherapy preparation and administration in appropriate, leak-proof, puncture-proof containers.
- Dispose of all chemotherapy wastes as hazardous materials.

When followed, these precautions greatly minimize the risk of exposure to chemotherapy agents.

spare the bone marrow from larger, ablative doses of chemotherapy or radiation therapy. The role of bone marrow transplantation (BMT) for malignant and some nonmalignant diseases continues to grow.

The process of obtaining donor cells has evolved over the years. Donor cells can be obtained by the traditional harvesting of large amounts of bone marrow tissue under general anesthesia in the operating room. However, a second method, referred to as peripheral blood stem cell transplantation (PBSCT), has gained widespread use. This method of collection uses apheresis of the donor to collect peripheral blood stem cells (PBSCs) for reinfusion. It is a safe and cost-effective means of collection rather than the traditional harvesting of marrow, which requires general anesthesia and an operative procedure.

Types of Bone Marrow Transplant

Types of BMT based on the source of donor cells include:

- Allogeneic: from a donor other than the patient; donor may be a related donor (ie, family member) or a matched unrelated donor (national bone marrow registry, cord blood registry)
- Autologous: from the patient
- Syngeneic: from an identical twin

Allogeneic BMT (AlloBMT), used primarily for disease of the bone marrow, depends on the availability of a human leukocyte antigen–matched donor. This greatly limits the number of possible transplants. An advantage of AlloBMT is that the transplanted cells should not be immunologically tolerant of a patient's malignancy and should cause a lethal **graft-versus-tumor effect,** in which the donor cells recognize the malignant cells and act to eliminate them.

AlloBMT may involve either ablative (high-dose) or nonablative (mini-dose) chemotherapy. In ablative AlloBMT, the recipient must undergo ablative doses of chemotherapy and possibly total body irradiation to destroy all existing bone marrow and malignant disease. The harvested donor marrow or PBSCs are infused intravenously into the recipients, and they travel to sites in the body where they produce bone marrow and establish themselves. Once engraftment is complete (2 to 4 weeks, sometimes longer), the new bone marrow becomes functional and begins producing RBCs, WBCs, and platelets. In nonablative AlloBMT, the chemotherapy doses are lower and are aimed at suppressing the recipient's immune system to allow engraftment of donor bone marrow or PBSCs. The lower doses of chemotherapy create less organ toxicity and thus can be offered to older patients or those with underlying organ dysfunction for whom high-dose chemotherapy would be prohibitive. After engraftment, it is hoped that the donor cells will create a graft-versus-tumor effect (Rodriguez, Tariman, Enecio, et al., 2007; Saria & Gosselin-Acomb, 2007). Before engraftment, patients are at high risk for infection, sepsis, and bleeding. Side effects of the high-dose chemotherapy and total body irradiation can be acute and chronic. Acute side effects include alopecia, hemorrhagic cystitis, nausea, vomiting, diarrhea, and severe stomatitis. Chronic side effects include sterility, pulmonary dysfunction, cardiac dysfunction, and liver disease.

To prevent **graft-versus-host disease (GVHD),** patients receive immunosuppressant drugs, such as cyclosporine (Sandimmune), methotrexate, tacrolimus (Prograf), or sirolimus (Rapamune). In allogeneic transplant recipients, GVHD occurs when the T lymphocytes from the transplanted donor marrow or PBSCs become activated and mount an immune response against the recipient's tissues (skin, gastrointestinal tract, liver). T lymphocytes respond in this manner because they view the recipient's tissue as "foreign," immunologically different from what they recognize as "self" in the donor. GVHD may occur acutely or chronically. Clinical manifestations of acute GVHD include diffuse rash progressing to blistering and desquamation similar to second-degree burns; mucosal shedding with subsequent diarrhea that may exceed 2 L per day; and biliary stasis with abdominal pain, hepatomegaly, and elevated liver enzymes progressing to obstructive jaundice. GVHD accounts for approximately 10% of all BMT deaths (Saria & Gosselin-Acomb, 2007).

The first 100 days or so after AlloBMT are crucial for patients; the immune system and blood-making capacity (hematopoiesis) must recover sufficiently to prevent infection and hemorrhage. Most acute side effects, such as nausea, vomiting, and mucositis, also resolve in the initial 100 days after transplantation. Patients are also at risk for venous occlusive disease (VOD), a vascular injury to the liver caused by high-dose chemotherapy, leading to hepatic outflow obstruction and portal hypertension, in the first 30 days or so after BMT, acute liver failure, and death (Saria & Gosselin-Acomb, 2007).

Autologous BMT (AuBMT) is considered for patients with disease of the bone marrow who do not have a suitable donor for AlloBMT and for patients who have healthy bone marrow but require bone marrow–ablative doses of chemotherapy to cure an aggressive malignancy. Conditions include non-Hodgkin and Hodgkin lymphoma, multiple myeloma, neuroblastoma, sarcoma, and germ cell tumors. Stem cells are collected from the patient and preserved for reinfusion; if necessary, they are treated to kill any malignant

cells within the marrow, called purging. The patient is then treated with ablative chemotherapy and, possibly, total body irradiation to eradicate any remaining tumor. Stem cells are then reinfused and engrafted. Until engraftment occurs in the bone marrow sites of the body, there is a high risk of infection, sepsis, and bleeding. Acute and chronic toxicities from chemotherapy and radiation therapy may be severe. The risk of VOD is also present after autologous transplantation. No immunosuppressant medications are necessary after AuBMT because the patient does not receive foreign tissue. A disadvantage of AuBMT is the risk that tumor cells may remain in the bone marrow despite high-dose chemotherapy (conditioning regimens).

Syngeneic transplants result in less incidence of GVHD and graft rejection; however, there is also less graft-versus-tumor effect to fight the malignancy. For this reason, even when an identical twin is available for marrow donation, another matched sibling or even an unrelated donor may be the most suitable donor to combat an aggressive malignancy.

Nursing Management in Bone Marrow Transplantation

Nursing care of patients undergoing BMT is complex and demands a high level of skill. Transplantation nursing can be extremely rewarding yet extremely stressful. The success of BMT is greatly influenced by nursing care throughout the transplantation process.

Implementing Pretransplantation Care

All patients must undergo extensive pretransplantation evaluations to assess the current clinical status of the disease. Nutritional assessments, extensive physical examinations, organ function tests, and psychological evaluations are conducted. Blood work includes assessing past antigen exposure (eg, hepatitis virus, cytomegalovirus, herpes simplex virus, human immunodeficiency virus [HIV], and syphilis). The patient's social support systems and financial and insurance resources are also evaluated. Informed consent and patient teaching about the procedure and pretransplantation and posttransplantation care are vital.

Providing Care During Treatment

Skilled nursing care is required during the treatment phase of BMT when high-dose chemotherapy (conditioning regimen) and total body irradiation are administered. The acute toxicities of nausea, diarrhea, mucositis, and hemorrhagic cystitis require close monitoring and constant attention by the nurse.

Nursing management during bone marrow infusion or stem cell reinfusions consists of monitoring the patient's vital signs and blood oxygen saturation; assessing for adverse effects, such as fever, chills, shortness of breath, chest pain, cutaneous reactions, nausea, vomiting, hypotension or hypertension, tachycardia, anxiety, and taste changes; and providing ongoing support and patient teaching. During stem cell reinfusion, patients may experience adverse reactions to the cryoprotectant dimethyl sulfoxide (DMSO) used to preserve the harvested stem cells. These reactions may include nausea, vomiting, chills, dyspnea, cardiac dysrhythmias, and hypotension progressing to cardiac or respiratory arrest (Rodriquez, et al., 2007).

Until engraftment of the new marrow occurs, the patient is at high risk for death from sepsis and bleeding. A cluster of symptoms referred to as engraftment syndrome occurs during the neutrophil recovery phase in both allogeneic and autologous transplants. Clinical features of this syndrome vary widely but may include noninfectious fever associated with skin rash, weight gain, diarrhea, and pulmonary infiltrates, with improvement noted after the initiation of corticosteroid therapy rather than antibiotic therapy (Saria & Gosselin-Acomb, 2007). Until engraftment is well established, the patient requires support with blood products and hemopoietic growth factors.

Potential infections may be bacterial, viral, fungal, or protozoan in origin. During the first 30 days following transplant, the patient is most at risk for developing reactivations of viral infections including herpes simplex, Epstein-Barr, cytomegalovirus, and varicella zoster. Mucosal denudement poses a risk for *Candida* yeast infection locally and systemically. Pulmonary toxicities offer the opportunity for fungal infections such as *Aspergillus*. Renal complications arise from the nephrotoxic chemotherapy agents used in the conditioning regimen or those used to treat infection (amphotericin B, aminoglycosides). Tumor lysis syndrome and acute tubular necrosis are also risks after BMT. Nursing assessment for signs of these complications is essential for early identification and treatment (Burcat & McAdams, 2007; Rodriguez, et al., 2007; Saria & Gosselin-Acomb, 2007).

GVHD requires skillful nursing assessment to detect early effects on the skin, liver, and gastrointestinal tract. VOD resulting from the conditioning regimens used in BMT can result in fluid retention, jaundice, abdominal pain, ascites, tender and enlarged liver, and encephalopathy. Pulmonary complications, such as pulmonary edema, interstitial pneumonia, and other pneumonias, often complicate the recovery after BMT (Saria & Gosselin-Acomb, 2007).

Providing Posttransplantation Care

Caring for Recipients. Ongoing nursing assessment in follow-up visits is essential to detect late effects of therapy after BMT, which occur 100 days or more after the procedure. Late effects include infections (eg, varicella zoster infection), restrictive pulmonary abnormalities, and recurrent pneumonias. Sterility often results due to total body irradiation as part of the ablative regimen. Chronic GVHD involves the skin, liver, intestine, esophagus, eyes, lungs, joints, and vaginal mucosa. Cataracts may also develop after total body irradiation.

Psychosocial assessments by nursing staff must be ongoing. In addition to the stressors affecting patients at each phase of the transplantation experience, marrow donors and family members also have psychosocial needs that must be addressed.

Caring for Donors. Like BMT recipients, donors also require nursing care. They commonly experience mood alterations, decreased self-esteem, and guilt from feelings of failure if the transplantation fails. Family members must be educated and supported to reduce anxiety and promote coping during this difficult time. In addition, they must also be assisted to maintain realistic expectations of themselves as well as of the patient. As BMT becomes more prevalent, many ethical issues

become apparent, including those related to informed consent, allocation of resources, and quality of life.

Hyperthermia

Hyperthermia (thermal therapy), the generation of temperatures greater than physiologic fever range (greater than 41.5°C [106.7°F]), has been used for many years to destroy cancerous tumors. Malignant cells may be more sensitive than normal cells to the harmful effects of high temperatures for several reasons. Malignant cells lack the mechanisms necessary to repair damage caused by elevated temperatures. Most tumor cells lack an adequate blood supply to provide needed oxygen during periods of increased cellular demand, such as during hyperthermia. Cancerous tumors lack blood vessels of adequate size for dissipation of heat. In addition, the body's immune system may be indirectly stimulated when hyperthermia is used.

Hyperthermia is most effective when combined with radiation therapy, chemotherapy, or biologic therapy. Hyperthermia and radiation therapy are thought to work well together because hypoxic tumor cells and cells in the S phase of the cell cycle are more sensitive to heat than radiation; the addition of heat damages tumor cells so that they cannot repair themselves after radiation therapy. Hyperthermia is thought to alter cellular membrane permeability when used with chemotherapy, allowing for an increased uptake of the chemotherapeutic agent. Hyperthermia may enhance the function of immune system cells, such as macrophages and T cells (Milani & Noessner, 2006; van der Zee & van Rhoon, 2006).

Heat can be produced by using radiowaves, ultrasound, microwaves, magnetic waves, hot-water baths, or even hot-wax immersions. Hyperthermia may be local or regional, or it may include the whole body. Local or regional hyperthermia may be delivered to a cancerous extremity (for malignant melanoma) by regional perfusion, in which the affected extremity is isolated by a tourniquet and an extracorporeal circulator heats the blood flowing through the affected part. Hyperthermia probes may also be inserted around a tumor in a local area and attached to a heat source during treatment. Chemotherapeutic agents, such as melphalan (Alkeran), may also be heated and instilled into the region's circulating blood. Local or regional hyperthermia may also include infusion of heated solutions into cancerous body organs. Whole body hyperthermia to treat disseminated disease may be achieved by extracorporeal circulation, immersion of the patient in heated water or paraffin, or enclosure in a heated suit (Bruner, et al., 2006; van der Zee & van Rhoon, 2006).

Side effects of hyperthermia treatments include skin burns and tissue damage, fatigue, hypotension, peripheral neuropathies, thrombophlebitis, nausea, vomiting, diarrhea, and electrolyte imbalances. Resistance to hyperthermia may develop during the treatment because cells adapt to repeated thermal insult. Research into the effectiveness of hyperthermia is ongoing.

Nursing Management in Hyperthermia

Although hyperthermia has been used for many years, many patients and their families are unfamiliar with this cancer treatment. Consequently, they need explanations about the procedure, its goals, and its effects. The nurse assesses the patient for adverse effects and acts to reduce the occurrence and severity of adverse effects. Local skin care at the site of the implanted probes is necessary.

Targeted Therapies

Recent scientific advances have led to an improved understanding of cancer development. Traditional therapies such as chemotherapy and radiation affect all actively proliferating cells. As a result, both healthy cells and malignant cells are subject to harmful systemic effects of treatment. **Targeted therapies** seek to minimize the negative effects on healthy tissues by disrupting specific cancer cell functions such as malignant transformation, cell communication pathways (called signal transduction), processes for growth and metastasis, and genetic coding. Actions of targeted therapies include stimulation or augmentation of immune responses through the use of biologic response modifiers, targeting of cancer cell growth factors, promotion of apoptosis, and genetic manipulation through gene therapy (Khoukaz, 2006; Rieger, 2006). Most of the currently available targeted therapies are categorized as either monoclonal antibodies or small molecule tyrosine kinase inhibitors.

Biologic Response Modifiers

Biologic response modifier (BRM) therapy involves the use of naturally occurring or recombinant (reproduced through genetic engineering) agents or treatment methods that can alter the immunologic relationship between the tumor and the cancer patient (host) to provide a therapeutic benefit. Although the mechanisms of action vary with each type of BRM, the goal is to destroy or stop the malignant growth. The basis of BRM treatment lies in the restoration, modification, stimulation, or augmentation of the body's natural immune defenses against cancer (Yarbro, et al., 2005).

Nonspecific Biologic Response Modifiers

Some of the early investigations of the stimulation of the immune system involved nonspecific agents such as bacille Calmette-Guérin (BCG) and *Corynebacterium parvum*. When injected into the patient, these agents serve as antigens that stimulate an immune response. The hope is that the stimulated immune system will then eradicate malignant cells. Extensive animal and human investigations with BCG have shown promising results, especially in treating localized malignant melanoma. In addition, BCG bladder instillation (intravesicular) is a standard form of treatment for localized bladder cancer (Creel, 2007). However, use of nonspecific agents in advanced cancer remains limited, and research is ongoing to identify other uses and other agents.

Monoclonal Antibodies

Monoclonal antibodies (MoAbs), another type of BRM, have become available through technologic advances, enabling investigators to grow and produce targeted antibodies for specific malignant cells. Theoretically, this type of specificity allows MoAbs to destroy the cancer cells and spare normal cells. The specificity of MoAbs is dependent on identifying key antigen proteins on the surface of tumors that are not present on normal tissues. These targets when

blocked lead to apoptosis by disrupting communication between cells. There are several categories of these tumor-associated antigens: oncofetal antigens such as CEA, a prominent tumor marker identified in colon cancer; growth factors such as EGFs and VEGFs; and oncogenes such as *C-erb* or *Bcr-Abl* (Kay, 2006). MoAbs bind with specific tumor cell antigens and block the ability of the tumor cell to reproduce, or deliver cytotoxic agents directly to the tumor cell causing cell death.

The production of MoAbs involves injecting tumor cells that act as antigens into mice. B cells in the spleen of the mouse produce immunoglobulin antibodies made in response to the injected antigens. Antibody-producing B cells are combined with a cancer cell that has the ability to grow indefinitely in culture medium and continue producing more antibodies.

The combination of spleen cells and the cancer cells is referred to as a hybridoma. From hybridomas that continue to grow in the culture medium, the desired antibodies are harvested, purified, and prepared for diagnostic or therapeutic use (Fig. 16-5). Recent advances in genetic engineering have led to the production of MoAbs with combinations of mouse and human components (*chimeric MoAbs*) or all-human components (*humanized MoAbs*). MoAbs made with human genes have greater immunologic properties and are less likely to cause allergic reactions (Yarbro, et al., 2005).

MoAbs are being used as aids in diagnostic evaluation of both primary and metastatic tumors through radiologic techniques. For example, MoAbs are used to assist in diagnosing ovarian and colorectal cancers. Their use in detecting breast, gastric, and prostate cancers and lymphoma is under investigation. MoAbs are also used in purging residual tumor cells from the bone marrow or peripheral blood of patients who are undergoing BMT or peripheral stem cell rescue after high-dose cytotoxic therapy.

Several MoAbs have been approved for treatment in cancer using a variety of extracellular (outside the cell membrane) and intracellular (inside the cell membrane) targets. Some of the MoAbs are used alone, whereas others are used in combination with agents that facilitate their antitumor actions. For example, gemtuzumab ozogamicin (Mylotarg) is used for the treatment of a specific type of acute myeloid leukemia (Wilkes & Barton-Burke, 2007);

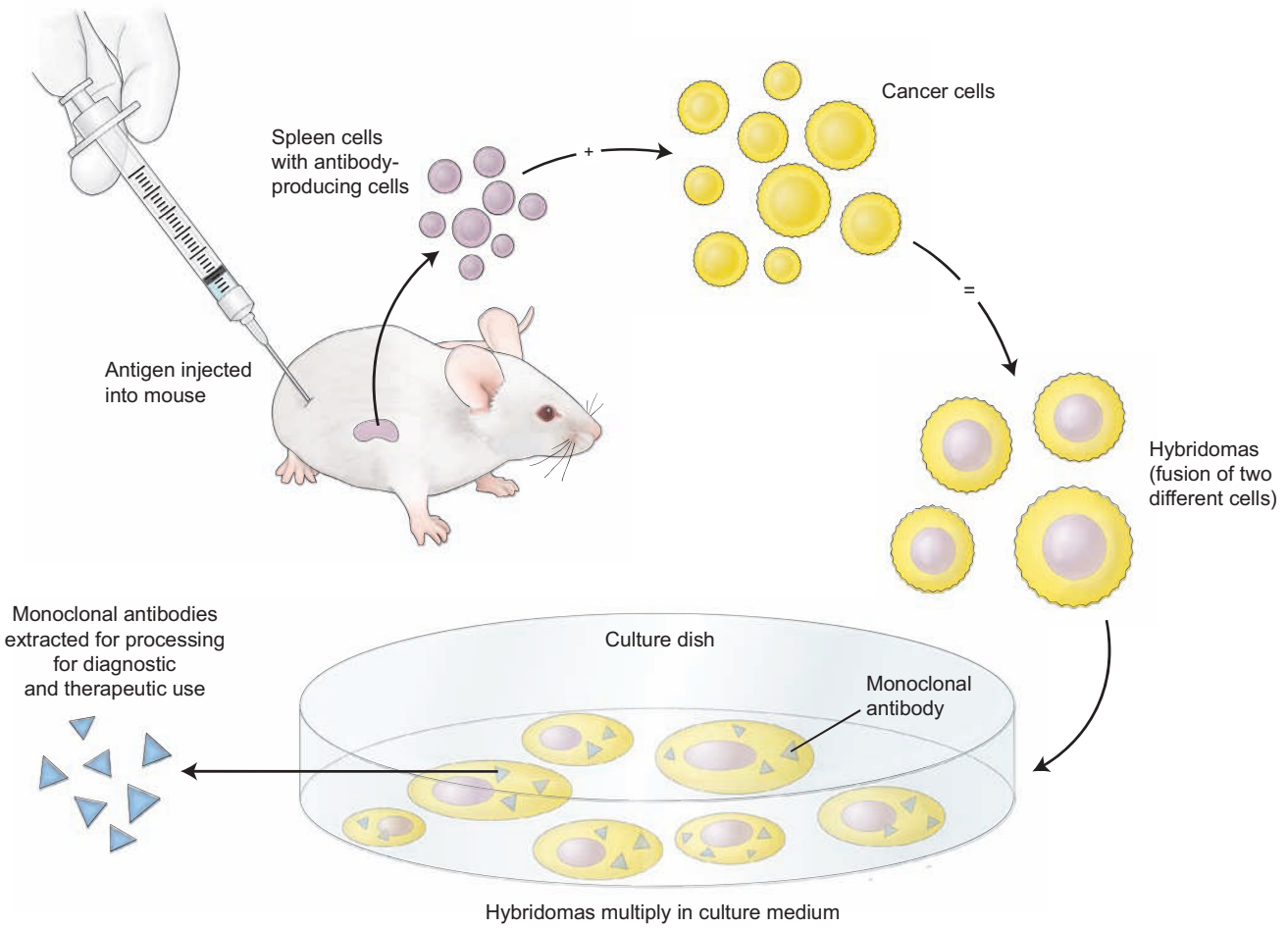

Figure 16-5 Antibody-producing spleen cells are fused with cancer cells. This process produces cells called hybridomas. These cells, which can grow indefinitely in a culture medium, produce antibodies that are harvested, purified, and prepared for diagnostic or treatment purposes.

ibritumomab-tiuxetan (Zevalin) and tositumomab (Bexxar) are used for the treatment of specific types of non-Hodgkin lymphoma. Some MoAbs target specific genetic mutations expressed by certain tumors, as in chronic myelogenous leukemia with the Philadelphia chromosome abnormality (Bcr-Abl). Imatinib mesylate (Gleevec) was developed to specifically bind with the Bcr-Abl abnormality, thus inhibiting cell proliferation.

Researchers continue to explore the development and use of MoAbs, either alone or in combination with other substances such as radioactive materials, chemotherapeutic agents, toxins, hormones, or other BRMs. Some specific targets for MoAbs under investigation include malignant cell growth factors, cell proteins, and substances that stimulate tumors to develop blood vessels (angiogenesis factors) (Kay, 2006; Wilkes & Barton-Burke, 2007).

Epidermal Growth Factor Receptors and Tyrosine Kinase Pathways. Normal cell growth is regulated by well-defined communication pathways between the environment surrounding the cell and the internal cell environment, the nucleus, and the intracellular cytoplasm. The cell membrane contains important protein receptors that respond to signals transmitted from the external environment and transmit that signal to the internal cell environment using enzymatic pathways called signal transduction pathways. Advances in understanding the genetic nature of cancers have resulted in these protein receptors and the cellular communication pathways to be used as targets for new cancer treatment agents. Much like a lock and key mechanism, new drugs are being developed that will target these specific receptors and pathways and prevent the continued growth of the cancer cells. The family of epidermal growth factor receptors (EGFR) have been proven to be a critical communication pathway. EGFRs are widely expressed by many normal cell types and in certain cancer tumors can be overexpressed or underexpressed (Franson & Lapka, 2005; Viele, 2005). The amount of EGFR that is expressed by a tumor can be measured by reliable laboratory testing. Overexpression of EGFR is associated with advanced tumor stage, more aggressive tumors, a predictor of resistance to standard chemotherapy, and a poor patient prognosis (Oishi, 2008). Recent scientific advances have enabled the development of numerous new targeted therapy drugs that bind to a specific protein receptor or block a specific signal transduction pathway expressed by a tumor but not by a normal cell, enabling a very targeted, specific cell kill. Monoclonal antibodies bind to the extracellular protein receptors and are larger molecules that are administered by IV. Tyrosine kinase inhibitors are smaller molecules that target the intracellular signaling pathways and are given orally. The efficacy of these new targeted agents depends on consistent and reliable delivery, and because they involve the patient's natural immune system they can precipitate very significant adverse events specific to each agent. It is important for nurses to be familiar with the administration issues related to patient education about self-administered oral agents and patient safety related to adverse events (Khoukaz, 2006).

Vascular Endothelial Growth Factors. Angiogenesis requires growth factors, cytokines, enzymes, and proteins, all generated by the tumor to stimulate the formation of new capillaries to deliver oxygen and other nutrients to the hypoxic tumor. The major pathway for angiogenesis is activation of the VEGF family of proteins (Franson & Lapka, 2005; Viele, 2005). VEGF is essential for the growth and proliferation of malignant cells and when activated stimulates growth of new blood vessels. These new blood vessels differ greatly from normal vessels with less well-organized structure, increased permeability allowing migration of tumor cells, and increased interstitial pressure preventing chemotherapy from reaching the tumor. VEGF is overexpressed in many solid tumors and is associated with advanced tumor stage and poor prognosis (Viele, 2005). In colorectal cancer, increased VEGF expression has been correlated with increased vascularity, invasiveness, metastasis, and poor prognosis (Franson & Lapka, 2005).

Bevacizumab (Avastin) is a MoAb directed toward VEGF to prevent the activation of endothelial cells and inhibit growth of new blood vessels. It is currently the only U.S. Food and Drug Administration (FDA)-approved angiogenesis inhibitor and is used for the treatment of colorectal cancer. Research is ongoing to evaluate its effectiveness with other solid tumors. Side effects of bevacizumab include delays in wound healing, hemorrhage, hypertension, thromboembolism and proteinuria. Newer agents such as sorafenib (Nexavar) and sunitinib (Sutent) have shown multitargeted activity against VEGF cell receptors and tyrosine kinase pathways and have been approved for metastatic renal cell carcinoma.

Cytokines

Cytokines, substances produced by cells of the immune system to enhance the production and functioning of components of the immune system, are also the focus of cancer treatment research. Cytokines are grouped into families, such as interferons, interleukins, colony-stimulating factors, and tumor necrosis factors. Colony-stimulating factors have been described earlier in this chapter for their supportive role in myelosuppressive treatment modalities. Refer to Chapter 50 for more detailed discussion of the immune system.

Interferons. Interferons (IFNs) are cytokines with both antiviral and antitumor properties. Multiple antitumor effects of IFNs include antiangiogenesis, direct destruction of tumor cells, inhibition of growth factors, and disruption of the cell cycle. IFN-α is used in treatment of hairy-cell leukemia, Kaposi's sarcoma, chronic myelogenous leukemia, high-grade non-Hodgkin lymphoma, renal cell cancer, cutaneous T-cell lymphoma, and melanoma. IFN is administered by subcutaneous, intramuscular, intravenous, and intracavitary routes. Efforts are under way to establish the effectiveness of IFN in combination with other treatment regimens for treatment of various malignancies.

Interleukins. Interleukins (ILs) are a subgroup of cytokines known as lymphokines and monokines produced by lymphocytes and monocytes. About 25 different ILs have been identified (Yarbro, et al., 2005) that act by signaling and coordinating other cells of the immune system and thus require an intact immune system to achieve their therapeutic effects. IL-2 is an approved treatment option for renal cell

cancer and metastatic melanoma in adults. IL-2 stimulates the production and activation of several different types of lymphocytes, enhances the production of other types of cytokines, and affects both humoral and cell-mediated immunity. Side effects of ILs include flulike symptoms, fatigue, and anorexia as well as serious side effects (eg, profound diarrhea, pulmonary edema, hypotension, and oliguria). When combined with other cytokines, IL-2 can cause hypersensitivity reactions or cardiac dysrhythmias and hypotension (Tyre & Quan, 2007).

Clinical trials are being conducted on the role of ILs in treatment of other cancers. Some early stage clinical trials are assessing their effects when combined with chemotherapy and as growth factors for treatment of myelosuppression after the use of some forms of chemotherapy.

Retinoids

Retinoids are vitamin A derivatives (retinol, all-*trans*-retinoic acid [ATRA], and 13-*cis*-retinoic acid) that play a role in growth, reproduction, apoptosis, epithelial cell differentiation, and immune function. Retinoids are believed to have a role in cancer prevention as well as treatment. Specific receptors in the cell nucleus are retinoid–dependent, thus when retinoids bind with these receptors, cell differentiation and replication are affected.

ATRA (tretinoin [Renova, Retin-A]) is used in treating acute promyelocytic leukemia, a rare form of leukemia, and cutaneous T-cell lymphoma. Synthetic retinoid agents such as 4HRP (Fenretinide) have been shown to play a role in cellular apoptosis and are being evaluated for prevention of second breast cancers. Retinoids are being tested for treatment of various epithelial cancers, leukemias, melanoma, and neuroblastoma, and for prevention of breast, lung, prostate, and brain cancers (Wilkes & Barton-Burke, 2007; Yarbro, et al., 2005).

Cancer Vaccines

Cancer vaccines are used to mobilize the body's immune response to recognize and attack cancer cells (Sinkovics & Horvath, 2006). Cancer vaccines contain either portions of cancer cells alone or portions of cells in combination with other substances (adjuvants) that can augment or boost immune responses. *Autologous* vaccines are made from the patient's own cancer cells, which are obtained during diagnostic biopsy or surgery. The cancer cells are killed and prepared for injection back into the patient. *Allogeneic* vaccines are made from cancer cells that are obtained from other people who have a specific type of cancer. These cancer cells are grown in a laboratory and eventually killed and prepared for injection.

Prophylactic vaccines are given to prevent disease. Quadrivalent human papillomavirus (HPV) recombinant vaccine (Gardasil) protects against HPV types 6, 11, 16, and 18 associated with common genital warts (type 6 and 11) and development of cervical cancer (type 16 and 18). It is administered over a series of three doses to females aged 9 to 26 (McLemore, 2006).

Therapeutic vaccines are given to kill existing cancer cells and to provide long-lasting immunity against further cancer development. Challenges to the therapeutic activity of cancer vaccines include the size of the tumor burden, the mechanisms that allow tumor cells to avoid recognition as "nonself" by the immune system, and immune tolerance as the result of previous exposure to the tumor antigens. Multiple clinical trials are being conducted to develop therapeutic vaccines for cancers of the prostate, breast, kidney, and lung, as well as for melanoma, myeloma, and lymphoma (Schlom, Arlen & Gulley, 2007).

Nursing Management in Biologic Response Modifier Therapy

Patients receiving BRM therapy have many of the same needs as patients undergoing other cancer treatment. However, manipulation and stimulation of the immune system create unique challenges. Consequently, it is essential for the nurse to assess the need for education, support, and guidance for both the patient and the family and assist in planning and evaluating patient care.

Monitoring Therapeutic and Adverse Effects. The nurse must become familiar with each agent given and its potential effects. Adverse effects such as fever, myalgia, nausea, and vomiting, as seen with IFN therapy, may not be life-threatening. However, the nurse must be aware of the impact of these side effects on the patient's quality of life. Other life-threatening adverse effects (eg, capillary leak syndrome, pulmonary edema, hypotension) may occur with IL-2 therapy.

Promoting Home and Community-Based Care. The nurse teaches patients self-care and assists in providing for continuing care. Some BRMs, such as IFN, EPO, and G-CSF, can be administered by the patient or family members at home. As needed, the nurse teaches the patient and family how to administer these agents through subcutaneous injections. The nurse also provides instructions about side effects and helps the patient and family identify strategies to manage many of the common side effects of BRM therapy, such as fatigue, anorexia, and flulike symptoms.

Referral for home care is usually indicated to monitor the patient's responses to treatment and to continue and reinforce patient and family teaching. During home visits, the nurse assesses the patient's and family members' technique in administering medications. The nurse collaborates with physicians, third-party payors, and pharmaceutical companies to help the patient obtain reimbursement for home administration of BRM therapies. The nurse also reminds the patient about the importance of keeping follow-up appointments with the physician and assesses the patient's need for changes in care.

Gene Therapy

Gene therapy includes approaches that correct genetic defects or manipulate genes to induce tumor cell destruction in the hope of preventing or combating disease. One of the challenges confronting cancer gene therapy is the multiple somatic mutations involved in the development of a cancer, making it difficult to identify the most effective gene therapy approach.

Considerable advances have been made in the identification of effective tumor cell targets and evaluation of the most appropriate vectors. Vectors serve as a vehicle or carrier that transports a gene into the target cell via the cell

membrane. With the improved understanding of cell surface proteins and signaling pathways, many phase I and phase II studies are currently evaluating the use of target specific vectors to disrupt tumor proliferation. The National Institutes of Health Web site identifies over 300 ongoing gene therapy trials targeting a variety of tumors and tumor cell markers. Examples include CEA, HER2/neu, and herpes simplex vaccine (NIH, 2007).

Viruses have long been hypothesized as an ideal delivery system because of the ease with which they cross the cell membrane and enter the intracellular space; however, their drawback includes their short-lived effect due to the strong immune response. Viruses used as vectors include retroviruses, adenoviruses (common cold virus), vaccinia virus (smallpox vaccine), fowlpox (avian poxvirus), herpes simplex viruses, and Epstein-Barr viruses (Yang, Wang, Zhao, et al., 2007). Clinical research studies are evaluating gene therapy across all cancer sites, including melanoma, prostate cancer, breast cancer, pancreatic cancer, head and neck squamous cell cancer, and non–small cell lung cancer. There are currently no FDA-approved cancer gene therapies in the United States.

Three general approaches have been used in the development of gene therapies, with adenoviruses showing effective promise in each approach.

- *Tumor-directed therapy* is introduction of a therapeutic gene (suicide gene) into tumor cells in an attempt to destroy them. This approach is very challenging because it is difficult to identify which gene would be the most beneficial. In addition, patients with widespread disease would require multiple injections to treat every site of disease.
- *Active immunotherapy* is the administration of genes that will invoke the antitumor responses of the immune system (Liu, 2003).
- *Adoptive immunotherapy* is the administration of genetically altered lymphocytes that are programmed to cause tumor destruction (Yang, et al., 2007).

Complementary and Alternative Medicine (CAM)

For many patients and their clinicians, a challenge in managing their cancer treatments is in finding the balance between achieving a reasonable quality of life while undergoing potentially toxic and life-saving modalities. Many patients seek a more holistic or nontraditional approach, turning to complementary and alternative therapies while continuing to utilize conventional medicine (Mumber, 2006).

The National Center for Complementary and Alternative Medicine (NCCAM) at the National Institutes of Health defines CAM as diverse medical and health care systems, practices, and products that are not presently considered to be part of conventional medicine. Complementary medicine denotes therapies in conjunction with conventional medicine, whereas alternative medicine denotes therapies used instead of conventional medicine. More recently, the term Integrative Medicine has been used, which denotes a combination of conventional medicine and CAM that have a strong scientific base for use and safety (NCCAM, 2007)

CAM is used by 28% to 85% of patients with cancer (Chart 16-6). More importantly, patients are using CAM but not communicating this to their health care providers either because they were never asked about its use or because they withheld the information fearing that their physicians would not approve (Chong, 2006; Rojas-Cooley & Grant, 2006). Many of the CAM modalities can be a source of comfort and emotional support for the patient, but assessment of CAM use is important for patient safety.

Mind–body and biofield therapies have a holistic focus on channeling positive energy, promoting relaxation, and reducing stress and have been reported as being beneficial to patients as measured by wound healing and reduction in pain, edema, and anxiety (Hibdon, 2005). There is, however, risk associated with some of the CAM modalities. Because of the possibility of herb-vitamin-drug interactions, there is concern about the use of biologicals and dietary supplements, which are not regulated by the FDA nor subjected to rigorous scientific evaluation. Patients often perceive vitamins and dietary supplements as harmless, natural products that have no side effects or potential toxicities. One example of herbal–drug interaction is the effect of St. John's wort on the efficacy of irinotecan (Camptosar), cyclophosphamide, tamoxifen, cyclosporine, warfarin (Coumadin), and indinavir (Crixivan). Each nursing assessment should include an open discussion with patients about their use of CAM. This requires that nurses develop the appropriate familiarity and knowledge related to CAM in order to direct patients to safe, reliable, and credible sources for information.

Unproven and Unconventional Therapies

Despite increasing 5-year survival rates with the use of traditional methods of treatment, a significant number of patients use or seriously consider using some form of unconventional treatment. Hopelessness, desperation, unmet needs, lack of factual information, and family or social pressures are major factors that motivate patients to seek unconventional methods of treatment and allow them to fall prey to deceptive practices and quackery.

Unconventional treatments are those without scientific evidence of the ability to cure or control cancer. In addition to being ineffective, some unconventional treatments may also be harmful to the patient and may cost thousands of dollars.

In the age of the Internet, patients have unlimited access to frequently unreliable claims of "miracle cures" that range from plant remedies to metabolic therapy using special diets, supplements, or "detoxification" regimens involving unconventional enemas and colonic cleansing procedures. The ACS established a clearinghouse along with NCCAM to investigate and identify potentially dangerous and harmful unproven therapies. ACS maintains this listing on its Web site.

Nursing Management in Unconventional Therapies

The most effective way to protect patients and families from fraudulent therapies and questionable cancer cures is to establish a trusting relationship, provide supportive care, and promote hope. Truthful responses given in a nonjudgmental manner to questions and inquiries about unproven methods of cancer treatments may alleviate the fear and guilt on the part of the patient and family that they are not "doing everything we can" to obtain a cure. The nurse should inform the patient and family of the characteristics common to fraudulent therapies so that they will be informed and

CHART 16-6 NURSING RESEARCH PROFILE

Use of Acupressure to Reduce Chemotherapy-induced Nausea and Vomiting

Dribble, S. L., Luce, J., Cooper, B. A., et al. (2007). Acupressure for chemotherapy-induced nausea and vomiting: A randomized clinical trial. *Oncology Nursing Forum, 34*(4), 813–820.

Purpose

The purpose of this study was to compare the effectiveness of acupressure, placebo acupressure, and usual care in reducing chemotherapy-induced nausea and vomiting (CINV) in women with breast cancer. Although significant advances have been made in the medications available to treat CINV, delayed nausea and vomiting continue to be problematic for many patients. Acupressure is a traditional Chinese nonpharmacologic, noninvasive pressure applied by the thumbs, fingers, and hands on the surface of the skin at specific points. The belief is that symptoms such as nausea may be lessened through the use of acupressure.

Design

This was a multicenter, longitudinal randomized clinical trial conducted throughout one cycle of highly emetogenic chemotherapy. Ten community oncology programs associated with a major cancer center and nine independent sites located throughout the United States served as the study sites. To be eligible, the women had to be receiving the second or third cycle of chemotherapy classified as moderate to highly emetogenic. In addition, during the previous cycle of chemotherapy, the women had to have experienced at least moderate nausea as measured by the Morrow Assessment of Nausea and Emesis.

A total of 160 women were randomly assigned to one of three intervention groups: acupressure, placebo acupressure, and usual care. Each woman received a prescription for antiemetic therapy to use at home; thus, acupressure was studied in the context of usual clinical care for nausea. The patients in the acupressure groups were taught how to use the actual or placebo acupressure techniques immediately prior to receiving chemotherapy. The acupressure and placebo acupressure groups completed daily logs for 3 weeks and recorded acupressure use as well as medications and other methods used to control their nausea. The usual care group also completed daily logs about efforts used to control nausea. On the eighth day of the chemotherapy cycle, women were reminded to complete the daily logs. Anxiety measures were obtained for all participants using the State-Trait Anxiety Inventory Scale at baseline and at the exit appointment at the time of the next chemotherapy cycle. Demographic data and diagnostic, cancer treatment, and treatment of nausea data were also collected. The occurrence of acute nausea and vomiting on the day of chemotherapy (study day 1) and its occurrence on the 2nd to 11th days after chemotherapy (delayed emesis) were analyzed.

Findings

The three groups were similar on demographic variables, disease, and treatment. There were no significant differences in episodes of acute CINV by treatment group. Although episodes of delayed CINV decreased for all three treatment groups over time, women in the acupressure group reported fewer episodes than both the placebo acupressure group and the usual care group. Many of the participants noted that acupressure was most effective when the nausea was mild, but that it was helpful in addition to pharmaceutical agents when the nausea was severe. The researchers also found that 30% of the participants experienced delayed nausea at 11 days after chemotherapy. They concluded that in future studies delayed nausea assessment should extend for at least 11 days versus the usual length of assessment of 5 days. The researchers reported differences in the incidence of CINV by age, with younger women reporting a greater intensity of nausea than older women.

Nursing Implications

The results of this study suggest that acupressure may be a valid addition to other interventions for the management of CINV, including CINV that occurred 2 to 11 days after chemotherapy treatment. Acupressure offers a nonpharmacologic, alternative approach to care of a significant problem for many patients. The technique is easily learned without significant expense or prolonged training. Future research might assess the role of acupressure in the treatment of CINV in both genders and for other types of cancer.

cautious when evaluating other forms of "therapy." The nurse also should encourage patients who use unconventional therapies to inform their physicians about such use. This knowledge can help prevent interactions with medications and other therapies that may be prescribed and avoid attributing the side effects of unconventional therapies to prescribed medications.

Nursing Care of Patients With Cancer

The outlook for patients with cancer has greatly improved because of scientific and technologic advances. However, as a result of the underlying disease or various treatment modalities, patients with cancer may experience a variety of secondary problems such as infection, reduced WBC counts, bleeding, skin problems, nutritional problems, pain, fatigue, and psychological stress. Chart 16-7 provides a nursing care plan for patients with cancer.

Maintaining Tissue Integrity

Some of the most frequently encountered disturbances of tissue integrity, in addition to stomatitis, include skin and tissue reactions to radiation therapy, alopecia, and metastatic skin lesions.

Stomatitis

Mucositis is a common side effect of radiation and some types of chemotherapy that may lead to inflammation and ulceration of any portion of the gastrointestinal tract from the oral cavity throughout the alimentary canal. One form of mucositis, stomatitis, is an inflammatory response of the oral tissues that is characterized by mild redness (erythema) and edema or, if severe, by painful ulcerations, bleeding, and secondary infection. Stomatitis commonly develops 5

CHART 16-7

PLAN OF NURSING CARE
The Patient With Cancer

NURSING DIAGNOSIS: Risk for infection related to inadequate defenses related to myelosuppression secondary to radiation or antineoplastic agents
GOAL: Prevention of infection

Nursing Interventions	Rationale	Expected Outcomes
1. Assess patient for evidence of infection: a. Check vital signs every 4 hours. b. Monitor white blood cell (WBC) count and differential each day. c. Inspect all sites that may serve as entry ports for pathogens (intravenous sites, wounds, skin folds, bony prominences, perineum, and oral cavity).	1. Signs and symptoms of infection may be diminished in the immunocompromised host. Prompt recognition of infection and subsequent initiation of therapy will reduce morbidity and mortality associated with infection.	• Demonstrates normal temperature and vital signs. • Exhibits absence of signs of inflammation: local edema, erythema, pain, and warmth. • Exhibits normal breath sounds on auscultation. • Takes deep breaths and coughs every 2 hours to prevent respiratory dysfunction and infection. • Exhibits absence of pathologic bacteria on cultures. • Avoids contact with others with infections. • Avoids crowds. • All personnel carry out hand hygiene after each voiding and bowel movement. • Excoriation and trauma of skin are avoided. • Trauma to mucous membranes is avoided (avoidance of rectal thermometers, suppositories, vaginal tampons, perianal trauma). • Uses recommended procedures and techniques if participating in management of invasive lines or catheters. • Uses electric razor. • Is free of skin breakdown and stasis of secretions. • Adheres to dietary and environmental restrictions. • Exhibits no signs of septicemia or septic shock. • Exhibits normal vital signs, cardiac output, and arterial pressures when monitored. • Demonstrates ability to administer colony-stimulating factor.
2. Report fever (≥38.3°C [101°F] or ≥38°C [100.4°F] for longer than 1 hour), chills, diaphoresis, swelling, heat, pain, erythema, exudate on any body surfaces. Also report change in respiratory or mental status, urinary frequency or burning, malaise, myalgias, arthralgias, rash, or diarrhea.	2. Early detection of infection facilitates early intervention.	
3. Obtain cultures and sensitivities as indicated before initiation of antimicrobial treatment (wound exudate, sputum, urine, stool, blood).	3. Tests identify the organism and indicate the most appropriate antimicrobial therapy. Use of inappropriate antibiotics enhances proliferation of additional flora and encourages growth of antibiotic-resistant organisms.	
4. Initiate measures to minimize infection. a. Discuss with patient and family (1) Placing patient in private room if absolute WBC count $<1000/mm^3$. (2) Importance of patient avoiding contact with people who have known or recent infection or recent vaccination.	4. Exposure to infection is reduced. a. Preventing contact with pathogens helps prevent infection.	
b. Instruct all personnel in careful hand hygiene before and after entering room.	b. Hands are significant source of contamination.	
c. Avoid rectal or vaginal procedures (rectal temperatures, examinations, suppositories; vaginal tampons).	c. Incidence of rectal and perianal abscesses and subsequent systemic infection is high. Manipulation may cause disruption of membrane integrity and enhance progression of infection.	
d. Use stool softeners to prevent constipation and straining.	d. Minimizes trauma to tissues.	
e. Assist patient in practice of meticulous personal hygiene.	e. Prevents skin irritation.	
f. Instruct patient to use electric razor.	f. Minimizes skin trauma.	
g. Encourage patient to ambulate in room unless contraindicated.	g. Minimizes chance of skin breakdown and stasis of pulmonary secretions.	
h. Avoid fresh fruits, raw meat, fish, and vegetables if absolute WBC count $<1000/mm^3$; remove fresh flowers and potted plants.	h. Fresh fruits and vegetables harbor bacteria not removed by ordinary washing. Flowers and potted plants are sources of organisms.	
i. Each day: change water pitcher, denture cleaning fluids, and respiratory equipment containing water.	i. Stagnant water is a source of infection.	

Continued on following page

CHART 16-7 PLAN OF NURSING CARE
The Patient With Cancer (Continued)

Nursing Interventions	Rationale	Expected Outcomes
5. Assess intravenous sites every day for evidence of infection: a. Change peripheral short-term intravenous sites every other day. b. Cleanse skin with povidone-iodine before arterial puncture or venipuncture. c. Change central venous catheter dressings every 48 hours. d. Change all solutions and infusion sets every 72–96 hours. e. Follow Infusion Nursing Society guidelines for care of peripheral and central venous access devices.	5. Nosocomial staphylococcal septicemia is closely associated with intravenous catheters. a. Incidence of infection is increased when catheter is in place >72 hours. b. Povidone-iodine is effective against many gram-positive and gram-negative pathogens. c. Allows observation of site and removes source of contamination. d. Once introduced into the system, microorganisms are capable of growing in infusion sets despite replacement of container and high flow rates. e. Infusion nursing society collaborates with other nursing subspecialties in determining guidelines for intravenous access care.	
6. Avoid intramuscular injections.	6. Reduces risk for skin abscesses.	
7. Avoid insertion of urinary catheters; if catheters are necessary, use strict aseptic technique.	7. Rates of infection greatly increase after urinary catheterization.	
8. Teach patient or family member to administer granulocyte (or granulocyte-macrophage) colony-stimulating factor when prescribed.	8. Granulocyte colony-stimulating factor decreases the duration of neutropenia and the potential for infection.	
9. Advise patient to avoid exposure to animal excreta; discuss dental procedures with physician; avoid vaginal douche; and avoid vaginal or rectal manipulation during sexual contact during period of neutropenia (Marrs, 2006; Zitella, et al., 2006).	9. Minimizes exposure to potential sources of infection and disruption of skin integrity.	
NURSING DIAGNOSIS: Impaired skin integrity: erythematous and wet desquamation reactions to radiation therapy		
1. In erythematous areas: a. Avoid the use of soaps, cosmetics, perfumes, powders, lotions and ointments, deodorants. b. Use only lukewarm water to bathe the area. c. Avoid rubbing or scratching the area. d. Avoid shaving the area with a straight-edged razor. e. Avoid applying hot-water bottles, heating pads, ice, and adhesive tape to the area. f. Avoid exposing the area to sunlight or cold weather. g. Avoid tight clothing in the area. Use cotton clothing. h. Apply vitamin A and D ointment to the area.	1. Care to the affected areas must focus on preventing further skin irritation, drying, and damage. a. These substances may cause pain and additional skin irritation and damage. b. Avoiding water of extreme temperatures minimizes additional skin damage, irritation and pain. c. Rubbing and or scratching will lead to additional skin irritation, damage and increased risk of infection. d. Use of razors may lead to additional irritation and disruption of skin integrity and increased risk of infection. e. Avoiding extreme temperatures minimizes additional skin damage, irritation, burns and pain. f. Sun exposure or extreme cold weather may lead to additional skin damage and pain. g. Allows air circulation to affected area. h. Aids healing.	• Avoids use of soaps, powders, and other cosmetics on site of radiation therapy. • States rationale for special care of skin. • Exhibits minimal change in skin. • Avoids trauma to affected skin region (avoids shaving, constricting and irritating clothing, extremes of temperature, and use of adhesive tape). • Reports change in skin promptly. • Demonstrates proper care of blistered or open areas. • Exhibits absence of infection of blistered and opened areas. • Wound is free from development of eschar.

Continued

CHART 16-7 PLAN OF NURSING CARE
The Patient With Cancer (Continued)

Nursing Interventions	Rationale	Expected Outcomes
2. If wet desquamation occurs:	2. Open weeping areas are susceptible to bacterial infection. Care must be taken to prevent introduction of pathogens.	
a. Do not disrupt any blisters that have formed.	a. Disruption of skin blisters disrupts skin integrity and may lead to increased risk of infection.	
b. Avoid frequent washing of the area.	b. Frequent washing may lead to increased irritation and skin damage, with increased risk for infection.	
c. Report any blistering.	c. Blistering of skin represents progression of skin damage.	
d. Use *prescribed* creams or ointments.	d. Decreases irritation and inflammation of the area.	
e. If area weeps, apply a nonadhesive absorbent dressing.	e. Enhances drying.	
f. If the area is without drainage, use moisture and vapor-permeable dressings such as hydrocolloids and hydrogels on noninfected areas (Swearingen, 2008).	f. Promotes healing.	
g. Consult with enterostomal therapist (ET) and physician if eschar forms.	g. Eschar must be removed to promote healing and prevent infection. ET nurses have expertise in the care of wounds.	

NURSING DIAGNOSIS: Impaired oral mucous membrane: stomatitis
GOAL: Maintenance of intact oral mucous membranes

Nursing Interventions	Rationale	Expected Outcomes
1. Assess oral cavity daily.	1. Provides baseline for later evaluation.	• States rationale for frequent oral assessment and hygiene.
2. Instruct patient to report oral burning, pain, areas of redness, open lesions on the lips, pain associated with swallowing, or decreased tolerance to temperature extremes of food.	2. Identification of initial stages of stomatitis will facilitate prompt interventions, including modification of treatment as prescribed by physician.	• Identifies signs and symptoms of stomatitis to report to nurse or physician.
3. Encourage and assist in oral hygiene.	3. Patients who are having discomfort or pain, or other symptoms related to the disease and treatment may require encouragement and assistance in performing oral hygiene.	• Participates in recommended oral hygiene regimen.
Preventive		• Avoids mouthwashes with alcohol.
a. Advise patient to avoid irritants such as commercial mouthwashes, alcoholic beverages, and tobacco.	a. Alcohol content of mouthwashes will dry oral tissues and potentiate breakdown.	• Brushes teeth and mouth with soft toothbrush.
b. Brush with soft toothbrush; use nonabrasive toothpaste after meals and bedtime; floss every 24 hours unless painful or platelet count falls below 40,000 cu/mm.	b. Limits trauma and removes debris.	• Uses lubricant to keep lips soft and nonirritated.
Mild stomatitis (generalized erythema, limited ulcerations, small white patches: *Candida*)		• Avoids hard-to-chew, spicy, and hot foods.
c. Use normal saline mouth rinses every 2 hours while awake; every 6 hours at night.	c. Assists in removing debris, thick secretions, and bacteria.	• Exhibits clean, intact oral mucosa.
d. Use soft toothbrush or toothette.	d. Minimizes trauma.	• Exhibits no ulcerations or infections of oral cavity.
e. Remove dentures except for meals; be certain dentures fit well.	e. Minimizes friction and discomfort.	• Exhibits no evidence of bleeding.
f. Apply water soluble lip lubricant.	f. Promotes comfort.	• Reports absent or decreased oral pain.
g. Avoid foods that are spicy or hard to chew and those with extremes of temperature.	g. Prevents local trauma.	• Reports no difficulty swallowing.
		• Exhibits healing (reepithelialization) of oral mucosa within 5 to 7 days (mild stomatitis).
		• Exhibits healing of oral tissues within 10 to 14 days (severe stomatitis).

Continued on following page

CHART 16-7 PLAN OF NURSING CARE
The Patient With Cancer (Continued)

Nursing Interventions	Rationale	Expected Outcomes
Severe stomatitis (confluent ulcerations with bleeding and white patches covering more than 25% of oral mucosa)		• Exhibits no bleeding or oral ulceration. • Consumes adequate fluid and food. • Exhibits absence of dehydration and weight loss.
h. Obtain tissue samples for culture and sensitivity tests of areas of infection.	h. Assists in identifying need for antimicrobial therapy.	
i. Assess ability to chew and swallow; assess gag reflex.	i. Patient may be in danger of aspiration.	
j. Use oral rinses (may combine in solution saline, anti-*Candida* agent, such as Mycostatin, and topical anesthetic agent as described below) as prescribed or place patient on side and irrigate mouth; have suction available.	j. Facilitates cleansing, provides for safety and comfort.	
k. Remove dentures.	k. Prevents trauma from ill-fitting dentures.	
l. Use toothette or gauze soaked with solution for cleansing.	l. Limits trauma, promotes comfort.	
m. Use water soluble lip lubricant.	m. Promotes comfort.	
n. Provide liquid or pureed diet.	n. Ensures intake of easily digestible foods.	
o. Monitor for dehydration.	o. Decreased oral intake and ulcerations potentiate fluid deficits.	
4. Minimize discomfort.		
a. Consult physician for use of topical anesthetic, such as dyclonine and diphenhydramine, or viscous lidocaine.	a. Alleviates pain and increases sense of well-being; promotes participation in oral hygiene and nutritional intake.	
b. Administer systemic analgesics as prescribed.	b. Adequate management of pain related to severe stomatitis can facilitate improved quality of life, participation in other aspects of activities of daily living, oral intake and verbal communication.	
c. Perform mouth care as described.	c. Promotes removal of debris, healing, and comfort.	
NURSING DIAGNOSIS: Impaired tissue integrity: alopecia **GOAL:** Maintenance of tissue integrity; coping with hair loss		
Nursing Interventions	**Rationale**	**Expected Outcomes**
1. Discuss potential hair loss and regrowth with patient and family; advise that hair loss may occur on body parts other than the head.	1. Provides information so patient and family can begin to prepare cognitively and emotionally for loss.	• Identifies alopecia as potential side effect of treatment. • Identifies positive and negative feelings and threats to self-image. • Verbalizes meaning that hair and possible hair loss have for him or her. • States rationale for modifications in hair care and treatment. • Uses mild shampoo and conditioner and shampoos hair only when necessary. • Avoids hair dryer, curlers, sprays, and other stresses on hair and scalp. • Wears hat or scarf over hair when exposed to sun. • Takes steps to deal with possible hair loss before it occurs; purchases wig or hairpiece.
2. Explore potential impact of hair loss on self-image, interpersonal relationships, and sexuality.	2. Facilitates coping.	
3. Prevent or minimize hair loss through the following:	3. Retains hair as long as possible.	
a. Use scalp hypothermia and scalp tourniquets, if appropriate.	a. Decreases hair follicle uptake of chemotherapy (not used for patients with leukemia or lymphoma because tumor cells may be present in blood vessels or scalp tissue).	
b. Cut long hair before treatment. c. Use mild shampoo and conditioner, gently pat dry, and avoid excessive shampooing. d. Avoid electric curlers, curling irons, dryers, clips, barrettes, hair sprays, hair dyes, and permanent waves. e. Avoid excessive combing or brushing; use wide-toothed comb.	b–e. Minimizes hair loss due to the weight and manipulation of hair.	

Continued

CHART 16-7 PLAN OF NURSING CARE
The Patient With Cancer (Continued)

Nursing Interventions	Rationale	Expected Outcomes
4. Prevent trauma to scalp. a. Lubricate scalp with vitamin A and D ointment to decrease itching. b. Have patient use sunscreen or wear hat when in the sun.	4. Preserves tissue integrity. a. Assists in maintaining skin integrity. b. Prevents ultraviolet light exposure.	• Maintains hygiene and grooming. • Interacts and socializes with others. • States that hair loss and necessity of wig are temporary.
5. Suggest ways to assist in coping with hair loss: a. Purchase wig or hairpiece before hair loss. b. If hair loss has occurred, take photograph to wig shop to assist in selection. c. Begin to wear wig before hair loss. d. Contact the American Cancer Society for donated wigs, or a store that specializes in this product. e. Wear hat, scarf, or turban.	5. Minimizes change in appearance. a. Wig that closely resembles hair color and style is more easily selected if hair loss has not begun. b. Facilitates adjustment. c. Enables patient to be prepared for loss and facilitates adjustment. d. Provides options to patient. e. Conceals loss.	
6. Encourage patient to wear own clothes and retain social contacts.	6. Assists in maintaining personal identity.	
7. Explain that hair growth usually begins again once therapy is completed.	7. Reassures patient that hair loss is usually temporary.	

NURSING DIAGNOSIS: Imbalanced nutrition, less than body requirements, related to nausea and vomiting
GOAL: Patient experiences less nausea and vomiting associated with chemotherapy; weight loss is minimized

Nursing Interventions	Rationale	Expected Outcomes
1. Assess the patient's previous experiences and expectations of nausea and vomiting, including causes and interventions used.	1. Identifies patient concerns, misinformation, potential strategies for intervention. Also gives patient sense of empowerment and control.	• Identifies previous triggers of nausea and vomiting. • Exhibits decreased apprehension and anxiety. • Identifies previously used successful interventions for nausea and vomiting. • Reports decrease in nausea. • Reports decrease in incidence of vomiting. • Consumes adequate fluid and food when nausea subsides. • Demonstrates use of distraction, relaxation, and imagery when indicated. • Exhibits normal skin turgor and moist mucous membranes. • Reports no additional weight loss.
2. Adjust diet before and after drug administration according to patient preference and tolerance.	2. Each patient responds differently to food after chemotherapy. A diet containing foods that relieve the patient's nausea or vomiting is most helpful.	
3. Prevent unpleasant sights, odors, and sounds in the environment.	3. Unpleasant sensations can stimulate the nausea and vomiting center.	
4. Use distraction, music therapy, biofeedback, self-hypnosis, relaxation techniques, and guided imagery before, during, and after chemotherapy.	4. Decreases anxiety, which can contribute to nausea and vomiting. Psychological conditioning may also be decreased.	
5. Administer prescribed antiemetics, sedatives, and corticosteroids before chemotherapy and afterward as needed.	5. Administration of antiemetic regimen before onset of nausea and vomiting limits the adverse experience and facilitates control. Combination drug therapy reduces nausea and vomiting through various triggering mechanisms.	
6. Ensure adequate fluid hydration before, during, and after drug administration; assess intake and output.	6. Adequate fluid volume dilutes drug levels, decreasing stimulation of vomiting receptors.	
7. Encourage frequent oral hygiene.	7. Reduces unpleasant taste sensations.	
8. Provide pain relief measures, if necessary.	8. Increased comfort increases physical tolerance of symptoms.	

Continued on following page

CHART 16-7 PLAN OF NURSING CARE
The Patient With Cancer (Continued)

Nursing Interventions	Rationale	Expected Outcomes
9. Consult with dietician as needed.	9. Interdisciplinary collaboration essential in addressing complex patient needs.	
10. Assess and address other contributing factors to nausea and vomiting, such as other symptoms, constipation, gastrointestinal irritation, electrolyte imbalance, radiation therapy, medications, and central nervous system metastasis.	10. Multiple factors may contribute nausea and vomiting.	

NURSING DIAGNOSIS: Imbalanced nutrition: less than body requirements, related to anorexia, cachexia, or malabsorption
GOAL: Maintenance of nutritional status and of weight within 10% of pretreatment weight

Nursing Interventions	Rationale	Expected Outcomes
1. Teach patient to avoid unpleasant sights, odors, sounds in the environment during mealtime.	1. Anorexia can be stimulated or increased with noxious stimuli.	• Patient and family identify minimal nutritional requirements.
2. Suggest foods that are preferred and well tolerated by the patient, preferably high-calorie and high-protein foods. Respect ethnic and cultural food preferences.	2. Foods preferred, well tolerated, and high in calories and protein maintain nutritional status during periods of increased metabolic demand.	• Exhibits weight loss no greater than 10% of pretreatment weight. • Reports decreasing anorexia and increased interest in eating. • Demonstrates normal skin turgor.
3. Encourage adequate fluid intake, but limit fluids at mealtime.	3. Fluids are necessary to eliminate wastes and prevent dehydration. Increased fluids with meals can lead to early satiety.	• Identifies rationale for dietary modifications. Patient and family verbalize strategies to address minimize nutritional deficits.
4. Suggest smaller, more frequent meals.	4. Smaller, more frequent meals are better tolerated because early satiety does not occur.	• Participates in calorie counts and diet histories. • Uses appropriate relaxation and imagery before meals.
5. Promote relaxed, quiet environment during mealtime with increased social interaction as desired.	5. A quiet environment promotes relaxation. Social interaction at mealtime increases appetite.	• Exhibits laboratory and clinical findings indicative of adequate nutritional intake: normal serum protein and transferrin levels; normal serum iron levels; normal hemoglobin, hematocrit, and lymphocyte levels; normal urinary creatinine levels.
6. If patient desires, serve wine at mealtime with foods.	6. Wine often may stimulate appetite and add calories.	
7. Consider cold foods, if desired.	7. Cold, high-protein foods are often more tolerable and less odorous than hot foods.	
8. Encourage nutritional supplements and high-protein foods between meals.	8. Supplements and snacks add protein and calories to meet nutritional requirements.	• Consumes diet high in required nutrients.
9. Encourage frequent oral hygiene.	9. Oral hygiene stimulates appetite and increases saliva production.	• Carries out oral hygiene before meals.
10. Provide pain relief measures.	10. Pain impairs appetite.	• Reports that pain does not interfere with meals.
11. Provide control of nausea and vomiting.	11. Nausea and vomiting increase anorexia.	• Reports decreasing episodes of nausea and vomiting.
12. Increase activity level as tolerated.	12. Increased activity promotes appetite.	• Participates in increasing levels of activity.
13. Decrease anxiety by encouraging verbalization of fears, concerns; use of relaxation techniques; imagery at mealtime.	13. Relief of anxiety may increase appetite.	
14. Position patient properly at mealtime.	14. Proper body position and alignment are necessary to aid chewing and swallowing.	• States rationale for use of tube feedings or parenteral nutrition. • Participates in management of tube feedings or parenteral nutrition, if prescribed.
15. For collaborative management, provide enteral tube feedings of commercial liquid diets, elemental diets, or blenderized foods as prescribed.	15. Tube feedings may be necessary in the severely debilitated patient who has a functioning gastrointestinal system.	
16. Provide parenteral nutrition with lipid supplements as prescribed.	16. Parenteral nutrition with supplemental fats supplies needed calories and proteins to meet nutritional demands, especially in the nonfunctional gastrointestinal system.	

Continued

CHART 16-7 PLAN OF NURSING CARE
The Patient With Cancer (Continued)

Nursing Interventions	Rationale	Expected Outcomes
17. Administer appetite stimulants as prescribed by physician.	17. Although the mechanism is unclear, medications such as megestrol acetate (Megace) have been noted to improve appetite in patients with cancer and human immunodeficiency virus (HIV) infection.	
18. Encourage family and friends not to nag or cajole patient about eating.	18. Pressuring patient to eat may cause conflict and unnecessary stress.	
19. Assess and address other contributing factors to nausea, vomiting, and anorexia such as other symptoms, constipation, GI irritation, electrolyte imbalance, radiation therapy, medications, and central nervous system metastasis.	19. Multiple factors contribute to anorexia and nausea.	
NURSING DIAGNOSIS: Fatigue **GOAL:** Increased activity tolerance and decreased fatigue level		
Nursing Interventions	**Rationale**	**Expected Outcomes**
1. Encourage rest periods during the day, especially before and after physical exertion.	1. During rest, energy is conserved and levels are replenished. Several shorter rest periods may be more beneficial than one longer rest period.	• Reports decreasing levels of fatigue. • Increases participation in activities gradually. • Rests when fatigued. • Reports restful sleep. • Requests assistance with activities appropriately. • Reports adequate energy to participate in activities important to him or her (eg, visiting with family, hobbies). • Consumes diet with recommended protein and caloric intake. • Uses relaxation exercises and imagery to decrease anxiety and promote rest. • Participates in planned exercise program gradually. • Reports no breathlessness during activities. • Exhibits acceptable hemoglobin and hematocrit levels. • Exhibits normal fluid and electrolyte balance. • Reports decreased discomfort. • Exhibits improved mobility.
2. At minimum, promote patient's normal sleep habits.	2. Sleep helps to restore energy levels. Prolonged napping during day may interfere with sleep habits.	
3. Rearrange daily schedule and organize activities to conserve energy expenditure.	3. Reorganization of activities can reduce energy losses and stressors.	
4. Encourage patient to ask for others' assistance with necessary chores, such as housework, child care, shopping, cooking.	4. Conserves energy.	
5. Encourage reduced job workload, if necessary and possible, by reducing number of hours worked per week.	5. Reducing workload decreases physical and psychological stress and increases periods of rest and relaxation.	
6. Encourage adequate protein and calorie intake.	6. Protein and calorie depletion decreases activity tolerance.	
7. Encourage use of relaxation techniques, mental imagery.	7. Promotion of relaxation and psychological rest decreases physical fatigue.	
8. Encourage participation in planned exercise programs.	8. Proper exercise programs increase endurance and stamina and lower fatigue.	
9. For collaborative management, administer blood products as prescribed.	9. Lowered hemoglobin and hematocrit predispose patient to fatigue due to decreased oxygen availability.	
10. Assess for fluid and electrolyte disturbances.	10. May contribute to altered nerve transmission and muscle function.	
11. Assess for sources of discomfort.	11. Coping with discomfort requires energy expenditure.	
12. Provide strategies to facilitate mobility.	12. Impaired mobility requires increased energy expenditure.	

Continued on following page

CHART 16-7 PLAN OF NURSING CARE
The Patient With Cancer (Continued)

NURSING DIAGNOSIS: Chronic pain
GOAL: Relief of pain and discomfort

Nursing Interventions	Rationale	Expected Outcomes
1. Use pain scale to assess pain and discomfort characteristics: location, quality, frequency, duration, etc.	1. Provides baseline for assessing changes in pain level and evaluation of interventions.	• Reports decreased level of pain and discomfort on pain scale.
2. Assure patient that you know that pain is real and will assist him or her in reducing it.	2. Fear that pain will not be considered real increases anxiety and reduces pain tolerance.	• Reports less disruption from pain and discomfort.
3. Assess other factors contributing to patient's pain: fear, fatigue, anger, etc.	3. Provides data about factors that decrease patient's ability to tolerate pain and increase pain level.	• Explains how fatigue, fear, anger, etc., contribute to severity of pain and discomfort.
4. Administer analgesics to promote optimum pain relief within limits of physician's prescription.	4. Analgesics tend to be more effective when administered early in pain cycle.	• Accepts analgesia as prescribed.
5. Assess patient's behavioral responses to pain and pain experience.	5. Provides additional information about patient's pain.	• Exhibits decreased physical and behavioral signs of pain and discomfort in acute pain (no grimacing, crying, moaning; displays interest in surroundings and activities around him).
6. Collaborate with patient, physician, and other health care team members when changes in pain management are necessary.	6. New methods of administering analgesia must be acceptable to patient, physician, and health care team to be effective; patient's participation decreases the sense of powerlessness.	• Takes an active role in administration of analgesia.
7. Encourage strategies of pain relief that patient has used successfully in previous pain experience.	7. Encourages success of pain relief strategies accepted by patient and family.	• Identifies additional effective pain relief strategies.
8. Teach patient new strategies to relieve pain and discomfort: distraction, imagery, relaxation, cutaneous stimulation, etc.	8. Increases number of options and strategies available to patient.	• Uses alternative pain relief strategies appropriately. • Reports effective use of new pain relief strategies and decrease in pain intensity. • Reports that decreased level of pain permits participation in other activities and events.

NURSING DIAGNOSIS: Anticipatory grieving related to loss; altered role functioning
GOAL: Appropriate progression through grieving process

Nursing Interventions	Rationale	Expected Outcomes
1. Encourage verbalization of fears, concerns, and questions regarding disease, treatment, and future implications.	1. An increased and accurate knowledge base decreases anxiety and dispels misconceptions.	• The patient and family progress through the phases of grief as evidenced by increased verbalization and expression of grief.
2. Explore previous successful coping strategies.	2. Provides frame of reference and examples of coping.	• The patient and family identify resources available to aid coping strategies during grieving.
3. Encourage active participation of patient or family in care and treatment decisions.	3. Active participation maintains patient independence and control.	• The patient and family use resources and supports appropriately.
4. Visit family frequently to establish and maintain relationships and physical closeness.	4. Frequent contacts promote trust and security and reduce feelings of fear and isolation.	• The patient and family discuss the future openly with each other.
5. Encourage ventilation of negative feelings, including projected anger and hostility, within acceptable limits.	5. This allows for emotional expression without loss of self-esteem.	• The patient and family discuss concerns and feelings openly with each other.
6. Allow for periods of crying and expression of sadness.	6. These feelings are necessary for separation and detachment to occur.	• The patient and family use nonverbal expressions of concern for each other.
7. Involve spiritual advisor as desired by the patient and family.	7. This facilitates the grief process and spiritual care.	
8. Advise professional counseling as indicated for patient or family to alleviate pathologic grieving.	8. This facilitates the grief process.	
9. Allow for progression through the grieving process at the individual pace of the patient and family.	9. Grief work is variable. Not every person uses every phase of the grief process, and the time spent in dealing with each phase varies with every person. To complete grief work, this variability must be allowed.	

Continued

CHART 16-7

PLAN OF NURSING CARE
The Patient With Cancer (Continued)

NURSING DIAGNOSIS: Disturbed body image and situational low self-esteem related to changes in appearance, function, and roles
GOAL: Improved body image and self-esteem

Nursing Interventions	Rationale	Expected Outcomes
1. Assess patient's feelings about body image and level of self- esteem.	1. Provides baseline assessment for evaluating changes and assessing effectiveness of interventions.	• Identifies concerns of importance. • Takes active role in activities. • Maintains previous role in decision making.
2. Identify potential threats to patient's self-esteem (eg, altered appearance, decreased sexual function, hair loss, decreased energy, role changes). Validate concerns with patient.	2. Anticipates changes and permits patient to identify importance of these areas to him or her.	• Verbalizes feelings and reactions to losses or threatened losses. • Participates in self-care activities. • Permits others to assist in care when he or she is unable to be independent.
3. Encourage continued participation in activities and decision making.	3. Encourages and permits continued control of events and self.	• Exhibits interest in appearance and uses aids (cosmetics, scarves, etc.) appropriately.
4. Encourage patient to verbalize concerns.	4. Identifying concerns is an important step in coping with them.	• Participates with others in conversations and social events and activities.
5. Individualize care for the patient.	5. Prevents or reduces depersonalization and emphasizes patient's self-worth.	• Verbalizes concern about sexual partner and/or significant others.
6. Assist patient in self-care when fatigue, lethargy, nausea, vomiting, and other symptoms prevent independence.	6. Physical well-being improves self-esteem.	• Explores alternative ways of expressing concern and affection.
7. Assist patient in selecting and using cosmetics, scarves, hair pieces, and clothing that increase his or her sense of attractiveness.	7. Promotes positive body image.	
8. Encourage patient and partner to share concerns about altered sexuality and sexual function and to explore alternatives to their usual sexual expression.	8. Provides opportunity for expressing concern, affection, and acceptance.	
9. Refer to collaborating specialists as needed.	9. Interdisciplinary collaboration essential in meeting patient needs.	

COLLABORATIVE PROBLEM: Potential complication: risk for bleeding problems
GOAL: Prevention of bleeding

Nursing Interventions	Rationale	Expected Outcomes
1. Assess for potential for bleeding: monitor platelet count.	1. Mild risk: 50,000–100,000/mm^3 (0.05–0.1 $\times$ 10^{12}/L) Moderate risk: 20,000–50,000/mm^3 (0.02–0.05 $\times$ 10^{12}/L) Severe risk: less than 20,000/mm^3 (0.02 $\times$ 10^{12}/L)	• Signs and symptoms of bleeding are identified. • Exhibits no blood in feces, urine, or emesis. • Exhibits no bleeding of gums or of injection or venipuncture sites. • Exhibits no ecchymosis (bruising). • Patient and family identify ways to prevent bleeding. • Uses recommended measures to reduce risk of bleeding (uses soft toothbrush, shaves with electric razor only). • Exhibits normal vital signs. • Reports that environmental hazards have been reduced or removed. • Consumes adequate fluid. • Reports absence of constipation. • Avoids substances interfering with clotting. • Absence of tissue destruction.
2. Assess for bleeding: a. Petechiae or ecchymosis b. Decrease in hemoglobin or hematocrit c. Prolonged bleeding from invasive procedures, venipunctures, minor cuts or scratches d. Frank or occult blood in any body excretion, emesis, sputum e. Bleeding from any body orifice f. Altered mental status	2. Early detection promotes early intervention. a. Indicates injury to microcirculation and larger vessels. b–e. Indicates blood loss. f. Indicates neurologic involvement.	

Continued on following page

CHART 16-7 PLAN OF NURSING CARE
The Patient With Cancer (Continued)

Nursing Interventions	Rationale	Expected Outcomes
3. Instruct patient and family about ways to minimize bleeding: a. Use soft toothbrush or toothette for mouth care. b. Avoid commercial mouthwashes. c. Use electric razor for shaving. d. Use emery board for nail care. e. Avoid foods that are difficult to chew.	3. Patient can participate in self-protection. a. Prevents trauma to oral tissues. b. Contain high alcohol content that will dry oral tissues. c. Prevents trauma to skin. d. Reduces risk of trauma to nailbeds. e. Prevents oral tissue trauma.	• Exhibits normal mental status and absence of signs of intracranial bleeding. • Avoids medications that interfere with clotting (eg, aspirin). • Absence of epistaxis and cerebral bleeding.
4. Initiate measures to minimize bleeding. a. Draw all blood for lab work with one daily venipuncture. b. Avoid taking temperature rectally or administering suppositories and enemas. c. Avoid intramuscular injections; use smallest needle possible. d. Apply direct pressure to injection and venipuncture sites for at least 5 minores. e. Lubricate lips with petrolatum. f. Avoid bladder catheterizations; use smallest catheter if catheterization is necessary. g. Maintain fluid intake of at least 3 L per 24 hours unless contraindicated. h. Use stool softeners or increase bulk in diet. i. Avoid medications that will interfere with clotting (eg, aspirin). j. Recommend use of water-based lubricant before sexual intercourse.	4. Preserves circulating blood volume. a. Minimizes trauma and blood loss. b. Prevents trauma to rectal mucosa. c. Prevents intramuscular bleeding. d. Minimizes blood loss. e. Prevents skin from drying. f. Prevents trauma to urethra. g. Hydration helps to prevent skin drying. h. Prevents constipation and straining that may injure rectal tissue. i. Minimizes risk of bleeding. j. Prevents friction and tissue trauma.	
5. When platelet count is less than 20,000/mm^3, institute the following: a. Bed rest with padded side rails. b. Avoidance of strenuous activity. c. Platelet transfusions as prescribed; administer prescribed diphenhydramine hydrochloride (Benadryl) or hydrocortisone sodium succinate (Solu-Cortef) to prevent reaction to platelet transfusion. d. Supervise activity when out of bed. e. Caution against forceful nose blowing.	5. Platelet count of less than 20,000/mm^3 (0.02×10^{12}/L) is associated with increased risk of spontaneous bleeding. a. Reduces risk of injury. b. Increases intracranial pressure and risk of cerebral hemorrhage. c. Allergic reactions to blood products are associated with antigen–antibody reaction that causes platelet destruction. d. Reduces risk of falls. e. Prevents trauma to nasal mucosa and increased intracranial pressure.	

to 14 days after patients receive certain chemotherapeutic agents, such as doxorubicin and 5-fluorouracil, and BRMs, such as IL-2 and IFN. As many as 40% of patients receiving chemotherapy experience some degree of stomatitis during treatment. Patients receiving high-dose chemotherapy, such as those patients undergoing stem cell transplant, are at increased risk. Stomatitis may also occur after radiation treatments to the head and neck. Oropharyngeal mucositis may be worse in patients with head and neck cancers who receive combined modality therapy of both radiation and chemotherapy (Cady, 2007).

As a result of normal everyday wear and tear, the epithelial cells that line the oral cavity undergo rapid turnover and slough off routinely. Both chemotherapy and radiation lead to the destruction of cells in the oral cavity (Sonis, 2004). This initiates the inflammatory process, leading to further tissue damage and ulceration of oral tissues. Normal flora invade the ulcerations and cause additional damage. Poor oral hygiene, existing dental disease, use of other medications that dry mucous membranes, advanced age, smoking, previous cancer treatment, diminished renal function, and impaired nutritional status further contribute to the severity of stomatitis. Radiation-induced xerostomia (dry mouth) associated with decreased function of the salivary glands may contribute to stomatitis in patients who have received radiation to the head and neck.

Myelosuppression (bone marrow depression), resulting from underlying disease or its treatment, predisposes the patient to oral bleeding and infection. Severe oral pain can significantly affect swallowing, nutritional intake, speech, and a willingness to maintain oral hygiene. As a result of the ability to give higher doses of chemotherapy due to improvements in managing neutropenia with growth factors, stomatitis is a common reason for treatment delays and dose reductions (Cawley & Benson, 2005). Advanced stomatitis may cause or prolong hospitalizations, significantly reduce the patient's quality of life, and ultimately lead to poor patient outcomes (Eilers & Million, 2007).

There is no standard of practice for the assessment of stomatitis. It is important for nurses to assess the oropharyngeal cavity of patients prior to, throughout the course of, and after treatment. It is important that the same tool or method of assessment is used by all clinicians involved in the patient's care. Nursing assessment begins with understanding the patient's usual practices for oral hygiene and identification of individuals at risk for stomatitis. Assessment of the patient's subjective experience and an objective assessment of the oropharyngeal tissues and teeth are important. The patient is also assessed for dehydration, infection, pain, and nutritional impairment resulting from stomatitis.

Optimal, evidence-based prevention and treatment approaches have not yet been identified (Eilers & Million, 2007). Ongoing studies are addressing the inflammation and release of chemical substances that lead to stomatitis. At this time, most clinicians agree that good oral hygiene, including brushing, flossing, and rinsing, is necessary to minimize the risk of oral complications associated with cancer therapies.

Palifermin (Kepivance), a synthetic form of human keratinocyte growth factor, is an IV medication approved by the FDA for treatment of oral mucositis in patients with hematologic cancer who are undergoing chemotherapy and radiation prior to hematopoietic stem cell support. Palifermin promotes epithelial cell repair and more rapid replacement of cells in the mouth and gastrointestinal tract (Oncology Nursing Society, 2006). It is not yet been approved for use in patients with other types of cancer. Careful timing of administration and monitoring are essential for maximum effectiveness and to detect adverse effects.

Radiation Associated Skin Impairment

Although advances in radiation therapy have resulted in decreased incidence and severity of skin impairments, patients may still develop skin reactions that lead to pain, irritation, pruritis, burning, and diminished quality of life. Nursing care for patients with skin reactions includes maintaining skin integrity, cleansing the skin, promoting comfort, reducing pain, preventing additional trauma, and preventing and managing infection (McQuestion, 2006). Although a variety of methods and products are used in clinical practice for patients with radiation-induced skin reactions, there is limited evidence to support their value. Patients with skin and tissue reactions to radiation therapy require careful skin care to prevent further skin irritation, drying, and damage, as discussed in the nursing care plan (see Chart 16-7), under Impaired skin integrity: erythematous and wet desquamation reactions to radiation therapy.

Alopecia

The temporary or permanent thinning or complete loss of hair is a potential adverse effect of radiation therapy to the brain and various chemotherapeutic agents. Alopecia usually begins 2 to 3 weeks after the initiation of treatment; regrowth usually begins within 8 weeks after the last treatment. Some patients who undergo radiation to the head may sustain permanent hair loss. Although health care providers may view hair loss as a minor issue, for many patients it is a major assault on body image, resulting in challenges to self-esteem, depression, anxiety, anger, rejection, and isolation. In some cases, patients may initially refuse treatment due to fears regarding hair loss (Nolte, Donnelly, Kelly, et al., 2006). To patients and families, hair loss can serve as a constant reminder of the challenges cancer places on their coping abilities, interpersonal relationships, and sexuality.

Although few studies have addressed methods to minimize the impact of alopecia, nurses provide information about hair loss and support the patient and family in coping with changes in body image, as discussed in the nursing care plan (see Chart 16-7) under Impaired tissue integrity: alopecia.

Malignant Skin Lesions

Skin lesions may occur with local extension or metastasis of the tumor into the epithelium and its surrounding lymph and blood vessels. Either locally invasive or metastatic cancer to the skin may result in redness (erythema), discolored nodules, or progression to wounds involving edema, exudates, and tissue necrosis. The most extensive lesions may ulcerate with an overgrowth of micro-organisms that result in a very distressing malodor. These lesions are a source of considerable pain, discomfort, and embarrassment.

Ulcerating skin lesions usually indicate widely disseminated disease that is unlikely to be eradicated. Managing these lesions becomes a nursing priority. Nurses carefully assess malignant skin lesions for the size, appearance, condition of the surrounding tissue, odor, bleeding, drainage, and associated pain or other symptoms including evidence of infection. The potential for serious complications such as hemorrhage, vessel compression/obstruction, or airway obstruction should be noted so that the caregiver can be instructed in palliative measures to maintain patient comfort (Seaman, 2006). Since this type of lesion is associated with advanced disease, the nurse assesses the wound for progression over time.

Nursing care also includes cleansing the skin, reducing superficial bacteria, controlling bleeding, reducing odor, protecting the skin from further trauma, and relieving pain. The patient and family require emotional support, assistance, and guidance to care for these skin lesions and to address comfort measures at home. Referral for home care is indicated.

Promoting Nutrition

Nutritional Problems

Most patients with cancer experience some weight loss during their illness. Anorexia, malabsorption, and cachexia are common examples of nutritional problems. Impaired nutritional status may contribute to both physical and psychosocial consequences (Chart 16-8). Nutritional concerns include decreased protein and caloric intake, metabolic or mechanical effects of the cancer, systemic disease, side effects of the treatment, or the patient's emotional status.

Anorexia

Among the many causes of anorexia in patients with cancer are alterations in taste, manifested by increased salty, sour, and metallic taste sensations, and altered responses to sweet and bitter flavors. Taste changes contribute to decreased appetite and nutritional intake and protein–calorie malnutrition. Taste alterations may result from mineral (eg, zinc) deficiencies, increases in circulating amino acids and cellular metabolites, or the administration of chemotherapeutic agents. Patients undergoing radiation therapy to the head and neck may experience "mouth blindness," which is a severe impairment of taste.

Chart 16-8 • *Potential Consequences of Impaired Nutrition in Patients With Cancer*

- Decreased survival
- Immune incompetence
- Anemia
- Increased incidence of infection
- Delayed tissue and wound healing
- Fatigue
- Diminished functional ability
- Decreased capacity to continue antineoplastic therapy
- Increased hospital admissions
- Increased length of hospital stay
- Impaired psychosocial functioning

Anorexia may occur because people feel full after eating only a small amount of food. This sense of fullness occurs secondary to a decrease in digestive enzymes, abnormalities in the metabolism of glucose and triglycerides, and prolonged stimulation of gastric volume receptors, which convey the feeling of being full. Psychological distress (eg, fear, pain, depression, and isolation) throughout illness may also have a negative impact on appetite. Patients may develop an aversion to food because of nausea and vomiting associated with treatment.

Malabsorption

Many patients with cancer are unable to absorb nutrients from the gastrointestinal system as a result of tumor activity and cancer treatment. Tumors can affect the gastrointestinal activity in several ways. They may impair enzyme production or produce fistulas. Some tumors secrete hormones and enzymes, such as gastrin, that lead to increased gastrointestinal irritation, peptic ulcer disease, and decreased fat digestion. Tumors may interfere with protein digestion.

Chemotherapy and radiation may irritate and damage mucosal cells of the bowel, inhibiting absorption. Radiation therapy has been associated with sclerosis of the blood vessels in the bowel and fibrotic changes in the gastrointestinal tissue. Surgical intervention may change peristaltic patterns, alter gastrointestinal secretions, and reduce the absorptive surfaces of the gastrointestinal mucosa, all leading to malabsorption.

Cachexia

Cachexia is common in patients with cancer, especially in advanced disease. Cancer cachexia is related to inadequate nutritional intake, along with increasing metabolic demand, increased energy expenditure due to anaerobic metabolism of the tumor, impaired glucose metabolism, competition of the tumor cells for nutrients, altered lipid metabolism, and a suppressed appetite. In addition, current literature suggests that cachexia in cancer may be related to a cytokine-induced inflammatory response (Tchekmedyian, 2006). Cachexia is characterized by loss of body weight, adipose tissue, visceral protein, and skeletal muscle. Patients with cachexia complain of loss of appetite, early satiety, and fatigue. As a result of protein losses, patients develop anemia and peripheral edema.

Nurses assess patients who are at risk of altered nutritional intake so that appropriate measures may be instituted prior to nutritional decline (Cady, 2007).

General Nutritional Considerations

Assessment of the patient's nutritional status is conducted at diagnosis and throughout the course of treatment and the disease process. The patient's weight and caloric intake are monitored closely. Diet history, episodes of anorexia, changes in appetite, situations and foods that aggravate or relieve anorexia, and medication history are assessed. Difficulty in chewing or swallowing and the presence of nausea, vomiting, or diarrhea are noted.

Clinical and laboratory data useful in assessing nutritional status include anthropometric measurements (triceps skin fold and middle-upper arm circumference), serum protein levels (albumin and transferrin), serum electrolytes,

lymphocyte count, skin response to intradermal injection of antigens, hemoglobin levels, hematocrit, urinary creatinine levels, and serum iron levels. Whenever possible, every effort is made to maintain adequate nutrition through the oral route. Prokinetic agents such as metoclopramide are used in some settings to increase gastric emptying in patients with early satiety and delayed gastric emptying.

If adequate nutrition cannot be maintained by oral intake, nutritional support via the enteral route may be necessary. Patients with head and neck cancers who receive radiation therapy or some combination of surgery, radiation, and chemotherapy are at particularly high risk for impaired oral intake and inadequate fluid and nutritional status. Increasingly, patients at risk for significantly impaired nutrition have prophylactic percutaneous endoscopic gastrostomy (PEG) tubes inserted prior to initiation of antineoplastic treatment and the onset of weight loss and other consequences of limited oral intake (Cady, 2007). When needed, the patient and family are taught to administer enteral nutrition in the home.

If malabsorption is a problem, enzyme and vitamin replacement may be instituted. Additional strategies include changing the feeding schedule, using simple diets, and relieving diarrhea. If malabsorption is severe, parenteral nutrition may be necessary. However, patients with advanced end-stage cancer, who have a life expectancy of less than 3 months, are usually not considered to be candidates for parenteral nutrition (Mirhosseini, Fainsinger & Baracos, 2005). Parenteral nutrition can be administered in several ways: by a long-term venous access device (eg, right atrial catheter), by an implanted venous port, or by a PICC (Fig. 16-6). The nurse teaches the patient and family to care for the venous access device and to administer parenteral nutrition. Home care nurses may assist with or supervise parenteral nutrition administration in the home.

Interventions to reduce cachexia usually do not prolong survival or improve nutritional status significantly. Before invasive nutritional strategies are instituted, the nurse should assess the patient carefully and discuss options with the patient and family. Creative dietary therapies, enteral (tube) feedings, or parenteral nutrition may be necessary to ensure adequate nutrition. Care is also directed toward preventing trauma, infection, and other complications that increase metabolic demands.

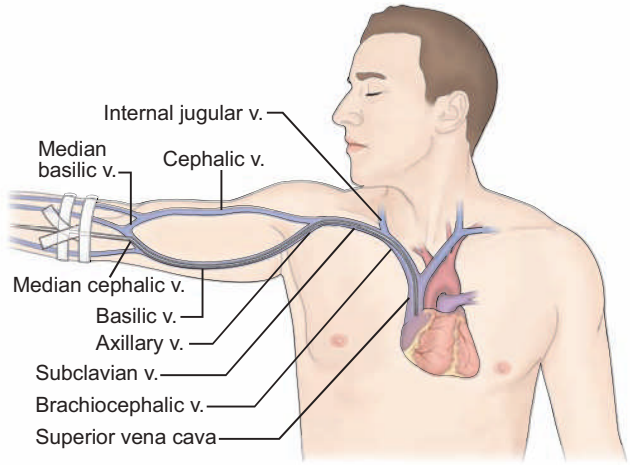

Figure 16-6 A peripherally inserted central catheter (PICC) is advanced through the cephalic or basilic vein to the axillary, subclavian, or brachiocephalic vein or the superior vena cava.

Relieving Pain

It is estimated that 90% to 95% of patients with progressive cancer experience pain (Stoneberg & von Gunten, 2006). Although the pain may be acute, it is more frequently characterized as chronic. (For more information on cancer-related pain, see Chapter 13.) As in other situations involving pain, the experience of cancer pain is influenced by physical, psychosocial, cultural, and spiritual factors.

Cancer can cause pain in various ways (Table 16-7). Pain related to the underlying cancer process accounts for the pain experienced by 75% of all patients with cancer (Abrahm, 2005). Pain is also associated with various cancer treatments. Acute pain is linked with trauma from surgery. Occasionally, chronic pain syndromes, such as postsurgical neuropathies (pain related to nerve tissue injury), occur. Some chemotherapeutic agents cause tissue necrosis, peripheral neuropathies, and stomatitis—all potential sources of pain—whereas radiation therapy can cause pain secondary to skin or organ inflammation. Cancer patients may have other sources of pain, such as arthritis or migraine headaches, that are unrelated to the underlying cancer or its treatment.

The nurse assesses the patient for the source and site of pain as well as those factors that increase the patient's perception of pain, such as fear and apprehension, fatigue, anger, and social isolation. Pain assessment scales (see Chapter 13) are useful for assessing the patient's pain before pain-relieving interventions are instituted and for evaluating the effectiveness of these interventions. Other symptoms that contribute to the pain experience, such as nausea and fatigue, are assessed as well.

Table 16-7 EXAMPLES OF SOURCES OF CANCER PAIN

Source	Descriptions	Underlying Cancer
Bone metastasis	Throbbing, aching	Breast, prostate, myeloma
Nerve compression, infiltration	Burning, sharp, tingling	Breast, prostate, lymphoma
Lymphatic or venous obstruction	Dull, aching, tightness	Lymphoma, breast, Kaposi's sarcoma
Ischemia	Sharp, throbbing	Kaposi's sarcoma
Organ obstruction	Dull, crampy, gnawing	Colon, gastric
Organ infiltration	Distention, crampy	Liver, pancreatic
Skin inflammation, ulceration, infection, necrosis	Burning, sharp	Breast, head and neck, Kaposi's sarcoma

In today's society, most people expect pain to disappear or resolve quickly, and in fact it usually does. Although it is often controllable, advanced cancer pain is commonly irreversible and not quickly resolved. For many patients, pain is often seen as a signal that the tumor is growing and that death is approaching. As patients anticipate the pain and their anxiety increases, pain perception heightens, producing fear and further pain. Chronic cancer pain, then, can lead to a cycle progressing from pain to anxiety to fear and back to pain, especially when the pain is not adequately managed. Inadequate pain management is most often the result of misconceptions and insufficient knowledge about pain assessment and pharmacologic interventions on the part of patients, families, and health care providers (Xue, Schulman-Green, Czaplinski, et al., 2007). Chapter 13 provides information concerning factors contributing to the pain experience, pain perception, and tolerance as well as pharmacologic and nonpharmacologic nursing interventions addressing pain. The nursing care plan (see Chart 16-7) also provides strategies for nursing assessment and management.

Inadequate pain management is most often the result of misconceptions and insufficient knowledge about pain assessment and pharmacologic interventions on the part of patients, families, and health care providers (Xue, et al., 2007).

The World Health Organization advocates a three-step approach to treat cancer pain (Fig. 16-7). Analgesics are administered based on the patient's level of pain. A cancer pain algorithm, developed as a set of analgesic guiding principles, is given in Figure 16-8.

No reasonable pharmacologic and nonpharmacologic approaches, even those that may be invasive, should be overlooked because of a poor or terminal prognosis. The nurse helps the patient and family take an active role in managing pain. The nurse provides education and support to correct fears and misconceptions about opioid use. Inadequate pain control leads to a diminished quality of life characterized by suffering, anxiety, fear, immobility, isolation, and depression. Improving the patient's quality of life through palliative care is as important as preventing a painful death.

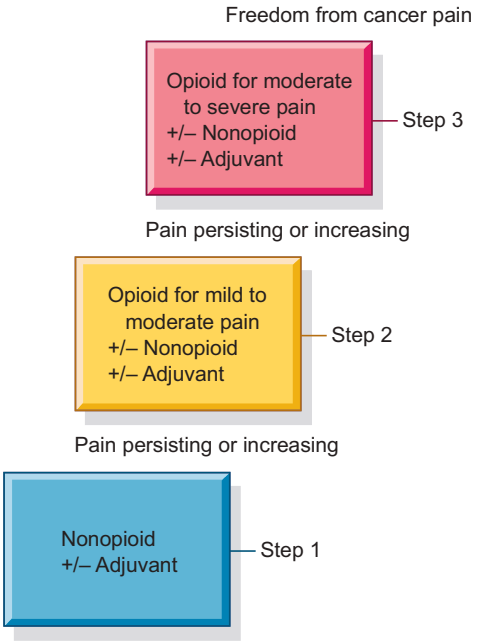

Figure 16-7 Adapted from the World Health Organization three-step ladder approach to relieving cancer pain. Various opioid (narcotic) and nonopioid medications may be combined with other medications to control pain.

Decreasing Fatigue

Fatigue is one of the most significant and frequent symptoms experienced by patients receiving cancer therapy. Fatigue also results from the stress of coping with cancer. It does not always signify that the cancer is advancing or that the treatment is failing. Potential factors contributing to the experience of fatigue are summarized in Chart 16-9.

In assessing fatigue the nurse distinguishes between acute fatigue, which occurs after an energy-demanding experience, and cancer-related fatigue, which is defined as "a distressing persistent, subjective sense of tiredness or exhaustion related to cancer or cancer treatment that is not proportional to recent activity and interferes with usual functioning" (National Comprehensive Cancer Network, 2006b). Acute fatigue serves a protective function, whereas cancer-related fatigue does not.

Fatigue is distressing for patients with cancer who are receiving treatment, for survivors, and for those in the late stages of disease (Mitchell, et al., 2007). Although patients may describe fatigue in a variety of ways, nurses assess for feelings of weariness, weakness, lack of energy, inability to carry out necessary and valued daily functions, lack of motivation, and inability to concentrate. Several assessment tools, such as a simple visual analogue scale, may be used to assess levels of fatigue (Madden & Newton, 2006). The nurse assesses physiologic and psychological stressors that can contribute to fatigue, including anemia, electrolyte imbalances, organ dysfunction, pain, nausea, dyspnea, constipation, fear, and anxiety.

The role of exercise as a helpful intervention has been supported by several controlled trials (Mitchell, et al., 2007; Young-McCaughan & Arzola, 2007). The nurse assists patients with additional nonpharmacologic strategies to minimize fatigue or help the patient cope with existing fatigue as described in the nursing care plan (see Chart 16-7) under Fatigue. Occasionally pharmacologic interventions are utilized, including antidepressants for patients with depression; anxiolytics for those with anxiety; hypnotics for patients with sleep disturbances; and psychostimulants for some patients with advanced cancer or fatigue that does not respond to other interventions (Abrahm, 2005).

The nurse assists patients with nonpharmacologic strategies to minimize fatigue or help the patient cope with existing fatigue.

Improving Body Image and Self-Esteem

The nurse identifies potential threats to the patient's body image and assesses the patient's ability to cope with the many assaults to body image experienced throughout the course of disease and treatment. Entry into the health care system is often accompanied by depersonalization. Threats to self-concept occur as the patient faces the realization of illness, disfigurement, possible disability, and death. To accommodate treatments or because of the disease, many patients with cancer are forced to alter their lifestyles. Priorities and values change when body image is threatened.

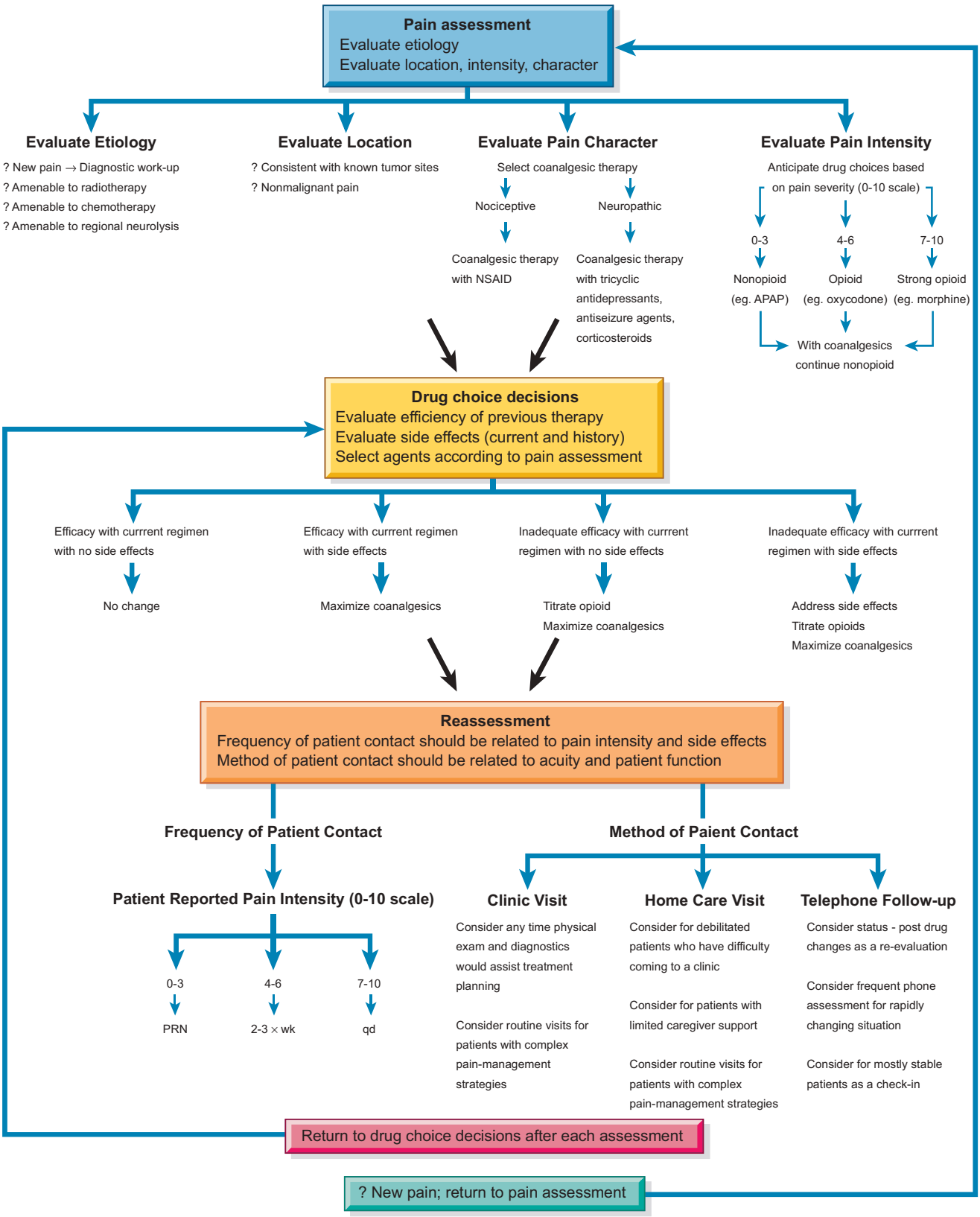

Figure 16-8 The cancer pain algorithm (highest-level view) is a decision-tree model for pain treatment that was developed as an interpretation of the AHCPR Guideline for Cancer Pain, 1994. Redrawn with permission from DuPen, A. R., DuPen, S., Hansberry, J., et al. (2000). An educational implementation of a cancer pain algorithm for ambulatory care. *Pain Management Nursing, 1*(4), 118.

Chart 16-9 • *Sources of Fatigue in Cancer Patients*

- Pain, pruritus
- Imbalanced nutrition related to anorexia, nausea, vomiting, cachexia
- Electrolyte imbalance related to vomiting, diarrhea
- Ineffective protection related to neutropenia, thrombocytopenia, anemia
- Impaired tissue integrity related to stomatitis, mucositis
- Impaired physical mobility related to neurologic impairments, surgery, bone metastasis, pain, and analgesic use
- Uncertainty and deficient knowledge related to disease process, treatment
- Anxiety related to fear, diagnosis, role changes, uncertainty of future
- Ineffective breathing patterns related to cough, shortness of breath, and dyspnea
- Disturbed sleep pattern related to cancer therapies, anxiety, and pain

Disfiguring surgery, hair loss, cachexia, skin changes, altered communication patterns, and sexual dysfunction treatment can threaten the patient's self-esteem and body image.

A creative and positive approach is essential when caring for patients with altered body image. Nursing approaches for addressing issues related to body image and self-esteem are and approaches for addressing issues related to body image and self-esteem are also included in the nursing care plan (see Chart 16-7). The nurse serves as a listener and counselor to both the patient and the family. Possible influences of the patient's culture and age are considered when discussing concerns and potential interventions (Romanek, McCaul & Sandgren, 2006).

As a result of the underlying cancer, treatments, and psychosocial responses to the experience, patients may experience a variety of sexuality-based issues. Patients who experience alterations in sexuality and sexual function are encouraged to discuss their concerns. Major barriers to addressing sexual dysfunction in patients with cancer include the lack of assessment tools and evidence-based interventions (Bakewell & Volker, 2005). In addition, nurses and other health care providers often fail to ask patients about sexual concerns, and patients may be hesitant to discuss them. However, standards of oncology nursing practice include the need for nurses to assess patients' sexuality and to help patients and their partners achieve the outcomes of importance to them (Wilmoth, 2006). Nurses who identify physiologic, psychological, or communication difficulties related to sexuality or sexual function are in a key position to help patients seek further specialized evaluation and intervention if necessary.

Assisting in the Grieving Process

Nurses assess the patient's psychological and mental status as the patient and family face this life-threatening experience, unpleasant diagnostic tests and treatment modalities, and progression of disease. The nurse assesses the patient's mood and emotional reaction to the results of diagnostic testing and prognosis and looks for evidence that the patient is progressing through the stages of grief and can talk about the diagnosis and prognosis with family members.

A cancer diagnosis need not indicate a fatal outcome. Many forms of cancer are curable, while others may be cured or controlled for long periods of time if treated early. Despite these facts, many patients and their families view cancer as a fatal disease that is inevitably accompanied by pain, suffering, debilitation, and emaciation. Grieving is a normal response to these fears and to actual or potential losses: loss of health, normal sensations, body image, social interaction, sexuality, and intimacy. Patients, families, and friends may grieve for the loss of quality time to spend with others, the loss of future and unfulfilled plans, and the loss of control over the patient's body and emotional reactions.

Patients and their families who have just been informed of a cancer diagnosis frequently respond with shock, numbness, and disbelief. It is often during this stage that the patient and family are called on to make important initial decisions about treatment. They require the support of physicians, nurses, and all members of the health care team to make these decisions. The nurse plays an important role in answering any questions the patient and family have and clarifying information provided by physicians. The plan of nursing care addresses anticipatory grieving and nursing strategies for promoting appropriate progression through the grieving process.

If the patient enters the terminal phase of disease, the nurse may realize that the patient and family members are at different stages of grief. In such cases, the nurse helps the patient and family to acknowledge and cope with their reactions and feelings. The nurse also helps the patient and family to explore preferences for issues related to end-of-life care, such as withdrawal of active disease treatment, desire for the use of life-support measures, and symptom management. Support, which can be as simple as holding a patient's hand or just being with a patient at home or at the bedside, often contributes to peace of mind. After the death of a patient with cancer, maintaining contact with surviving family members may help them work through their feelings of loss and grief. See Chapter 17 for further discussion of end-of-life issues.

Monitoring and Managing Potential Complications

Infection

For patients in all stages of cancer, the nurse assesses factors that can promote infection. Although the infection-associated mortality rate has decreased, infection remains a major cause of morbidity and mortality in patients with cancer (Zitella, Friese, Hauser, et al., 2006). Factors predisposing patients to infection are summarized in Table 16-8. Often, more than one predisposing factor is present in patients with cancer (Friese, 2007). The nurse monitors laboratory studies to detect early changes in WBC counts. Common sites of infection, such as the pharynx, skin, perianal area, urinary tract, and respiratory tract, are assessed on a regular basis. However, the typical signs of infection (swelling, redness, drainage, and pain) may not occur in immunosuppressed patients because of decreased circulating white blood cells (WBC) (the cause purulent drainage) and a diminished local inflammatory response. Fever may be the only sign of infection (Marrs, 2006). The nurse monitors the patient for sepsis, particularly if invasive catheters or infusion lines are in place.

Table 16-8 FACTORS PREDISPOSING CANCER PATIENTS TO INFECTION

Factors	Underlying Mechanisms
Impaired skin and mucous membrane integrity	Loss of body's first line of defense against invading organisms.
Chemotherapy	Chemotherapy agents that cause mucositis impair skin and mucous membrane integrity. Organ damage associated with certain agents may also predispose patients to infection. Organ damage such as pulmonary fibrosis or cardiomyopathy that is associated with certain agents may also predispose patients to infection.
Radiation therapy	Radiation involving sites of bone marrow production may result in bone marrow suppression. May also lead to impaired tissue integrity.
Biologic response modifiers	Some biologic response modifiers may cause bone marrow suppression and organ dysfunction.
Malignancy	Malignant cells may infiltrate the bone marrow and interfere with production of white blood cells and lymphocytes. Hematologic malignancies (leukemias and lymphomas) are associated with impaired function and production of blood cells.
Malnutrition	Malnutrition results in impaired production and function of cells of the immune response. It may contribute to impaired skin integrity.
Medications	Antibiotics disturb the balance of normal flora, allowing them to become pathogenic. This process occurs most commonly in the gastrointestinal tract. Corticosteroids and nonsteroidal anti-inflammatory drugs (NSAIDs) mask inflammatory responses.
Urinary catheter	The catheter creates a port and mechanism of entry for organisms.
Intravenous catheter	The catheter is a site of entry for organisms.
Other invasive procedures (eg, surgery, paracentesis, thoracentesis, drainage tubes, endoscopy, mechanical ventilation)	These procedures create a port of entry and possible introduction of exogenous organisms into the system.
Contaminated equipment	Stagnant water in oxygen equipment is associated with growth of microorganisms.
Age	Increasing age is associated with declining organ function and decreased production and functioning of the cells of the immune system.
Chronic illness	Chronic illness is associated with impaired organ function and altered immune responses.
Prior infections	Recent infection may be associated with depleted immune responses and debilitation; prior infections may not have completely resolved; previous use of antibiotics may have altered normal flora allowing flora to become pathogenic.
Recent travel	Travel, especially to less developed areas, may have lead to exposure to microbial infection and disease.
Pet excreta	Exposure to pet excreta, especially in the immunocompromised patient, may be associated with infections such as *Toxoplasma gondii* (toxoplasmosis).
Prolonged hospitalization	Hospitalization allows increased exposure to nosocomial infection and colonization by new organisms.

WBC function is often impaired in patients with cancer. There are five types of WBCs: neutrophils (granulocytes), lymphocytes, monocytes, basophils, and eosinophils. Neutrophils, which comprise 60% to 70% of all the body's WBCs, serve as the body's primary defense against invading organisms by engulfing and destroying infective organisms through phagocytosis. Both the total WBC count and the concentration of neutrophils are important in determining the patient's ability to fight infection. A decrease in circulating WBCs is referred to as leukopenia. Granulocytopenia is a decrease in neutrophils.

A differential WBC count identifies the relative numbers of WBCs and permits tabulation of polymorphonuclear neutrophils or segmented neutrophils (mature neutrophils, reported as "polys," PMNs, or "segs") and immature forms of neutrophils (reported as bands, metamyelocytes, and "stabs"). The absolute neutrophil count (ANC) is calculated by the following formula:

$$\text{ANC} = \frac{(\text{Total WBC}) \times [\%\ \text{segmented neutrophils} + \text{bands}]}{100}$$

For example, if the total WBC count is 6000 cells/mm^3, with segmented neutrophils 25% and bands 25%, the ANC is 3000 cells/mm^3.

Neutropenia, an abnormally low ANC, is associated with an increased risk for infection. The risk for infection rises as the ANC decreases. As the ANC declines below 1500 cells/mm^3, the risk for infection increases. An ANC less than 500 cells/ mm^3 reflects a severe risk of infection (Marrs, 2006). **Nadir** is the lowest ANC after myelosuppressive chemotherapy or radiation therapy that suppresses bone marrow function. Febrile patients who are neutropenic are assessed for factors that increase the risk for infection and for sources of infection through cultures of blood, sputum, urine, stool, IV or urinary catheters, and wounds, if appropriate. In addition, a chest x-ray is often obtained to assess for pulmonary infections.

Defense against infection is compromised in many different ways. The integrity of the skin and mucous membranes is challenged by multiple invasive diagnostic and therapeutic procedures, by adverse effects of radiation and chemotherapy, and by the detrimental effects of immobility.

Impaired nutrition as a result of anorexia, nausea, vomiting, diarrhea, and the underlying disease alters the body's ability to combat invading organisms. Medications such as antibiotics disturb the balance of normal flora, allowing the overgrowth of normal flora and pathogenic organisms. Other medications can also alter the immune response (see Chapter 50). Cancer itself may lead to defects in cellular and humoral immunity. Advanced cancer can cause obstruction of the hollow viscera (eg, intestines), blood vessels, and lymphatic vessels, creating a favorable environment for proliferation of pathogenic organisms. In some patients, tumor cells infiltrate bone marrow

and prevent normal production of WBCs. However, most often, a decrease in WBCs is a result of bone marrow suppression after chemotherapy or radiation therapy. Severe neutropenia may necessitate delays in administration of myelosuppressive therapies or treatment dose adjustments, although the use of the hematopoietic growth factors, also called colony-stimulating factors (see previous discussion), has reduced the severity and duration of neutropenia associated with myelosuppressive chemotherapy and radiation therapy. The administration of these factors assists in reducing the risk for infection and, possibly, in maintaining treatment schedules, drug dosages, treatment effectiveness, and quality of life.

Nurses are in a key position to assist in preventing and identifying symptoms of infection, as discussed in the nursing care plan (see Chart 16-7). Although multiple infection control practices are employed, there is a significant lack of evidence to support many of them (Zitella, et al., 2006). Clinical practice guidelines developed by the Oncology Nursing Society (ONS), the Infusion Nurses Society (INS), the National Comprehensive Cancer Network (NCCN), and the American Society of Clinical Oncology (ASCO) are used to guide interventions. Interventions to prevent infection and education formats to teach patients and families about infection are high research priorities.

Gram-positive bacteria (*Streptococcus*, enterococci, and *Staphylococcus* species) and gram-negative organisms (*Escherichia coli, Klebsiella pneumoniae,* and *Pseudomonas aeruginosa*) are the most frequently isolated causes of infection. Fungal organisms, such as *Candida albicans,* also contribute to the incidence of serious infection. Viral infections in immunocompromised patients are caused most often by herpes viruses and respiratory viruses.

Fever is probably the most important sign of infection in immunocompromised patients. In neutropenic patients, any one-time temperature of 38.3°C (101°F) or higher or 38.0°C (100.4°F) or higher for 1 hour or more is reported and dealt with promptly (NCCN, 2008c). Antibiotics may be prescribed to treat infections after cultures of wound drainage, exudates, sputum, urine, stool, or blood are obtained. Careful consideration is given to the underlying malignancy, prior antineoplastic treatment, absolute neutrophil count, comorbidities, and other patient-related factors prior to the identification of the most appropriate initial antibiotic therapy. The NCCN provides guidelines for prevention and treatment of cancer-related infections (NCCN, 2008c). Patients with neutropenia are treated with broad-spectrum antibiotics before the infecting organism is identified because of the high incidence of mortality associated with untreated infection. Broad-spectrum antibiotic therapy targets the most likely major pathogenic organisms. It is important for the nurse to administer these medications promptly and as scheduled to achieve adequate blood levels of the medications. Once the offending organism is identified, more specific antimicrobial therapy is prescribed if appropriate.

Septic Shock

The nurse assesses the patient frequently for infection and inflammation throughout the course of the disease. Septicemia and septic shock are life-threatening complications that must be prevented or detected and treated promptly. Although all patients with cancer are at risk, patients who are neutropenic or who have hematologic malignancies are at the greatest risk. Patients with signs and symptoms of impending sepsis and septic shock require immediate hospitalization and aggressive treatment in the intensive care setting.

Signs and symptoms of septic shock (see Chapter 15) include altered mental status, either subnormal or elevated temperature, cool and clammy skin, decreased urine output, hypotension, tachycardia, other dysrhythmias, electrolyte imbalances, tachypnea, and abnormal arterial blood gas values. Patients and family members are instructed about signs of septicemia, methods for preventing infection, and actions to take if signs and symptoms of infection or septicemia occur.

Although septic shock is most often associated with overwhelming gram-negative bacterial infections, there is an increasing incidence of gram-positive infections related to the use of long-term venous access devices. Patients with prolonged neutropenia or hematologic malignancies are also more susceptible to fungal and viral sources of sepsis as well. In a patient with impending shock, the nurse monitors temperature and hemodynamic and respiratory status on a frequent basis. Neurologic assessments are carried out to detect changes in orientation and responsiveness. Fluid and electrolyte status is monitored by measuring fluid intake and output and serum electrolytes. Arterial blood gas values and pulse oximetry are monitored to determine tissue oxygenation. Nurses administer IV fluids, blood products, vasopressor and inotropic agents as prescribed to maintain blood pressure and tissue perfusion, as well as broad-spectrum antibiotics, which may be prescribed initially to combat the underlying infection (see Chapter 15). Supplemental oxygen and mechanical ventilation are often necessary. Systemic steroids and drotrecogin alfa (Xigris; recombinant human activated protein C) have been used in some centers for patients who have severe and prolonged septic shock or those that at risk for development of clotting disturbances (Gobel & Peterson, 2006).

Bleeding and Hemorrhage

Platelets are essential for normal blood clotting and coagulation (hemostasis). Thrombocytopenia, a decrease in the circulating platelet count, is the most common cause of bleeding in patients with cancer and is usually defined as a platelet count of less than 100,000/mm^3 (0.1×10^{12}/L). When the platelet count decreases to between 20,000 and 50,000/mm^3 (0.02 to 0.05×10^{12}/L), the risk of bleeding increases. Platelet counts lower than 20,000/mm^3 (0.02×10^{12}/L) are associated with an increased risk for spontaneous bleeding, for which patients require a platelet transfusion.

Thrombocytopenia often results from bone marrow depression after certain types of chemotherapy and radiation therapy and with tumor infiltration of the bone marrow. In some cases, platelet destruction is associated with an enlarged spleen (hypersplenism) and abnormal antibody function, which occur with leukemia and lymphoma. The nursing care plan addresses nursing assessment parameters and interventions for patients at risk for bleeding (see Chart 16-7).

In limited circumstances, the nurse may administer IL-11, which has been approved by the FDA to prevent severe thrombocytopenia and to reduce the need for platelet transfusions after myelosuppressive chemotherapy in patients with nonmyeloid malignancies, as previously described. Additional

medications may be prescribed to address bleeding due to disorders of coagulation.

Promoting Home and Community-Based Care

Teaching Patients Self-Care

Increasingly, patients with cancer are diagnosed and treated in the outpatient setting with minimal or no period of hospitalization. Nurses in outpatient settings often have the responsibility for patient teaching and for coordinating care in the home. The shift of care from acute care to the home or outpatient setting places a great deal of the responsibility for care on the patient and family; this requires teaching that enables them to provide care. Teaching initially focuses on the most immediate care needs likely to be encountered at home.

Side effects of treatments and changes in the patient's status that should be reported are reviewed verbally and reinforced with written information. Strategies to deal with side effects of treatment are discussed with the patient and family. Other learning needs are based on the priorities conveyed by the patient and family as well as on the complexity of care required in the home.

Technologic advances allow home administration of chemotherapy, parenteral nutrition, blood products, parenteral antibiotics, and analgesics, as well as management of symptoms and care of vascular access devices. Although home care nurses provide care and support for patients receiving this type of care, patients and families need instruction and support to enable them to feel comfortable and proficient in managing these treatments at home. Follow-up visits and telephone calls from the nurse assist in identifying problems and are often reassuring, increasing the patient's and family's comfort in dealing with complex and new aspects of care. Continued contact facilitates evaluation of the patient's progress and assessment of the ongoing needs of the patient and family.

Continuing Care

Referral for home care is often indicated for patients with cancer. The responsibilities of the home care nurse include assessing the home environment, suggesting modifications in the home or in care to help the patient and family address the patient's physical needs, providing physical care, and assessing the psychological and emotional impact of the illness on the patient and family.

Assessing changes in the patient's physical status and reporting relevant changes to the physician help ensure that appropriate and timely modifications in therapy are made. The home care nurse also assesses the adequacy of pain management and the effectiveness of other strategies to prevent or manage the side effects of treatment modalities and disease progression.

It is necessary to assess the patient's and family's understanding of the treatment plan and management strategies and to reinforce previous teaching. The nurse facilitates coordination of patient care by maintaining close communication with all involved health care providers. The nurse may make referrals and coordinate available community resources (eg, local office of the American Cancer Society, home aides, church groups, parish nurses, support groups) to assist patients and caregivers.

Gerontologic Considerations

More than 60% of all new cancers occur in people older than 65 years of age, and about 70% of all cancer deaths occur in people 65 years of age and older (Lynch, Marcone & Kagan, 2007). The rising numbers of individuals over age 65 with cancer has led to the emergence of geriatric oncology, a multidimensional and multidisciplinary approach to treating growing numbers of older adults with cancer (Lynch, et al., 2007).

Nurses working with the elderly population must understand the normal physiologic changes that occur with aging and the implications for the patient with cancer (Table 16-9). These changes that affect all body systems may ultimately influence elderly patients' responses to cancer treatment (Lichtman, 2006). In addition, many elderly patients have other chronic diseases requiring multiple medications. The existence of comorbidities and multiple medications may contribute to drug interactions and toxicities in elderly patients (Extermann & Hurria, 2007).

The understanding of the effects and tolerance of chemotherapy, biotherapy, and radiation in the elderly is limited because there have been few studies of the effects of cancer treatments in this population (Lichtman, 2006). In addition, the elderly have been underrepresented in oncology clinical trials (Lichtman, Wildiers, Chatelut, et al., 2007). Potential chemotherapy-related toxicities, such as renal impairment, myelosuppression, fatigue, and cardiomyopathy, may increase as a result of declining organ function and diminished physiologic reserves. The recovery of normal tissues after radiation therapy may be delayed, and older patients may experience more severe adverse effects, such as mucositis, nausea and vomiting, and myelosuppression. Because of impaired healing and declining pulmonary and cardiovascular functioning, older patients are slower to recover from surgery. Elderly patients are also at increased risk for complications such as atelectasis, pneumonia, and wound infections.

Several studies have shown that when compared to younger patients, some elderly patients with cancer have received substandard or suboptimal treatment (Bouchardy, Rapiti, Blagojevic, et al., 2007). Access to quality cancer care for elderly patients may be limited by discriminatory or fatalistic attitudes of health care providers, caregivers, and patients themselves. Issues such as the gradual loss of supportive resources, declining health or loss of a spouse, and unavailability of relatives or friends may result in limited access to care and unmet needs for assistance with activities of daily living. In addition, the economic impact of health care may be difficult for those living on fixed incomes.

It is not uncommon for elderly patients to delay reporting symptoms, attributing them to "old age." Many elderly people do not want to report illness for fear of losing their independence or financial security. Sensory losses (eg, hearing and visual losses) and memory deficits are considered when planning patient education, because they may affect the patient's ability to process and retain information. In such cases, the nurse acts as a patient advocate, encouraging independence and identifying resources for support when indicated. Nurses must be aware of the

Table 16-9 AGE-RELATED CHANGES AND THEIR EFFECTS ON PATIENTS WITH CANCER

Age-Related Changes	Implications
Impaired immune system	Use special precautions to avoid infection; monitor for atypical signs and symptoms of infection.
Altered drug absorption, distribution, metabolism, and elimination	Mandates careful calculation of chemotherapy and frequent assessment for drug response and side effects; dose adjustments may be necessary.
Increased prevalence of other chronic diseases	Monitor for effect of cancer or its treatment on patient's other chronic diseases; monitor patient's tolerance for cancer treatment; monitor for interactions with medications used to treat chronic diseases.
Diminished renal, respiratory, and cardiac reserve	Be proactive in prevention of decreased renal function, atelectasis, pneumonia, and cardiovascular compromise; monitor for side effects of cancer treatment.
Decreased skin and tissue integrity; reduction in body mass; delayed healing	Prevent pressure ulcers secondary to immobility; monitor skin and mucous membranes for changes related to radiation or chemotherapy; monitor nutritional status.
Decreased musculoskeletal strength	Prevent falls; assess support for performing activities of daily living in home setting; encourage safe use of assistive mobility devices.
Decreased neurosensory functioning: loss of vision, hearing, and distal extremity tactile senses	Provide teaching and instructions modified for patient's hearing and vision changes; provide instruction concerning safety and skin care for distal extremities; assess home for safety.
Altered social and economic resources	Assess for financial concerns, living conditions, and resources for support.
Potential changes in cognitive and emotional capacity	Provide teaching and support modified for patient's level of functioning and safety.

special needs of the aging population and work collaboratively with other disciplines to address identified needs.

Providing Care in Oncologic Emergencies

Table 16-10 discusses nursing and medical care of oncologic emergencies.

Providing Care of the Patient With Advanced Cancer

Patients with advanced cancer are likely to experience many of the problems previously described, but all to a greater degree. Symptoms of gastrointestinal disturbances, nutritional problems, weight loss, and cachexia make patients more susceptible to skin breakdown, fluid and electrolyte problems, and infection.

Although not all cancer patients experience pain, those who do commonly fear that it will not be adequately treated. Although treatment at this stage of illness is likely to be palliative rather than curative, prevention and appropriate management of problems can improve the patient's quality of life considerably. For example, use of analgesia at set intervals is recommended rather than on an "as needed" basis. Working with the patient and family as well as with other health care providers to manage pain frequently increases the patient's comfort and sense of control. Other medications (eg, sedatives, tranquilizers, muscle relaxants, antiemetics) are added to assist in promoting patient comfort.

If the patient is a candidate for radiation therapy or surgical intervention to relieve pain, the consequences of these procedures (eg, percutaneous nerve block, cordotomy) are explained to the patient and family. Measures are taken to prevent complications that result from altered sensation, immobility, and changes in bowel and bladder function.

With the appearance of each new symptom, patients may fear that the disease is progressing. However, one cannot assume that all symptoms are related to the cancer. The new symptoms are evaluated and treated aggressively if possible to increase the patient's comfort and improve quality of life.

Weakness, immobility, fatigue, and inactivity typically increase with advanced cancer as a result of the disease, treatment, inadequate nutritional intake, or dyspnea. The nurse works with the patient and family to set realistic goals and promote comfort. Measures include use of energy-conserving methods to accomplish tasks and activities that the patient values most.

Efforts are made to provide the patient with as much control and independence as desired but with assurance that support and assistance are available when needed. In addition, health care teams work with the patient and family to ascertain and comply with the patient's wishes about treatment methods and care as the terminal phase of illness and death approach.

Hospice

For many years, society was unable to cope appropriately with patients in the most advanced stages of cancer, and patients died in acute care settings rather than at home or in facilities designed to meet their needs. The needs of patients with terminal illnesses are best met by a comprehensive multidisciplinary specialty program that focuses on quality of life, palliation of symptoms, and provision of psychosocial and spiritual support for patients and families when cure and control of the disease are no longer possible. The concept of hospice best addresses these needs. Most important, the focus of care is on the family, as well as the patient. Hospice care can be provided in several settings: free-standing, hospital-based, and comm unity or home-based settings.

Table 16-10 ONCOLOGIC EMERGENCIES: MANIFESTATIONS AND MANAGEMENT

Emergency	Clinical Manifestations and Diagnostic Findings	Management
Superior Vena Cava Syndrome (SVCS) Compression or invasion of the superior vena cava by tumor, enlarged lymph nodes, intraluminal thrombus that obstructs venous circulation, or drainage of the head, neck, arms, and thorax. Typically associated with lung cancer, SVCS can also occur with breast cancer, Kaposi's sarcoma, thymoma, lymphoma, and mediastinal metastases (Kuzin, 2006). If untreated, SVCS may lead to cerebral anoxia (because not enough oxygen reaches the brain), laryngeal edema, bronchial obstruction, and death.	**Clinical** Gradually or suddenly impaired venous drainage giving rise to: Progressive shortness of breath (dyspnea), cough, hoarseness, chest pain, and facial swelling Edema of the neck, arms, hands, and thorax and reported sensation of skin tightness and difficulty swallowing Possibly engorged and distended jugular, temporal, and arm veins Dilated thoracic vessels causing prominent venous patterns on the chest wall Increased intracranial pressure, associated visual disturbances, headache, and altered mental status **Diagnostic** Diagnosis is confirmed by: Clinical findings Chest x-ray Thoracic computed tomography (CT) scan Thoracic magnetic resonance imaging (MRI) Intraluminal thrombosis is identified by venogram.	**Medical** • Radiation therapy to shrink tumor size and relieve symptoms • Chemotherapy for chemosensitive cancers (eg, lymphoma, small cell lung cancer) or when the mediastinum has been irradiated to maximum tolerance (Kuzin, 2006) • Anticoagulant or thrombolytic therapy for intraluminal thrombosis • Percutaneously placed intravascular stents are increasingly being used to reopen the occluded SVC (Kuzin, 2006) • Surgery (less common), such as vena cava bypass graft (synthetic or autologous), to redirect blood flow around the obstruction • Supportive measures such as oxygen therapy, corticosteroids, and diuretics **Nursing** • Identify patients at risk for SVCS • Monitor and report clinical manifestations of SVCS • Monitor cardiopulmonary and neurologic status • Avoid upper extremity venipuncture and blood pressure measurement • Facilitate breathing by positioning the patient properly; this helps to promote comfort and reduce anxiety produced by difficulty breathing resulting from progressive edema • Promote energy conservation to minimize shortness of breath • Monitor the patient's fluid volume status and administer fluids cautiously to minimize edema • Assess for thoracic radiation-related problems such as dysphagia (difficulty swallowing) and esophagitis • Monitor for chemotherapy-related problems, such as myelosuppression • Provide postoperative care as appropriate
Spinal Cord Compression Potentially leading to permanent neurologic impairment and associated morbidity and mortality; compression of the cord and its nerve roots may result from tumor, lymphomas, intervertebral collapse, or interruption of blood supply to the nerve tissues (Kaplan, 2006b). The prognosis depends on the severity and rapidity of onset. About 70% of compressions occur at the thoracic level, 20% in the lumbosacral level, and 10% in the cervical region (Marrs, 2006). Metastasis from breast, lung, kidney, prostate cancers, myeloma, lymphoma to the bone or between the bone and the epidural space are associated with spinal cord compression (Kaplan, 2006b).	**Clinical** • Local inflammation, edema, venous stasis, and impaired blood supply to nervous tissues • Local or radicular back or neck pain along the dermatomal areas innervated by the affected nerve root (Marrs, 2006) (eg, thoracic radicular pain extends in a band around the chest or abdomen) • Pain exacerbated by movement, supine recumbent position, coughing, sneezing, or the Valsalva maneuver • Neurologic dysfunction, and related motor and sensory deficits (numbness, tingling, feelings of coldness in the affected area, inability to detect vibration, loss of positional sense) • Motor loss ranging from subtle weakness to flaccid paralysis • Bladder and/or bowel dysfunction depending on level of compression (above S2, overflow incontinence; from S3 to S5, flaccid sphincter tone, and bowel incontinence)	**Medical** • Radiation therapy to reduce tumor size to halt progression and corticosteroid therapy to decrease inflammation and swelling at the compression site • Surgery to debulk tumor and stabilize the spine if symptoms progress despite radiation therapy or if vertebral fracture or bone fragments lead to additional nerve damage; surgery is also an option when the tumor is not radiosensitive or is located in an area that was previously irradiated (Kaplan, 2006b) • Vertebroplasty is used to stabilize vertebrae when patients have pain without neurologic dysfunction; vertebroplasty involves percutaneous injection of polymethyl methacrylate (PMMA), a bone cement filler, into the vertebral body (Kaplan, 2006b) • Chemotherapy as adjuvant to radiation therapy for patients with lymphoma or small cell lung cancer • *Note:* Despite treatment, patients with poor neurologic function before treatment are less likely to regain complete motor and sensory function; patients who develop complete paralysis usually do not regain all neurologic function (Kaplan, 2006b)

Continued on following page

Table 16-10 ONCOLOGIC EMERGENCIES: MANIFESTATIONS AND MANAGEMENT (Continued)

Emergency	Clinical Manifestations and Diagnostic Findings	Management
	Diagnostic • Percussion tenderness at the level of compression • Abnormal reflexes • Sensory and motor abnormalities • MRI, spinal cord x-rays, bone scans, and CT scan. CT-guided myelogram is reserved for patients who are unable to undergo MRI (Kaplan, 2006b).	**Nursing** • Perform ongoing assessment of neurologic function to identify existing and progressing dysfunction • Control pain with pharmacologic and nonpharmacologic measures • Prevent complications of immobility resulting from pain and decreased function (eg, skin breakdown, urinary stasis, thrombophlebitis, decreased clearance of pulmonary secretions) • Maintain muscle tone by assisting with range-of-motion exercises in collaboration with physical and occupational therapists • Institute intermittent urinary catheterization and bowel training programs for patients with bladder or bowel dysfunction • Provide encouragement and support to patient and family coping with pain and altered functioning, lifestyle, roles, and independence
Hypercalcemia In patients with cancer, hypercalcemia is a potentially life-threatening metabolic abnormality resulting when the calcium released from the bones is more than the kidneys can excrete or the bones can reabsorb. It may result from: • Production of cytokines, hormonal substances and growth factors by cancer cells, or by the body in response to substances produced by cancer cells; which lead to bone breakdown and calcium release (Kaplan, 2006a). • Excessive use of vitamins and minerals and conditions unrelated to cancer, such as dehydration, renal impairment, primary hyperparathyroidism, thyrotoxicosis, thiazide diuretics, and hormone therapy.	**Clinical** Fatigue, weakness, confusion, decreased level of responsiveness, hyporeflexia, nausea, vomiting, constipation, ileus, polyuria (excessive urination), polydipsia (excessive thirst), dehydration, and dysrhythmias **Diagnostic** Serum calcium level exceeding 11 mg/dL (2.74 mmol/L)	**Medical** See Chapter 14. **Nursing** • Identify patients at risk for hypercalcemia and assess for signs and symptoms of hypercalcemia • Educate patient and family; prevention and early detection can prevent fatality • Teach at-risk patients to recognize and report signs and symptoms of hypercalcemia • Encourage patients to consume 2–4 L of fluid daily unless contraindicated by existing renal or cardiac disease • Explain the use of dietary and pharmacologic interventions such as stool softeners and laxatives for constipation • Advise patients to maintain nutritional intake without restricting normal calcium intake • Discuss antiemetic therapy if nausea and vomiting occur • Promote mobility by emphasizing its importance in preventing demineralization and breakdown of bones • Institute safety precautions for patients with impaired mental and mobility status
Pericardial Effusion and Cardiac Tamponade Pericardial effusion is an accumulation of fluid in the pericardial space. Cardiac tamponade occurs when the accumulation compresses the heart and thereby impedes expansion of the ventricles and cardiac filling during diastole. As ventricular volume and cardiac output fall, the heart pump fails, and circulatory collapse develops. With gradual onset, fluid accumulates gradually, and the outer layer of the pericardial space stretches to compensate for rising pressure. Large amounts of fluid accumulate before symptoms of heart failure occur. With rapid onset, pressures rise too quickly for the pericardial space to compensate.	**Clinical** • Neck vein distention during inspiration (Kussmaul's sign) • Pulsus paradoxus (systolic blood pressure decrease exceeding 10 mm Hg during inspiration; pulse gets stronger on expiration) • Distant heart sounds, rubs and gallops, cardiac dullness • Compensatory tachycardia (heart beats faster to compensate for decreased cardiac output) • Increased venous and vascular pressures **Diagnostic** • Electrocardiography (ECG) helps diagnose pericardial effusion • In small effusion, chest x-rays show small amounts of fluid in the pericardium; in large effusions, x-ray films disclose "water-bottle" heart (obliteration of vessel contour and cardiac chambers) • CT scans help diagnose pleural effusions and evaluate effect of treatment • Narrow pulse pressure • Shortness of breath and tachypnea	**Medical** • Patients with small effusions who are not symptomatic do not require treatment. These patients are monitored for signs and symptoms of increasing fluid accumulation (Higdon & Higdon, 2006) • Pericardiocentesis (the aspiration or withdrawal of pericardial fluid by a large-bore needle inserted into the pericardial space); in malignant effusions, pericardiocentesis provides only temporary relief; fluid may reaccumulate (Story, 2006); windows or openings in the pericardium can be created surgically as a palliative measure to drain fluid into the pleural space; catheters may also be placed in the pericardial space and sclerosing agents (such as bleomycin or thiotepa) injected to prevent fluid from reaccumulating (Story, 2006) • Radiation therapy or antineoplastic agents, depending on how sensitive the primary tumor is to these treatments and the degree of symptoms that exist; in mild effusions, prednisone and diuretic medications may be prescribed and the patient's status carefully monitored

Continued

Table 16-10 ONCOLOGIC EMERGENCIES: MANIFESTATIONS AND MANAGEMENT (Continued)

Emergency	Clinical Manifestations and Diagnostic Findings	Management
Cancerous tumors, particularly from adjacent thoracic tumors (lung, esophagus, breast cancers), and cancer treatment are the most common causes of cardiac tamponade. Radiation therapy of 4000 cGy or more to the mediastinal area has also been implicated in pericardial fibrosis, pericarditis, and resultant cardiac tamponade. Untreated pericardial effusion and cardiac tamponade lead to circulatory collapse and cardiac arrest (Story, 2006).	• Weakness, chest pain, orthopnea, anxiety, diaphoresis, lethargy, and altered consciousness from decreased cerebral perfusion	**Nursing** • Monitor vital signs and oxygen saturation frequently • Assess for pulsus paradoxus • Monitor ECG tracings • Assess heart and lung sounds, neck vein filling, level of consciousness, respiratory status, and skin color and temperature • Monitor and record intake and output • Review laboratory findings (eg, arterial blood gas and electrolyte levels) • Elevate the head of the patient's bed to ease breathing • Minimize patient's physical activity to reduce oxygen requirements; administer supplemental oxygen as prescribed • Provide frequent oral hygiene • Reposition and encourage the patient to cough and take deep breaths every 2 hours • As needed, maintain patent intravenous access, reorient the patient, and provide supportive measures and appropriate patient instruction
Disseminated Intravascular Coagulation (DIC; also called consumption coagulopathy) Complex disorder of coagulation or fibrinolysis (destruction of clots), which results in thrombosis or bleeding. DIC is most commonly associated with hematologic cancers (leukemia and lymphoma); cancer of prostate, gastrointestinal (GI) tract, and lungs; chemotherapy (methotrexate, prednisone, L-asparaginase, vincristine, 5-fluorouracil, cyclophosphamide; targeted agents bevacizumab, thalidomide, interferon; hormonal agents (tamoxifen, Megace); and other processes such as trauma, sepsis, hepatic failure, and anaphylaxis (Enzone, 2006; Viable, 2005). Blood clots form when normal coagulation mechanisms are triggered. Once activated, the clotting cascade continues to consume clotting factors and platelets faster than the body can replace them. Clots are deposited in the microvasculature, placing the patient at great risk for impaired circulation, tissue hypoxia, and necrosis. In addition, fibrinolysis occurs, breaking down clots and increasing the circulating levels of anticoagulant substances, thereby placing the patient at risk for hemorrhage (Enzone, 2006).	**Clinical** *Chronic DIC*: Few or no observable symptoms or easy bruising, prolonged bleeding from venipuncture and injection sites, bleeding of the gums, and slow GI bleeding *Acute DIC*: Life-threatening hemorrhage and infarction; clinical symptoms of this syndrome are varied and depend on the organ system involved in thrombus and infarction or bleeding episodes **Diagnostic** • Prolonged prothrombin time (PT or protime) • Prolonged partial thromboplastin time (PTT) • Prolonged thrombin time (TT) • Decreased fibrinogen level • Decreased platelet level • Decrease in clotting factors • Decreased hemoglobin • Decreased hematocrit • Elevated fibrin split products • Positive protamine sulfate precipitation test (thrombin activation test) • Elevated D-dimer • Prolonged international normalized ratio (INR) • Decreased plasminogen levels	**Medical** • Chemotherapy, biologic response modifier therapy, radiation therapy, or surgery is used to treat the underlying cancer • Antibiotic therapy is used for sepsis • Anticoagulants, such as heparin or antithrombin III, decrease the stimulation of the coagulation pathways • Drotrecogin alfa is used with caution in patients with DIC related to sepsis (Ezzone, 2006) • Transfusion of fresh-frozen plasma or cryoprecipitates (which contain clotting factors and fibrinogen), packed red blood cells, and platelets may be used as replacement therapy to prevent or control bleeding • Although controversial, antifibrinolytic agents such as aminocaproic acid (Amicar), which is associated with increased thrombus formation, may be used **Nursing** • Monitor vital signs • Measure and document intake and output • Assess skin color and temperature; lung, heart, and bowel sounds; level of consciousness, headache, visual disturbances, chest pain, decreased urine output, and abdominal tenderness • Inspect all body orifices, tube insertion sites, incisions, and bodily excretions for bleeding • Review laboratory test results • Minimize physical activity to decrease injury risks and oxygen requirements • Prevent bleeding; apply pressure to all venipuncture sites, and avoid nonessential invasive procedures; provide electric rather than straight-edged razors; avoid tape on the skin and advise gentle but adequate oral hygiene • Assist the patient to turn, cough, and take deep breaths on regular schedule • Reorient the patient, if needed; maintain a safe environment; and provide appropriate patient education and supportive measures

Continued on following page

Table 16-10 ONCOLOGIC EMERGENCIES: MANIFESTATIONS AND MANAGEMENT (Continued)

Emergency	Clinical Manifestations and Diagnostic Findings	Management
Syndrome of Inappropriate Secretion of Antidiuretic Hormone (SIADH) The continuous, uncontrolled release of antidiuretic hormone (ADH), produced by tumor cells or by the abnormal stimulation of the hypothalamic–pituitary network, leads to increased extracellular fluid volume, water intoxication, hyponatremia, and increased excretion of urinary sodium. As fluid volume increases, stretch receptors in the right atrium respond by releasing a second hormone, atrial natriuretic factor (ANF). The release of ANF causes increased renal excretion of sodium, which worsens hyponatremia. The most common cause of SIADH is cancer, especially small cell cancers of the lung. A variety of nonmalignant diseases, trauma and medications are associated with SIADH. Antineoplastics including vincristine, vinblastine, cisplatin, and cyclophosphamide, as well as morphine stimulate ADH secretion, which promotes conservation and reabsorption of water by the kidneys. As more fluid is absorbed, the circulatory volume increases, ANF is released, and sodium is actively excreted by the kidneys in compensation (Clancey, 2006)	**Clinical** *Serum sodium levels lower than 125 mEq/L* (125 mmol/L): symptoms of hyponatremia including personality changes, irritability, nausea, anorexia, vomiting, weight gain, fatigue, muscular pain (myalgia), headache, lethargy, and confusion *Serum sodium levels lower than 115 mEq/L* (11 mmol/L): seizure, abnormal reflexes and gait, papilledema, coma, and death; edema is rare **Diagnostic** • Decreased serum sodium level • Increased urine osmolality • Increased urinary sodium level • Decreased blood urea nitrogen (BUN), creatinine, and serum albumin levels secondary to dilution • Abnormal water load test results	**Medical** • Treat underlying disease process or eliminate contributing medications • Fluid intake range limited to 500–1000 mL/day to increase the serum sodium level and decrease fluid overload. If water restriction alone is not effective in correcting or controlling serum sodium levels, demeclocycline is often prescribed to interfere with the antidiuretic action of ADH and ANF; if neurologic symptoms are severe, parenteral sodium replacement and diuretic therapy are indicated; electrolyte levels are monitored carefully to detect secondary magnesium, potassium, and calcium imbalances; after the symptoms of SIADH are controlled, the underlying cancer is treated; if water excess continues despite treatment, pharmacologic intervention (urea and furosemide) may be indicated (Clancey, 2006) **Nursing** • Recognize individuals at risk • Maintain intake and output measurements as often as hourly for severe hyponatremia (Clancey, 2006) • Assess level of consciousness, lung and heart sounds, vital signs, daily weight, and urine specific gravity; also assess for nausea, vomiting, anorexia, edema, fatigue, and lethargy • Monitor laboratory test results, including serum electrolyte levels, osmolality, and BUN, creatinine, and urinary sodium levels • Minimize the patient's activity; provide appropriate oral hygiene; maintain environmental safety; and restrict fluid intake if necessary • Reorient the patient and provide instruction and encouragement as needed
Tumor Lysis Syndrome Potentially fatal complication associated with radiation, biotherapy, or chemotherapy-induced cell destruction of large or rapidly growing cancers such as leukemia, lymphoma, and small cell lung cancer (Higdon & Higdon, 2006). The release of intracellular contents from the tumor cells leads to electrolyte imbalances—hyperkalemia, hypocalcemia, hyperphosphatemia, and hyperuricemia—because the kidneys can no longer excrete large volumes of the released intracellular metabolites.	**Clinical** Clinical manifestations depend on the extent of metabolic abnormalities • *Neurologic:* Fatigue, weakness, memory loss, altered mental status, muscle cramps, tetany, paresthesias (numbness and tingling), seizures • *Cardiac:* Elevated blood pressure, shortened QT complexes, widened QRS waves, altered T waves, dysrhythmias, cardiac arrest • *GI:* Anorexia, nausea, vomiting, abdominal cramps, diarrhea, increased bowel sounds • *Renal:* Flank pain, oliguria, anuria, renal failure, acidic urine pH Other: Gout, malaise, pruritis **Diagnostic** Electrolyte imbalances identified by serum electrolyte measurement and urinalysis; EKG necessary to monitor cardiac abnormalities (Gobel, 2006)	**Medical** • To prevent renal failure and restore electrolyte balance, aggressive fluid hydration is initiated 24–48 hours before and after the initiation of cytotoxic therapy to increase urine volume and eliminate uric acid and electrolytes; urine is alkalinized by adding sodium bicarbonate to intravenous fluid to maintain a urine pH of 7 to 7.5; this prevents renal failure secondary to uric acid precipitation in the kidneys (Gobel, 2006) • Diuresis with a loop diuretic or osmotic diuretic if urine output is not sufficient (Gobel, 2006) • Allopurinol therapy to inhibit the conversion of nucleic acids to uric acid or rasburicase to oxidizes uric acid to allantoin that has higher solubility than uric acid (Gobel, 2006) • Administration of a cation-exchange resin, such as sodium polystyrene sulfonate (Kayexalate) to treat hyperkalemia by binding and eliminating potassium through the bowel • Administration of intravenous sodium bicarbonate, hypertonic dextrose, and regular insulin temporarily shifts potassium into cells and lowers serum potassium levels • Administration of phosphate-binding gels, such as aluminum hydroxide, to treat hyperphosphatemia by promoting phosphate excretion in the feces • Hemodialysis when patients are unresponsive to the standard approaches for managing uric acid and electrolyte abnormalities

Continued

Table 16-10 ONCOLOGIC EMERGENCIES: MANIFESTATIONS AND MANAGEMENT (Continued)

Emergency	Clinical Manifestations and Diagnostic Findings	Management
		Nursing • Identify at-risk patients, including those in whom tumor lysis syndrome may develop up to 1 week after therapy has been completed • Institute essential preventive measures (eg, fluid hydration, allopurinol) • Assess patient for signs and symptoms of electrolyte imbalances • Assess urine pH to confirm alkalization • Monitor serum electrolyte and uric acid levels for evidence of fluid volume overload secondary to aggressive hydration • Instruct patients to report symptoms indicating electrolyte disturbances

Because of the high costs associated with maintaining free-standing hospices, care is often delivered through coordination of services provided by hospitals, home care programs, and the community. The view that palliative care services are necessary only in extreme circumstances prevents appropriate and timely consultation with palliative care specialists (MacDonald, 2005; Pavlish & Ceronsky, 2007). Patients should be referred to palliative care and hospice services in a timely fashion so that complex patient needs can be addressed. Although physicians, social workers, clergy, dietitians, pharmacists, physical therapists, and volunteers are involved in patient care, nurses often coordinate hospice services.

Hospice programs strive to facilitate clear communication among family members and health care providers. Most patients and families are informed of the prognosis and are encouraged to participate in decisions regarding pursuing or terminating cancer treatment. Through collaboration with other support disciplines, the nurse helps the patient and family cope with changes in role identity, family structure, grief, and loss. Hospice nurses are actively involved in bereavement counseling. See Chapter 17 for detailed discussion of end-of-life care.

Cancer Survivorship

The National Cancer Institute (2007) estimates that over 10 million individuals are alive today who have been previously diagnosed with cancer. Largely as a result of increased screening programs for breast, cervical, and prostate cancers and advances in treatment, the numbers of cancer survivors has tripled over the past 37 years. Cancer survivorship refers to a distinct phase of cancer care that follows primary treatment for cancer and lasts until cancer recurrence or end of life (Hewitt, Greenfield & Stovall, 2006). Although individuals vary and there are many types of cancers and treatments, the acute, long-term, and late effects of cancer and its treatment may have multiple physical and psychosocial consequences.

Approaches to survivorship care are often based on expert opinion and experiences rather than evidence-based interventions. Knowledge regarding survivorship concerns continues to evolve. The Institute of Medicine identified four components of survivorship care (Hewitt, et al., 2006), listed in Table 16-11. Multiple professional and advocacy organizations across the country have recommended that a survivorship care plan be provided to all cancer patients and their primary care physician at the completion of treatment. The survivorship care plan includes a summary of cancer diagnosis and treatment, recommendations for follow-up and care, including approaches to treat symptoms, rehabilitative needs, monitoring for late effects, and surveillance and screening for new and recurrent cancer. Referrals for specific services such as lymphedema therapy, support groups, and genetic counseling are also provided. Nurses assist in the development of the survivorship care plan and provide education and care for cancer survivors. Nurses, other health care providers, public health professionals, and patient advocates design and conduct research in order to identify needs of cancer survivors and evidence-based approaches to care.

Table 16-11 COMPONENTS OF SURVIVORSHIP CARE

Component	Examples of Care
Prevention and detection of new and recurrent cancer.	• Mammography (per ACS guidelines) • Papanicolaou (Pap) smears (per ACS guidelines) • Smoking cessation programs • Nutrition counseling
Surveillance for cancer spread, recurrence or second cancers	• Colonoscopy post–colon cancer • Mammography post–breast cancer • Liver function tests post–colon cancer • Prostate specific antigen post–prostate cancer
Intervention for consequences of cancer and its treatments	• Lymphedema therapy • Pain management • Enterostomal therapy • Fertility care
Coordination between specialists and primary care providers to meet health needs	• Care for comorbidities (eg, diabetes) • Influenza vaccination • Bone densitometry

ACS, American Cancer Society.

From Hewitt, M., Greenfield, S. & Stovall, E. (Eds.). (2006). *From cancer patient to cancer survivor*. Washington, DC: Institute of Medicine and National Research Council. The National Academies Press. Components of survivorship care provided by the Institute of Medicine report on cancer survivorship.

CRITICAL THINKING EXERCISES

EBP **1** Your patient has just completed treatment planning for receiving external beam radiation for an aggressive nasopharyngeal cancer. The patient has expressed concerns about what side effects of this treatment can be anticipated. What would your response be to him? What evidence-based nursing interventions would you implement to minimize side effects? Are there any preventive measures to protect the patient's oral mucosa? What nutritional needs would this patient experience and what approaches should be used to address them? What is the evidence for the interventions you identified? How strong is that evidence, and what criteria did you use to assess the strength of that evidence?

2 A 58-year-old patient with bone metastasis from an unknown primary cancer has been receiving an opioid through a continuous subcutaneous infusion of analgesia with an infusion pump to relieve his severe pain. His wife tells you that both she and her husband fear that he will become addicted to the opioid; his adult children report that his pain remains severe and unrelieved. As a home care nurse, what assessments would be of highest priority to you during your initial visit to this patient? What nursing interventions would be indicated for the patient and his wife?

3 A 33-year-old man has presented to the cancer center for treatment of colorectal cancer. In reviewing his family history, you note that his father and grandfather (who are both deceased) had metastatic colon cancer and his father's sister had endometrial cancer at age 45. You also note that he has two younger sisters. What information is important in this family history and why? What type of referral would be appropriate for this man and his family? How would you best advise this man and his family regarding cancer risks and screening practices?

4 Your 28-year-old patient with acute leukemia, hospitalized for high-dose chemotherapy, has developed tumor lysis syndrome and acute renal failure. Describe the underlying pathology that can lead to the signs and symptoms of tumor lysis syndrome. What patient monitoring will be essential during this patient's care? Describe the medical and nursing management strategies that will be used for this patient.

The Smeltzer suite offers these additional resources to enhance learning and facilitate understanding of this chapter:

- thePoint on line resource, thepoint.lww.com/Smeltzer12E
- Student CD-ROM included with the book
- *Study Guide to Accompany Brunner & Suddarth's Textbook of Medical-Surgical Nursing*
- *Handbook for Brunner & Suddarth's Textbook of Medical-Surgical Nursing*

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**Asterisk indicates nursing research.*
***Double asterisk indicates classic reference.*

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pulmonary artery catheter, which is used to obtain the hemodynamic data essential for diagnosis and treatment (see Chapter 26). Measurements of intracardiac pressures, pulmonary artery pressures, and cardiac output are made at intervals. Therapy, especially intravenous (IV) medication, is adjusted based on the hemodynamic data. The patient with an invasive hemodynamic catheter is usually managed in an intensive care environment because of the need for frequent nursing assessments and interventions.

HEART FAILURE

HF is the inability of the heart to pump sufficient blood to meet the needs of the tissues for oxygen and nutrients. In the past, HF was often referred to as **congestive heart failure (CHF),** because many patients experience pulmonary or peripheral congestion. Currently HF is recognized as a clinical syndrome characterized by signs and symptoms of fluid overload or of inadequate tissue perfusion. Fluid overload and decreased tissue perfusion result when the heart cannot generate a CO sufficient to meet the body's demands. The term HF indicates myocardial disease in which there is a problem with contraction of the heart (systolic dysfunction) or filling of the heart (diastolic dysfunction) that may or may not cause pulmonary or systemic congestion. Some cases of HF are reversible, depending on the cause. Most often, HF is a progressive, life-long condition that is managed with lifestyle changes and medications to prevent episodes of **acute decompensated heart failure**. These episodes are characterized by an increase in symptoms, decreased CO, and low perfusion (Varughese, 2007). They are associated with increased hospitalizations, increased health care costs, and decreased quality of life.

Chronic Heart Failure

As with coronary artery disease, the incidence of HF increases with age. More than 5 million people in the United States have HF, and 550,000 new cases are diagnosed each year (American Heart Association [AHA], 2007). Although HF can affect people of all ages, it is most common in people older than 75 years of age; as the U.S. population ages, HF has become an epidemic that challenges the country's health care resources. HF is the most common reason for hospitalization of people older than 65 years of age and is the second most common reason for visits to a physician's office. The rate of emergency department visits and hospital readmissions for this condition remains very high. The estimated economic burden caused by HF is more than $33 billion annually in direct and indirect costs and is expected to increase (AHA, 2007).

The increase in the incidence of HF reflects not only the increased number of elderly people but also improvements in treatment of cardiac diseases such as MI, resulting in increased survival rates. Many hospitalizations for HF can be prevented by appropriate outpatient care. Prevention and early intervention to arrest the progression of HF are major health initiatives in the United States.

Two major types of HF are identified by assessment of left ventricular functioning, usually by echocardiogram. The more common type is an alteration in ventricular contraction called **systolic heart failure,** which is characterized by a weakened heart muscle. The less common type is **diastolic heart failure,** which is characterized by a stiff and noncompliant heart muscle, making it difficult for the ventricle to fill. An assessment of the **ejection fraction (EF)** is performed to assist in determining the type of HF. EF is calculated by subtracting the amount of blood present in the left ventricle at the end of systole from the amount present at the end of diastole and calculating the percentage of blood that is ejected. A normal EF is 55% to 65% of the ventricular volume; the ventricle does not completely empty between contractions. The EF is normal in diastolic HF but severely reduced in systolic HF.

Although a low EF is a hallmark of systolic HF, which is the most common type of HF, the severity of HF is frequently classified according to the patient's symptoms. The New York Heart Association (NYHA) Classification is described in Table 30-1. The American College of Cardiology and the American Heart Association (ACC/AHA) have developed another a HF classification system (Hunt, Abraham, Chin, et al., 2005). This system, described in Table 30-2, takes into consideration the natural history and progressive nature of HF. Treatment guidelines have been developed for each stage.

Pathophysiology

HF results from a variety of cardiovascular conditions, including chronic hypertension, coronary artery disease, and valvular disease. These conditions can result in systolic failure, diastolic failure, or both. Significant myocardial dysfunction usually occurs before the patient experiences signs and symptoms of HF such as shortness of breath, edema, or fatigue.

Table 30-1 NEW YORK HEART ASSOCIATION (NYHA) CLASSIFICATION OF HEART FAILURE

Classification	Signs and Symptoms	Prognosis
I	Ordinary physical activity does not cause undue fatigue, dyspnea, palpitations, or chest pain No pulmonary congestion or peripheral hypotension Patient is considered asymptomatic Usually no limitations of activities of daily living (ADLs)	Good
II	Slight limitation on ADLs Patient reports no symptoms at rest but increased physical activity will cause symptoms Basilar crackles and S_3 murmur may be detected	Good
III	Marked limitation on ADL Patient feels comfortable at rest but less than ordinary activity will cause symptoms	Fair
IV	Symptoms of cardiac insufficiency at rest	Poor

Table 30-2 AMERICAN COLLEGE OF CARDIOLOGY AND AMERICAN HEART ASSOCIATION (ACC/AHA) CLASSIFICATION OF HEART FAILURE

Classification	Criteria
Stage A	Patients at high risk for developing left ventricular dysfunction but without structural heart disease or symptoms of heart failure
Stage B	Patients with left ventricular dysfunction or structural heart disease who have not developed symptoms of heart failure
Stage C	Patients with left ventricular dysfunction or structural heart disease with current or prior symptoms of heart failure
Stage D	Patients with refractory end-stage heart failure requiring specialized interventions

As HF develops, the body activates neurohormonal compensatory mechanisms. These mechanisms represent the body's attempt to cope with the HF and are responsible for the signs and symptoms that eventually develop. Understanding these mechanisms is important because the treatment of HF is aimed at opposing them and relieving symptoms.

Systolic HF results in decreased blood volume being ejected from the ventricle. The decreased ventricular stretch is sensed by baroreceptors in the aortic and carotid bodies. The sympathetic nervous system is then stimulated to release epinephrine and norepinephrine (Fig. 30-2) . The purpose of this initial response is to increase heart rate and contractility and support the failing myocardium, but the

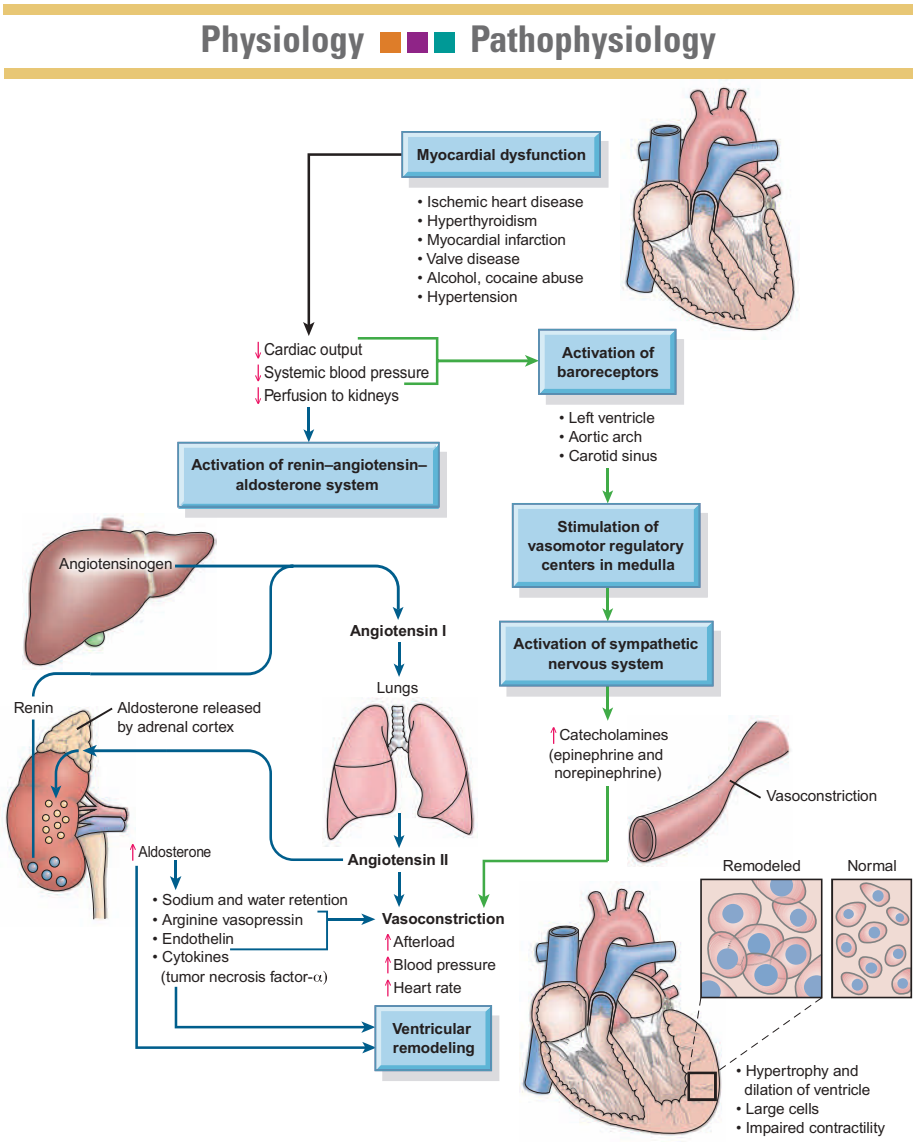


Figure 30-2 The pathophysiology of heart failure. A decrease in cardiac output activates multiple neurohormonal mechanisms that ultimately result in the signs and symptoms of heart failure.

continued response has multiple negative effects. Sympathetic stimulation causes vasoconstriction in the skin, gastrointestinal tract, and kidneys. A decrease in renal perfusion due to low CO and vasoconstriction then causes the release of renin by the kidneys. Renin promotes the formation of angiotensin I, a benign, inactive substance. Angiotensin-converting enzyme (ACE) in the lumen of pulmonary blood vessels converts angiotensin I to angiotensin II, a potent vasoconstrictor, which then increases the blood pressure and afterload. Angiotensin II also stimulates the release of aldosterone from the adrenal cortex, resulting in sodium and fluid retention by the renal tubules and stimulation of antidiuretic hormone. These mechanisms lead to the fluid volume overload commonly seen in HF. Angiotensin, aldosterone, and other neurohormones (eg, endothelin, prostacyclin) lead to an increase in preload and afterload, which increases stress on the ventricular wall, causing an increase in the workload of the heart. A counterregulatory mechanism is attempted through the release of natriuretic peptides. Atrial natriuretic peptide (ANP) and B-type (ie, brain type) natriuretic peptide (BNP) are released from the overdistended cardiac chambers. These substances promote vasodilation and diuresis. However, their effect is usually not strong enough to overcome the negative effects of the other mechanisms.

As the heart's workload increases, contractility of the myocardial muscle fibers decreases. Decreased contractility results in an increase in end-diastolic blood volume in the ventricle, stretching the myocardial muscle fibers and increasing the size of the ventricle (ventricular dilation). The increased size of the ventricle further increases the stress on the ventricular wall, adding to the workload of the heart. One way the heart compensates for the increased workload is to increase the thickness of the heart muscle (ventricular hypertrophy). However, hypertrophy results in an abnormal proliferation of myocardial cells, a process known as ventricular remodeling. Under the influence of neurohormones (eg, angiotensin II), large myocardial cells are produced that are dysfunctional and die early, leaving the other normal myocardial cells to struggle to maintain CO. The compensatory mechanisms of HF have been called the "vicious cycle of HF" because the heart does not pump sufficient blood to the body, which causes the body to stimulate the heart to work harder; thus, the heart cannot respond and failure becomes worse.

Diastolic HF develops because of continued increased workload on the heart, which responds by increasing the number and size of myocardial cells (ie, ventricular hypertrophy and altered cellular functioning). These responses cause resistance to ventricular filling, which increases ventricular filling pressures despite a normal or reduced blood volume. Less blood in the ventricles causes decreased CO. The low CO and high ventricular filling pressures can cause the same neurohormonal responses as described for systolic HF.

Etiology

Myocardial dysfunction is most often caused by coronary artery disease, cardiomyopathy, hypertension, or valvular disorders. Patients with diabetes mellitus are also at high risk for HF. Atherosclerosis of the coronary arteries is the primary cause of HF, and coronary artery disease is found in more than 60% of the patients with HF (Zipes, Libby & Bonow, 2005). Ischemia causes myocardial dysfunction because it deprives heart cells of oxygen and leads to acidosis from the accumulation of lactic acid. MI causes focal heart muscle necrosis, the death of myocardial cells, and a loss of contractility; the extent of the infarction correlates with the severity of HF. Revascularization of the coronary artery by a percutaneous coronary intervention (PCI) or by coronary artery bypass surgery (CABG) may improve myocardial oxygenation and ventricular function.

Cardiomyopathy is a disease of the myocardium. There are three types: dilated, hypertrophic, and restrictive (see Chapter 29). Dilated cardiomyopathy, the most common type of cardiomyopathy, causes diffuse cellular necrosis and fibrosis, leading to decreased contractility (systolic failure). Dilated cardiomyopathy can be idiopathic (unknown cause) or it can result from an inflammatory process, such as myocarditis, or from a cytotoxic agent, such as alcohol or doxorubicin (Adriamycin). Hypertrophic cardiomyopathy and restrictive cardiomyopathy lead to decreased distensibility and ventricular filling (diastolic failure). Usually, HF due to cardiomyopathy becomes chronic and progressive. However, cardiomyopathy and HF may resolve following removal of the causative agent, such as with the cessation of alcohol ingestion.

Systemic or pulmonary hypertension increases afterload (resistance to ejection), which increases the workload of the heart and leads to hypertrophy of myocardial muscle fibers. This can be considered a compensatory mechanism because it increases contractility. However, the hypertrophy may impair the heart's ability to fill properly during diastole, and the hypertrophied ventricle may eventually dilate and fail.

Valvular heart disease is also a cause of HF. The valves ensure that blood flows in one direction. With valvular dysfunction, blood has increasing difficulty moving forward, increasing pressure within the heart and increasing cardiac workload, leading to HF. Chapter 29 discusses the effects of valvular heart disease.

Several systemic conditions, including progressive renal failure and uncontrolled hypertension, contribute to the development and severity of HF (Varughese, 2007). Acute illness such as pneumonia with fever and hypoxia increase the metabolic rate and may precipitate HF. All of these conditions require an increase in CO to satisfy the systemic oxygen demand, and they stress the compromised myocardium. Cardiac dysrhythmias may cause HF or may be a result of HF; either way, the altered electrical stimulation impairs myocardial contraction and decreases the overall efficiency of myocardial function. Other factors, such as acidosis (respiratory or metabolic), electrolyte abnormalities, and antiarrhythmic medications, can worsen myocardial function.

Clinical Manifestations

The clinical manifestations produced by the different types of HF (systolic, diastolic, or both) are similar (Chart 30-1) and therefore do not assist in differentiating the types of HF. The signs and symptoms of HF can be related to which ventricle is affected. **Left-sided heart failure (left ventricular failure)** causes different manifestations than **right-sided**

CHART 30-1 *Assessing for Heart Failure*

Be alert for the following signs and symptoms:

General

- Fatigue
- Decreased activity tolerance
- Dependent edema
- Weight gain

Cardiovascular

- Third heart sound (S_3)
- Apical impulse enlarged with left lateral displacement
- Pallor and cyanosis
- Jugular venous distention (JVD)

Respiratory

- Dyspnea on exertion
- Pulmonary crackles that do not clear with cough
- Orthopnea
- Paroxysmal nocturnal dyspnea (PND)
- Cough on exertion or when supine

Cerebrovascular

- Unexplained confusion or altered mental status
- Lightheadedness

Renal

- Oliguria and decreased frequency during the day
- Nocturia

Gastrointestinal

- Anorexia and nausea
- Enlarged liver
- Ascites
- Hepatojugular reflux

heart failure (right ventricular failure). In chronic HF, patients may have signs and symptoms of both left and right ventricular failure.

Left-Sided Heart Failure

Pulmonary congestion occurs when the left ventricle cannot effectively pump blood out of the ventricle into the aorta and the systemic circulation. The increased left ventricular end-diastolic blood volume increases the left ventricular end-diastolic pressure, which decreases blood flow from the left atrium into the left ventricle during diastole. The blood volume and pressure in the left atrium increases, which decreases blood flow from the pulmonary vessels. Pulmonary venous blood volume and pressure increase, forcing fluid from the pulmonary capillaries into the pulmonary tissues and alveoli, causing pulmonary interstitial edema and impaired gas exchange. The clinical manifestations of pulmonary congestion include dyspnea, cough, pulmonary crackles, and low oxygen saturation levels. An extra heart sound, the S_3, or "ventricular gallop," may be detected on auscultation. It is caused by a large volume of fluid entering the ventricle at the beginning of diastole.

Dyspnea, or shortness of breath, may be precipitated by minimal to moderate activity (dyspnea on exertion [DOE]); dyspnea also can occur at rest. The patient may report **orthopnea,** difficulty breathing when lying flat. Patients with orthopnea usually prefer not to lie flat. They may need pillows to prop themselves up in bed, or they may sit in a chair and even sleep sitting up. Some patients have sudden attacks of dyspnea at night, a condition known as **paroxysmal nocturnal dyspnea (PND).** Fluid that accumulates in the dependent extremities during the day may be reabsorbed into the circulating blood volume when the patient lies down. Because the impaired left ventricle cannot eject the increased circulating blood volume, the pressure in the pulmonary circulation increases, shifting fluid into the alveoli. The fluid-filled alveoli cannot exchange oxygen and carbon dioxide. Without sufficient oxygen, the patient experiences dyspnea and has difficulty sleeping.

The cough associated with left ventricular failure is initially dry and nonproductive. Most often, patients complain of a dry hacking cough that may be mislabeled as asthma or chronic obstructive pulmonary disease (COPD). The cough may become moist over time. Large quantities of frothy sputum, which is sometimes pink (blood-tinged), may be produced, usually indicating severe pulmonary congestion (pulmonary edema).

Adventitious breath sounds may be heard in various areas of the lungs. Usually, bibasilar crackles that do not clear with coughing are detected in the early phase of left ventricular failure. As the failure worsens and pulmonary congestion increases, crackles may be auscultated throughout all lung fields. At this point, oxygen saturation may decrease.

In addition to increased pulmonary pressures that cause decreased oxygenation, the amount of blood ejected from the left ventricle decreases. The dominant feature in HF is inadequate tissue perfusion. The diminished CO has widespread manifestations because not enough blood reaches all the tissues and organs (low perfusion) to provide the necessary oxygen. The decrease in SV can also lead to stimulation of the sympathetic nervous system, which further impedes perfusion to many organs.

A reduction in CO decreases blood flow to the kidneys, reducing urine output (**oliguria**). Renal perfusion pressure falls, which results in the release of renin from the kidney. Release of renin leads to aldosterone secretion and increased intravascular volume. However, when the patient is sleeping, the cardiac workload is decreased, improving renal perfusion, which in some patients leads to frequent urination at night (nocturia).

As HF progresses, decreased CO may cause other symptoms. Decreased gastrointestinal perfusion causes altered digestion. Decreased brain perfusion causes dizziness, lightheadedness, confusion, restlessness, and anxiety due to decreased oxygenation and blood flow. As anxiety increases, so does dyspnea, increasing anxiety and creating a vicious cycle. Stimulation of the sympathetic system also causes the peripheral blood vessels to constrict, so the skin appears pale or ashen and feels cool and clammy.

A decrease in SV causes the sympathetic nervous system to increase the HR (tachycardia), often causing the patient to complain of palpitations. The pulses become weak and thready. Without adequate CO, the body cannot respond to increased energy demands, and the patient becomes easily

fatigued and has decreased activity tolerance. Fatigue also results from the increased energy expended in breathing and the insomnia that results from respiratory distress, coughing, and nocturia.

Right-Sided Heart Failure

When the right ventricle fails, congestion in the peripheral tissues and the viscera predominates. This occurs because the right side of the heart cannot eject blood and cannot accommodate all the blood that normally returns to it from the venous circulation. Increased venous pressure leads to JVD and increased capillary hydrostatic pressure throughout the venous system.

The systemic clinical manifestations include edema of the lower extremities (dependent edema), hepatomegaly (enlargement of the liver), **ascites** (accumulation of fluid in the peritoneal cavity), anorexia and nausea, and weakness and weight gain due to retention of fluid.

Edema usually affects the feet and ankles and worsens when the patient stands or sits for a long period. The edema decreases when the patient elevates the legs. The edema can gradually progress up the legs and thighs and eventually into the external genitalia and lower trunk. Edema in the abdomen, as evidenced by increased abdominal girth, may be the only edema present. Sacral edema is common in patients who are on bed rest, because the sacral area is dependent. Pitting edema, in which indentations in the skin remain after even slight compression with the fingertips (Fig. 30-3), is obvious only after retention of at least 4.5 kg (10 lb) of fluid (4.5 L).

Hepatomegaly and tenderness in the right upper quadrant of the abdomen result from venous engorgement of the liver. The increased pressure may interfere with the liver's ability to function (secondary liver dysfunction). As hepatic dysfunction progresses, increased pressure within the portal vessels may force fluid into the abdominal cavity, causing ascites. Ascites may increase pressure on the stomach and intestines and cause gastrointestinal distress. Hepatomegaly may also increase pressure on the diaphragm, causing respiratory distress.

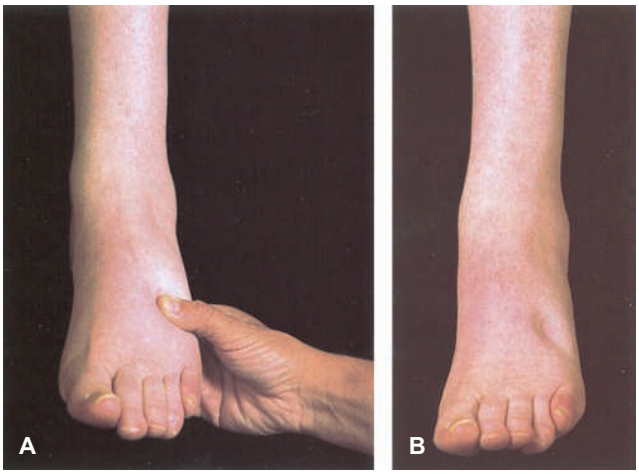


Figure 30-3 Example of pitting edema. **A,** The nurse applies finger pressure to an area near the ankle. **B,** When the pressure is released, an indentation remains in the edematous tissue. From Bickley, L. S. (2007). *Bates' guide to physical examination and history taking* (9th ed.). Philadelphia: Lippincott Williams & Wilkins.

Anorexia (loss of appetite) and nausea or abdominal pain result from the venous engorgement and venous stasis within the abdominal organs. The weakness that accompanies right-sided HF results from reduced CO, impaired circulation, and inadequate removal of catabolic waste products from the tissues.

Assessment and Diagnostic Findings

HF may go undetected until the patient presents with signs and symptoms of pulmonary and peripheral edema. However, the physical signs that suggest HF may also occur with other diseases, such as renal failure and COPD. Assessment of ventricular function is an essential part of the initial diagnostic workup.

An echocardiogram is usually performed to confirm the diagnosis of HF, identify the underlying cause, and determine the EF, which helps identify the type and severity of HF. This information may also be obtained noninvasively by radionuclide ventriculography or invasively by ventriculography as part of a cardiac catheterization procedure. A chest x-ray and an electrocardiogram (ECG) are obtained to assist in the diagnosis. Laboratory studies usually performed during the initial workup include serum electrolytes, blood urea nitrogen (BUN), creatinine, thyroid-stimulating hormone, complete blood cell count, BNP, and routine urinalysis. The BNP level is a key diagnostic indicator of HF; high levels are a sign of high cardiac filling pressure and can aid in the diagnosis of HF (Heart Failure Society of America [HFSA], 2006). The results of these laboratory studies assist in determining the underlying cause and can also be used to establish a baseline to assess effects of treatment. Cardiac stress testing or cardiac catheterization may be performed to determine whether coronary artery disease and cardiac ischemia are causing the HF.

Medical Management

The overall goals of management of HF are to relieve patient symptoms, to improve functional status and quality of life, and to extend survival. Medical management is based on the type, severity, and cause of HF. Specific objectives of medical management include the following:

- Eliminate or reduce any etiologic contributory factors, such as uncontrolled hypertension or atrial fibrillation with a rapid ventricular response
- Optimize pharmacologic and other therapeutic regimens
- Reduce the workload on the heart by reducing preload and afterload
- Promote a lifestyle conducive to cardiac health
- Prevent episodes of acute decompensated HF

Treatment options vary according to the severity of the patient's condition and may include oral and IV medications, major lifestyle changes, supplemental oxygen, implantation of assistive devices, and surgical approaches, including cardiac transplantation.

Managing the patient with HF includes providing comprehensive education and counseling to the patient and family. It is important that the patient and family understand the nature of HF and the importance of their participation

in the treatment regimen. Lifestyle recommendations include restriction of dietary sodium; avoidance of excessive fluid intake, alcohol, and smoking; weight reduction when indicated; and regular exercise. The patient must know how to recognize signs and symptoms that need to be reported to a health care professional.

Pharmacologic Therapy

Several medications are routinely prescribed for systolic HF, including ACE inhibitors, beta-blockers, diuretics, and digitalis (Table 30-3). Target doses for these medications are identified in the AHA/ACC guidelines, and nurses and physicians work collaboratively toward achieving effective dosing of these medications (Albert, 2006).

Angiotensin-Converting Enzyme Inhibitors

ACE inhibitors play a pivotal role in the management of systolic HF. They have been found to relieve the signs and symptoms of HF and significantly decrease mortality and morbidity. ACE inhibitors (eg, lisinopril [Prinivil, Zestril]) slow the progression of HF, improve exercise tolerance, and decrease the number of hospitalizations for HF (Institute for Clinical Systems Improvement [ICSI], 2006). Available as oral and IV medications, ACE inhibitors promote vasodilation and diuresis by decreasing afterload and preload. By doing so, they decrease the workload of the heart. Vasodilation reduces resistance to left ventricular ejection of blood, diminishing the heart's workload and improving ventricular emptying. In promoting diuresis, ACE inhibitors decrease the secretion of aldosterone, a hormone that causes the kidneys to retain sodium and water. ACE inhibitors stimulate the kidneys to excrete sodium and fluid (while retaining potassium), thereby reducing left ventricular filling pressure and decreasing pulmonary congestion. ACE inhibitors may be the first medication prescribed for patients in mild failure—patients with fatigue or DOE but without signs of fluid overload and pulmonary congestion. These agents are also recommended for prevention of heart failure in patients who are at risk (Hunt, et al., 2005).

ACE inhibitors are started at a low dose that is increased every 2 weeks until the optimal dose is achieved and the patient is hemodynamically stable. The final maintenance

Table 30-3 MEDICATIONS USED TO TREAT HEART FAILURE

Medication	Therapeutic Effects	Key Nursing Considerations
Angiotensin-Converting Enzyme (ACE) Inhibitors		
Lisinopril (Prinivil, Zestril) Benazepril (Lotensin) Captopril (Capoten) Enalapril/enalaprilat (Vasotec) Fosinopril (Monopril) Moexipril (Univasc) Perindopril (Aceon) Quinapril (Accupril) Ramipril (Altace) Trandolapril (Mavik)	↓ BP and ↓ afterload Relieves signs and symptoms of HF Prevents progression of HF	Observe for symptomatic hypotension, increased serum K^+, cough, and worsening renal function.
Angiotensin Receptor Blockers (ARBs)		
Valsartan (Diovan) Candesartan (Atacand) Eprosartan (Teveten) Irbesartan (Avapro) Telmisartan (Micardis) Losartan (Cozaar)	↓ BP and ↓ afterload Relieves signs and symptoms of HF Prevents progression of HF	Observe for symptomatic hypotension, increased serum K^+, and worsening renal function.
Beta-adrenergic Blocking Agents (beta-blockers)		
Metoprolol (Lopressor, Toprol) Atenolol (Tenormin) Carvedilol (Coreg)	Dilates blood vessels and ↓ afterload ↓ signs and symptoms of HF Improves exercise capacity	Observe for decreased heart rate, symptomatic hypotension, and fatigue.
Diuretics		
Loop diuretic: Furosemide (Lasix) *Thiazide diuretic:* Metolazone (Zaroxolyn) Hydrochlorothiazide (HCTZ) *Aldosterone antagonist:* Spironolactone (Aldactone)	↓ fluid volume overload ↓ signs and symptoms of HF	Observe for electrolyte abnormalities, renal dysfunction, diuretic resistance, and decreased BP. Carefully monitor I&O and daily weight (see Chart 30-2).
Digitalis		
Digoxin (Lanoxin)	Improves contractility ↓ signs and symptoms of HF	Observe for bradycardia and digitalis toxicity (see Chart 30-3).
Calcium Channel Blockers		
Dihydropyridines: Amlodipine (Norvasc) Felodipine (Plendil)	Vasodilation and reduction of systemic vascular resistance	Observe for symptomatic hypotension, drowsiness, or dizziness.

BP, blood pressure; HF, heart failure; I&O, input and output; ↓, decreases

dose depends on the patient's blood pressure, fluid status, and renal status, as well as the severity of the HF.

Patients receiving ACE inhibitors are monitored for hypotension, hypovolemia, hyperkalemia, and alterations in renal function, especially if they are also receiving diuretics. Hypotension is most likely to develop from ACE inhibitor therapy in patients older than 75 years of age and in those with a systolic blood pressure of 100 mm Hg or less, a serum sodium level of lower than 135 mEq/L, or severe HF. Adjusting the dose or type of diuretic in response to the patient's blood pressure and renal function may result in continued increases in the dosage of ACE inhibitors.

Because ACE inhibitors cause the kidneys to retain potassium, the patient who is also receiving a diuretic may not need to take oral potassium supplements. However, patients receiving potassium-sparing diuretics (which do not cause potassium loss with diuresis) must be carefully monitored for hyperkalemia, an increased level of potassium in the blood. Before the initiation of the ACE inhibitor, hyperkalemic and hypovolemic states must be corrected. ACE inhibitors may be discontinued if the potassium level remains greater than 5.5 mEq/L or if the serum creatinine rises.

Other side effects of ACE inhibitors include a dry, persistent cough that may not respond to cough suppressants. However, cough can also indicate a worsening of ventricular function and failure. Rarely, the cough indicates angioedema. If angioedema affects the oropharyngeal area and impairs breathing, the ACE inhibitor must be stopped immediately.

If the patient cannot continue taking an ACE inhibitor because of development of cough, an elevated creatinine level, or hyperkalemia, an angiotensin II receptor blocker (ARB) or a combination of hydralazine and isosorbide dinitrate is prescribed.

Angiotensin II Receptor Blockers

Although the action of ARBs is different from that of ACE inhibitors, ARBs (eg, valsartan [Diovan]) have similar hemodynamic effects: decreased blood pressure, decreased systemic vascular resistance, and improved cardiac output (ICSI, 2006). Whereas ACE inhibitors block the conversion of angiotensin I to angiotensin II, ARBs block the effects of angiotensin II at the angiotensin II receptor. ACE inhibitors and ARBs also have similar side effects: hyperkalemia, hypotension, and renal dysfunction. ARBs are prescribed as an alternative to ACE inhibitors, especially when patients cannot tolerate ACE inhibitors because of cough.

Hydralazine and Isosorbide Dinitrate

A combination of hydralazine and isosorbide dinitrate may be another alternative for patients who cannot take ACE inhibitors (Hunt, et al., 2005). Nitrates (eg, isosorbide dinitrate) cause venous dilation, which reduces the amount of blood return to the heart and lowers preload. Hydralazine lowers systemic vascular resistance and left ventricular afterload. This combination of medications is also recommended in heart failure guidelines.

Beta-Blockers

Beta-blockers, such as carvedilol (Coreg) and metoprolol (Lopressor, Toprol), have been found to reduce mortality and morbidity in patients with HF by reducing the adverse effects from the constant stimulation of the sympathetic nervous system. Beta-blockers are routinely prescribed in addition to ACE inhibitors, diuretics, and digitalis (ICSI, 2006). In addition, they have been recommended for patients with asymptomatic systolic dysfunction, such as those with a decreased EF, to prevent the onset of symptoms of HF.

Beta-blockers may produce a number of side effects and may potentially exacerbate symptoms of HF. Side effects are most common in the initial few weeks of treatment. The most frequent side effects are dizziness, hypotension, and bradycardia. Because of these side effects, beta-blockers are started at a low dose and only after the patient is stabilized and euvolemic (ie, state of normal volume). The dose is titrated slowly (every 2 weeks), with close monitoring after each dosage increase. If symptoms of HF increase during the titration phase, treatment options include increasing the dose of the diuretic, reducing the dose of the ACE inhibitor, or decreasing the dose of the beta-blocker.

An important nursing role during titration is educating the patient about the potential worsening of symptoms during the early phase of treatment and stressing that improvement may take several weeks. It is very important for nurses to provide support to patients going through this symptom-provoking phase of treatment. Because beta-blockade can cause bronchiole constriction, a beta-1–selective beta-blocker (ie, one that primarily blocks the beta-adrenergic receptor sites in the heart), such as metoprolol, is recommended for patients with well-controlled, mild to moderate asthma. Patients need to be monitored closely for increased asthma symptoms nonetheless, because even cardioselective beta-blockers retain some modest beta-2 effects. Any type of beta-blocker is contraindicated in patients with severe or uncontrolled asthma.

Diuretics

Diuretics are prescribed to remove excess extracellular fluid by increasing the rate of urine produced in patients with signs and symptoms of fluid overload. Of the types of diuretics prescribed for patients with edema from HF, three are most common: loop, thiazide, and aldosterone blocking (potassium-sparing) diuretics. These medications are classified according to their site of action in the kidney and their effects on renal electrolyte excretion and reabsorption.

Loop diuretics, such as furosemide (Lasix), inhibit sodium and chloride reabsorption mainly in the ascending loop of Henle. Patients are generally treated with a loop diuretic first (HFSA, 2006). Thiazide diuretics, such as metolazone (Zaroxolyn), inhibit sodium and chloride reabsorption in the early distal tubules. Both of these classes of diuretics increase the excretion of potassium; therefore, patients treated with these medications must have their serum potassium levels closely scrutinized. A medication from each of these classes may be used for patients in severe HF who are unresponsive to a single diuretic. Diuretics may be most effective if the patient assumes a supine position for 1 or 2 hours after taking them. The need for diuretics can be decreased if the patient avoids excessive fluid intake (eg, more than 2 qt/day) and adheres to a low-sodium diet (eg, less than 2 g/day).

Spironolactone (Aldactone) is a potassium-sparing diuretic that blocks the effects of aldosterone in the late distal tubule and collecting duct. It has been found to be effective in reducing mortality and morbidity in patients with moderate to severe HF (ICSI, 2006). Serum creatinine and potassium levels are monitored frequently (eg, within the first week and then every 4 weeks) when this medication is first administered.

Side effects of diuretics include electrolyte imbalances, symptomatic hypotension (especially with overdiuresis), hyperuricemia (causing gout), and ototoxicity. The type and dose of diuretic prescribed depend on clinical signs and symptoms and renal function. Careful patient monitoring and dose adjustments are necessary to balance the effectiveness of these medications with the side effects (Chart 30-2). Diuretics are administered IV for exacerbations of HF when rapid diuresis is necessary. Diuretics improve the patient's symptoms, provided that renal function is adequate (ICSI, 2006). As HF progresses, there may be a decline in renal function known as cardiorenal syndrome. Patients with this syndrome are resistant to diuretics and may require other interventions to deal with congestive signs and symptoms (Varughese, 2007).

Digitalis

The most commonly prescribed form of digitalis for patients with HF is digoxin (Lanoxin). Until recently, this medication was considered an essential agent for the treatment of HF, but with the advent of new medications, it may or may not be prescribed. Digoxin increases the force of myocardial contraction and slows conduction through the atrioventricular node. It improves contractility, increasing left ventricular output, which also enhances diuresis. Although the use of digitalis does not result in decreased mortality rates among patients with HF, it is effective in decreasing the symptoms of systolic HF and may help prevent hospitalization (ICSI, 2006).

A key concern associated with digitalis therapy is digitalis toxicity. Chart 30-3 summarizes the actions and uses of digoxin along with the nursing surveillance required when it is administered. The patient is observed for indications that digitalis therapy is effective: lessening dyspnea and orthopnea, decrease in pulmonary crackles on auscultation, relief of peripheral edema, weight loss, and increase in activity tolerance. The serum potassium level is measured at intervals because the effect of digoxin is enhanced in the presence of hypokalemia and digoxin toxicity may occur. A serum digoxin level is obtained if there have been changes in the patient's renal function or symptoms.

Calcium Channel Blockers

First-generation calcium channel blockers, such as verapamil (Calan), nifedipine (Procardia), and diltiazem (Cardizem), are contraindicated in patients with systolic HF, although they may be used in patients with diastolic HF (ICSI, 2006). Amlodipine (Norvasc) and felodipine (Plendil), which are dihydropyridine calcium channel blockers, cause vasodilation, reducing systemic vascular resistance. They may be used to improve symptoms, especially in patients with nonischemic cardiomyopathy.

Intravenous Infusions

Nesiritide. Nesiritide (Natrecor), a BNP that is made using recombinant technology, is indicated for patients with acute decompensated HF. BNPs are produced by the failing myocardium to mount a compensatory response in the presence of the myocardial demands, including increased ventricular end-diastolic pressure, myocardial wall stress, and increased release of neurohormones (eg, norepinephrine, renin, aldosterone) that occur with HF. Specifically, BNP binds to vascular smooth muscle and endothelial cells, causing dilation of arteries and veins. It also suppresses the neurohormones responsible for fluid retention, thus promoting diuresis. The result is reduced preload and afterload and increased SV (Fontana, 2006).

Nesiritide causes rapid improvement in the symptoms of HF. It may be used to treat hospitalized patients with acute exacerbations or to prevent exacerbations in outpatients when given as a series of intermittent IV infusions. The most common side effect is dose-related hypotension.

CHART 30-2

PHARMACOLOGY

Administering and Monitoring Diuretic Therapy

When nursing care involves diuretic therapy for conditions such as heart failure, the nurse needs to administer the medication and monitor the patient's response carefully, as follows:

- Administer the diuretic at a time conducive to the patient's lifestyle; for example, early in the day to avoid nocturia.
- Give supplementary potassium with thiazide and loop diuretics as prescribed to replace potassium loss.
- Check laboratory results for electrolyte depletion, especially potassium, magnesium, and sodium; and for electrolyte elevation, especially potassium with potassium-sparing agents.
- Monitor daily weights, intake, and output to assess response. Monitor serum blood urea nitrogen and creatinine for increasing levels, which indicate renal dysfunction.
- Assess lung sounds, jugular vein distention, daily weight, and peripheral, abdominal, or sacral edema to identify response to therapy.
- Monitor for adverse reactions, such as nausea and gastrointestinal distress, vomiting, diarrhea, weakness, headache, fatigue, anxiety or agitation, and cardiac dysrhythmias.
- Assess for signs of volume depletion, such as postural hypotension, dizziness, and balance problems.
- Monitor for glucose intolerance in patients with and without diabetes mellitus who are receiving thiazide diuretics.
- Monitor for potential ototoxicity in patients, especially those with renal failure, who are receiving a loop diuretic.
- Advise patients to avoid prolonged exposure to the sun because of the risk of photosensitivity.
- Monitor for elevated serum uric acid levels and the development of gout.
- Implement nursing actions to facilitate effect of medication, such as positioning patient supine after dose is taken.

CHART 30-3

PHARMACOLOGY

Digoxin Use and Toxicity in Heart Failure

The therapeutic level of digoxin is usually 0.5 to 2.0 mg/mL. Blood samples are usually obtained and analyzed to determine digitalis concentration at least 6 to 10 hours after the last dose. Toxicity may occur despite normal serum levels, and recommended dosages vary considerably.

Preparations

Digoxin

- Tablets: 0.125, 0.25, 0.5 mg (Lanoxin)
- Capsules: 0.05, 0.1, 0.2 mg (Lanoxicaps)
- Elixir: 0.05 mg/mL (Lanoxin Pediatric elixir)
- Injection: 0.25 mg/mL, 0.1 mg/mL (Lanoxin)

Digoxin Toxicity

A serious complication of digoxin therapy is toxicity. Diagnosis of digoxin toxicity is based on the patient's clinical symptoms, which include the following:

- Anorexia, nausea, vomiting, fatigue, depression, and malaise (early effects of digitalis toxicity)
- Changes in heart rate or rhythm; onset of irregular rhythm
- ECG changes indicating SA or AV block; new onset of irregular rhythm indicating ventricular dysrhythmias; and atrial tachycardia with block, junctional tachycardia, and ventricular tachycardia

Reversal of Toxicity

Digoxin toxicity is treated by holding the medication while monitoring the patient's symptoms and serum digoxin level. If the toxicity is severe, digoxin immune FAB (Digibind) may be prescribed. Digibind binds with digoxin and makes it unavailable for use. The Digibind dosage is based on the digoxin level and the patient's weight. Serum digoxin values are not accurate for several days after administration of Digibind because they do not differentiate between bound and unbound digoxin. Because Digibind quickly decreases the amount of available digoxin, an increase in ventricular rate due to atrial fibrillation and worsening of symptoms of HF may ensue shortly after its administration.

Nursing Considerations and Actions

1. Assess the patient's clinical response to digoxin therapy by evaluating relief of symptoms such as dyspnea, orthopnea, crackles, hepatomegaly, and peripheral edema.
2. Monitor the patient for factors that increase the risk of toxicity:
   - Decreased potassium level (hypokalemia), which may be caused by diuretics. Hypokalemia increases the action of digoxin and predisposes patients to digoxin toxicity and dysrhythmias.
   - Use of medications that enhance the effects of digoxin, including oral antibiotics and cardiac drugs that slow AV conduction and can further decrease heart rate.
   - Impaired renal function, particularly in patients age 65 and older. Because digoxin is eliminated by the kidneys, renal function (serum creatinine) is monitored and doses of digoxin are adjusted accordingly.
3. Before administering digoxin, it is standard nursing practice to assess apical heart rate. When the patient's rhythm is atrial fibrillation and the heart rate is less than 60 bpm, or the rhythm becomes regular, the nurse may withhold the medication and notify the physician, because these signs indicate the development of AV conduction block. Although withholding digoxin is a common practice, the medication does not need to be withheld for a heart rate of less than 60 bpm if the patient is in sinus rhythm because digoxin does not affect SA node automaticity. Measuring the PR interval for a patient with cardiac monitoring is more important than the apical pulse in determining whether digoxin should be held.
4. Monitor for gastrointestinal side effects: anorexia, nausea, vomiting, abdominal pain, and distention.
5. Monitor for neurologic side effects: headache, malaise, nightmares, forgetfulness, social withdrawal, depression, agitation, confusion, paranoia, hallucinations, decreased visual acuity, yellow or green halo around objects (especially lights), or "snowy" vision.

AV, atrioventricular; bpm, beats per minute; ECG, electrocardiogram; HF, heart failure; SA, sinoatrial.

Milrinone. Milrinone (Primacor) is a phosphodiesterase inhibitor that delays the release of calcium from intracellular reservoirs and prevents the uptake of extracellular calcium by the cells. This promotes vasodilation, resulting in decreased preload and afterload and reduced cardiac workload. Milrinone is administered intravenously if the patient has not responded to other therapies. The major side effects are hypotension, gastrointestinal dysfunction, increased ventricular dysrhythmias, and, rarely, decreased platelet counts. Blood pressure is monitored closely during and following infusions of milrinone.

Dobutamine. Dobutamine (Dobutrex) is an IV medication administered to patients with significant left ventricular dysfunction and hypoperfusion. A catecholamine, dobutamine stimulates the beta-1–adrenergic receptors. Its major action is to increase cardiac contractility. However, at high doses, it also increases the heart rate and can precipitate ectopic beats and tachydysrhythmias. Because it also increases atrioventricular conduction, care must be taken in patients who have underlying atrial fibrillation.

IV inotropes such as dobutamine and milrinone are reserved for patients with severe ventricular dysfunction, who must be monitored continuously via ECG. Some patients require admission to the intensive care unit and hemodynamic monitoring with a pulmonary artery catheter or impedance cardiography. A form of noninvasive monitoring, impedance cardiography uses external electrodes placed on the chest that generate a small current between the electrodes to assess fluid status and cardiac output (Folan & Funk, 2008). Hemodynamic data guide therapy and assess patient response (Varughese, 2007).

Medications for Diastolic Dysfunction

Patients with predominant diastolic HF may be treated with different medications than indicated for patients with systolic HF. After contributing causes such as hypertension and

ischemic heart disease are evaluated and treated, patients may be started on beta-blockers and other HF medications. An effort is made to restore normal sinus rhythm in patients with concomitant atrial fibrillation (Hunt, et al., 2005).

Other Medications for Heart Failure

Anticoagulants may be prescribed, especially if the patient has a history of atrial fibrillation or a thromboembolic event. Medications that manage hyperlipidemia (eg, statins) are also routinely prescribed for the HF population (Albert, 2006). Nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen (Motrin) should be avoided because they decrease renal perfusion, especially in the elderly.

Nutritional Therapy

A low-sodium (2 to 3 g/day) diet and avoidance of drinking excessive amounts of fluid are usually recommended. Dietary restriction of sodium reduces fluid retention and the symptoms of peripheral and pulmonary congestion. The purpose of sodium restriction is to decrease the amount of circulating blood volume, which decreases myocardial work. A balance needs to be achieved between the ability of the patient to comply with the diet and the recommended dietary restriction. Any change in diet needs to be made with consideration of good nutrition as well as the patient's likes, dislikes, and cultural food patterns. Patient compliance is important because dietary indiscretions may result in severe exacerbations of HF requiring hospitalization (Albert, 2005). However, behavioral changes in this area are difficult for many patients.

Additional Therapy

Supplemental Oxygen

Oxygen therapy may become necessary as HF progresses. The need is based on the degree of pulmonary congestion and resulting hypoxia. Some patients require supplemental oxygen only during periods of activity.

Other Interventions

A number of procedures and surgical approaches may benefit patients with HF. If the patient has underlying coronary artery disease, coronary artery revascularization with percutaneous coronary intervention or coronary artery bypass surgery (see Chapter 28) may be considered. Ventricular function may improve in some patients when coronary flow is increased.

Patients with HF are at high risk for dysrhythmias. Sudden cardiac death is a common cause of death among patients with advanced HF (Jensen, Galvin, Thompson, et al., 2007). In patients with severe left ventricular dysfunction and the possibility of life-threatening dysrhythmias, placement of an implantable cardioverter defibrillator (ICD) can prevent sudden cardiac death and extend survival (Ezekowitz, Rowe, Dryden, et al., 2007).

In the patient with HF who does not improve with standard therapy, **cardiac resynchronization therapy (CRT)** is another treatment that may be beneficial. CRT involves the use of a biventricular pacemaker to treat electrical conduction defects. Left bundle branch block, a type of delayed conduction that is frequently seen in patients with HF, results in dyssynchronous conduction and contraction of the right and left ventricles, which can further decrease EF (Jensen, et al., 2007). Use of a pacing device with leads placed in the right atrium, right ventricle, and left ventricular cardiac vein can synchronize the contractions of the right and left ventricles. This intervention has been shown to improve cardiac output, optimize myocardial energy consumption, reduce mitral regurgitation, and slow the ventricular remodeling process. For selected patients, this results in fewer symptoms and increased functional status (Saul, 2007). Combination devices are available for patients who require CRT and an ICD.

Ultrafiltration is an alternative intervention for patients with severe fluid overload. A dual-lumen central IV catheter is used, which could be a peripherally inserted central catheter or a midline or central catheter, and the patient's blood is circulated through a small bedside machine. Liters of excess fluid and plasma are removed slowly from the patient's intravascular circulating volume over a number of hours. This fluid is then discarded, and the filtered blood products are returned to the patient (Constanzo, Saltzberg, O'Sullivan, et al., 2005; Soat, 2008; Walsh & Wagemester, 2007).

For some patients with end-stage HF, cardiac transplantation is the only option for long-term survival. Some of these patients require mechanical circulatory assistance with an implanted ventricular assist device as a bridge therapy to cardiac transplantation (see Chapter 29).

Gerontologic Considerations

Several normal age-related changes increase the frequency of HF: increased systolic blood pressure, increased ventricular wall thickness, and increased myocardial fibrosis. Elderly people may present with atypical signs and symptoms: fatigue, weakness, and somnolence. Decreased renal function makes the elderly patient resistant to diuretics and more sensitive to changes in volume. The administration of diuretics to elderly men requires nursing surveillance for bladder distention caused by urethral obstruction from an enlarged prostate gland. The bladder may be assessed with an ultrasound scanner or the suprapubic area palpated for an oval mass and percussed for dullness, indicative of bladder fullness. Urinary frequency and urgency may be particularly stressful to the elderly patient.

NURSING PROCESS

The Patient With Heart Failure

Despite advances in treatment of HF, morbidity and mortality remains high. Nurses have a major impact on outcomes for patients with HF.

Assessment

The nursing assessment for the patient with HF focuses on observing for effectiveness of therapy and for the patient's ability to understand and implement self-management strategies. Signs and symptoms of pulmonary and systemic fluid overload are recorded and reported immediately so that adjustments can be made in therapy. The nurse also

explores the patient's emotional response to the diagnosis of HF because it is a chronic and often progressive condition.

Health History

The health history focuses on the signs and symptoms of HF, such as dyspnea, shortness of breath, fatigue, and edema. Sleep disturbances, particularly sleep suddenly interrupted by shortness of breath, may be reported. Patients are asked about the number of pillows needed for sleep, edema, abdominal symptoms, altered mental status, activities of daily living, and the activities that cause fatigue. The nurse explores each patient's understanding of HF, self-management strategies, and the ability and willingness to adhere to those strategies. Patients are asked to identify the impact that HF has had on their quality of life and successful coping skills that they have used. Family and significant others are often included in these discussions.

Physical Examination

The lungs are auscultated to detect crackles and wheezes. Crackles are produced by the sudden opening of edematous small airways and alveoli. They may be heard at the end of inspiration and are not cleared with coughing. Wheezing may also be heard in some patients. The rate and depth of respirations are also documented.

The heart is auscultated for an S_3 heart sound, a sign that the heart is beginning to fail and that increased blood volume fills the ventricle with each beat. HR and rhythm are also documented. When the heart rate is rapid, the SV decreases because the ventricle has less time to fill. This in turn produces increased pressure in the atria and eventually in the pulmonary vascular bed.

JVD is also assessed; distention greater than 3 cm above the sternal angle is considered abnormal. This is an estimate, not a precise measurement, of central venous pressure.

Sensorium and level of consciousness must be evaluated. As the volume of blood ejected by the heart decreases, so does the amount of oxygen transported to the brain.

The nurse assesses dependent parts of the patient's body for perfusion and edema. With significant decreases in SV, there is a decrease in perfusion to the periphery, causing the skin to feel cool and appear pale or cyanotic. The feet and lower legs are examined for edema; if the patient is supine in bed, the sacrum and back are also assessed for edema. Fingers and hands may also become edematous.

The liver is assessed for hepatojugular reflux. The patient is asked to breathe normally while manual pressure is applied over the right upper quadrant of the abdomen for 30 to 60 seconds. If neck vein distention increases more than 1 cm, the finding is positive for increased venous pressure.

If the patient is hospitalized, the nurse measures urinary output carefully to establish a baseline against which to assess the effectiveness of diuretic therapy. Intake and output records are rigorously maintained. It is important to know whether the patient has ingested more fluid than he or she has excreted (positive fluid balance), which is then compared with any gain in weight. The patient must be monitored for oliguria (diminished urine output, less than 500 mL/24 h) or **anuria** (urine output less than 50 mL/24 h).

The patient is weighed daily in the hospital or at home, at the same time of day, with the same type of clothing, and on the same scale. If there is a significant change in weight (ie, 2- to 3-lb increase in a day or 5-lb increase in a week), the physician is notified and medications are adjusted (eg, the diuretic dose is increased).

Diagnosis

Nursing Diagnoses

Based on the assessment data, major nursing diagnoses for the patient with HF may include the following:

- Activity intolerance and fatigue related to decreased CO
- Excess fluid volume related to the HF syndrome
- Anxiety related to breathlessness from inadequate oxygenation
- Powerlessness related to chronic illness and hospitalizations
- Ineffective therapeutic regimen management related to lack of knowledge

Collaborative Problems/Potential Complications

Based on the assessment data, potential complications that may develop include the following:

- Hypotension, poor perfusion, and cardiogenic shock (see Chapter 15)
- Dysrhythmias (see Chapter 27)
- Thromboembolism (see Chapter 31)
- Pericardial effusion and cardiac tamponade (see Chapter 29)

Planning and Goals

Major goals for the patient may include promoting activity and reducing fatigue, relieving fluid overload symptoms, decreasing anxiety or increasing the patient's ability to manage anxiety, encouraging the patient to verbalize his or her ability to make decisions and influence outcomes, and teaching the patient about the self-care program.

Nursing Interventions

Promoting Activity Tolerance

Reduced physical activity caused by HF symptoms leads to physical deconditioning that worsens the patient's symptoms and exercise tolerance. Prolonged bed rest, which may be self-imposed, should be avoided because of its deconditioning effects and risks such as pressure ulcers (especially in edematous patients), venous thrombosis, and pulmonary embolism. An acute illness that exacerbates HF symptoms or that requires hospitalization may be an indication for temporary bed rest. Otherwise, a total of 30 minutes of physical activity every day should be encouraged (HFSA, 2006). Exercise training has many favorable effects for HF, including increasing functional capacity and decreasing dyspnea. The exercise regimen should include 5 minutes of warm-up activities followed by about 30 minutes of exercise at the prescribed intensity level. A typical program for a patient with HF might include a daily walking regimen, with duration increased over a 6-week period. The physician, nurse, and patient collaborate to develop a schedule that promotes pacing and prioritization of activities. The schedule should alternate activities with periods of rest and avoid having two significant energy-consuming activities occur on the same day or in immediate succession.

Before undertaking physical activity, the patient should be given the following safety guidelines:

- Begin with a few minutes of warm-up activities.
- Avoid performing physical activities outside in extreme hot, cold, or humid weather.
- Ensure that you are able to talk during the physical activity; if you cannot do so, decrease the intensity of activity.
- Wait 2 hours after eating a meal before performing the physical activity.
- Stop the activity if severe shortness of breath, pain, or dizziness develops.
- End with cool-down activities and a cool-down period.

Because some patients may be severely debilitated, they may need to limit physical activities to only 3 to 5 minutes at a time, one to four times per day. The patient should increase the duration of the activity, then the frequency, before increasing the intensity of the activity.

Barriers to performing other activities are identified, and methods of adjusting an activity are discussed. For example, vegetables can be chopped or peeled while sitting at the kitchen table rather than standing at the kitchen counter. Small, frequent meals decrease the amount of energy needed for digestion while providing adequate nutrition. The nurse helps the patient identify peak and low periods of energy, planning energy-consuming activities for peak periods. For example, the patient may prepare the meals for the entire day in the morning. Pacing and prioritizing activities help maintain the patient's energy to allow participation in regular physical activity.

The patient's response to activities needs to be monitored. If the patient is hospitalized, vital signs and oxygen saturation level are monitored before, during, and immediately after an activity to identify whether they are within the desired range. HR should return to baseline within 3 minutes following the activity. If the patient is at home, the degree of fatigue felt after the activity can be used to assess the response. If the patient tolerates the activity, short-term and long-term goals can be developed to gradually increase the intensity, duration, and frequency of activity.

Adherence to exercise training is essential if the patient is to benefit from it, but it may be difficult for patients with other conditions (eg, arthritis) and longer duration of heart failure. Referral to a cardiac rehabilitation program may be needed, especially for HF patients with recent myocardial infarction, recent open heart surgery, or increased anxiety. A supervised program may also benefit those who need a structured environment, significant educational support, regular encouragement, and interpersonal contact.

Managing Fluid Volume

Patients with severe HF may receive IV diuretic therapy, but patients with less severe symptoms may receive oral diuretic medication. Oral diuretics should be administered early in the morning so that diuresis does not interfere with the patient's nighttime rest. Discussing the timing of medication administration is especially important for elderly patients who may have urinary urgency or incontinence. A single dose of a diuretic may cause the patient to excrete a large volume of fluid shortly after its administration.

The patient's fluid status is monitored closely, auscultating the lungs, monitoring daily body weight, and assisting the patient to adhere to a low-sodium diet by reading food labels and avoiding high-sodium foods such as canned, processed, and convenience foods (Chart 30-4). If the diet

CHART 30-4

HEALTH PROMOTION

Facts About Dietary Sodium

Although the major source of sodium in the average American diet is salt, many types of natural foods contain varying amounts of sodium. Even if no salt is added in cooking and if salty foods are avoided, the daily diet will still contain about 2000 mg of sodium. Fresh fruits and vegetables are low in sodium and should be encouraged.

Additives in Food

In general, food prepared at home is lower in sodium than restaurant or processed foods. Added food substances (additives), such as sodium alginate, which improves food texture; sodium benzoate, which acts as a preservative; and disodium phosphate, which improves cooking quality in certain foods, increase the sodium intake when included in the daily diet. Therefore, patients on low-sodium diets should be advised to check labels carefully for such words as "salt" or "sodium," especially on canned foods. For example, without looking at the sodium content per serving found on the nutrition labels, when given a choice between a serving of potato chips and a cup of canned cream of mushroom soup, most would think that soup is lower in sodium. However, when the labels are examined, the lower sodium choice is found to be the chips. Although potato chips are *not* recommended in a low sodium diet, this example illustrates that it is important to read food labels to determine both sodium content and serving size.

Nonfood Sodium Sources

Sodium is also contained in municipal water. Water softeners also increase the sodium content of drinking water. Patients on sodium-restricted diets should be cautioned against using nonprescription medications such as antacids, cough syrups, and laxatives. Salt substitutes may be allowed, but it is recognized that they are high in potassium. Over-the-counter medications should not be used without first consulting the physician.

Promoting Dietary Adherence

If patients find food unpalatable because of the dietary sodium restrictions and/or the taste disturbances caused by the medications, they may refuse to eat or to comply with the dietary regimen. For this reason, severe sodium restrictions should be avoided and the amount of medication should be balanced with the patient's ability to restrict dietary sodium. A variety of flavorings, such as lemon juice, vinegar, and herbs, may be used to improve the taste of the food and facilitate acceptance of the diet. The patient's food preferences should be taken into account—diet counseling and educational handouts can be geared to individual and ethnic preferences. It is very important to involve the family in the dietary teaching.

includes fluid restriction, the nurse can assist the patient to plan fluid intake throughout the day while respecting the patient's dietary preferences. If the patient is receiving IV fluids, the amount of fluid needs to be monitored closely, and the physician or pharmacist can be consulted about the possibility of maximizing the amount of medication in the same volume of IV fluid (eg, double concentrating to decrease the fluid volume administered).

The patient is positioned or taught how to assume a position that facilitates breathing. The number of pillows may be increased, the head of the bed may be elevated, or the patient may sit in a comfortable armchair. In these positions, the venous return to the heart (preload) is reduced, pulmonary congestion is alleviated, and pressure on the diaphragm is minimized. The lower arms are supported with pillows to eliminate the fatigue caused by the pull of the patient's weight on the shoulder muscles.

Because decreased circulation in edematous areas increases the risk of pressure ulcers, the nurse assesses for skin breakdown and institutes preventive measures. Frequent changes of position, positioning to avoid pressure, and leg exercises may help prevent pressure ulcers.

Controlling Anxiety

Because patients with HF have difficulty maintaining adequate oxygenation, they are likely to be restless and anxious and feel overwhelmed by breathlessness. These symptoms tend to intensify at night and may interfere with sleep. Emotional stress stimulates the sympathetic nervous system, which causes vasoconstriction, elevated arterial pressure, and increased heart rate. This sympathetic response increases the cardiac workload. By decreasing anxiety, the patient's cardiac workload also is decreased. Oxygen may be administered during an acute event to diminish the work of breathing and to increase the patient's comfort.

When the patient exhibits anxiety, the nurse takes steps to promote physical comfort and provide psychological support. In many cases, a family member's presence provides reassurance. Pet visitation or animal-assisted therapy can also be beneficial (Cole, 2007).

Along with reassurance, the nurse can begin teaching the patient ways to control anxiety and avoid anxiety-provoking situations. The patient learns how to identify factors that contribute to anxiety and how to use relaxation techniques to control anxious feelings. As the patient's anxiety decreases, cardiac function may improve and symptoms of HF may decrease.

NURSING ALERT

In cases of confusion and anxiety reactions that affect the patient's safety, the use of restraints should be avoided. Restraints are likely to be resisted, and resistance inevitably increases the cardiac workload.

The patient who insists on getting out of bed at night can be seated comfortably in an armchair. As cerebral and systemic circulation improves, the degree of anxiety decreases and the quality of sleep improves.

In addition to anxiety, patients with HF have a high incidence of depression; depression is associated with increased morbidity and mortality in patients with HF (Jiang, Kuchibhatia, Clary, et al., 2007). Patients with HF should be screened for this condition.

Minimizing Powerlessness

Patients need to recognize that they are not helpless and that they can influence the direction of their lives and the outcomes of treatment. The patient is assessed for factors that contribute to a sense of powerlessness. Contributing factors may include lack of knowledge and lack of opportunities to make decisions, particularly if health care providers and family members behave in controlling ways. If the patient is hospitalized, hospital policies may limit the patient's ability to make decisions (eg, what time to have meals or to take medications).

Taking time to listen actively to patients encourages them to express their concerns and ask questions. Other strategies include providing the patient with decision-making opportunities, such as when activities are to occur or encouraging food and fluid choices consistent with the dietary restrictions. Encouragement is provided, progress is identified, and the patient is assisted to differentiate between factors that can and cannot be controlled.

Monitoring and Managing Potential Complications

Many potential problems associated with HF therapy relate to the use of diuretics:

- Excessive and repeated diuresis can lead to hypokalemia (ie, potassium depletion). Signs include ventricular dysrhythmias, hypotension, muscle weakness, and generalized weakness. Hypokalemia poses problems for the patient with HF because it markedly weakens cardiac contractions. In patients receiving digoxin, hypokalemia can lead to digitalis toxicity. Digitalis toxicity and hypokalemia increase the likelihood of dangerous dysrhythmias (see Chart 30-3). Patients with HF may also develop low levels of magnesium, which can add to the risk of dysrhythmias.
- Hyperkalemia may occur, especially with the use of ACE inhibitors, ARBs, or spironolactone.
- Prolonged diuretic therapy may produce hyponatremia (deficiency of sodium in the blood), which results in disorientation, apprehension, weakness, fatigue, malaise, and muscle cramps.
- Volume depletion from excessive fluid loss may lead to dehydration and hypotension. ACE inhibitors and beta-blockers may contribute to the hypotension.
- Other problems associated with diuretics include increased serum creatinine and hyperuricemia (excessive uric acid in the blood), which leads to gout.

Promoting Home and Community-Based Care

TEACHING PATIENTS SELF-CARE. The nurse provides patient education and involves the patient in the therapeutic regimen to promote understanding and adherence to the plan. When the patient recognizes that the diagnosis of HF can be successfully managed with lifestyle changes and medications, recurrences of acute HF lessen, unnecessary hospitalizations decrease, and life expectancy increases. The Joint Commission and other agencies have established standards pertaining to the education of patients with HF.

Nurses teach patients and their families about medication management, low-sodium diets, activity and exercise recommendations, smoking cessation, learning to recognize the signs and symptoms of worsening HF, and when to contact a health care provider (Albert, 2006; Varughese, 2007). Although noncompliance is not well understood, interventions that may promote compliance include teaching to ensure accurate understanding. A basic home teaching plan for the patient with HF is presented in Chart 30-5.

The patient and family members are supported and encouraged to ask questions so that information can be clarified and understanding enhanced. It is important to be aware of cultural factors and adapt the teaching plan accordingly. Patients and their families need to be informed that the progression of the disease is influenced in part by choices made about health care and the decisions about following the treatment plan. They also need to be informed that health care providers are available to assist them in reaching their health care goals. Patients and family members need to make the decisions about the treatment plan and need to understand the possible outcomes of those decisions. The treatment plan is based on the patient's goals rather than on what health care providers think is needed.

CONTINUING CARE. Success in management of HF requires a complex medical regimen and multiple lifestyle changes. Assistance may be provided through home health care, an HF clinic, or telehealth management.

Depending on the patient's physical status and the availability of family assistance, a home care referral may be indicated for a patient who has been hospitalized. Elderly patients and those who have long-standing heart disease with compromised physical stamina often require assistance with the transition to home after hospitalization for an acute episode of HF. The home care nurse assesses the physical environment of the home and makes suggestions for adapting the home environment to meet the patient's activity limitations. If stairs are a concern, the patient can plan the day's activities so that stair climbing is minimized; for some patients, a temporary bedroom may be set up on the main level of the home. The home care nurse works with the patient and family to maximize the benefits of these changes.

The home care nurse also reinforces and clarifies information about dietary changes and fluid restrictions, the

CHART 30-5 HOME CARE CHECKLIST

The Patient With Heart Failure

At the completion of the home care instruction, the patient or caregiver will be able to:	PATIENT	CAREGIVER
• Identify heart failure as a chronic disease that can be managed with medications and specific self-management behaviors.	✔	✔
• Take or administer medications daily, exactly as prescribed.	✔	✔
• Monitor effects of medication such as changes in breathing and edema.	✔	✔
• Know signs and symptoms of orthostatic hypotension and how to prevent it.	✔	✔
• Weigh self daily at the same time with same clothes.	✔	
• Restrict sodium intake to 2000–3000 mg daily: adapt diet by examining nutrition labels to check sodium content per serving; avoid canned or processed foods; eat fresh or frozen foods; consult the written diet plan and the list of permitted and restricted foods; avoid salt use; and avoid excesses in eating and drinking.	✔	✔

• Review activity program.
  • Participate in a daily exercise program.
  • Increase walking and other activities gradually, provided they do not cause unusual fatigue or dyspnea.
  • Conserve energy by balancing activity with rest periods.
  • Avoid activity in extremes of heat and cold, which increase the work of the heart.
  • Recognize that air conditioning may be essential in a hot, humid environment.
	✔	

• Develop methods to manage and prevent stress.
  • Avoid tobacco.
  • Avoid alcohol.
  • Engage in diversional activities, meditation, guided imagery, or music therapy.
	✔	

• Keep regular appointments with physician or clinic.	✔	✔
• Be alert for symptoms that may indicate recurring heart failure. • Know how to contact health care provider.	✔	✔

• Report immediately to the physician or clinic any of the following:
  • Gain in weight of 2–3 lb (0.9–1.4 kg) in 1 day, or 5 lb (2.3 kg) in 1 week.
  • Loss of appetite.
  • Unusual shortness of breath with activity.
  • Swelling of ankles, feet, or abdomen.
  • Persistent cough.
  • Development of restless sleep; increase in number of pillows needed to sleep.
	✔	✔

need to monitor symptoms and daily body weights, and the importance of obtaining follow-up health care. Assistance may be given in scheduling and keeping appointments as well. The patient is encouraged to gradually increase his or her self-care and responsibility for carrying out the therapeutic regimen. Assistance from home health nurses can result in fewer exacerbations of HF, lower costs, and improved quality of life (Anderson, 2007).

Disease management programs are important components in the successful management of HF (HFSA, 2006). HF clinics offer individualized disease management strategies. Referral to an HF clinic gives the patient access to education, professional staff, and timely adjustments to treatment regimens. Research has shown that patients managed through HF clinics have fewer exacerbations of HF, fewer hospitalizations, decreased costs of medical care, and increased quality of life (HFSA, 2006).

Telehealth management can provide the frequent contact necessary to manage HF without requiring frequent visits to health care providers. A variety of techniques ranging from simple telephone monitoring to sophisticated computer and video connections that monitor daily weight, vital signs, and symptoms may be used. Telehealth management can also decrease costs and hospitalizations for exacerbations of HF.

END-OF-LIFE CONSIDERATIONS. Because HF is a chronic and often progressive condition, patients and families need to consider issues related to the end of life. Although the prognosis in HF patients may be uncertain, issues often arise sooner or later related to the patient's thoughts and possible concerns about the use of complex treatment options (eg, ultrafiltration for fluid overload, implantation of a ventricular assist device). Discussions concerning the use of technology, preferences for end-of-life care, and advance directives should take place while the patient is able to participate and express preferences (Dougherty, Pyper, Au, et al., 2007).

Evaluation

Expected Patient Outcomes

1. Demonstrates tolerance for increased activity
   a. Describes adaptive methods for usual activities
   b. Schedules activities to conserve energy and reduce fatigue and dyspnea
   c. Maintains heart rate, blood pressure, respiratory rate, and pulse oximetry within the targeted range
2. Maintains fluid balance
   a. Exhibits decreased peripheral and sacral edema
   b. Demonstrates methods for preventing edema
3. Is less anxious
   a. Avoids situations that produce stress
   b. Sleeps comfortably at night
   c. Reports decreased stress and anxiety
   d. Denies symptoms of depression
4. Makes sound decisions regarding care and treatment
   a. Demonstrates ability to influence outcomes
5. Adheres to self-care regimen
   a. Performs and records daily weights
   b. Ensures dietary intake includes no more than 2 to 3 g of sodium per day
   c. Takes medications as prescribed
   d. Reports any unusual symptoms or side effects

Pulmonary Edema

Pulmonary edema is the abnormal accumulation of fluid in the interstitial spaces of the lungs that diffuses into the alveoli.

Pathophysiology

Pulmonary edema is an acute event that results from left ventricular failure. It can occur acutely, such as with MI, or it can occur as an exacerbation of chronic HF. Myocardial scarring can limit the distensibility of the ventricle and render it vulnerable to a sudden increase in workload. With increased resistance to left ventricular filling, blood backs up into the pulmonary circulation. The patient quickly develops pulmonary edema, sometimes called "flash pulmonary edema," from the blood volume overload in the lungs. Pulmonary edema can also be caused by noncardiac disorders, such as renal failure, and other conditions that cause the body to retain fluid. The pathophysiology is similar to that seen in HF, in that the left ventricle cannot handle the volume overload and blood volume and pressure buildup in the left atrium. The rapid increase in atrial pressure results in an acute increase in pulmonary venous pressure, which produces an increase in hydrostatic pressure that forces fluid out of the pulmonary capillaries into the interstitial spaces and alveoli. Lymphatic drainage of the excess fluid is ineffective.

The fluid within the alveoli mixes with air, producing the classic symptom of pulmonary edema—frothy pink (blood-tinged) sputum. The fluid within the alveoli creates a diffusion block that severely impairs gas exchange. The result is hypoxemia, which is often severe. The onset may be preceded by premonitory symptoms of pulmonary congestion, but the condition also may develop quickly in the patient with a ventricle that has little reserve to meet increased oxygen needs.

Clinical Manifestations

As a result of decreased cerebral oxygenation, the patient becomes increasingly restless and anxious. Along with a sudden onset of breathlessness and a sense of suffocation, the patient's hands become cold and moist, the nail beds become cyanotic (bluish), and the skin turns ashen (gray). The pulse is weak and rapid, and the neck veins are distended. Incessant coughing may occur, producing increasing quantities of foamy sputum. As pulmonary edema progresses, the patient's anxiety and restlessness increase; the patient becomes confused, then stuporous. Breathing is rapid, noisy, and moist sounding. The patient's oxygen saturation is significantly decreased. The patient, nearly suffocated by the blood-tinged, frothy fluid filling the alveoli, is literally drowning in secretions. The situation demands emergent action.

Assessment and Diagnostic Findings

The diagnosis is made by evaluating the clinical manifestations resulting from pulmonary congestion. A chest x-ray may be obtained to confirm the extent of pulmonary vessel and tissue engorgement. Abrupt onset of signs of left-sided HF (eg, crackles on auscultation of the lungs) may occur without evidence of right-sided HF (eg, no JVD, no dependent edema).

to these classifications has been reached. Three categories are frequently cited: hyperacute, acute, and subacute liver failure. In hyperacute liver failure, the duration of jaundice before the onset of encephalopathy is 0 to 7 days; in acute liver failure, it is 8 to 28 days; and in subacute liver failure, it is 28 to 72 days. The prognosis for fulminant hepatic failure is much worse than for chronic liver failure. However, in fulminant failure, the hepatic lesion is potentially reversible, and survival rates are approximately 20% to 50%, depending greatly on the cause of liver failure. Those who do not survive die of massive hepatocellular injury and necrosis (Wolfe, 2006).

Viral hepatitis is a common cause of fulminant hepatic failure; other causes include toxic medications (eg, acetaminophen) and chemicals (eg, carbon tetrachloride), metabolic disturbances (eg, Wilson's disease, a hereditary syndrome with deposition of copper in the liver), and structural changes (eg, Budd-Chiari syndrome, an obstruction to outflow in major hepatic veins).

Jaundice and profound anorexia may be the initial reasons the patient seeks health care. Fulminant hepatic failure is often accompanied by coagulation defects, renal failure and electrolyte disturbances, cardiovascular abnormalities, infection, hypoglycemia, encephalopathy, and cerebral edema.

The key to optimized treatment is rapid recognition of acute liver failure and intensive intervention. Supporting the patient in the ICU and assessing the indications for and feasibility of liver transplantation are hallmarks of management of this population. The use of antidotes for certain conditions may be indicated such as *N*-acetylcysteine for acetaminophen toxicity and penicillin for mushroom poisoning. Treatment modalities may include plasma exchanges (plasmapheresis) to correct coagulopathy and to stabilize the patient awaiting liver transplantation and prostaglandin therapy to enhance hepatic blood flow; however, more clinical trials are needed to determine the effects or outcomes of these treatments. Hepatocytes within synthetic fiber columns have been tested as liver support systems (liver assist devices) to provide a bridge to transplantation.

Research into interventions for acute liver failure has begun to focus on techniques that combine the efficacy of a whole liver with the convenience and biocompatibility of hemodialysis. The acronyms ELAD (*e*xtracorporeal *l*iver *a*ssist *d*evices) and BAL (*bio*artificial *l*iver) have been used to describe these hybrid devices. These short-term devices, which remain experimental, may help patients survive until transplantation is possible. The BAL device exposes separated plasma to a cartridge containing porcine liver cells after the plasma has flowed through a charcoal column that removes substances toxic to hepatocytes. The ELAD exposes whole blood to cartridges containing human hepatoblastoma cells, resulting in removal of toxic substances. In the near future, similar extracorporeal circuits using **xenografts** may be studied as a bridge to liver transplantation. These approaches appear promising and have had success in animal studies. In human clinical application, the use of various BAL systems has resulted in improved neurologic and biochemical parameters. Adding albumin to the dialysate is effective in removing protein-bound toxins and is potentially useful in unstable patients with fulminant liver failure (Rodes, et al., 2007). To fully determine the clinical applicability of such systems on outcomes and survival rates, controlled, randomized clinical trials in large patient groups are required.

In patients who have fulminant liver failure with stage 4 encephalopathy, there is a high risk of cerebral edema, a life-threatening complication. The cause is not fully understood, although disruption of the blood–brain barrier and plasma leakage into the cerebrospinal fluid may be one cause. An increase in the intracellular osmolarity within cerebral astrocyte cells, possibly related to increased sodium and glutamine in these cells, may be another (Rodes, et al., 2007). These patients require intracranial pressure monitoring. Measures to promote adequate cerebral perfusion include careful fluid balance and hemodynamic assessments, a quiet environment, and diuresis with mannitol (Osmitrol), an osmotic diuretic.

Use of barbiturate anesthesia or pharmacologic paralysis and sedation is indicated to prevent surges in intracranial pressure related to agitation. Other support measures include monitoring for and treating hypoglycemia, coagulopathies, and infection. Despite these treatment modalities, the mortality rate remains high. Consequently, liver transplantation (discussed later) has become the treatment of choice for fulminant hepatic failure.

HEPATIC CIRRHOSIS

Cirrhosis is a chronic disease characterized by replacement of normal liver tissue with diffuse fibrosis that disrupts the structure and function of the liver. There are three types of cirrhosis or scarring of the liver:

- Alcoholic cirrhosis, in which the scar tissue characteristically surrounds the portal areas. This is most frequently caused by chronic alcoholism and is the most common type of cirrhosis.
- Postnecrotic cirrhosis, in which there are broad bands of scar tissue. This is a late result of a previous bout of acute viral hepatitis.
- Biliary cirrhosis, in which scarring occurs in the liver around the bile ducts. This type of cirrhosis usually results from chronic biliary obstruction and infection (cholangitis); it is much less common than the other two types.

The portion of the liver chiefly involved in cirrhosis consists of the portal and the periportal spaces, where the bile canaliculi of each lobule communicate to form the liver bile ducts. These areas become the sites of inflammation, and the bile ducts become occluded with inspissated (thickened) bile and pus. The liver attempts to form new bile channels; hence, there is an overgrowth of tissue made up largely of disconnected, newly formed bile ducts and surrounded by scar tissue.

Pathophysiology

Although several factors have been implicated in the etiology of cirrhosis, alcohol consumption is considered the major causative factor. Cirrhosis occurs with greatest frequency among people with alcoholism. Although nutritional deficiency with reduced protein intake contributes to liver

destruction in cirrhosis, excessive alcohol intake is the major causative factor in fatty liver and its consequences. However, cirrhosis has also occurred in people who do not consume alcohol and in those who consume a normal diet and have a high alcohol intake.

Some people appear to be more susceptible than others to this disease, whether or not they have alcoholism or are malnourished. Other factors may play a role, including exposure to certain chemicals (carbon tetrachloride, chlorinated naphthalene, arsenic, or phosphorus) or infectious schistosomiasis. Twice as many men as women are affected, although, for unknown reasons, women are at greater risk for development of alcohol-induced liver disease. Most patients are between 40 and 60 years of age. Each year more than 27,000 people die of chronic liver diseases and cirrhosis in the United States (Rodes, et al., 2007).

Alcoholic cirrhosis is characterized by episodes of necrosis involving the liver cells, which sometimes occur repeatedly throughout the course of the disease. The destroyed liver cells are gradually replaced by scar tissue. Eventually, the amount of scar tissue exceeds that of the functioning liver tissue. Islands of residual normal tissue and regenerating liver tissue may project from the constricted areas, giving the cirrhotic liver its characteristic hobnail appearance. The disease usually has an insidious onset and a protracted course, occasionally proceeding over a period of 30 or more years.

The prognoses for different forms of cirrhosis caused by various liver diseases have been investigated in several studies. Of the many prognostic indicators, the Child-Pugh classification seems most useful in predicting the outcome of patients with liver disease (Table 39-5). It is also used in choosing management approaches.

Clinical Manifestations

Signs and symptoms of cirrhosis increase in severity as the disease progresses. Their severity is used to categorize the disorder as compensated or decompensated cirrhosis (Chart 39-11). Compensated cirrhosis, with its less severe, often vague symptoms, may be discovered secondarily at a routine physical examination. The hallmarks of decompensated cirrhosis result from failure of the liver to synthesize proteins, clotting factors, and other substances and manifestations of portal hypertension (see earlier sections of this chapter for clinical manifestations and management of portal hypertension, ascites, varices, and hepatic encephalopathy).

Table 39-5 MODIFIED CHILD-PUGH CLASSIFICATION OF THE SEVERITY OF LIVER DISEASE*

Parameter	Points Assigned		
	1	2	3
Ascites	Absent	Slight	Moderate
Bilirubin (mg/dL)	≤ 2	2–3	> 3
Albumin (g/dL)	> 3.5	2.8–3.5	< 2.8
Prothrombin time (seconds over control)	1–3	4–6	> 6
Encephalopathy	None	Grade 1–2	Grade 3–4

*Total score of 1–6, grade A; 7–9, grade B; 10–15, grade C.

Schiff, E. R., Somell, M. F. & Maddrey, W. C. (Eds.) (2006). *Schiff's diseases of the liver* (10th ed.). Philadelphia: Lippincott Williams & Wilkins.

CHART 39-11 *Assessing for Cirrhosis*

Be alert for the following signs and symptoms:

Compensated

- Intermittent mild fever
- Vascular spiders
- Palmar erythema (reddened palms)
- Unexplained epistaxis
- Ankle edema
- Vague morning indigestion
- Flatulent dyspepsia
- Abdominal pain
- Firm, enlarged liver
- Splenomegaly

Decompensated

- Ascites
- Jaundice
- Weakness
- Muscle wasting
- Weight loss
- Continuous mild fever
- Clubbing of fingers
- Purpura (due to decreased platelet count)
- Spontaneous bruising
- Epistaxis
- Hypotension
- Sparse body hair
- White nails
- Gonadal atrophy

Liver Enlargement

Early in the course of cirrhosis, the liver tends to be large, and the cells are loaded with fat. The liver is firm and has a sharp edge that is noticeable on palpation. Abdominal pain may be present because of recent, rapid enlargement of the liver, which produces tension on the fibrous covering of the liver (Glisson's capsule). Later in the disease, the liver decreases in size as scar tissue contracts the liver tissue. The liver edge, if palpable, is nodular.

Portal Obstruction and Ascites

Portal obstruction and ascites, late manifestations of cirrhosis, are caused partly by chronic failure of liver function and partly by obstruction of the portal circulation. Almost all of the blood from the digestive organs is collected in the portal veins and carried to the liver. Because a cirrhotic liver does not allow free blood passage, blood backs up into the spleen and the GI tract, and these organs become the seat of chronic passive congestion; that is, they are stagnant with blood and therefore cannot function properly. Indigestion and altered bowel function result. Fluid rich in protein may accumulate in the peritoneal cavity, producing ascites. This can be detected through percussion for shifting dullness or a fluid wave (see Fig. 39-5).

Infection and Peritonitis

Bacterial peritonitis may develop in patients with cirrhosis and ascites in the absence of an intra-abdominal source of infection or an abscess. This condition is referred to as spontaneous bacterial peritonitis (SBP). Bacteremia due to translocation of intestinal flora is believed to be the most likely route of infection. Clinical signs may be absent, necessitating paracentesis for diagnosis. Antibiotic therapy is effective in the treatment and prevention of recurrent episodes of SBP. The most severe complication of SBP is hepatorenal syndrome, a form of renal failure unresponsive to administration of fluid or diuretics. This type of renal failure is characterized by a lack of pathologic changes in the kidney; there is no evidence of dehydration or obstruction of the urinary tract or any other renal disorder.

Gastrointestinal Varices

The obstruction to blood flow through the liver caused by fibrotic changes also results in the formation of collateral blood vessels in the GI system and shunting of blood from the portal vessels into blood vessels with lower pressures. As a result, the patient with cirrhosis often has prominent, distended abdominal blood vessels, which are visible on abdominal inspection (caput medusae) and distended blood vessels throughout the GI tract. The esophagus, stomach, and lower rectum are common sites of collateral blood vessels. These distended blood vessels form varices or hemorrhoids, depending on their location (see Fig. 39-6).

Because these vessels were not intended to carry the high pressure and volume of blood imposed by cirrhosis, they may rupture and bleed. Therefore, assessment must include observation for occult and frank bleeding from the GI tract.

Edema

Another late symptom of cirrhosis is edema, which is attributed to chronic liver failure. A reduced plasma albumin concentration predisposes the patient to the formation of edema. Although edema is generalized, it often affects the lower extremities, the upper extremities, and the presacral area. Facial edema is not typical. Overproduction of aldosterone occurs, causing sodium and water retention and potassium excretion.

Vitamin Deficiency and Anemia

Because of inadequate formation, use, and storage of certain vitamins (notably vitamins A, C, and K), signs of deficiency are common, particularly hemorrhagic phenomena associated with vitamin K deficiency. Chronic gastritis and impaired GI function, together with inadequate dietary intake and impaired liver function, account for the anemia that is often associated with cirrhosis. The patient's anemia, poor nutritional status, and poor state of health result in severe fatigue, which interferes with the ability to carry out routine activities of daily living (ADLs).

Mental Deterioration

Additional clinical manifestations include deterioration of mental and cognitive function with impending hepatic encephalopathy and hepatic coma, as previously described. Neurologic assessment is indicated, including assessment of the patient's general behavior, cognitive abilities, orientation to time and place, and speech patterns.

Assessment and Diagnostic Findings

The extent of liver disease and the type of treatment are determined after review of the laboratory findings. The functions of the liver are complex, and many diagnostic tests provide information about liver function (see Table 39-1). The patient needs to know why these tests are being performed and how to cooperate.

In severe parenchymal liver dysfunction, the serum albumin level tends to decrease, and the serum globulin level rises. Enzyme tests indicate liver cell damage: serum alkaline phosphatase, AST, ALT, and GGT levels increase, and the serum cholinesterase level may decrease. Bilirubin tests are performed to measure bile excretion or retention; increased levels of bilirubin can occur with cirrhosis and other liver disorders. Prothrombin time is prolonged.

Ultrasound scanning is used to measure the difference in density of parenchymal cells and scar tissue. CT, MRI, and radioisotope liver scans give information about liver size and hepatic blood flow and obstruction. Diagnosis is confirmed by liver biopsy. Arterial blood gas analysis may reveal a ventilation–perfusion imbalance and hypoxia.

Medical Management

The management of the patient with cirrhosis is usually based on the presenting symptoms. For example, antacids or histamine-2 (H_2) antagonists are prescribed to decrease gastric distress and minimize the possibility of GI bleeding. Vitamins and nutritional supplements promote healing of damaged liver cells and improve the patient's general nutritional status. Potassium-sparing diuretics such as spironolactone or triamterene (Dyrenium) may be indicated to decrease ascites, if present; these diuretics are preferred because they minimize the fluid and electrolyte changes commonly seen with other agents. An adequate diet and avoidance of alcohol are essential. Although the fibrosis of the cirrhotic liver cannot be reversed, its progression may be halted or slowed by such measures.

Preliminary studies indicate that colchicine, an anti-inflammatory agent used to treat the symptoms of gout, may increase survival time in patients with mild to moderate cirrhosis. Many medications have been shown to possess antifibrotic activity for the treatment of cirrhosis. Some of these medications include angiotensin system inhibitors, statins, diuretics, immunosuppressants, and glitazones. These medications have reasonable safety profiles, but their long-term safety and efficacy in patients with cirrhosis has yet to be demonstrated (Schuppan & Afdhal, 2008).

Many patients who have end-stage liver disease (ESLD) with cirrhosis use the herb milk thistle (*Silybum marianum*) to treat jaundice and other symptoms. This herb has been used for centuries because of its healing and regenerative properties for liver disease. Silymarin from milk thistle has anti-inflammatory and antioxidant properties that may have beneficial effects, especially in hepatitis. The natural compound, SAM-e (s-adenosylmethionine), may improve outcomes in liver disease by improving liver function, possibly through enhancing antioxidant function. Primary

biliary cirrhosis has been treated with ursodeoxycholic acid (Actigall, URSO) to improve liver function.

Nursing Management

Nursing management for the patient with cirrhosis of the liver is described in detail in the Plan of Nursing Care for the Patient with Impaired Liver Function (Chart 39-12). Nursing interventions are directed toward promoting patient's rest, improving nutritional status, providing skin care, reducing risk of injury, and monitoring and managing potential complications.

Promoting Rest

The patient with cirrhosis requires rest and other supportive measures to permit the liver to reestablish its functional ability. If the patient is hospitalized, weight and fluid intake and output are measured and recorded daily. The nurse adjusts the patient's position in bed for maximal respiratory efficiency, which is especially important if ascites is marked, because it interferes with adequate thoracic excursion. Oxygen therapy may be required in liver failure to oxygenate the damaged cells and prevent further cell destruction.

Rest reduces the demands on the liver and increases the liver's blood supply. Because the patient is susceptible to the hazards of immobility, efforts to prevent respiratory, circulatory, and vascular disturbances are initiated. These measures may help prevent such problems as pneumonia, thrombophlebitis, and pressure ulcers. After nutritional status improves and strength increases, the nurse encourages the patient to increase activity gradually. Activity and mild exercise, as well as rest, are planned.

Improving Nutritional Status

The patient with cirrhosis without ascites, edema, or signs of impending hepatic coma should receive a nutritious, high-protein diet, if tolerated, supplemented by vitamins of the B complex, as well as A, C, and K. The nurse encourages the patient to eat. If ascites is present, small, frequent meals may be better tolerated than three large meals because of the abdominal pressure exerted by ascites. Patient preferences are considered. Patients with prolonged or severe anorexia and those who are vomiting or eating poorly for any reason may receive nutrients by the enteral or parenteral route.

Patients with fatty stools (steatorrhea) should receive water-soluble forms of fat-soluble vitamins A, D, and E (Aquasol A, D, and E). Folic acid and iron are prescribed to prevent anemia. If the patient shows signs of impending or advancing coma, the amount of protein in the diet is decreased temporarily. Protein is restricted if encephalopathy develops. Incorporating vegetable protein to meet protein needs may decrease the risk for encephalopathy. Sodium restriction is also indicated to prevent ascites.

Providing Skin Care

Providing careful skin care is important because of subcutaneous edema, the patient's immobility, jaundice, and increased susceptibility to skin breakdown and infection. Frequent changes in position are necessary to prevent pressure ulcers. Irritating soaps and the use of adhesive tape are avoided to prevent trauma to the skin. Lotion may be soothing to irritated skin; the nurse takes measures to minimize scratching by the patient.

Reducing Risk of Injury

The nurse protects the patient with cirrhosis from falls and other injuries. The side rails should be in place and padded with blankets or other materials in case the patient becomes agitated or restless. To minimize agitation, the nurse orients the patient to time and place and explains all procedures. The nurse instructs the patient to ask for assistance to get out of bed. The nurse carefully evaluates any injury because of the possibility of internal bleeding.

Because of the risk for bleeding from abnormal clotting, the patient should use an electric razor rather than a safety razor. A soft-bristled toothbrush helps minimize bleeding gums, and pressure applied to all venipuncture sites helps minimize bleeding.

Monitoring and Managing Potential Complications

A major role of the nurse is monitoring of the patient with cirrhosis for complications.

Bleeding and Hemorrhage

The patient is at increased risk for bleeding and hemorrhage because of decreased production of prothrombin and decreased ability of the diseased liver to synthesize the necessary substances for blood coagulation. This was discussed earlier in the section on esophageal varices.

Hepatic Encephalopathy

As previously described, hepatic encephalopathy and coma, complications of cirrhosis, may manifest as deteriorating mental status and dementia or as physical signs such as abnormal voluntary and involuntary movements. Hepatic encephalopathy was discussed earlier in the chapter in detail and in Chart 39-12.

Monitoring is an essential nursing function to identify early deterioration in mental status. The nurse monitors the patient's mental status closely and reports changes so that treatment of encephalopathy can be initiated promptly. An extensive neurologic evaluation is key to identify progression through the four stages of encephalopathy.

Each advancing stage demands more intensive nursing interventions aimed at providing for patient safety and prevention and early identification of life-threatening complications such as respiratory failure and cerebral edema, which would necessitate interventions in an ICU. Because electrolyte disturbances can contribute to encephalopathy, serum electrolyte levels are carefully monitored and corrected if abnormal. Oxygen is administered if oxygen desaturation occurs. The nurse monitors for fever or abdominal pain, which may signal the onset of bacterial peritonitis or other infection (see earlier discussion of hepatic encephalopathy).

Fluid Volume Excess

Patients with advanced chronic liver disease develop cardiovascular abnormalities. These occur due to an increased cardiac output and decreased peripheral vascular resistance, possibly resulting from the release of vasodilators.

CHART 39-12

PLAN OF NURSING CARE
The Patient With Impaired Liver Function

NURSING DIAGNOSIS: Activity intolerance related to fatigue, lethargy, and malaise
GOAL: Patient reports decrease in fatigue and reports increased ability to participate in activities

Nursing Interventions	Rationale	Expected Outcomes
1. Assess level of activity tolerance and degree of fatigue, lethargy, and malaise when performing routine activities of daily living.	1. Provides baseline for further assessment and criteria for assessment of effectiveness of interventions.	• Exhibits increased interest in activities and events. • Participates in activities and gradually increases exercise within physical limits. • Reports increased strength and well-being. • Reports absence of abdominal pain and discomfort. • Plans activities to allow ample periods of rest. • Takes vitamins as prescribed.
2. Assist with activities and hygiene when fatigued.	2. Promotes exercise and hygiene within patient's level of tolerance.	
3. Encourage rest when fatigued or when abdominal pain or discomfort occurs.	3. Conserves energy and protects the liver.	
4. Assist with selection and pacing of desired activities and exercise.	4. Stimulates patient's interest in selected activities.	
5. Provide diet high in carbohydrates with protein intake consistent with liver function.	5. Provides calories for energy and protein for healing.	
6. Administer supplemental vitamins (A, B complex, C, and K).	6. Provides additional nutrients.	

NURSING DIAGNOSIS: Imbalanced nutrition: less than body requirements, related to abdominal distention and discomfort and anorexia
GOAL: Positive nitrogen balance, no further loss of muscle mass; meets nutritional requirements

Nursing Interventions	Rationale	Expected Outcomes
1. Assess dietary intake and nutritional status through diet history and diary, daily weight measurements, and laboratory data.	1. Identifies deficits in nutritional intake and adequacy of nutritional state.	• Exhibits improved nutritional status by increased weight (without fluid retention) and improved laboratory data. • States rationale for dietary modifications. • Identifies foods high in carbohydrates and within protein requirements (moderate to high protein in cirrhosis and hepatitis, low protein in hepatic failure). • Reports improved appetite. • Participates in oral hygiene measures. • Reports increased appetite; identifies rationale for smaller, frequent meals. • Demonstrates intake of high-calorie diet; adheres to protein restriction. • Identifies foods and fluids that are nutritious and permitted on diet. • Gains weight without increased edema or ascites formation. • Reports increased appetite and well-being. • Excludes alcohol from diet. • Takes medications for gastrointestinal disorders as prescribed. • Reports normal gastrointestinal function with regular bowel function.
2. Provide diet high in carbohydrates with protein intake consistent with liver function.	2. Provides calories for energy, sparing protein for healing.	
3. Assist patient in identifying low-sodium foods.	3. Reduces edema and ascites formation.	
4. Elevate the head of the bed during meals.	4. Reduces discomfort from abdominal distention and decreases sense of fullness produced by pressure of abdominal contents and ascites on the stomach.	
5. Provide oral hygiene before meals and pleasant environment for meals at meal time.	5. Promotes positive environment and increased appetite; reduces unpleasant taste.	
6. Offer smaller, more frequent meals (6 per day).	6. Decreases feeling of fullness, bloating.	
7. Encourage patient to eat meals and supplementary feedings.	7. Encouragement is essential for the patient with anorexia and gastrointestinal discomfort.	
8. Provide attractive meals and an aesthetically pleasing setting at meal time.	8. Promotes appetite and sense of well-being.	
9. Eliminate alcohol.	9. Eliminates "empty calories" and further damage from alcohol.	
10. Apply an ice collar for nausea.	10. May reduce incidence of nausea.	
11. Administer medications prescribed for nausea, vomiting, diarrhea, or constipation.	11. Reduces gastrointestinal symptoms and discomforts that decrease the appetite and interest in food.	
12. Encourage increased fluid intake and exercise if the patient reports constipation.	12. Promotes normal bowel pattern and reduces abdominal discomfort and distention.	

Continued

CHART 39-12

PLAN OF NURSING CARE
The Patient With Impaired Liver Function (Continued)

NURSING DIAGNOSIS: Impaired skin integrity related to pruritus from jaundice and edema
GOAL: Decrease potential for pressure ulcer development; breaks in skin integrity

Nursing Interventions	Rationale	Expected Outcomes
1. Assess degree of discomfort related to pruritus and edema.	1. Assists in determining appropriate interventions.	• Exhibits intact skin without redness, excoriation, or breakdown.
2. Note and record degree of jaundice and extent of edema.	2. Provides baseline for detecting changes and evaluating effectiveness of interventions.	• Reports relief from pruritus.
3. Keep patient's fingernails short and smooth.	3. Prevents skin excoriation and infection from scratching.	• Exhibits no skin excoriation from scratching.
4. Provide frequent skin care; avoid use of soaps and alcohol-based lotions.	4. Removes waste products from skin while preventing dryness of skin.	• Uses nondrying soaps and lotions. States rationale for use of nondrying soaps and lotions.
5. Massage every 2 h with emollients; turn every 2 h.	5. Promotes mobilization of edema.	• Turns self periodically. Exhibits reduced edema of dependent parts of the body.
6. Initiate use of alternating-pressure mattress or low air loss bed.	6. Minimizes prolonged pressure on bony prominences susceptible to breakdown.	• Exhibits no areas of skin breakdown.
7. Recommend avoiding use of harsh detergents.	7. May decrease skin irritation and need for scratching.	• Exhibits decreased edema; normal skin turgor.
8. Assess skin integrity every 4–8 h. Instruct patient and family in this activity.	8. Edematous skin and tissue have compromised nutrient supply and are vulnerable to pressure and trauma.	
9. Restrict sodium as prescribed.	9. Minimizes edema formation.	
10. Perform range of motion exercises every 4 h; elevate edematous extremities whenever possible.	10. Promotes mobilization of edema.	

NURSING DIAGNOSIS: High risk for injury related to altered clotting mechanisms and altered level of consciousness
GOAL: Reduced risk of injury

Nursing Interventions	Rationale	Expected Outcomes
1. Assess level of consciousness and cognitive level.	1. Assists in determining patient's ability to protect self and comply with required self-protective actions; may detect deterioration of hepatic function.	• Is oriented to time, place, and person.
		• Exhibits no hallucinations, and demonstrates no efforts to get up unassisted or to leave hospital.
2. Provide safe environment (pad side rails, remove obstacles in room, prevent falls).	2. Minimizes falls and injury if falls occur.	• Exhibits no ecchymoses (bruises), cuts, or hematoma.
3. Provide frequent surveillance to orient patient and avoid use of restraints.	3. Protects patient from harm while stimulating and orienting patient; use of restraints may disturb patient further.	• Uses electric razor rather than sharp-edged razor.
		• Exhibits absence of frank bleeding from gastrointestinal tract.
4. Replace sharp objects (razors) with safer items.	4. Avoids cuts and bleeding.	• Exhibits absence of restlessness, epigastric fullness, and other indicators of hemorrhage and shock.
5. Observe each stool for color, consistency, and amount.	5. Permits detection of bleeding in gastrointestinal tract.	• Exhibits negative results of test for occult gastrointestinal bleeding.
6. Be alert for symptoms of anxiety, epigastric fullness, weakness, and restlessness.	6. May indicate early signs of bleeding and shock.	• Is free of ecchymotic areas or hematoma formation.
		• Exhibits normal vital signs.
7. Test each stool and emesis for occult blood.	7. Detects early evidence of bleeding.	• Maintains rest and remains quiet if active bleeding occurs.
8. Observe for hemorrhagic manifestations: ecchymosis, epistaxis, petechiae, and bleeding gums.	8. Indicates altered clotting mechanisms.	• Identifies rationale for blood transfusions and measures to treat bleeding.
		• Uses measures to prevent trauma (eg, uses soft toothbrush, blows nose gently, avoids bumps and falls, avoids straining during defecation).
9. Record vital signs at frequent intervals, depending on patient acuity (every 1–4 h).	9. Provides baseline and evidence of hypovolemia, and hemorrhagic shock.	• Experiences no side effects of medications.

Continued on following page

CHART 39-12 PLAN OF NURSING CARE

The Patient With Impaired Liver Function (Continued)

Nursing Interventions	Rationale	Expected Outcomes
10. Keep patient quiet and limit activity.	10. Minimizes risk of bleeding and straining.	• Takes all medications as prescribed. • Identifies rationale for precautions with use of all medications. • Cooperates with treatment modalities.
11. Assist physician in passage of tube for esophageal balloon tamponade, if its insertion is indicated.	11. Promotes nontraumatic insertion of tube in anxious and combative patient for immediate treatment of bleeding.	
12. Observe during blood transfusions.	12. Permits detection of transfusion reactions (risk is increased with multiple blood transfusions needed for active bleeding from esophageal varices).	
13. Measure and record nature, time, and amount of vomitus.	13. Assists in evaluating extent of bleeding and blood loss.	
14. Maintain patient in fasting state, if indicated.	14. Reduces risk of aspiration of gastric contents and minimizes risk of further trauma to esophagus and stomach by preventing vomiting.	
15. Administer vitamin K as prescribed.	15. Promotes clotting by providing fat-soluble vitamin necessary for clotting.	
16. Remain with patient during episodes of bleeding.	16. Reassures anxious patient and permits monitoring and detection of further needs of the patient.	
17. Offer cold liquids by mouth when bleeding stops (if prescribed).	17. Minimizes risk of further bleeding by promoting vasoconstriction of esophageal and gastric blood vessels.	
18. Institute measures to prevent trauma: a. Maintain safe environment.	18. Promotes safety of patient a. Minimizes risk of trauma and bleeding by avoiding falls and cuts, etc.	
b. Encourage *gentle* blowing of nose.	b. Reduces risk of nosebleed (epistaxis) secondary to trauma and decreased clotting.	
c. Provide soft toothbrush and avoid use of toothpicks.	c. Prevents trauma to oral mucosa while promoting good oral hygiene.	
d. Encourage intake of foods with high content of vitamin C.	d. Promotes healing.	
e. Apply cold compresses where indicated.	e. Minimizes bleeding into tissues by promoting local vasoconstriction.	
f. Record location of bleeding sites.	f. Permits detection of new bleeding sites and monitoring of previous sites of bleeding.	
g. Use small-gauge needles for injections.	g. Minimizes oozing and blood loss from repeated injections.	
19. Administer medications carefully; monitor for side effects.	19. Reduces risk of side effects secondary to damaged liver's inability to detoxify (metabolize) medications normally.	

NURSING DIAGNOSIS: Disturbed body image related to changes in appearance, sexual dysfunction, and role function
GOAL: Patient verbalizes feelings consistent with improvement of body image and self-esteem

Nursing Interventions	Rationale	Expected Outcomes
1. Assess changes in appearance and the meaning these changes have for patient and family.	1. Provides information for assessing impact of changes in appearance, sexual function, and role on the patient and family.	• Verbalizes concerns related to changes in appearance, life, and lifestyle.

Continued

CHART 39-12 PLAN OF NURSING CARE

The Patient With Impaired Liver Function (Continued)

Nursing Interventions	Rationale	Expected Outcomes
2. Encourage patient to verbalize reactions and feelings about these changes.	2. Enables patient to identify and express concerns; encourages patient and significant others to share these concerns.	• Shares concerns with significant others. • Identifies past coping strategies that have been effective.
3. Assess patient's and family's previous coping strategies.	3. Permits encouragement of those coping strategies that are familiar to patient and have been effective in the past.	• Uses past effective coping strategies to deal with changes in appearance, life, and lifestyle.
4. Assist and encourage patient to maximize appearance (such as strategies to limit the appearance of jaundice and ascites through careful selection of colors and type of clothing) and explore alternatives to previous sexual and role functions.	4. Encourages patient to continue safe roles and functions while encouraging exploration of alternatives.	• Maintains good grooming and hygiene. • Identifies short-term goals and strategies to achieve them. • Takes an active role in decision making about self and care. • Identifies resources that are not harmful.
5. Assist patient in identifying short-term goals.	5. Accomplishing these goals serves as positive reinforcement and increases self-esteem.	• Verbalizes that some of previous lifestyle practices have been harmful.
6. Encourage and assist patient in decision making about care.	6. Promotes patient's control of life and improves sense of well-being and self-esteem.	• Uses healthy expressions of frustration, anger, anxiety.
7. Identify with patient resources to provide additional support (counselor, spiritual advisor).	7. Assists patient in identifying resources and accepting assistance from others when indicated.	
8. Assist patient in identifying previous practices that may have been harmful to self (alcohol and drug abuse). Involve patient in goal-setting and provide positive feedback for accomplishments.	8. Recognition and acknowledgment of the harmful effects of these practices are necessary for identifying a healthier lifestyle.	

NURSING DIAGNOSIS: Chronic pain and discomfort related to enlarged tender liver and ascites
GOAL: Increased level of comfort

Nursing Interventions	Rationale	Expected Outcomes
1. Maintain bed rest when patient experiences abdominal discomfort.	1. Reduces metabolic demands and protects the liver.	• Reports pain and discomfort if present.
2. Administer antispasmodic and analgesic agents as prescribed.	2. Reduces irritability of the gastrointestinal tract and decreases abdominal pain and discomfort.	• Maintains bed rest and decreases activity in presence of pain. • Takes antispasmodic and analgesics as indicated and as prescribed.
3. Observe, record, and report presence and character of pain and discomfort.	3. Provides baseline to detect further deterioration of status and to evaluate interventions.	• Reports decreased pain and abdominal discomfort.
4. Reduce sodium and fluid intake if prescribed.	4. Minimizes further formation of ascites.	• Reduces sodium and fluid intake to prescribed levels if indicated to treat ascites.
5. Prepare patient and assist with paracentesis.	5. Removal of ascites fluid may decrease abdominal discomfort.	• Exhibits decreased abdominal girth and appropriate weight changes.
6. Encourage the use of distracting activities such as music, reading or meditation.	6. Distraction may limit the perception of pain.	• Reports decreased discomfort after paracentesis.

NURSING DIAGNOSIS: Fluid volume excess related to ascites and edema formation
GOAL: Restoration of normal fluid volume

Nursing Interventions	Rationale	Expected Outcomes
1. Restrict sodium and fluid intake if prescribed.	1. Minimizes formation of ascites and edema.	• Consumes diet low in sodium and within prescribed fluid restriction.
2. Administer diuretics, potassium, and protein supplements as prescribed.	2. Promotes excretion of fluid through the kidneys and maintenance of normal fluid and electrolyte balance.	• Takes diuretics, potassium, and protein supplements as indicated without experiencing side effects.

Continued on following page

CHART 39-12 PLAN OF NURSING CARE
The Patient With Impaired Liver Function (Continued)

Nursing Interventions	Rationale	Expected Outcomes
3. Record intake and output every 1 to 8 h depending on response to interventions and on patient acuity.	3. Indicates effectiveness of treatment and adequacy of fluid intake.	• Exhibits increased urine output. • Exhibits decreasing abdominal girth. • Exhibits no rapid increase in weight.
4. Measure and record abdominal girth and weight daily.	4. Monitors changes in ascites formation and fluid accumulation.	
5. Explain rationale for sodium and fluid restriction.	5. Promotes patient's understanding of restriction and cooperation with it.	• Identifies rationale for sodium and fluid restriction.
6. Prepare patient and assist with paracentesis.	6. Paracentesis will temporarily decrease amount of ascites present.	• Shows a decrease in ascites with decreased weight.

NURSING DIAGNOSIS: Disturbed thought processes and potential for mental deterioration related to abnormal liver function and increased serum ammonia level
GOAL: Improved mental status; safety maintained; ability to cope with cognitive and behavioral changes

Nursing Interventions	Rationale	Expected Outcomes
1. Restrict dietary protein as prescribed for transient period.	1. Reduces source of ammonia (protein foods).	• Adheres to protein restriction.
2. Give frequent, small feedings of carbohydrates.	2. Promotes consumption of adequate carbohydrates for energy requirements and spares protein from breakdown for energy.	• Demonstrates an interest in events and activities in environment. • Demonstrates normal attention span. • Follows and participates in conversation appropriately.
3. Protect from infection.	3. Minimizes risk for further increase in metabolic requirements.	• Is oriented to person, place, and time.
4. Keep environment warm and draft-free.	4. Minimizes shivering, which would increase metabolic requirements.	• Remains in bed when indicated.
5. Pad the side rails of the bed.	5. Provides protection for the patient should hepatic coma and seizure activity occur.	• Reports no urinary or fecal incontinence. • Experiences no seizures.
6. Limit visitors.	6. Minimizes patient's activity and metabolic requirements.	• No neurological or respiratory depression. • Develops no cognitive impairments but if they develop they are quickly identified and treated enhancing the potential of recovery.
7. Provide careful nursing surveillance to ensure patient's safety.	7. Provides close monitoring of new symptoms and minimizes trauma to the confused patient.	
8. Avoid opioids and barbiturates.	8. Prevents masking of symptoms of hepatic coma and prevents drug overdose secondary to reduced ability of the damaged liver to metabolize opioids and barbiturates. Prevents respiratory depression.	• Patient and family describe adequate feelings of coping and lowered anxiety. They demonstrate ability to listen and to make decisions as able.
9. Awaken at intervals (every 2–4 h) to assess cognitive status.	9. Provides stimulation to the patient and opportunity for observing the patient's level of consciousness.	• Patient and family communicate their feelings and their needs in a secure and caring environment.
10. Identify subtle changes in behavior or sleep–wake pattern (consistent staff caring for the patient enhances this assessment as they become familiar with patient's baseline).	10/11. These changes may herald worsening of encephalopathy which requires rapid intervention including medication.	
11. Assess handwriting or drawing skill daily as indication of cognitive ability.		
12. Encourage patient and family to participate in therapeutic strategies to enhance coping with episodes of mental deterioration.	12. Promoting activities such as listening to music, relaxation techniques or preillness coping strategies can reduce anxiety.	
13. Encourage patient and family to discuss feeling of fear, powerlessness or emotional distress related to patient's mental deterioration.	13. Actively listening demonstrates caring and concern.	

Continued

CHART 39-12 PLAN OF NURSING CARE

The Patient With Impaired Liver Function (Continued)

NURSING DIAGNOSIS: Risk for imbalanced body temperature: hyperthermia related to inflammatory process of cirrhosis or hepatitis
GOAL: Maintenance of normal body temperature, free from infection

Nursing Interventions	Rationale	Expected Outcomes
1. Record temperature regularly (every 4 h).	1. Provides baseline to detect fever and to evaluate interventions.	• Exhibits normal temperature and reports absence of chills or sweating.
2. Encourage fluid intake.	2. Corrects fluid loss from perspiration and fever and increases patient's level of comfort.	• Demonstrates adequate intake of fluids.
3. Apply cool sponges or ice bag for elevated temperature.	3. Promotes reduction of fever and increases patient's comfort.	• Exhibits no evidence of local or systemic infection.
4. Administer antibiotics as prescribed.	4. Ensures appropriate serum concentration of antibiotics to treat infection.	• Develops no nosocomial infections related to invasive procedures/lines.
5. Avoid exposure to infections.	5. Minimizes risk of further infection and further increases in body temperature and metabolic rate.	
6. Keep patient at rest while temperature is elevated.	6. Reduces metabolic rate.	
7. Assess for abdominal pain, tenderness.	7. May occur with bacterial peritonitis.	
8. Use sterile technique for all invasive procedures.	8. Many evidence-based practice guidelines (for example central venous catheter care) recommend the use of sterile technique to prevent nosocomial infections.	

NURSING DIAGNOSIS: Ineffective breathing pattern related to ascites and restriction of thoracic excursion secondary to ascites, abdominal distention, and fluid in the thoracic cavity
GOAL: Improved respiratory status

Nursing Interventions	Rationale	Expected Outcomes
1. Elevate head of bed to at least 30 degrees.	1. Reduces abdominal pressure on the diaphragm and permits fuller thoracic excursion and lung expansion.	• Experiences improved respiratory status.
2. Conserve patient's strength by providing rest periods and assisting with activities.	2. Reduces metabolic and oxygen requirements.	• Reports decreased shortness of breath.
3. Change position every 2 h.	3. Promotes expansion and oxygenation of all areas of the lungs.	• Reports increased strength and sense of well-being.
4. Assist with paracentesis or thoracentesis.	4. Paracentesis and thoracentesis (performed to remove fluid from the abdominal and thoracic cavities, respectively) may be frightening to the patient.	• Exhibits normal respiratory rate (12–18/min) with no adventitious sounds.
a. Explain procedure and its purpose to patient.	a. Helps obtain patient's cooperation with procedures.	• Exhibits full thoracic excursion without shallow respirations.
b. Have patient void before paracentesis.	b. Prevents inadvertent bladder injury.	• Exhibits normal arterial blood gases.
c. Support and maintain position during procedure.	c. Prevents inadvertent organ or tissue injury.	• Exhibits adequate oxygen saturation by pulse oximetry.
d. Record both the amount and the character of fluid aspirated.	d. Provides record of fluid removed and indication of severity of limitation of lung expansion by fluid.	• Experiences absence of confusion or cyanosis.
e. Observe for evidence of coughing, increasing dyspnea, or pulse rate.	e. Indicates irritation of the pleural space and evidence of pneumothorax or hemothorax.	

Continued on following page

CHART 39-12 PLAN OF NURSING CARE

The Patient With Impaired Liver Function (Continued)

COLLABORATIVE PROBLEM: Gastrointestinal bleeding and hemorrhage
GOAL: Absence of episodes of gastrointestinal bleeding and hemorrhage

Nursing Interventions	Rationale	Expected Outcomes
1. Assess patient for evidence of gastrointestinal bleeding or hemorrhage. If bleeding does occur: a. Monitor vital signs (blood pressure, pulse, respiratory rate) every 4 h or more frequently, depending on acuity. b. Assess skin temperature, level of consciousness every 4 h or more frequently, depending on acuity. c. Monitor gastrointestinal secretions and output (emesis, stool for occult or obvious bleeding). Test emesis for blood once per shift and with any color change. Hematest each stool. d. Monitor hematocrit and hemoglobin for trends and changes.	1. Allows early detection of signs and symptoms of bleeding and hemorrhage.	• Experiences no episodes of bleeding and hemorrhage. • Vital signs are within acceptable range for patient. • No evidence of bleeding from gastrointestinal tract. • Hematocrit and hemoglobin levels within acceptable limits. • Turns and moves without straining and increasing intra-abdominal pressure. • No straining with bowel movements. • No further bleeding episodes if aggressive treatment of bleeding and hemorrhage was needed. • Patient and family state rationale for treatments. • Patient and family identify supports available to them. • Patient and family describe signs and symptoms of a recurrent bleeding episode and identify needed action.
2. Avoid activities that increase intra-abdominal pressure (straining, turning). a. Avoid coughing/sneezing. b. Assist patient to turn. c. Keep all needed items within easy reach. d. Use measures to prevent constipation such as adequate fluid intake; stool softeners. e. Ensure small meals.	2. Minimizes increases in intra-abdominal pressure that could lead to rupture and bleeding of esophageal or gastric varices.	
3. Have equipment (Blakemore tube, medications, IV fluids) available if indicated.	3. Equipment, medications, and supplies will be readily available if patient experiences bleeding from ruptured esophageal or gastric varices.	
4. Assist with procedures and therapy needed to treat gastrointestinal bleeding and hemorrhage.	4. Gastrointestinal bleeding and hemorrhage require emergency measures (eg, insertion of Blakemore tube, administration of fluids and medications).	
5. Monitor respiratory status every hour and minimize risk of respiratory complications if balloon tamponade is needed.	5. The patient is at high risk for respiratory complications, including asphyxiation if gastric balloon of tamponade tube ruptures or migrates upward.	
6. Prepare patient physically and psychologically for other treatment modalities if needed.	6. The patient who experiences hemorrhage is very anxious and fearful; minimizing anxiety assists in control of hemorrhage.	
7. Monitor patient for recurrence of bleeding and hemorrhage.	7. Risk of rebleeding is high with all treatment modalities used to halt gastrointestinal bleeding.	
8. Keep family informed of patient's status.	8. Family members are likely to be anxious about the patient's status; providing information will reduce their anxiety level and promote more effective coping.	
9. Once recovered from bleeding episode, provide patient and family with information regarding signs and symptoms of gastrointestinal bleeding.	9. Risk of rebleeding is high. Subtle signs may be more quickly identified.	

Continued

CHART 39-12 PLAN OF NURSING CARE

The Patient With Impaired Liver Function (Continued)

COLLABORATIVE PROBLEM: Hepatic encephalopathy
GOAL: Absence of changes in cognitive status and of injury

Nursing Interventions	Rationale	Expected Outcomes
1. Assess cognitive status every 4–8 h: a. Assess patient's orientation to person, place, and time. b. Monitor patient's level of activity, restlessness, and agitation. Assess for presence of flapping hand tremors (asterixis). c. Obtain and record daily sample of patient's handwriting or ability to construct a simple figure (eg, star). d. Assess neurologic signs (deep tendon reflexes, ability to follow instructions).	1. Data will provide baseline of patient's cognitive status and enable detection of changes.	• Remains awake, alert, and aware of surroundings. • Is oriented to time, place, and person. • Exhibits no restlessness or agitation. • Record of handwriting demonstrates no deterioration in cognitive function. • States rationale for treatment used to prevent or treat hepatic encephalopathy. • Demonstrates stable serum ammonia level within acceptable limits. • Consumes adequate caloric intake and adheres to protein restriction. • Takes medications as prescribed. • Breath sounds are normal without adventitious sounds. • Skin and tissue intact without evidence of pressure or breaks in integrity. • Verbalizes understanding of need for treatments and procedures to promote recovery.
2. Monitor medications to prevent administration of those that may precipitate hepatic encephalopathy (sedatives, hypnotics, analgesics).	2. Medications are a common precipitating factor in development of hepatic encephalopathy in patients at risk.	
3. Monitor laboratory data, especially serum ammonia level.	3. Increases in serum ammonia level are associated with hepatic encephalopathy and coma.	
4. Notify physician of even subtle changes in patient's neurologic assessment, cognitive function, sleep pattern, or mood.	4. Allows early initiation of treatment of hepatic encephalopathy and prevention of hepatic coma.	
5. Limit sources of protein from diet if indicated.	5. Reduces breakdown and conversion of protein to ammonia.	
6. Administer medications prescribed to reduce serum ammonia level (eg, lactulose, antibiotics, glucose, benzodiazepine antagonist [Flumazenil] if indicated).	6. Reduces serum ammonia level.	
7. Assess respiratory status and initiate measures to prevent complications.	7. The patient who develops hepatic coma is at risk for respiratory complications (ie, pneumonia, atelectasis, infection).	
8. Protect patient's skin and tissue from pressure and breakdown.	8. The patient in coma is at risk for skin breakdown and pressure ulcer formation.	
9. Provide support and active listening for patient and family as patient's mental status deteriorates.	9. The patient with hepatic encephalopathy can experience episodes of mental deterioration due to liver failure. This can produce feelings of fear and anxiety.	

A hyperdynamic circulatory state develops in patients with cirrhosis, and plasma volume increases. This increase in circulating plasma volume is probably multifactorial, but some studies have implicated excess production of nitrous oxide, like that seen in sepsis, as one causative factor (Rodes, et al., 2007). The greater the degree of hepatic decompensation, the more severe the hyperdynamic state. Close assessment of cardiovascular and respiratory status is of key importance for the care of patients with this disorder. Pulmonary compromise, which is always a potential complication of ESLD because of plasma volume excess, makes prevention of pulmonary complications an important role for the nurse. Administering diuretics, implementing fluid restrictions, and enhancing patient positioning can optimize pulmonary function. Fluid retention may be noted in the development of ascites, lower extremity swelling, and dyspnea. Monitoring of intake and output, daily weight changes, changes in abdominal girth, and edema formation is part of nursing assessment in the hospital or in the home setting. Patients are also monitored for nocturia and, later, for oliguria, because these states indicate increasing severity of liver dysfunction (Rodes, et al., 2007).

Promoting Home and Community-Based Care

Teaching Patients Self-Care

During the hospital stay, the nurse and other health care providers prepare the patient with cirrhosis for discharge, focusing on dietary instruction. Of greatest importance is the exclusion of alcohol from the diet. The patient may need referral to Alcoholics Anonymous, psychiatric care, or counseling or may benefit from support from a spiritual advisor. The patient should also avoid the consumption of raw shellfish.

Sodium restriction will continue for a considerable time, if not permanently. The patient will require written instructions, teaching, reinforcement, and support from the staff as well as family members.

Successful treatment depends on convincing the patient of the need to adhere completely to the therapeutic plan. This includes rest, lifestyle changes, adequate dietary intake, and the elimination of alcohol. The nurse also instructs the patient and family about symptoms of impending encephalopathy, possible bleeding tendencies, and susceptibility to infection.

Recovery is neither rapid nor easy; there are frequent setbacks and apparent lack of improvement. Many patients find it difficult to refrain from using alcohol for comfort or escape. The nurse has a significant role in offering support and encouragement to the patient and in providing positive feedback when the patient experiences success.

Continuing Care

Referral for home care may assist the patient in dealing with the transition from hospital to home. The use of alcohol may have been an important part of normal home and social life in the past. The home care nurse assesses the patient's progress at home and the manner in which the patient and family are coping with the elimination of alcohol and the dietary restrictions. The nurse also reinforces previous teaching and answers questions that may not have occurred to the patient or family until the patient is back home and trying to establish new patterns of eating, drinking, and lifestyle.

CANCER OF THE LIVER

Hepatic tumors may be malignant or benign. Benign liver tumors were uncommon until oral contraceptives were in widespread use. Now benign liver tumors occur most frequently in women in their reproductive years who are taking oral contraceptives.

Primary Liver Tumors

Few cancers originate in the liver. Primary liver tumors usually are associated with chronic liver disease, hepatitis B and C infections, and cirrhosis. Hepatocellular carcinoma (HCC) is the most common type of primary liver cancer, with more than half a million cases diagnosed each year on a worldwide basis. HCC is the third leading cause of cancer-related mortality worldwide. It is rare in the United States and Northern Europe, accounting for less than 5 cases per 100,000 inhabitants (Rodes, et al., 2007). Other types of primary liver cancer include cholangiocellular carcinoma and combined hepatocellular and cholangiocellular carcinoma. HCC is usually nonresectable because of rapid growth and metastasis. If found early, resection of primary liver cancer may be possible, but early detection is unlikely.

Cirrhosis, chronic infection with hepatitis B and C, and exposure to certain chemical toxins (eg, vinyl chloride, arsenic) have been implicated as causes of HCC. Cigarette smoking has also been identified as a risk factor, especially when combined with alcohol use. Some evidence suggests that aflatoxin, a metabolite of the fungus *Aspergillus flavus*, may be a risk factor for HCC. This is especially true in areas where HCC is endemic (ie, Asia and Africa). Aflatoxin and other similar toxic molds can contaminate food such as ground nuts and grains and may act as co-carcinogens with hepatitis B. The risk of contamination is greatest when these foods are stored unrefrigerated in tropical or subtropical climates.

Liver Metastases

Metastases from other primary sites, particularly the digestive system, breast, and lung, are found in the liver 2.5 times more frequently than tumors due to primary liver cancers (Rodes, et al., 2007). Malignant tumors are likely to reach the liver eventually, by way of the portal system or lymphatic channels, or by direct extension from an abdominal tumor. Moreover, the liver apparently is an ideal place for these malignant cells to thrive. Often the first evidence of cancer in an abdominal organ is the appearance of liver metastases; unless exploratory surgery or an autopsy is performed, the primary tumor may never be identified.

Clinical Manifestations

The early manifestations of malignancy of the liver include pain—a continuous dull ache in the right upper quadrant, epigastrium, or back. Weight loss, loss of strength, anorexia, and anemia may also occur. The liver may be enlarged and irregular on palpation. Jaundice is present only if the larger bile ducts are occluded by the pressure of malignant nodules in the hilum of the liver. Ascites develops if such nodules obstruct the portal veins or if tumor tissue is seeded in the peritoneal cavity.

Assessment and Diagnostic Findings

The diagnosis of liver cancer is based on clinical signs and symptoms, the history and physical examination, and the results of laboratory and x-ray studies. Increased serum levels of bilirubin, alkaline phosphatase, AST, GGT, and lactic dehydrogenase may occur. Leukocytosis (increased white blood cells), erythrocytosis (increased red blood cells), hypercalcemia, hypoglycemia, and hypocholesterolemia may also be seen on laboratory assessment.

The serum level of alpha-fetoprotein (AFP), which serves as a tumor marker, is elevated in 30% to 40% of patients with primary liver cancer. The level of carcinoembryonic antigen (CEA), a marker of advanced cancer of the digestive tract, may be elevated. These two markers together

chapter 41

Assessment and Management of Patients With Diabetes Mellitus

LEARNING OBJECTIVES

On completion of this chapter, the learner will be able to:

1. Differentiate between type 1 and type 2 diabetes.
2. Describe etiologic factors associated with diabetes.
3. Relate the clinical manifestations of diabetes to the associated pathophysiologic alterations.
4. Identify the diagnostic and clinical significance of blood glucose test results.
5. Explain the dietary modifications used for management of people with diabetes.
6. Describe the relationships among diet, exercise, and medication (ie, insulin or oral antidiabetic agents) for people with diabetes.
7. Develop a plan for teaching insulin self-management.
8. Identify the role of oral antidiabetic agents in diabetic therapy.
9. Differentiate between hyperglycemia with diabetic ketoacidosis and hyperosmolar nonketotic syndrome.
10. Describe management strategies for a person with diabetes to use during "sick days."
11. Describe the major macrovascular, microvascular, and neuropathic complications of diabetes and the self-care behaviors that are important in their prevention.
12. Identify the teaching aids and community support groups available for people with diabetes.

GLOSSARY

continuous glucose monitoring system (CGMS): a device worn for 72 hours that continuously monitors blood glucose levels; the data are downloaded and analyzed for blood glucose patterns for that time period; presently used diagnostically to elicit patterns and tailor treatment
continuous subcutaneous insulin infusion, insulin pump: a small device that delivers insulin on a 24-hour basis as basal insulin; it is also programmed by the patient to deliver a bolus dose before eating a meal in an attempt to mimic normal pancreatic function
diabetes mellitus: a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both
diabetic ketoacidosis (DKA): a metabolic derangement in type 1 diabetes that results from a deficiency of insulin; highly acidic ketone bodies are formed, resulting in acidosis; usually requires hospitalization for treatment and is usually caused by nonadherence to the insulin regimen, concurrent illness, or infection
fasting plasma glucose (FPG): blood glucose determination obtained in the laboratory after fasting for more than 8 hours
gestational diabetes mellitus (GDM): any degree of glucose intolerance with its onset during pregnancy
glycated hemoglobin (glycosylated hemoglobin, Hgb A_{1C} or A1C): a long-term measure of glucose control that is a result of glucose attaching to hemoglobin for the life of the red blood cell (120 days). The goal of diabetes therapy is a normal to near-normal level of glycated hemoglobin, the same as in the nondiabetic population
hyperglycemia: elevated blood glucose level; fasting level greater than 110 mg/dL (6.1 mmol/L); 2-hour postprandial level greater than 140 mg/dL (7.8 mmol/L)
hyperglycemic hyperosmolar nonketotic syndrome (HHNS): a metabolic disorder of type 2 diabetes resulting from a relative insulin deficiency initiated by an intercurrent illness that raises the demand for insulin; associated with polyuria and severe dehydration
hypoglycemia: low blood glucose level (less than 60 mg/dL [less than 2.7 mmol/L])
impaired fasting glucose (IFG), impaired glucose tolerance (IGT): a metabolic stage intermediate between normal glucose homeostasis and diabetes; now referred to as prediabetes
insulin: a hormone secreted by the beta cells of the islets of Langerhans of the pancreas that is necessary for the metabolism of carbohydrates, proteins, and fats; a deficiency of insulin results in diabetes mellitus
islet cell transplantation: an investigational procedure in which purified islet cells from cadaver donors are injected into the portal vein of the liver, with the goal of having these cells secrete insulin and cure type 1 diabetes
ketone: a highly acidic substance formed when the liver breaks down free fatty acids in the absence of insulin; the result is diabetic ketoacidosis
medical nutrition therapy (MNT): nutritional therapy prescribed by the physician for management of diabetes
nephropathy: a long-term complication of diabetes in which the kidney cells are damaged; characterized by microalbuminuria in early stages and progressing to end-stage renal disease
neuropathy: a long-term complication of diabetes resulting from damage to the nerve cell
prediabetes: impaired glucose metabolism in which blood glucose concentrations fall between normal levels and those considered diagnostic for diabetes; includes IFG and IGT, not clinical entities in their own right but risk factors for future diabetes and cardiovascular disease
retinopathy: a long-term complication of diabetes in which the microvascular system of the eye is damaged
self-monitoring of blood glucose (SMBG): a method of capillary blood glucose testing in which the patient pricks his or her finger and applies a drop of blood to a test strip that is read by a meter
sulfonylurea: a classification of oral antidiabetic medication for treating type 2 diabetes; stimulates insulin secretion and insulin action
thiazolidinedione: a class of oral antidiabetic medications that reduce insulin resistance in target tissues, enhancing insulin action without directly stimulating insulin secretion
type 1 diabetes: a metabolic disorder characterized by an absence of insulin production and secretion from autoimmune destruction of the beta cells of the islets of Langerhans in the pancreas; formerly called insulin-dependent, juvenile, or type I diabetes
type 2 diabetes: a metabolic disorder characterized by the relative deficiency of insulin production and a decreased insulin action and increased insulin resistance; Formerly called non–insulin-dependent, adult-onset, or type II diabetes

GLOSSARY *(Continued)*

Diabetes mellitus is a group of metabolic diseases characterized by increased levels of glucose in the blood (**hyperglycemia**) resulting from defects in insulin secretion, insulin action, or both (American Diabetes Association [ADA], 2009a). Normally, a certain amount of glucose circulates in the blood. The major sources of this glucose are absorption of ingested food in the gastrointestinal tract and formation of glucose by the liver from food substances.

DIABETES

Epidemiology

Diabetes is becoming more common in the United States. From 1980 through 2002, the number of Americans with diabetes more than doubled and increased in all age groups. Currently, it is estimated that more than 23 million people in the United States have diabetes, although almost one third of these cases are undiagnosed. The number of people newly diagnosed with diabetes increases by about 1 million people per year (Centers for Disease Control and Prevention [CDC], 2008). By 2030, the number of cases is expected to exceed 30 million. In 2000, the worldwide estimate of the prevalence of diabetes was 171 million people, and by 2030, this is expected to increase to more than 360 million (World Health Organization, 2008). Diabetes is especially prevalent in the elderly; as many as 50% of people older than 65 years of age have some degree of glucose intolerance. People 65 years and older account for almost 40% of people with diabetes.

Minority populations are disproportionately affected by diabetes. From 1980 through 2002, the age-adjusted prevalence of diabetes increased among all gender and race groups, but compared to Caucasians, African Americans and members of other racial and ethnic groups (Native Americans and persons of Hispanic origin) are more likely to develop diabetes, are at greater risk for many of the complications, and have higher death rates due to diabetes (CDC, 2008). Chart 41-1 summarizes risk factors for diabetes mellitus.

CHART 41-1 *Risk Factors for Diabetes Mellitus*

- Family history of diabetes (ie, parents or siblings with diabetes)
- Obesity (ie, ≥20% over desired body weight or BMI ≥27 kg/m^2)
- Race/ethnicity (eg, African Americans, Hispanic Americans, Native Americans, Asian Americans, Pacific Islanders)
- Age ≥45 y
- Previously identified impaired fasting glucose or impaired glucose tolerance
- Hypertension (≥140/90 mm Hg)
- HDL cholesterol level ≤35 mg/dL (0.90 mmol/L) and/or triglyceride level ≥250 mg/dL (2.8 mmol/L)
- History of gestational diabetes or delivery of babies over 9 lb

Used with permission of American Diabetes Association. (2009a). Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care, 32*(Suppl 1), S62–S67.

Diabetes has far-reaching and devastating physical, social, and economic consequences, including the following:

- In the United States, diabetes is the leading cause of nontraumatic amputations, blindness in working-age adults, and end-stage renal disease (CDC, 2008).
- Diabetes is the third leading cause of death from disease, primarily because of the high rate of cardiovascular disease (myocardial infarction, stroke, and peripheral vascular disease) among people with diabetes.
- Hospitalization rates for people with diabetes are 2.4 times greater for adults and 5.3 times greater for children than for the general population.

The economic cost of diabetes continues to increase because of increasing health care costs and an aging population. Half of all people who have diabetes and are older than 65 years of age are hospitalized each year, and severe and life-threatening complications often contribute to the increased rates of hospitalization. Costs related to diabetes are estimated to be almost $174 billion annually, including direct medical care expenses and indirect costs attributable to disability and premature death (ADA, 2008a).

Classification

The major classifications of diabetes are type 1 diabetes, type 2 diabetes, gestational diabetes, and diabetes mellitus associated with other conditions or syndromes (ADA, 2009a). The different types of diabetes mellitus vary in cause, clinical course, and treatment (Table 41-1). The classification system is dynamic in two ways. First, research findings suggest many differences among individuals within each category. Second, except for people with type 1 diabetes, patients may move from one category to another. For example, a woman with gestational diabetes may, after delivery, move into the type 2 category. **Prediabetes** is classified as **impaired glucose tolerance (IGT)** or **impaired fasting glucose (IFG)** and refers to a condition in which blood glucose concentrations fall between normal levels and those considered diagnostic for diabetes.

Pathophysiology

Insulin is secreted by beta cells, which are one of four types of cells in the islets of Langerhans in the pancreas. Insulin is an anabolic, or storage, hormone. When a person eats a meal, insulin secretion increases and moves glucose from the blood into muscle, liver, and fat cells. In those cells, insulin

- Transports and metabolizes glucose for energy
- Stimulates storage of glucose in the liver and muscle (in the form of glycogen)
- Signals the liver to stop the release of glucose
- Enhances storage of dietary fat in adipose tissue
- Accelerates transport of amino acids (derived from dietary protein) into cells

Insulin also inhibits the breakdown of stored glucose, protein, and fat.

During fasting periods (between meals and overnight), the pancreas continuously releases a small amount of insulin (basal insulin); another pancreatic hormone called glucagon (secreted by the alpha cells of the islets of Langerhans) is released when blood glucose levels decrease and stimulates the liver to release stored glucose. The insulin

Table 41-1 CLASSIFICATION OF DIABETES MELLITUS AND RELATED GLUCOSE INTOLERANCES

Current Classification	Clinical Characteristics and Clinical Implications
Type 1 (5–10% of all diabetes) (Previously classified as juvenile diabetes, juvenile-onset diabetes, ketosis-prone diabetes, brittle diabetes, and insulin-dependent diabetes mellitus [IDDM])	Onset any age, but usually young (<30 y) Usually thin at diagnosis; recent weight loss Etiology includes genetic, immunologic, and environmental factors (eg, virus) Often have islet cell antibodies Often have antibodies to insulin even before insulin treatment Little or no endogenous insulin Need insulin to preserve life Ketosis prone when insulin absent Acute complication of hyperglycemia: diabetic ketoacidosis
Type 2 (90–95% of all diabetes: obese—80% of type 2; nonobese—20% of type 2) (Previously classified as adult-onset diabetes, maturity-onset diabetes, ketosis-resistant diabetes, stable diabetes, and non–insulin-dependent diabetes [NIDDM])	Onset any age, usually over 30 y Usually obese at diagnosis Causes include obesity, heredity, and environmental factors No islet cell antibodies Decrease in endogenous insulin, or increased with insulin resistance Most patients can control blood glucose through weight loss if obese Oral antidiabetic agents may improve blood glucose levels if dietary modification and exercise are unsuccessful May need insulin on a short-term or long-term basis to prevent hyperglycemia Ketosis uncommon, except in stress or infection Acute complication: hyperglycemic hyperosmolar nonketotic syndrome
Diabetes mellitus associated with other conditions or syndromes (Previously classified as secondary diabetes)	Accompanied by conditions known or suspected to cause the disease: pancreatic diseases, hormonal abnormalities, medications such as corticosteroids and estrogen-containing preparations Depending on the ability of the pancreas to produce insulin, the patient may require treatment with oral antidiabetic agents or insulin
Gestational diabetes	Onset during pregnancy, usually in the second or third trimester Due to hormones secreted by the placenta, which inhibit the action of insulin Above-normal risk for perinatal complications, especially macrosomia (abnormally large babies) Treated with diet and, if needed, insulin to strictly maintain normal blood glucose levels Occurs in about 2–5% of all pregnancies Glucose intolerance transitory but may recur: • In subsequent pregnancies • 30–40% will develop overt diabetes (usually type 2) within 10 years (especially if obese) Risk factors include obesity, age older than 30 years, family history of diabetes, previous large babies (>9 lb) Screening tests (glucose challenge test) should be performed on all pregnant women between 24- and 28-weeks gestation Should be screened for diabetes periodically
Prediabetes (Previously classified as previous abnormality of glucose tolerance [PrevAGT])	Previous history of hyperglycemia (eg, during pregnancy or illness) Current normal glucose metabolism Impaired glucose tolerance or impaired fasting glucose screening after age 40 years if there is a family history of diabetes or if symptomatic Encourage ideal body weight, because loss of 10–15 lb may improve glycemic control

and the glucagon together maintain a constant level of glucose in the blood by stimulating the release of glucose from the liver.

Initially, the liver produces glucose through the breakdown of glycogen (glycogenolysis). After 8 to 12 hours without food, the liver forms glucose from the breakdown of noncarbohydrate substances, including amino acids (gluconeogenesis).

Type 1 Diabetes

Type 1 diabetes affects approximately 5% to 10% of people with the disease; it is characterized by an acute onset, usually before 30 years of age (CDC, 2008). Type 1 diabetes is characterized by destruction of the pancreatic beta cells. Combined genetic, immunologic, and possibly environmental (eg, viral) factors are thought to contribute to beta-cell destruction. Although the events that lead to beta-cell destruction are not fully understood, it is generally accepted that a genetic susceptibility is a common underlying factor in the development of type 1 diabetes. People do not inherit type 1 diabetes itself but rather a genetic predisposition, or tendency, toward development of type 1 diabetes. This genetic tendency has been found in people with certain human leukocyte antigen (HLA) types. There is also evidence of an autoimmune response in type 1 diabetes. This is an abnormal response in which antibodies are directed against normal tissues of the body, responding to these tissues as if they were foreign. Autoantibodies against islet cells and against endogenous (internal) insulin have been detected in people at the time of diagnosis and even several years before the development of clinical signs of type 1 diabetes. In addition to genetic and immunologic components, environmental factors, such as viruses or toxins, that may initiate destruction of the beta cell are being investigated.

Regardless of the specific cause, the destruction of the beta cells results in decreased insulin production, unchecked glucose production by the liver, and fasting

hyperglycemia. In addition, glucose derived from food cannot be stored in the liver but instead remains in the bloodstream and contributes to postprandial (after meals) hyperglycemia. If the concentration of glucose in the blood exceeds the renal threshold for glucose, usually 180 to 200 mg/dL (9.9 to 11.1 mmol/L), the kidneys may not reabsorb all of the filtered glucose; the glucose then appears in the urine (glycosuria). When excess glucose is excreted in the urine, it is accompanied by excessive loss of fluids and electrolytes. This is called osmotic diuresis.

Because insulin normally inhibits glycogenolysis (breakdown of stored glucose) and gluconeogenesis (production of new glucose from amino acids and other substrates), these processes occur in an unrestrained fashion in people with insulin deficiency and contribute further to hyperglycemia. In addition, fat breakdown occurs, resulting in an increased production of **ketone** bodies, which are the byproducts of fat breakdown.

NURSING ALERT

Ketone bodies are acids that disturb the acid–base balance of the body when they accumulate in excessive amounts. The resulting diabetic ketoacidosis (DKA) may cause signs and symptoms such as abdominal pain, nausea, vomiting, hyperventilation, a fruity breath odor, and, if left untreated, altered level of consciousness, coma, and death. Initiation of insulin treatment, along with fluid and electrolytes as needed, is essential to treat hyperglycemia and DKA and rapidly improves the metabolic abnormalities.

Type 2 Diabetes

Type 2 diabetes affects approximately 90% to 95% of people with the disease (CDC, 2008). It occurs more commonly among people who are older than 30 years of age and obese (National Institute of Diabetes and Digestive and Kidney Diseases [NIDDK], 2005), although its incidence is rapidly increasing in younger people because of the growing epidemic of obesity in children, adolescents, and young adults (CDC, 2008). The two main problems related to insulin in type 2 diabetes are insulin resistance and impaired insulin secretion. Insulin resistance refers to a decreased tissue sensitivity to insulin. Normally, insulin binds to special receptors on cell surfaces and initiates a series of reactions involved in glucose metabolism. In type 2 diabetes, these intracellular reactions are diminished, making insulin less effective at stimulating glucose uptake by the tissues and at regulating glucose release by the liver (Fig. 41-1). The exact mechanisms that lead to insulin resistance and impaired insulin secretion in type 2 diabetes are unknown, although genetic factors are thought to play a role.

To overcome insulin resistance and to prevent the buildup of glucose in the blood, increased amounts of insulin must be secreted to maintain the glucose level at a normal or slightly elevated level. This is called metabolic syndrome, which includes hypertension, hypercholesterolemia, and abdominal obesity. However, if the beta cells cannot keep up with the increased demand for insulin, the glucose level rises and type 2 diabetes develops.

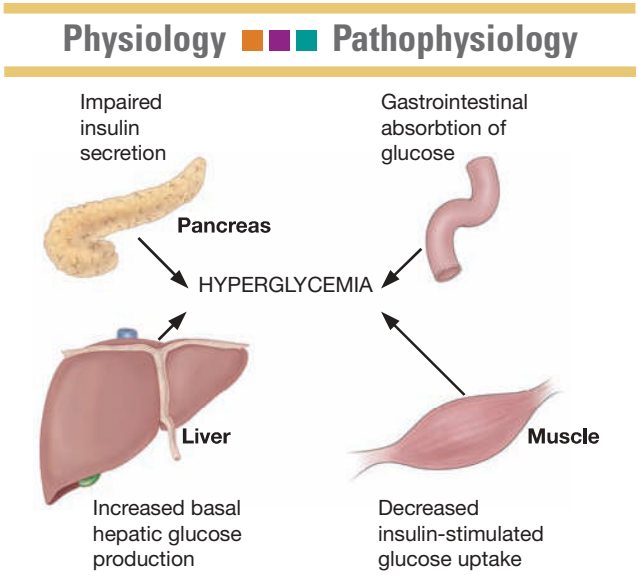


Figure 41-1 Pathogenesis of type 2 diabetes.

Despite the impaired insulin secretion that is characteristic of type 2 diabetes, there is enough insulin present to prevent the breakdown of fat and the accompanying production of ketone bodies. Therefore, DKA does not typically occur in type 2 diabetes. However, uncontrolled type 2 diabetes may lead to another acute problem—hyperglycemic hyperosmolar nonketotic syndrome (see later discussion).

Because type 2 diabetes is associated with a slow, progressive glucose intolerance, its onset may go undetected for many years. If the patient experiences symptoms, they are frequently mild and may include fatigue, irritability, polyuria, polydipsia, poorly healing skin wounds, vaginal infections, or blurred vision (if glucose levels are very high).

For most patients (approximately 75%), type 2 diabetes is detected incidentally (eg, when routine laboratory tests or ophthalmoscopic examinations are performed). One consequence of undetected diabetes is that long-term diabetes complications (eg, eye disease, peripheral neuropathy, peripheral vascular disease) may have developed before the actual diagnosis of diabetes is made (ADA, 2009a), signifying that the blood glucose has been elevated for a time before diagnosis.

Gestational Diabetes

Gestational diabetes mellitus (GDM) is any degree of glucose intolerance with its onset during pregnancy. Hyperglycemia develops during pregnancy because of the secretion of placental hormones, which causes insulin resistance. Gestational diabetes occurs in as many as 14% of pregnant women and increases their risk for hypertensive disorders during pregnancy (ADA, 2009a).

Women who are considered to be at high risk for GDM and who should be screened by blood glucose testing at their

first prenatal visit are those with marked obesity, a personal history of GDM, glycosuria, or a strong family history of diabetes. High-risk ethnic groups include Hispanic Americans, Native Americans, Asian Americans, African Americans, and Pacific Islanders. If these high-risk women do not have GDM at initial screening, they should be retested between 24 and 28 weeks of gestation. All women of average risk should be tested at 24 to 28 weeks of gestation. Testing is not specifically recommended for women identified as being at low risk. Low-risk women are those who meet all of the following criteria: age younger than 25 years, normal weight before pregnancy, member of an ethnic group with low prevalence of GDM, no history of abnormal glucose tolerance, no known history of diabetes in first-degree relatives, and no history of poor obstetric outcome (ADA, 2009a). Women considered to be at high risk or average risk should have either an oral glucose tolerance test (OGTT) or a glucose challenge test (GCT) followed by OGTT in women who exceed the glucose threshold value of 140 mg/dL (7.8 mmol/L) (ADA, 2009a).

Initial management includes dietary modification and blood glucose monitoring. If hyperglycemia persists, insulin is prescribed. Goals for blood glucose levels during pregnancy are 105 mg/dL (5.8 mmol/L) or less before meals and 130 mg/dL (7.2 mmol/L) or less 2 hours after meals (ADA, 2009a).

After delivery, blood glucose levels in women with GDM usually return to normal. However, many women who have had GDM develop type 2 diabetes later in life. For this reason, a woman who has had GDM should be counseled to maintain her ideal body weight and to exercise regularly to reduce her risk for type 2 diabetes (Kitzmiller, Dang-Kilduff & Taslimi, 2007).

Prevention

In 2002 the Diabetes Prevention Program Research Group reported that type 2 diabetes can be prevented with appropriate changes in lifestyle. Persons at high risk for type 2 diabetes (BMI 24 or greater, fasting and postprandial plasma glucose levels elevated but not to levels diagnostic of diabetes) received either standard lifestyle recommendations plus metformin, standard lifestyle recommendations plus placebo, or an intensive program of lifestyle modifications. The 16-lesson curriculum of the intensive program of lifestyle modifications focused on weight reduction of greater than 7% of initial body weight and physical activity of moderate intensity. It also included behavior modification strategies designed to help patients achieve the goals of weight reduction and participation in exercise. The lifestyle intervention group had a 58% lower incidence of diabetes and the metformin group had a 31% lower incidence of diabetes compared to the placebo group. These findings were found in both genders and all racial and ethnic groups. These findings demonstrate that type 2 diabetes can be prevented or delayed in persons at high risk for the disease.

Clinical Manifestations

Clinical manifestations depend on the patient's level of hyperglycemia. Classic clinical manifestations of all types of diabetes include the "three Ps": polyuria, polydipsia, and polyphagia. Polyuria (increased urination) and polydipsia (increased thirst) occur as a result of the excess loss of fluid associated with osmotic diuresis. Patients also experience polyphagia (increased appetite) that results from the catabolic state induced by insulin deficiency and the breakdown of proteins and fats. Other symptoms include fatigue and weakness, sudden vision changes, tingling or numbness in hands or feet, dry skin, skin lesions or wounds that are slow to heal, and recurrent infections. The onset of type 1 diabetes may also be associated with sudden weight loss or nausea, vomiting, or abdominal pains, if DKA has developed.

Assessment and Diagnostic Findings

An abnormally high blood glucose level is the basic criterion for the diagnosis of diabetes. **Fasting plasma glucose (FPG),** random plasma glucose, and glucose level 2 hours after receiving glucose (2-hour postload) may be used. The OGTT and the intravenous (IV) glucose tolerance test are no longer recommended for routine clinical use. See Chart 41-2 for the ADA's diagnostic criteria for diabetes mellitus (ADA, 2009a).

In addition to the assessment and diagnostic evaluation performed to diagnose diabetes, ongoing specialized assessment of patients with known diabetes and evaluation for complications in patients with newly diagnosed diabetes are important components of care. Parameters that should be regularly assessed are discussed in Chart 41-3.

Chart 41-2 • *Criteria for the Diagnosis of Diabetes Mellitus*

1. Symptoms of diabetes plus casual plasma glucose concentration equal to or greater than 200 mg/dL (11.1 mmol/L). Casual is defined as any time of day without regard to time since last meal. The classic symptoms of diabetes include polyuria, polydipsia, and unexplained weight loss.

or

2. Fasting plasma glucose greater than or equal to 126 mg/dL (7.0 mmol/L). Fasting is defined as no caloric intake for at least 8 hours.

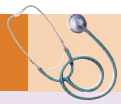
or

3. Two-hour postload glucose equal to or greater than 200 mg/dL (11.1 mmol/L) during an oral glucose tolerance test. The test should be performed as described by the World Health Organization, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water.

In the absence of unequivocal hyperglycemia with acute metabolic decompensation, these criteria should be confirmed by repeat testing on a different day. The third measure is not recommended for routine clinical use.

Used with permission of American Diabetes Association. (2009a). Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, *Diabetes Care, 32*(Suppl. 1), S62–S67.

CHART 41-3



Assessing the Patient With Diabetes

History

- Symptoms related to the diagnosis of diabetes:
  Symptoms of hyperglycemia
  Symptoms of hypoglycemia
  Frequency, timing, severity, and resolution
- Results of blood glucose monitoring
- Status, symptoms, and management of chronic complications of diabetes:
  Eye; kidney; nerve; genitourinary and sexual, bladder, and gastrointestinal
  Cardiac; peripheral vascular; foot complications associated with diabetes
- Adherence to/ability to follow prescribed dietary management plan
- Adherence to prescribed exercise regimen
- Adherence to/ability to follow prescribed pharmacologic treatment (insulin or oral antidiabetic agents)
- Use of tobacco, alcohol, and prescribed and over-the-counter medications/drugs
- Lifestyle, cultural, psychosocial, and economic factors that may affect diabetes treatment
- Effects of diabetes or its complications on functional status (eg, mobility, vision)

Physical Examination

- Blood pressure (sitting and standing to detect orthostatic changes)
- Body mass index (height and weight)
- Fundoscopic examination and visual acuity
- Foot examination (lesions, signs of infection, pulses)
- Skin examination (lesions and insulin-injection sites)
- Neurologic examination
  Vibratory and sensory examination using monofilament
  Deep tendon reflexes
- Oral examination

Laboratory Examination

- $HgbA_{1C}$ (A1C)
- Fasting lipid profile
- Test for microalbuminuria
- Serum creatinine level
- Urinalysis
- Electrocardiogram

Need for Referrals

- Ophthalmology
- Podiatry
- Dietitian
- Diabetes educator
- Others if indicated

Gerontologic Considerations

Elevated blood glucose levels appear to be age related and occur in both men and women throughout the world. Elevated blood glucose levels commonly appear in the fifth decade of life and increase in frequency with advancing age. Approximately 10% to 30% of elderly people have age-related hyperglycemia, not counting those with overt diabetes. What causes age-related changes in carbohydrate metabolism is not known. Possibilities include poor diet, physical inactivity, a decrease in the lean body mass in which ingested carbohydrate may be stored, altered insulin secretion, and increase in fat tissue, which increases insulin resistance (ADA, 2009b).

Medical Management

The main goal of diabetes treatment is to normalize insulin activity and blood glucose levels to reduce the development of vascular and neuropathic complications. The Diabetes Control and Complications Trial (DCCT), a 10-year prospective clinical trial conducted from 1983 to 1993, demonstrated the importance of achieving blood glucose control in the normal, nondiabetic range. This landmark trial demonstrated that intensive glucose control dramatically reduced the development and progression of complications such as **retinopathy, nephropathy,** and **neuropathy.** Intensive treatment is defined as three or four insulin injections per day or **continuous subcutaneous insulin infusion, insulin pump** therapy plus frequent blood glucose monitoring and weekly contacts with diabetes educators. The American Diabetes Association now recommends that all patients with diabetes strive for glucose control to reduce their risk of complications (ADA, 2009b).

Intensive therapy must be initiated with caution and must be accompanied by thorough education of the patient and family and by responsible behavior of the patient. Careful screening of patients is a key step in initiating intensive therapy.

A study conducted in the United Kingdom demonstrated a decrease in complications among patients with type 2 diabetes receiving intensive therapy compared to those receiving conventional therapy (United Kingdom Prospective Diabetes Study Group [UKPDS], 1998; see also ADA, 2009b).

The results of the DCCT and UKPDS have been supported by follow-up studies, including the Epidemiology of Diabetes Interventions and Complications (EDIC) study (Nathan, Cleary, Backlund, et al., 2005). Therefore, the therapeutic goal for diabetes management is to achieve normal blood glucose levels (euglycemia) without hypoglycemia while maintaining a high quality of life. Diabetes management has five components: nutritional therapy, exercise, monitoring, pharmacologic therapy, and education. Diabetes management involves constant assessment and modification of the treatment plan by health professionals and daily adjustments in therapy by the patient. Although the health care team directs the treatment, it is the individual patient who must manage the complex therapeutic regimen. For this reason, patient and family education is an

essential component of diabetes treatment and is as important as all other components of the regimen.

Nutritional Therapy

Nutrition, meal planning, and weight control are the foundation of diabetes management. The most important objectives in the dietary and nutritional management of diabetes are control of total caloric intake to attain or maintain a reasonable body weight, control of blood glucose levels, and normalization of lipids and blood pressure to prevent heart disease. Success in this area alone is often associated with reversal of hyperglycemia in type 2 diabetes. However, achieving these goals is not always easy. Because **medical nutrition therapy** (MNT, nutritional management) of diabetes is complex, a registered dietitian who understands diabetes management has the major responsibility for designing and teaching this aspect of the therapeutic plan. Nurses and all other members of the health care team must be knowledgeable about nutritional therapy and supportive of patients who need to implement nutritional and lifestyle changes. Nutritional management of diabetes includes the following goals (ADA, 2008b).

1. To achieve and maintain
   - Blood glucose levels in the normal range or as close to normal as is safely possible
   - A lipid and lipoprotein profile that reduces the risk for vascular disease
   - Blood pressure levels in the normal range or as close to normal as is safely possible
2. To prevent, or at least slow, the rate of development of the chronic complications of diabetes by modifying nutrient intake and lifestyle
3. To address individual nutrition needs, taking into account personal and cultural preferences and willingness to change
4. To maintain the pleasure of eating by only limiting food choices when indicated by scientific evidence.

For obese patients with diabetes (especially those with type 2 diabetes), weight loss is the key to treatment. (It is also a major factor in preventing diabetes.) In general, overweight is considered to be a body mass index (BMI) of 25 to 29; obesity is defined as 20% above ideal body weight or a BMI equal to or greater than 30 (National Institutes of Health, 2000). BMI is a weight-to-height ratio calculated by dividing body weight (in kilograms) by the square of the height (in meters). Calculation of BMI is discussed in Chapter 5. Obese patients who have type 2 diabetes and who require insulin or oral agents to control blood glucose levels may be able to reduce or eliminate the need for medication through weight loss. A weight loss as small as 5% to 10% of total weight may significantly improve blood glucose levels (ADA, 2009b). For obese patients with diabetes who do not take insulin or sulfonylureas, consistent meal content or timing is important but not as critical. Rather, decreasing the overall caloric intake assumes more importance. However, meals should not be skipped. Pacing food intake throughout the day places more manageable demands on the pancreas.

Consistently following a meal plan is one of the most challenging aspects of diabetes management. It may be more realistic to restrict calories only moderately. For patients who have lost weight, maintaining the weight loss may be difficult. To help these patients incorporate new dietary habits into their lifestyles, diet education, behavioral therapy, group support, and ongoing nutrition counseling are encouraged.

Meal Planning and Related Teaching

The meal plan must consider the patient's food preferences, lifestyle, usual eating times, and ethnic and cultural background. For patients who require insulin to help control blood glucose levels, maintaining as much consistency as possible in the amount of calories and carbohydrates ingested at each meal is essential. In addition, consistency in the approximate time intervals between meals, with the addition of snacks if necessary, helps prevent hypoglycemic reactions and maintain overall blood glucose control. For patients who can master the insulin-to-carbohydrate calculations, lifestyle can be more flexible and diabetes control more predictable. For those using intensive insulin therapy, there may be greater flexibility in the timing and content of meals by allowing adjustments in insulin dosage for changes in eating and exercise habits. Advances in insulin management (new insulin analogues, insulin algorithms, insulin pumps) permit greater flexibility of schedules than was previously possible. This contrasts with the older concept of maintaining a constant dose of insulin, which required strict scheduling of meals to match the actions and duration of the insulin.

The first step in preparing a meal plan is a thorough review of the patient's diet history to identify his or her eating habits and lifestyle. This includes a thorough assessment of the patient's need for weight loss, gain, or maintenance. In most instances, people with type 2 diabetes require weight reduction.

In teaching about meal planning, clinical dietitians use various educational tools, materials, and approaches. Initial education addresses the importance of consistent eating habits, the relationship of food and insulin, and the provision of an individualized meal plan. In-depth follow-up education then focuses on management skills, such as eating at restaurants, reading food labels, and adjusting the meal plan for exercise, illness, and special occasions. The nurse plays an important role in communicating pertinent information to the dietitian and reinforcing the patient's understanding.

Certain aspects of meal planning, such as the food exchange system, may be difficult to learn. This may be related to limitations in the patient's intellectual level or to emotional issues, such as difficulty accepting the diagnosis of diabetes or feelings of deprivation and undue restriction in eating. In any case, it helps to emphasize that using the exchange system (or any food classification system) provides a new way of thinking about food rather than a new way of eating. It is also important to simplify information as much as possible and to provide opportunities for the patient to practice and repeat activities and information.

Caloric Requirements. Calorie-controlled diets are planned by first calculating a person's energy needs and caloric requirements based on age, gender, height, and weight. An activity element is then factored in to provide

the actual number of calories required for weight maintenance. To promote a 1- to 2-pound weight loss per week, 500 to 1000 calories are subtracted from the daily total. The calories are distributed into carbohydrates, proteins, and fats, and a meal plan is then developed, taking into account the patient's lifestyle and food preferences.

In contrast to the priority for the obese person with type 2 diabetes, the priority for a young patient with type 1 diabetes should be a diet with enough calories to maintain normal growth and development. Some patients may be underweight at the onset of type 1 diabetes because of rapid weight loss from severe hyperglycemia. The goal initially may be to provide a higher-calorie diet to regain lost weight and blood glucose control.

Caloric Distribution. A meal plan for diabetes focuses on the percentages of calories that come from carbohydrates, proteins, and fats.

Carbohydrates. The caloric distribution currently recommended is higher in carbohydrates than in fat and protein. In general, carbohydrate foods have the greatest effect on blood glucose levels because they are more quickly digested than other foods and are converted into glucose rapidly. However, research into the appropriateness of a higher-carbohydrate diet in patients with decreased glucose tolerance is ongoing, and recommendations may change accordingly. Currently, the ADA and the American Dietetic Association recommend that for all levels of caloric intake, 50% to 60% of calories should be derived from carbohydrates, 20% to 30% from fat, and the remaining 10% to 20% from protein. The majority of the selections for carbohydrates should come from whole grains. These recommendations are also consistent with those of the American Heart Association and American Cancer Society.

Carbohydrates consist of sugars (eg, sucrose) and starches (eg, rice, pasta, bread). Low glycemic index diets (described later) may reduce postprandial glucose levels. Therefore, the nutrition guidelines recommend that all carbohydrates should be eaten in moderation to avoid high postprandial blood glucose levels (ADA, 2008b).

Foods high in carbohydrates, such as sucrose (concentrated sweets), are not totally eliminated from the diet but should be eaten in moderation (up to 10% of total calories), because they are typically high in fat and lack vitamins, minerals, and fiber.

Fats. The recommendations regarding fat content of the diabetic diet include both reducing the total percentage of calories from fat sources to less than 30% of total calories and limiting the amount of saturated fats to 10% of total calories. Additional recommendations include limiting the total intake of dietary cholesterol to less than 300 mg/day. This approach may help reduce risk factors such as increased serum cholesterol levels, which are associated with the development of coronary artery disease, the leading cause of death and disability among people with diabetes (ADA, 2008c).

Protein. The meal plan may include the use of some nonanimal sources of protein (eg, legumes, whole grains), to help reduce saturated fat and cholesterol intake. In addition, the amount of protein intake may be reduced in patients with early signs of renal disease.

Fiber. Increased fiber in the diet may improve blood glucose levels, decrease the need for exogenous insulin, and lower total cholesterol and low-density lipoprotein levels in the blood (ADA, 2008b).

There are two types of dietary fibers: soluble and insoluble. Soluble fiber—in foods such as legumes, oats, and some fruits—plays more of a role in lowering blood glucose and lipid levels than does insoluble fiber, although the clinical significance of this effect is probably small (ADA, 2008b). Soluble fiber slows stomach emptying and the movement of food through the upper digestive tract. The potential glucose-lowering effect of fiber may be caused by the slower rate of glucose absorption from foods that contain soluble fiber. Insoluble fiber is found in whole-grain breads and cereals and in some vegetables. This type of fiber along with soluble fiber increases satiety, which is helpful for weight loss. At least 25 g of fiber should be ingested daily.

One risk involved in suddenly increasing fiber intake is that it may require adjusting the dosage of insulin or oral agents to prevent hypoglycemia. Other problems may include abdominal fullness, nausea, diarrhea, increased flatulence, and constipation if fluid intake is inadequate. If fiber is added to or increased in the meal plan, it should be done gradually and in consultation with a dietitian. The exchange lists (ADA, 2008b) serve as an excellent guide for increasing fiber intake. Fiber-rich food choices within the vegetable, fruit, and starch/bread exchanges are highlighted in the lists.

Food Classification Systems. To teach diet principles and to help in meal planning, several systems have been developed in which foods are organized into groups with common characteristics, such as number of calories, composition of foods (ie, amount of protein, fat, or carbohydrate in the food), or effect on blood glucose levels. Several of these are listed here.

Exchange Lists. A commonly used tool for nutritional management is the exchange lists for meal planning (ADA, 2008b). There are six main exchange lists: bread/starch, vegetable, milk, meat, fruit, and fat. Foods within one group (in the portion amounts specified) contain equal numbers of calories and are approximately equal in grams of protein, fat, and carbohydrate. Meal plans can be based on a recommended number of choices from each exchange list. Foods on one list may be interchanged with one another, allowing for variety while maintaining as much consistency as possible in the nutrient content of foods eaten. Table 41-2 presents three sample lunch menus that are interchangeable in terms of carbohydrate, protein, and fat content.

Exchange list information on combination foods such as pizza, chili, and casseroles, as well as convenience foods, desserts, snack foods, and fast foods, is available from the American Diabetes Association (see Resources). Some food manufacturers and restaurants publish exchange lists that describe their products.

Nutrition Labels. Food manufacturers are required to have the nutrition content of foods listed on package labels, and reading food labels is an important skill for patients to learn and use when food shopping. The label includes information about how many grams of carbohydrate are in a serving

Table 41-2 SELECTED SAMPLE MENUS FROM EXCHANGE LISTS

Exchanges	Sample Lunch #1	Sample Lunch #2	Sample Lunch #3
2 starch	2 slices bread	Hamburger bun	1 cup cooked pasta
3 meat	2 oz sliced turkey and 1 oz lowfat cheese	3 oz lean beef patty	3 oz boiled shrimp
1 vegetable	Lettuce, tomato, onion	Green salad	½ cup plum tomatoes
1 fat	1 tsp mayonnaise	1 tbsp salad dressing	1 tsp olive oil
1 fruit	1 medium apple	1¼ cup watermelon	1¼ cup fresh strawberries
"Free" items (optional)	Unsweetened iced tea Mustard, pickle, hot pepper	Diet soda 1 tbsp catsup, pickle, onions	Ice water with lemon Garlic, basil

of food. This information can be used to determine how much medication is needed. For example, a patient who takes premeal insulin may use the algorithm, 1 unit of insulin for 15 g of carbohydrate. Patients can also be taught to have a "carbohydrate budget" per meal (eg, 45 to 60 g).

Carbohydrate counting is a nutritional tool used for blood glucose management because carbohydrates are the main nutrients in food that influence blood glucose levels. This method provides flexibility in food choices, can be less complicated to understand than the diabetic food exchange list, and allows more accurate management with multiple daily injections (insulin before each meal). However, if carbohydrate counting is not used with other meal-planning techniques, weight gain can result. A variety of methods are used to count carbohydrates. When developing a diabetic meal plan using carbohydrate counting, all food sources should be considered.

Once digested, 100% of carbohydrates are converted to glucose. Approximately 50% of protein foods (meat, fish, and poultry) are also converted to glucose, but this has minimal effect on blood glucose levels.

Carbohydrate counting consists of counting grams of carbohydrates. If target goals are not reached by counting carbohydrates alone, protein is factored into the calculations. This is especially true if the meal consists only of meat, fish, and nonstarchy vegetables.

Although carbohydrate counting is now commonly used for blood glucose management with type 1 and type 2 diabetes, it is not a perfect system. All carbohydrates affect the blood glucose level to different degrees, regardless of equivalent serving size. When carbohydrate counting is used, reading labels on food items is the key to success. Knowing what the "carbohydrate budget" for the meal is and knowing how many grams of carbohydrate are in a serving of a food, the patient can calculate the amount in one serving.

Healthy Food Choices. An alternative to counting grams of carbohydrate is measuring servings or choices. This method is used more often by people with type 2 diabetes. It is similar to the food exchange list and emphasizes portion control of total servings of carbohydrate at meals and snacks. One carbohydrate serving is equivalent to 15 g of carbohydrate. Examples of one serving are an apple 2 inches in diameter and one slice of bread. Vegetables and meat are counted as one third of a carbohydrate serving. This system works well for those who have difficulty with the other complicated systems.

Food Guide Pyramid. The Food Guide Pyramid (ie, MyPyramid) is another tool used to develop meal plans. It is commonly used for patients with type 2 diabetes who have a difficult time following a calorie-controlled diet. The 2005 food pyramid consists of the following food groups: (1) grains, (2) vegetables, (3) fruits, (4) milk and other dairy products, and (5) meats and beans. Oils and other high-fat foods comprise another food group on the pyramid (see Chapter 5). Foods (starches, fruits, and vegetables) that are lowest in calories and fat and highest in fiber should make up the basis of the diet. For those with diabetes, as well as for the general population, 50% to 60% of the daily caloric intake should be from these three groups. Foods higher in fat (particularly saturated fat) should account for a smaller percentage of the daily caloric intake. Fats, oils, and sweets should be used sparingly to obtain weight and blood glucose control and to reduce the risk for cardiovascular disease. Reliance on the MyPyramid may result in fluctuations in blood glucose levels, however, because high-carbohydrate foods may be grouped with low-carbohydrate foods. The pyramid is appropriately used only as a first-step teaching tool for patients who are learning how to control food portions and how to identify which foods contain carbohydrate, protein, and fat.

Glycemic Index. One of the main goals of diet therapy in diabetes is to avoid sharp, rapid increases in blood glucose levels after food is eaten. The term glycemic index is used to describe how much a given food increases the blood glucose level compared with an equivalent amount of glucose. The effects of use of the glycemic index on blood glucose levels and on long-term patient outcomes are unclear, but it may be beneficial (ADA, 2008b). Although more research is necessary, the following guidelines may be helpful when making dietary recommendations:

- Combining starchy foods with protein-containing and fat-containing foods tends to slow their absorption and lower the glycemic response.
- In general, eating foods that are raw and whole results in a lower glycemic response than eating chopped, puréed, or cooked foods.
- Eating whole fruit instead of drinking juice decreases the glycemic response, because fiber in the fruit slows absorption.
- Adding foods with sugars to the diet may result in a lower glycemic response if these foods are eaten with foods that are more slowly absorbed.

Patients can create their own glycemic index by monitoring their blood glucose level after ingestion of a particular food. This can help improve blood glucose control through individualized manipulation of the diet. Many

patients who use frequent monitoring of blood glucose levels can use this information to adjust their insulin doses in accordance with variations in food intake.

Other Dietary Concerns

Alcohol Consumption. Patients with diabetes do not need to give up alcoholic beverages entirely, but they and health care professionals must be aware of the potential adverse effects of alcohol specific to diabetes. Alcohol is absorbed before other nutrients and does not require insulin for absorption. Large amounts can be converted to fats, increasing the risk for DKA. In general, the same precautions regarding the use of alcohol by people without diabetes should be applied to patients with diabetes. Moderation is recommended. A major danger of alcohol consumption by the patient with diabetes is hypoglycemia, especially for patients who take insulin or insulin secretagogues (medications that increase the secretion of insulin by the pancreas). Alcohol may decrease the normal physiologic reactions in the body that produce glucose (gluconeogenesis). Therefore, if a patient with diabetes consumes alcohol on an empty stomach, there is an increased likelihood of hypoglycemia. In addition, excessive alcohol intake may impair the patient's ability to recognize and treat hypoglycemia or to follow a prescribed meal plan to prevent hypoglycemia. To reduce the risk of hypoglycemia, the patient should be cautioned to consume food along with the alcohol; however, carbohydrate consumed with alcohol may raise blood glucose (ADA, 2008b).

Alcohol consumption may lead to excessive weight gain (from the high caloric content of alcohol), hyperlipidemia, and elevated glucose levels (especially with mixed drinks and liqueurs). Patient teaching regarding alcohol intake must emphasize moderation in the amount of alcohol consumed. Moderate intake is considered to be one alcoholic beverage per day for women and two per day for men. Lower-calorie or less sweet drinks (eg, light beer, dry wine) and food intake along with alcohol consumption are advised. Especially for patients with type 2 diabetes who wish to control their weight, it is important to incorporate the calories from alcohol into the overall meal plan.

Sweeteners. Use of artificial sweeteners is acceptable, especially if it assists in overall dietary adherence. Moderation in the amount of sweetener used is encouraged to avoid potential adverse effects. There are two main types of sweeteners: nutritive and nonnutritive. The nutritive sweeteners contain calories, and the nonnutritive sweeteners have few or no calories in the amounts normally used.

Nutritive sweeteners include fructose (fruit sugar), sorbitol, and xylitol, all of which provide calories in amounts similar to those in sucrose (table sugar). They cause less elevation in blood sugar levels than sucrose does and are often used in "sugar-free" foods. Sweeteners containing sorbitol may have a laxative effect.

Nonnutritive sweeteners have minimal or no calories. They are used in food products and are also available for table use. They produce minimal or no elevation in blood glucose levels and have been approved by the U.S. Food and Drug Administration (FDA) as safe for people with diabetes. Nonnutritive sweeteners include saccharin, aspartame (NutraSweet), acesulfame-K (Sunnette), and sucralose (Splenda) (ADA, 2009b).

Misleading Food Labels. Foods labeled "sugarless" or "sugar-free" may still provide calories equal to those of the equivalent sugar-containing products if they are made with nutritive sweeteners. Therefore, these foods should not be considered "free" foods to be eaten in unlimited quantity, because they can elevate blood glucose levels. Foods labeled "dietetic" are not necessarily reduced-calorie foods. Patients are advised that foods labeled "dietetic" may still contain significant amounts of sugar or fat.

It is important that patients read the labels of "health foods"—especially snacks—because they often contain carbohydrates (eg, honey, brown sugar, corn syrup) and saturated vegetable fats (eg, coconut or palm oil), hydrogenated vegetable fats, or animal fats, which may be contraindicated in people with elevated blood lipid levels.

Exercise

Exercise is extremely important in diabetes management because of its effects on lowering blood glucose and reducing cardiovascular risk factors. Exercise lowers blood glucose levels by increasing the uptake of glucose by body muscles and by improving insulin utilization. It also improves circulation and muscle tone. Resistance (strength) training, such as weight lifting, can increase lean muscle mass, thereby increasing the resting metabolic rate. These effects are useful in diabetes in relation to losing weight, easing stress, and maintaining a feeling of well-being. Exercise also alters blood lipid concentrations, increasing levels of high-density lipoproteins and decreasing total cholesterol and triglyceride levels. This is especially important for people with diabetes because of their increased risk of cardiovascular disease (Nathan, et al., 2005).

Exercise Recommendations

Ideally, a person with diabetes should exercise at the same time (preferably when blood glucose levels are at their peak) and in the same amount each day. Regular daily exercise, rather than sporadic exercise, should be encouraged. Exercise recommendations must be altered as necessary for patients with diabetic complications such as retinopathy, autonomic neuropathy, sensorimotor neuropathy, and cardiovascular disease (ADA, 2009b). Increased blood pressure associated with exercise may aggravate diabetic retinopathy and increase the risk of a hemorrhage into the vitreous or retina. Patients with ischemic heart disease risk triggering angina or a myocardial infarction, which may be silent. Avoiding trauma to the lower extremities is especially important in patients with numbness related to neuropathy.

In general, a slow, gradual increase in the exercise period is encouraged. For many patients, walking is a safe and beneficial form of exercise that requires no special equipment (except for proper shoes) and can be performed anywhere. People with diabetes should discuss an exercise program with their health care providers and undergo a careful medical evaluation with appropriate diagnostic studies before beginning program (ADA, 2009b).

For patients who are older than 30 years and who have two or more risk factors for heart disease, an exercise stress

test is recommended. Risk factors for heart disease include hypertension, obesity, high cholesterol levels, abnormal resting electrocardiogram (ECG), sedentary lifestyle, smoking, male gender, and a family history of heart disease. An abnormal stress test may indicate cardiac ischemia. Typically, an abnormal stress test is followed up with a cardiac catheterization and, in some cases, with an intervention such as angioplasty, stent replacement, or cardiac surgery.

Exercise Precautions

Patients who have blood glucose levels exceeding 250 mg/dL (14 mmol/L) and who have ketones in their urine should not begin exercising until the urine test results are negative for ketones and the blood glucose level is closer to normal. Exercising with elevated blood glucose levels increases the secretion of glucagon, growth hormone, and catecholamines. The liver then releases more glucose, and the result is an increase in the blood glucose level (ADA, 2009b).

The physiologic decrease in circulating insulin that normally occurs with exercise cannot occur in patients treated with insulin. Initially, patients who require insulin should be taught to eat a 15-g carbohydrate snack (a fruit exchange) or a snack of complex carbohydrates with a protein before engaging in moderate exercise to prevent unexpected hypoglycemia. The exact amount of food needed varies from person to person and should be determined by blood glucose monitoring.

Another potential concern for patients who take insulin is hypoglycemia that occurs many hours after exercise. To avoid postexercise hypoglycemia, especially after strenuous or prolonged exercise, the patient may need to eat a snack at the end of the exercise session and at bedtime and monitor the blood glucose level more frequently. Patients who are capable, knowledgeable, and responsible can learn to adjust their own insulin doses by working closely with a diabetes educator. Others need specific instructions on what to do when they exercise.

Patients taking insulin and participating in extended periods of exercise should test their blood glucose levels before, during, and after the exercise period, and they should snack on carbohydrates as needed to maintain blood glucose levels (ADA, 2006a). Other participants or observers should be aware that the person exercising has diabetes, and they should know what assistance to give if severe hypoglycemia occurs.

In obese people with type 2 diabetes, exercise in addition to dietary management both improves glucose metabolism and enhances loss of body fat. Exercise coupled with weight loss improves insulin sensitivity and may decrease the need for insulin or oral antidiabetic agents (ADA, 2006a). Eventually, the patient's glucose tolerance may return to normal. Patients with type 2 diabetes who are not taking insulin or an oral agent may not need extra food before exercise.

General precautions for exercise in diabetes are presented in Chart 41-4.

CHART 41-4 PATIENT EDUCATION

General Precautions for Exercise in People With Diabetes

- Use proper footwear and, if appropriate, other protective equipment.
- Avoid exercise in extreme heat or cold.
- Inspect feet daily after exercise.
- Avoid exercise during periods of poor metabolic control.

Gerontologic Considerations

Physical activity that is consistent and realistic is beneficial to elderly people with diabetes. Physical fitness in the elderly population with diabetes may lead to improved glycemic control, decreased risk for chronic vascular disease, and an improved quality of life (ADA, 2006a). Advantages of exercise in this population include a decrease in hyperglycemia, a general sense of well-being, and better use of ingested calories, resulting in weight reduction. Because there is an increased incidence of cardiovascular problems in the elderly, a physical examination and exercise stress test may be warranted before an exercise program is initiated. A pattern of gradual, consistent exercise, including resistance exercise, should be planned that does not exceed the patient's physical capacity. Physical impairment due to other chronic diseases must also be considered. In some cases, a physical therapy evaluation may be indicated, with the goal of determining exercises specific to the patient's needs and abilities. Tools such as the "Armchair Fitness" video may be helpful. For more information about age-related changes that affect diabetes management, see Chart 41-5.

Monitoring Glucose Levels and Ketones

Blood glucose monitoring is a cornerstone of diabetes management, and **self-monitoring of blood glucose (SMBG)** levels has dramatically altered diabetes care.

Self-Monitoring of Blood Glucose

Using frequent SMBG and learning how to respond to the results enable people with diabetes to adjust their treatment regimen to obtain optimal blood glucose control. This allows for detection and prevention of hypoglycemia and hyperglycemia and plays a crucial role in normalizing blood glucose levels, which in turn may reduce the risk of long-term diabetic complications.

Various methods for SMBG are available. Most involve obtaining a drop of blood from the fingertip, applying the blood to a special reagent strip, and allowing the blood to stay on the strip for the amount of time specified by the manufacturer (usually 5 to 30 seconds). The meter gives a digital readout of the blood glucose value. The meters available for SMBG offer various features and benefits such as monthly averages, tracking of events like exercise and food consumption, and downloading capacity. Some meters are biosensors that can use blood obtained from alternative test sites, such as the forearm. They have a special lancing device that is useful for patients who have painful fingertips or experience pain with fingersticks.

Because laboratory methods measure plasma glucose, most blood glucose monitors approved for patients' use in the home and some test strips calibrate blood glucose readings to plasma values. Plasma glucose values are 10% to 15% higher than whole blood glucose values, and it is

CHART 41-5

GERONTOLOGIC CONSIDERATIONS
Age-Related Changes That May Affect Diabetes and Its Management

Sensory Changes

Decreased vision
Decreased smell
Taste changes
Decreased proprioception
Diminished thirst

Gastrointestinal Changes

Dental problems
Appetite changes
Delayed gastric emptying
Decreased bowel motility

Activity/exercise Pattern Changes

More sedentary

Renal Function Changes

Decreased function
Decreased drug clearance

Affective/cognitive Changes

Medications/meals omitted or taken erratically

Socioeconomic Factors

Fad diets
Loneliness/living alone
Lack of money/lack of support system

Chronic Diseases

Hypertension
Arthritis
Neoplasms
Acute/chronic infections

Potential Drug Interactions

Use of another person's medications
Consulting multiple physicians for different illnesses
Alcohol use/abuse

crucial for patients with diabetes to know whether their monitor and strips provide whole blood or plasma results.

Methods for SMBG must match the skill level of patients. Factors affecting SMBG performance include visual acuity, fine motor coordination, cognitive ability, comfort with technology and willingness to use it, and cost. Some meters can be used by patients with visual impairments that have audio components to assist in performing the test and obtaining the result. In addition, meters are available to check both blood glucose and blood ketone levels by those who are particularly susceptible to DKA. Most insurance companies cover some or all of the costs of meters and strips.

A potential hazard of all methods of SMBG is that the patient may obtain and report erroneous blood glucose values as a result of using incorrect techniques. Some common sources of error include improper application of blood (eg, drop too small), damage to the reagent strips caused by heat or humidity, use of outdated strips, and improper meter cleaning and maintenance.

Nurses play an important role in providing initial teaching about SMBG techniques. Equally important is evaluating the techniques of patients who are experienced in self-monitoring. Every 6 to 12 months, patients should conduct a comparison of their meter result with a simultaneous laboratory-measured blood glucose level in their physician's office and have their technique observed. The accuracy of the meter and strips can also be assessed with control solutions specific to that meter whenever a new vial of strips is used and whenever the validity of the reading is in doubt.

Candidates for Self-Monitoring of Blood Glucose. SMBG is a useful tool for managing self-care for everyone with diabetes. It is a key component of treatment for any intensive insulin therapy regimen (ie, two to four injections per day or use of an insulin pump) and for diabetes management during pregnancy. It is also recommended for patients with the following conditions:

- Unstable diabetes (severe swings from very high to very low blood glucose levels within a 24-hour day)
- A tendency to develop severe ketosis or hypoglycemia
- Hypoglycemia without warning symptoms

For patients not taking insulin, SMBG is helpful for monitoring the effectiveness of exercise, diet, and oral antidiabetic agents. It can also help motivate patients to continue with treatment. For patients with type 2 diabetes, SMBG is recommended during periods of suspected hyperglycemia (eg, illness) or hypoglycemia (eg, unusual increased activity levels) and when the medication or dosage of medication is modified (ADA, 2009b).

Frequency of Self-Monitoring of Blood Glucose. For most patients who require insulin, SMBG is recommended two to four times daily (usually before meals and at bedtime). For patients who take insulin before each meal, SMBG is required at least three times daily before meals to determine each dose (ADA, 2009b). Those not receiving insulin may be instructed to assess their blood glucose levels at least two or three times per week, including a 2-hour postprandial test. For all patients, testing is recommended whenever hypoglycemia or hyperglycemia is suspected, with changes in medications, activity, or diet, and with stress or illness.

Responding to Self-Monitoring of Blood Glucose Results. Patients are asked to keep a record or logbook of blood glucose levels so that they can detect patterns. Testing is done at the peak action time of the medication to evaluate the need for dosage adjustments. To evaluate basal insulin and determine bolus insulin doses, testing is performed before meals. To determine bolus doses of regular or rapid-acting insulin (lispro [Humalog], aspart [Novolog], or glulisine [Apidra]), testing is done 2 hours after meals. Patients with type 2 diabetes are encouraged to test daily before and 2 hours after the largest meal of the day until stabilized. Thereafter, testing should be done periodically before and after meals. Patients who take insulin at bedtime or who use an insulin infusion pump should also test at 3 AM once a

week to document that the blood glucose level is not decreasing during the night. If the patient is unwilling or cannot afford to test frequently, then once or twice a day may be sufficient if the time of testing is varied (eg, before breakfast one day and before lunch the next day).

A tendency to discontinue SMBG is more likely to occur if the patient does not receive instruction about using the results to alter the treatment regimen, if positive reinforcement is not given, and if costs of testing increase. At the very least, the patient should be given parameters for contacting the physician. Patients using intensive insulin therapy regimens may be instructed in the use of algorithms (rules or decision trees) for changing the insulin doses based on patterns of values greater or less than the target range and the amount of carbohydrate to be consumed. Baseline patterns should be established by SMBG for 1 to 2 weeks.

Using a Continuous Glucose Monitoring System

A **continuous glucose monitoring system (CGMS)** can be used to continuously monitor blood glucose levels (Fig. 41-2). A sensor attached to an infusion set, which is similar to an insulin pump infusion set, is inserted subcutaneously in the abdomen and connected to the device worn on a belt. After 72 hours, the data from the device are downloaded, and blood glucose readings are analyzed. Although the CGMS cannot be used for making decisions about specific insulin doses, it can be used to determine whether treatment is adequate over a 24-hour period. This device will be refined in the future so that it can be used by patients to make daily treatment decisions.

Testing for Glycated Hemoglobin

Glycated hemoglobin (also referred to as **glycosylated hemoglobin, $HgbA_{1C}$, or A1C**) is a blood test that reflects average blood glucose levels over a period of approximately 2 to 3 months (ADA, 2009b). When blood glucose levels are elevated, glucose molecules attach to hemoglobin in red blood cells. The longer the amount of glucose in the blood remains above normal, the more glucose binds to hemoglobin and the higher the glycated hemoglobin level becomes. This complex (hemoglobin attached to the glucose) is permanent and lasts for the life of an individual red blood cell, approximately 120 days. If near-normal blood glucose levels are maintained, with only occasional increases, the overall value will not be greatly elevated. However, if the blood glucose values are consistently high, then the test result is also elevated. If the patient reports mostly normal SMBG results but the glycated hemoglobin is high, there may be errors in the methods used for glucose monitoring, errors in recording results, or frequent elevations in glucose levels at times during the day when the patient is not usually monitoring blood sugar levels. Normal values typically range from 4% to 6% and indicate consistently near-normal blood glucose concentrations. The target range for people with diabetes is less than 7% (ADA, 2009b).

Figure 41-2 MiniMed CGMS System Gold Continuous Glucose Monitoring System. (Courtesy of Medtronic Diabetes.)

Testing for Ketones

Ketones (or ketone bodies) are byproducts of fat breakdown, and they accumulate in the blood and urine. Ketones in the urine signal that there is a deficiency of insulin and control of type 1 diabetes is deteriorating. The risk of DKA is high. When there is almost no effective insulin available, the body starts to break down stored fat for energy. Urine testing is the most common method used for self-testing of ketone bodies by patients. A meter that enables testing of blood for ketones is available.

Most commonly, the patient uses a urine dipstick (Ketostix or Chemstrip uK) to detect ketonuria. The reagent pad on the strip turns purple when ketones are present. (One of the ketone bodies is called acetone, and this term is frequently used interchangeably with the term ketones.) Other strips are available for measuring both urine glucose and ketones (Keto-Diastix or Chemstrip uGK). Large amounts of ketones may depress the color response of the glucose test area.

Urine ketone testing should be performed whenever patients with type 1 diabetes have glycosuria or persistently elevated blood glucose levels (more than 240 mg/dL or 13.2 mmol/L for two testing periods in a row) and during illness, in pregnancy with preexisting diabetes, and in gestational diabetes (ADA, 2009b).

Pharmacologic Therapy

As previously stated, insulin is secreted by the beta cells of the islets of Langerhans and works to lower the blood glucose level after meals by facilitating the uptake and utilization of glucose by muscle, fat, and liver cells. In the absence of adequate insulin, pharmacologic therapy is essential.

Insulin Therapy

In type 1 diabetes, exogenous insulin must be administered for life because the body loses the ability to produce insulin. In type 2 diabetes, insulin may be necessary on a long-term basis to control glucose levels if meal planning and oral agents are ineffective. In addition, some patients in whom type 2 diabetes is usually controlled by meal planning alone

or by meal planning and an oral antidiabetic agent may require insulin temporarily during illness, infection, pregnancy, surgery, or some other stressful event. In many cases, insulin injections are administered two or more times daily to control the blood glucose level. Because the insulin dose required by the individual patient is determined by the level of glucose in the blood, accurate monitoring of blood glucose levels is essential; thus, SMBG is a cornerstone of insulin therapy.

Preparations. A number of insulin preparations are available. They vary according to three main characteristics: time course of action, species (source), and manufacturer.

Time Course of Action. Insulins may be grouped into several categories based on the onset, peak, and duration of action (Table 41-3). Human insulin preparations have a shorter duration of action than insulin from animal sources because the presence of animal proteins triggers an immune response that results in the binding of animal insulin, which slows its availability.

Rapid-acting insulins produce a more rapid effect that is of shorter duration than regular insulin. Because of their rapid onset, the patient should be instructed to eat no more than 5 to 15 minutes after injection. Because of the short duration of action of these insulin analogues, patients with type 1 diabetes and some patients with type 2 or gestational diabetes also require a long-acting insulin (basal insulin) to maintain glucose control. Basal insulin is necessary to maintain blood glucose levels irrespective of meals. A constant level of insulin is required at all times. Intermediate-acting insulins function as basal insulins but may have to be split into two injections to achieve 24-hour coverage.

Short-acting insulins are called regular insulin (marked R on the bottle). Regular insulin is a clear solution and is usually administered 20 to 30 minutes before a meal, either alone or in combination with a longer-acting insulin. Regular insulin is the only insulin approved for IV use.

Intermediate-acting insulins are called NPH insulin (neutral protamine Hagedorn) or Lente insulin. Intermediate-acting insulins, which are similar in their time course of action, appear white and cloudy. If NPH or Lente insulin is taken alone, it is not crucial that it be taken 30 minutes before the meal. However, it is important that patients eat some food around the time of the onset and peak of these insulins.

"Peakless" basal or very long-acting insulins are approved by the FDA for use as a basal insulin—that is, the insulin is absorbed very slowly over 24 hours and can be given once a day. Because the insulin is in a suspension with a pH of 4, it cannot be mixed with other insulins because this would cause precipitation. It was originally approved to be given once a day at bedtime; however, it has now been approved to be given once a day at any time of the day but must be given at the same time each day to prevent overlap of action. Many patients fall asleep, forgetting to take their bedtime insulin or may be wary of taking insulin before going to sleep. Having these patients take their insulin in the morning ensures that the dose is taken.

NURSING ALERT

When administering insulin, it is very important to read the label carefully and to be sure that the correct type of insulin is administered. It is also important to avoid mistaking Lantus insulin for Lente insulin and vice versa.

The nurse should emphasize which meals—and snacks—are being "covered" by which insulin doses. In general, the rapid-acting and short-acting insulins are expected to cover the increase in glucose levels after meals, immediately after the injection; the intermediate-acting insulins are expected to cover subsequent meals; and the long-acting insulins provide a relatively constant level of insulin and act as a basal insulin.

Species (Source). In the past, all insulins were obtained from beef (cow) and pork (pig) pancreases. Human insulins are now widely available. They are produced by recombinant

Table 41-3 CATEGORIES OF INSULIN

Time Course	Agent	Onset	Peak	Duration	Indications
Rapid-acting	Lispro (Humalog)	10–15 min	1 h	2–4 h	Used for rapid reduction of glucose level, to treat postprandial hyperglycemia, and/or to prevent nocturnal hypoglycemia
	Aspart (Novolog)	5–15 min	40–50 min	2–4 h	
	Glulisine (Apidra)	5–15 min	30–60 min	2 h	
Short-acting	Regular (Humalog R, Novolin R, Iletin II Regular)	½–1 h	2–3 h	4–6 h	Usually administered 20–30 min before a meal; may be taken alone or in combination with longer-acting insulin
Intermediate-acting	NPH (neutral protamine Hagedorn)	2–4 h	4–12 h	16–20 h	Usually taken after food
	(Humulin N, Iletin II Lente, Iletin II NPH, Novolin L [Lente], Novolin N [NPH])	3–4 h	4–12 h	16–20 h	
Very long-acting	Glargine (Lantus)	1 h	Continuous (no peak)	24 h	Used for basal dose
	Detemir (Levemir)				

DNA technology and have largely replaced insulin from animal sources. These insulins are preferable to animal source insulins because they are not antigenic and do not depend on sufficient animal sources. Human insulin preparations have a shorter duration of action than insulin from animal sources because the presence of animal proteins triggers an immune response that results in the binding of animal insulin, which slows its availability.

Insulin Regimens. Insulin regimens vary from one to four injections per day. Usually there is a combination of a short-acting insulin and a longer-acting insulin. The normally functioning pancreas continuously secretes small amounts of insulin during the day and night. In addition, whenever blood glucose increases after ingestion of food, there is a rapid burst of insulin secretion in proportion to the glucose-raising effect of the food. The goal of all but the simplest, one-injection insulin regimens is to mimic this normal pattern of insulin secretion in response to food intake and activity patterns. Table 41-4 describes several insulin regimens and the advantages and disadvantages of each.

There are two general approaches to insulin therapy: conventional and intensive (described in detail below). The patient can learn to use SMBG results and carbohydrate counting to vary the insulin doses. This allows more flexibility in timing and content of meals and exercise periods. However, complex insulin regimens require a strong level of commitment, intensive education, and close follow-up by the health care team.

The patient should be very involved in the decision regarding which insulin regimen to use. The patient should compare the potential benefits of different regimens with the potential costs (eg, time involved, number of injections or fingersticks for glucose testing, amount of record keeping). There are no set guidelines as to which insulin regimen should be used for which patient. It must not be assumed that elderly patients should automatically be given a simplified regimen. Likewise, it must not be assumed that all people want to be involved in a complex treatment regimen. The nurse plays an important role in educating the patient about the various approaches to insulin therapy. The nurse should refer the patient to a diabetes specialist or a diabetes education center, if available, for further training and education in the insulin treatment regimens.

Conventional Regimen. One approach is to simplify the insulin regimen as much as possible, with the aim of avoiding the acute complications of diabetes (hypoglycemia and symptomatic hyperglycemia). With this type of simplified regimen (eg, one or more injections of a mixture of short-acting and intermediate-acting insulins per day), the patient should not vary meal patterns and activity levels. The simplified regimen would be appropriate for the terminally ill, the frail elderly with limited self-care abilities, or patients who are completely unwilling or unable to engage in the self-management activities that are part of a more complex insulin regimen.

Intensive Regimen. The second approach is to use a more complex insulin regimen to achieve as much control over blood glucose levels as is safe and practical. A more complex insulin regimen allows the patient more flexibility to change the insulin doses from day to day in accordance with changes in eating and activity patterns, with stress and illness, and as needed for variations in the prevailing glucose level.

Although the DCCT Research Group (1993) found that intensive treatment (three or four injections of insulin per day) reduced the risk of complications, not all people with diabetes are candidates for very tight control of blood glucose. The DCCT also found that the risk of severe hypoglycemia was increased threefold in patients receiving intensive treatment (ADA, 2009b). Patients who have received a kidney transplant because of nephropathy and chronic renal failure should follow an intensive insulin regimen to preserve function of the new kidney.

Those who are not candidates include those with:

- Nervous system disorders rendering them unaware of hypoglycemic episodes (eg, those with autonomic neuropathy)
- Recurring severe hypoglycemia
- Irreversible diabetic complications, such as blindness or end-stage renal disease
- Cerebrovascular or cardiovascular disease
- Ineffective self-care skills

Complications of Insulin Therapy

Local Allergic Reactions. A local allergic reaction (redness, swelling, tenderness, and induration or a 2- to 4-cm wheal) may appear at the injection site 1 to 2 hours after the insulin administration. These reactions, which usually occur during the beginning stages of therapy and disappear with continued use of insulin, are becoming rare because of the increased use of human insulins. The physician may prescribe an antihistamine to be taken 1 hour before the injection if such a local reaction occurs.

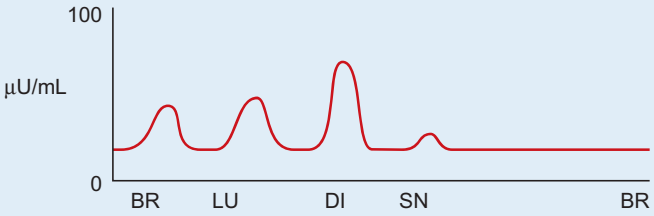
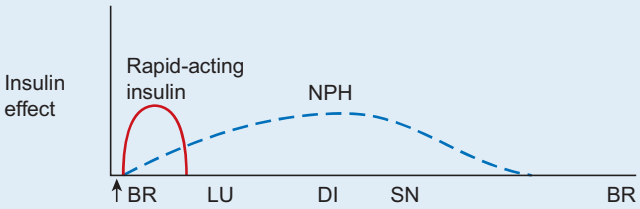
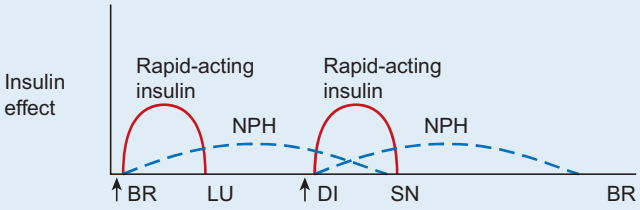
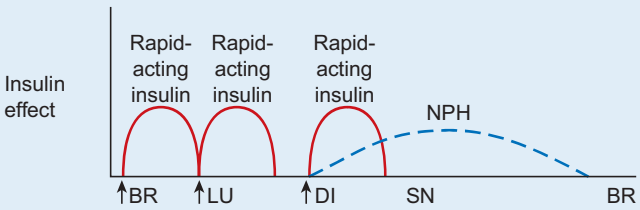
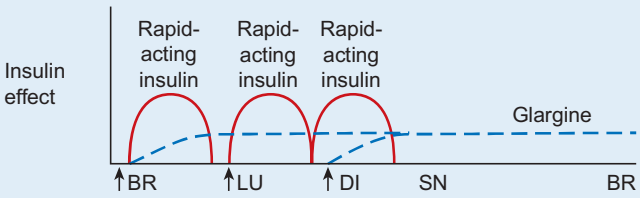
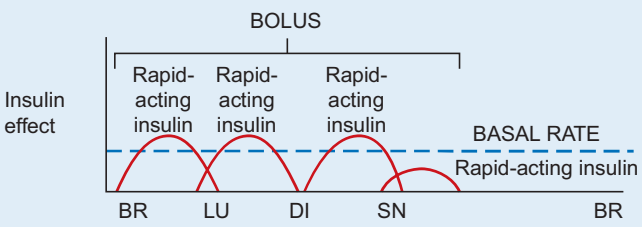
Systemic Allergic Reactions. Systemic allergic reactions to insulin are rare. When they do occur, there is an immediate local skin reaction that gradually spreads into generalized urticaria (hives). These rare reactions are occasionally associated with generalized edema or anaphylaxis. The treatment is desensitization, with small doses of insulin administered in gradually increasing amounts using a desensitization kit.

Insulin Lipodystrophy. Lipodystrophy refers to a localized reaction, in the form of either lipoatrophy or lipohypertrophy, occurring at the site of insulin injections. Lipoatrophy is loss of subcutaneous fat; it appears as slight dimpling or more serious pitting of subcutaneous fat. The use of human insulin has almost eliminated this disfiguring complication.

Lipohypertrophy, the development of fibrofatty masses at the injection site, is caused by the repeated use of an injection site. If insulin is injected into scarred areas, absorption may be delayed. This is one reason that rotation of injection sites is so important. Patients should avoid injecting insulin into these areas until the hypertrophy disappears.

Resistance to Injected Insulin. Most patients have some degree of insulin resistance at one time or another. This may occur for various reasons, the most common being obesity, which can be overcome by weight loss. Clinical insulin resistance has been defined as a daily insulin requirement of

Table 41-4 INSULIN REGIMENS

Schematic Representation	Description	Advantages	Disadvantages
Normal pancreas 	Insulin release increases when blood glucose levels rise and continues at a low steady rate between meals.		
One injection per day 	Before breakfast: • NPH or • NPH with rapid acting insulin	Simple regimen	Difficult to control fasting blood glucose if effects of NPH do not last Afternoon hypoglycemia may result from attempts to control fasting glucose level by increasing NPH dose
Two injections per day–mixed 	Before breakfast and dinner: • NPH or • NPH with rapid-acting insulin or • Premixed (rapid-acting insulin) insulin	Simplest regimen that attempts to mimic normal pancreas	Need relatively fixed schedule of meals and exercise Cannot independently adjust NPH or regular if premixed insulin is used
Three or four injections per day 	Rapid-acting insulin before each meal with: • NPH at dinner or • NPH at bedtime or • Glargine one or two times per day	More closely mimics normal pancreas than two-injection regimen Each premeal dose of regular insulin decided independently More flexibility with meals and exercise	Requires more injections than other regimens Requires multiple blood glucose tests on a daily basis Requires intensive education and follow-up
			
Insulin pump 	Uses ONLY rapid-acting, (Lispro, Aspart, or Apidra) insulin infused at continuous, low rate called *basal rate* (commonly 0.5–1.5 units/h) and premeal *bolus doses* activated by pump wearer	Most closely mimics normal pancreas Decreases unpredictable peaks of intermediate-acting and long-acting insulins Increases meal and exercise flexibility	Requires intensive training and frequent follow-up Potential for mechanical problems Requires multiple blood glucose tests on a daily basis Potential increase in expenses (depending on insurance coverage)

BR, breakfast; LU, lunch; DI, dinner; SN, snack; REG, regular; ↑ indicates insulin injections.
Rapid acting insulin; lispro, aspart, or glulisine [Apidra]

200 units or more. In most patients with diabetes who take insulin, immune antibodies develop and bind the insulin, thereby decreasing the insulin available for use. All animal insulins, and human insulins to a lesser degree, cause antibody production in humans.

Very few resistant patients develop high levels of antibodies. Many of these patients have a history of insulin therapy interrupted for several months or longer. Treatment consists of administering a more concentrated insulin preparation, such as U500, which is available by special order. Occasionally, prednisone is needed to block the production of antibodies. This may be followed by a gradual reduction in the insulin requirement. Therefore, patients must monitor their blood for hypoglycemia.

Morning Hyperglycemia. An elevated blood glucose level on arising in the morning is caused by an insufficient level of insulin, which may be caused by several factors: the dawn phenomenon, the Somogyi effect, or insulin waning. The dawn phenomenon is characterized by a relatively normal blood glucose level until approximately 3 AM, when blood glucose levels begin to rise. The phenomenon is thought to result from nocturnal surges in growth hormone secretion, which create a greater need for insulin in the early morning hours in patients with type 1 diabetes. It must be distinguished from insulin waning (the progressive increase in blood glucose from bedtime to morning) and from the Somogyi effect (nocturnal hypoglycemia followed by rebound hyperglycemia). Insulin waning is frequently seen if the evening NPH dose is administered before dinner; it is prevented by moving the evening dose of NPH insulin to bedtime.

It may be difficult to tell from a patient's history which of these causes is responsible for morning hyperglycemia. To determine the cause, the patient must be awakened once or twice during the night to test blood glucose levels. Testing at bedtime, at 3 AM, and on awakening provides information that can be used to make adjustments in insulin to avoid morning hyperglycemia. Table 41-5 summarizes the differences among insulin waning, the dawn phenomenon, and the Somogyi effect.

Table 41-5 CAUSES OF MORNING HYPERGLYCEMIA

Characteristic	Treatment
Insulin Waning	
Progressive rise in blood glucose from bedtime to morning	Increase evening (predinner or bedtime) dose of intermediate-acting or long-acting insulin, or institute a dose of insulin before the evening meal if one is not already part of the treatment regimen.
Dawn Phenomenon	
Relatively normal blood glucose until about 3 AM, when the level begins to rise	Change time of injection of evening intermediate-acting insulin from dinnertime to bedtime.
Somogyi Effect	
Normal or elevated blood glucose at bedtime, a decrease at 2–3 AM to hypoglycemic levels, and a subsequent increase caused by the production of counterregulatory hormones	Decrease evening (predinner or bedtime) dose of intermediate-acting insulin, or increase bedtime snack.

Methods of Insulin Delivery. Methods of insulin delivery include traditional subcutaneous injections, insulin pens, jet injectors, and insulin pumps. See the nursing management section for discussion of traditional subcutaneous injections.

Insulin Pens. Insulin pens use small (150- to 300-unit) prefilled insulin cartridges that are loaded into a penlike holder. A disposable needle is attached to the device for insulin injection. Insulin is delivered by dialing in a dose or pushing a button for every 1- or 2-unit increment administered. People using these devices still need to insert the needle for each injection (Fig. 41-3); however, they do not need to carry insulin bottles or draw up insulin before each injection. These devices are most useful for patients who need to inject only one type of insulin at a time (eg, premeal rapid-acting insulin three times a day and bedtime NPH insulin) or who can use the premixed insulins. These pens are convenient for those who administer insulin before dinner if eating out or traveling. They are also useful for patients with impaired manual dexterity, vision, or cognitive function, which makes the use of traditional syringes difficult.

Jet Injectors. As an alternative to needle injections, jet injection devices deliver insulin through the skin under pressure in an extremely fine stream. These devices are more expensive and require thorough training and supervision when first used. In addition, patients should be cautioned

Figure 41-3 Prefilled insulin syringe.

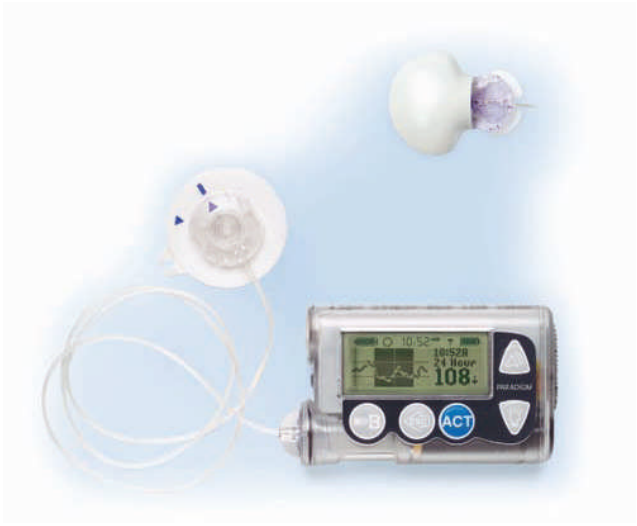


Figure 41-4 MiniMed Paradigm real-time insulin pump and continuous glucose monitoring system. (Courtesy of Medtronic Diabetes.)

that absorption rates, peak insulin activity, and insulin levels may be different when changing to a jet injector. (Insulin administered by jet injector is usually absorbed faster.) Use of jet injectors has been associated with bruising in some patients.

Insulin Pumps. Continuous subcutaneous insulin infusion involves the use of small, externally worn devices (insulin pumps) that closely mimic the functioning of the normal pancreas (ADA, 2009b). Insulin pumps contain a 3-mL syringe attached to a long (24- to 42-in), thin, narrow-lumen tube with a needle or Teflon catheter attached to the end (Fig. 41-4). The patient inserts the needle or catheter into subcutaneous tissue (usually on the abdomen) and secures it with tape or a transparent dressing. The needle or catheter is changed at least every 3 days. The pump is then worn either on a belt or in a pocket. Some women keep the pump tucked into the front or side of the bra or wear it on a garter belt on the thigh.

When an insulin pump is used, insulin is delivered by subcutaneous infusion at a basal rate (eg, 0.5 to 2.0 units/h). When a meal is consumed, the patient calculates a dose of insulin to metabolize the meal by counting the total amount of carbohydrate for the meal using a predetermined insulin-to-carbohydrate ratio; for example, a ratio of 1 unit of insulin for every 15 g of carbohydrate would require 3 units for a meal with 45 g of carbohydrate. This allows flexibility of meal timing and content.

Possible disadvantages of insulin pumps are unexpected disruptions in the flow of insulin from the pump that may occur if the tubing or needle becomes occluded, if the supply of insulin runs out, or if the battery is depleted, increasing the risk of DKA. Effective teaching to produce knowledgeable patients minimizes this risk. Another disadvantage is the potential for infection at needle insertion sites. Hypoglycemia may occur with insulin pump therapy; however, this is usually related to the lowered blood glucose levels many patients achieve rather than to a specific problem with the pump itself. The tight diabetes control associated with use of an insulin pump may increase the incidence of hypoglycemia unawareness because of the very gradual decline in serum glucose level, from more than 70 mg/dL (3.9 mmol/L) to less than 60 mg/dL (3.3 mmol/L).

Some patients find that wearing the pump for 24 hours each day is inconvenient. However, the pump can easily be disconnected, per patient preference, for limited periods, such as for showering, exercise, or sexual activity.

Candidates for the insulin pump must be willing to assess their blood glucose level several times daily. In addition, they must be psychologically stable and open about having diabetes, because the insulin pump is often a visible sign to others and a constant reminder to patients that they have diabetes. Most important, patients using insulin pumps must have extensive education in the use of the pump and in self-management of blood glucose and insulin doses. They must work closely with a team of health care professionals who are experienced in insulin pump therapy—specifically, a diabetologist/endocrinologist, a dietitian, and a certified diabetes educator.

The most common risk of insulin pump therapy is ketoacidosis, which can occur if there is an occlusion in the infusion set or tubing. Because only rapid-acting insulin is used in the pump, any interruption in the flow of insulin may rapidly cause the patient to be without insulin. The patient should be taught to administer insulin by manual injection if an insulin interruption is suspected (eg, no response in blood glucose level after a meal bolus).

Many insurance companies cover the cost of pump therapy. If not, the extra expense of the pump and associated supplies may be a deterrent for some patients. Medicare covers insulin pump therapy for patients with type 1 diabetes.

Insulin pumps have been used in patients with type 2 diabetes whose beta-cell function has diminished and who require insulin. Patients with a hectic lifestyle often do well with an insulin pump. There is no risk of DKA when there is an interruption of the flow of insulin in people with type 2 diabetes wearing an insulin pump.

Future Insulin Delivery. Research into mechanical delivery of insulin has involved implantable insulin pumps that can be externally programmed according to blood glucose test results. Clinical trials with these devices are continuing. In addition, there is research into the development of implantable devices that both measure the blood glucose level and deliver insulin as needed. Methods of administering insulin by the oral route (oral spray or capsule) and skin patch are undergoing intensive study.

Transplantation of Pancreatic Cells. Transplantation of the whole pancreas or a segment of the pancreas is being performed on a limited population (mostly patients with diabetes who are receiving a kidney transplantation simultaneously). One main issue is weighing the risks of antirejection medications against the advantages of pancreas transplantation. Implantation of insulin-producing pancreatic islet cells is another approach under investigation. This latter approach involves a less extensive surgical procedure and a potentially lower incidence of immunogenic problems (ADA, 2006b). However, thus far, independence from exogenous insulin has been limited to 2 years after transplantation of islet cells. Results of recent studies of patients with

islet cell transplants using less toxic antirejection drugs have shown some promise (NIDDK, 2007).

Oral Antidiabetic Agents

Oral antidiabetic agents may be effective for patients who have type 2 diabetes that cannot be treated effectively with MNT and exercise alone. In the United States, oral antidiabetic agents include first-generation and second-generation **sulfonylureas,** biguanides, alpha-glucosidase inhibitors, non-sulfonylurea insulin secretogogues (meglitinides and phenylalanine derivatives), **thiazolidinediones** (glitazones), and dipeptide-peptidase-4 (DPP-4) inhibitors (Table 41-6). Sulfonylureas and meglitinides are considered insulin secretagogues because their action increases the secretion of insulin by the pancreatic beta cells.

Patients must understand that oral agents are prescribed as an addition to (not as a substitute for) other treatment modalities, such as MNT and exercise. Use of oral antidiabetic medications may need to be halted temporarily and insulin prescribed if hyperglycemia develops that is attributable to infection, trauma, or surgery.

In time, oral antidiabetic agents may no longer be effective in controlling diabetes because of decline in function of beta cells. In such cases, the patient is treated with insulin. Approximately half of all patients who initially use oral antidiabetic agents eventually require insulin. This is referred to as a secondary failure. Primary failure occurs when the blood glucose level remains high 1 month after initial medication use.

Because mechanisms of action vary (Fig. 41-5), effects may be enhanced with the use of multidose, multiple medications. Use of multiple medications with different mechanisms of action is very common today. A combination of oral agents with insulin, usually glargine at bedtime, has also been used frequently as a treatment for some patients with type 2 diabetes.

Other Pharmacologic Therapy

Two new medications became available in 2005 for use in the pharmacologic management of diabetes. Both are injectable medications; neither is a substitute for insulin if insulin is required to control diabetes.

Pramlintide (Symlin), a synthetic analogue of human amylin, a hormone that is secreted by the beta cells of the pancreas, has recently been approved for treatment of both type 1 and type 2 diabetes. It is used to control hyperglycemia in adults who have not achieved acceptable levels of glucose control despite the use of insulin at mealtimes. It is used with insulin, not in place of insulin. Although pramlintide is not yet widely used, it is anticipated that it will be useful to minimize fluctuations in daily glucose levels and provide better glucose control. Risks associated with pramlintide include hypoglycemia; therefore, a source of glucose must be available if hypoglycemia occurs. Pramlintide must be injected into the abdomen or thigh because of variable absorption rates if it is injected into the arm. It should not be injected close to an insulin injection site. Caution must be exercised in preparing and administering pramlintide to avoid errors in dosing. Patients are instructed to monitor their blood glucose level before each meal, 2 hours afterward, and at bedtime during the initial period of use of pramlintide.

Exenatide (Byetta) is another medication approved for the treatment of type 2 diabetes in combination with metformin or sulfonylureas. It is derived from a hormone that is produced in the small intestine and has been found to be deficient in type 2 diabetes. It is normally released after food is ingested to delay gastric emptying and enhance insulin secretion, resulting in dampening of the rise in blood glucose levels after meals and a feeling of satiety. The return of the blood glucose level to normal results in decreased production of the hormone. Hypoglycemia is not a side effect of exenatide if adjustments are made in the sulfonylurea dose. Exenatide has been shown to result in weight loss because of the increased satiety produced. Exenatide must be injected twice a day within 1 hour before breakfast and dinner. It is not a substitute for insulin in patients who require insulin to control their diabetes.

Nursing Management

Nursing management of patients with diabetes can involve treatment of a wide variety of physiologic disorders, depending on the patient's health status and whether the patient is newly diagnosed or seeking care for an unrelated health problem. Nursing management of patients with DKA and hyperglycemic hyperosmolar nonketotic syndrome and of those with diabetes as a secondary diagnosis is discussed in subsequent sections of this chapter.

Because all patients with diabetes must master the concepts and skills necessary for long-term management and avoidance of potential complications of diabetes, a solid educational foundation is necessary for competent self-care and is an ongoing focus of nursing care.

Providing Patient Education

Diabetes mellitus is a chronic illness that requires a lifetime of special self-management behaviors. Because MNT, physical activity, and physical and emotional stress affect diabetic control, patients must learn to balance a multitude of factors.

Developing a Diabetic Teaching Plan

Changes in the health care system as a whole have had a major impact on diabetes education and training. Patients with new-onset type 1 diabetes are hospitalized for much shorter periods or may be managed completely on an outpatient basis. Patients with new-onset type 2 diabetes are rarely hospitalized for initial care. There has been a proliferation of outpatient diabetes education and training programs, with increasing support of third-party reimbursement. All encounters with patients with diabetes are opportunities for reinforcement of self-management skills, regardless of the setting.

Many hospitals employ nurses who specialize in diabetes education and management and who are certified by the National Certification Board of Diabetes Educators as Certified Diabetes Educators (CDEs). However, because of the large number of patients with diabetes who are admitted to every unit of a hospital for reasons other than diabetes or its complications, staff nurses play a vital role in identifying patients with diabetes, assessing self-care skills, providing

Table 41-6 ORAL ANTIDIABETIC AGENTS

Generic (Trade) Name	Action/Indications	Side Effects	Implications
First-Generation Sulfonylureas			
Acetohexamide (Dymelor) Chlorpropamide (Diabinese) Tolazamide (Tolinase) Tolbutamide (Orinase)	Used infrequently in U.S. today Used in type 2 diabetes to control blood glucose levels Stimulate beta cells of the pancreas to secrete insulin; may improve binding between insulin and insulin receptors or increase the number of insulin receptors	Hypoglycemia Mild GI symptoms Weight gain Drug–drug interactions (NSAIDs, warfarin, sulfonamides) Sulfa allergy Skin reactions	Monitor patient for hypoglycemia Monitor blood glucose and urine ketone levels to assess effectiveness of therapy Patients at high risk for hypoglycemia: advanced age, renal insufficiency When taken with beta-adrenergic blocking agents may mask usual warning signs and symptoms of hypoglycemia Instruct patients to avoid use of alcohol Check for interactions with other medications
Second-Generation Sulfonylureas			
Glipizide (Glucotrol, Glucotrol XL) Glyburide (Micronase, Glynase, Dia-Beta) Glimepiride (Amaryl)	Stimulate beta cells of the pancreas to secrete insulin; may improve binding between insulin and insulin receptors or increase the number of insulin receptors Used in type 2 diabetes to control blood glucose levels Have more potent effects than first-generation sulfonylureas May be used in combination with metformin or insulin to improve glucose control	Hypoglycemia Mild GI symptoms Weight gain Drug–drug interactions (NSAIDs, warfarin, sulfonamides) Sulfa allergy	Monitor patient for hypoglycemia Monitor blood glucose and urine ketone levels to assess effectiveness of therapy Patients at high risk for hypoglycemia: advanced age, renal insufficiency When taken with beta-adrenergic blocking agents, may mask usual warning signs and symptoms of hypoglycemia Instruct patients to avoid use of alcohol
Biguanides			
Metformin (Glucophage, Glucophage XL, Fortamet) Metformin with glyburide (Glucovance)	Inhibit production of glucose by the liver Increase body tissues' sensitivity to insulin Decrease hepatic synthesis of cholesterol Used in type 2 diabetes to control blood glucose levels	Lactic acidosis Hypoglycemia if metformin is used in combination with insulin or other antidiabetic agents Drug–drug interaction GI disturbances Contraindicated in patients with impaired renal or liver function, respiratory insufficiency, severe infection, or alcohol abuse	Monitor for lactic acidosis and hypoglycemia Monitor renal function Patients taking metformin are at increased risk of acute renal failure and lactic acidosis with use of iodinated contrast material for diagnostic studies; metformin should be stopped 48 h prior to and for 48 h after use of contrast agent or until renal function is evaluated and normal Check for interactions with other medications
Alpha-Glucosidase Inhibitors			
Acarbose (Precose) Miglitol (Glyset)	Delay absorption of complex carbohydrates in the intestine and slow entry of glucose into systemic circulation Do not increase insulin secretion Used in type 2 diabetes to control blood glucose levels Can be used alone or in combination with sulfonylureas, metformin, or insulin to improve glucose control	Hypoglycemia (risk increased if used with insulin or other antidiabetic agents) GI side effects (abdominal discomfort or distention, diarrhea, flatulence) Drug–drug interactions	Must be taken with first bite of food to be effective Monitor for GI side effects (diarrhea, abdominal distention) Monitor for blood glucose levels to assess effectiveness of therapy Monitor liver function studies every 3 mo for 1 y, then periodically Contraindicated in patients with GI or renal dysfunction, or cirrhosis **Alert: Hypoglycemia must be treated with glucose, not sucrose**

Continued on following page

Table 41-6 ORAL ANTIDIABETIC AGENTS (Continued)

Generic (Trade) Name	Action/Indications	Side Effects	Implications
Non-Sulfonylurea Insulin Secretagogues			
Repaglinide (Prandin) categorized as a meglitinide Nateglinide (Starlix) categorized as a D-phenylalanine derivative	Stimulate pancreas to secrete insulin Used in type 2 diabetes to control blood glucose levels Can be used alone or in combination with metformin or thiazolidinediones to improve glucose control	Hypoglycemia/weight gain less likely than sulfonylureas Drug–drug interactions (with ketoconazole, fluconazole, erythromycin, rifampin, isoniazid)	Monitor blood glucose levels to assess effectiveness of therapy Has rapid action and short half-life Should be taken only if able to eat a meal immediately Teach patients symptoms of hypoglycemia Monitor patients with impaired liver function and renal impairment Has no effect on plasma lipids Is taken before each meal Check for interactions with other medications
Thiazolidinediones (or glitazones)			
Pioglitazone (Actos) Rosiglitazone (Avandia)	Sensitize body tissue to insulin; stimulate insulin receptor sites to lower blood glucose and improve action of insulin May be used alone or in combination with sulfonylurea, metformin, or insulin	Hypoglycemia (risk increased with use of insulin or other antidiabetic agents) Anemia Weight gain, edema Decrease effectiveness of oral contraceptives Possible liver dysfunction Drug–drug interactions Hyperlipidemia (has variable effect on lipids; pioglitazone may be preferred choice in patients with lipid abnormalities) Impaired platelet function	Monitor blood glucose levels to assess effectiveness of therapy Monitor liver function tests Arrange dietary teaching to establish weight control program Instruct patient taking oral contraceptives about increased risk of pregnancy
Dipeptidyl Peptidase-4 (DPP-4) Inhibitor			
Sitagliptin (Januvia) Vildagliptin (Galvus)	Increase and prolongs the action of incretin, a hormone that increases insulin release and decreases glucagon levels, with the result of improved glucose control	Upper respiratory infection Stuffy or runny nose and sore throat Headache Stomach discomfort and diarrhea Hypoglycemia, if used with sulfonylurea	Usually administered once a day Used alone or with other oral antidiabetic agents Instruct patient about signs and symptoms of hypoglycemia and other adverse effects to report Monitor renal function

basic education, reinforcing the teaching provided by the specialist, and referring patients for follow-up care after discharge. Diabetes patient education programs that have been peer-reviewed by the ADA as meeting National Standards for Diabetes Education can be reimbursed for education.

Organizing Information. There are various strategies for organizing and prioritizing the vast amount of information that must be taught to patients with diabetes. In addition, many hospitals and outpatient diabetes centers have devised written guidelines, care plans, and documentation forms (often based on ADA guidelines) that may be used to document and evaluate teaching. One approach is to organize education using the seven tips for managing diabetes identified and developed by the American Association of Diabetes Educators (AADE, 2005): healthy eating, being active, monitoring, taking medication, problem solving, healthy coping, and reducing risks. The AADE can be contacted for additional information about assessment and documentation of outcomes of this approach to teaching.

Another general approach is to organize information and skills into two main types: basic, initial, or "survival" skills and information, and in-depth (advanced) or continuing education.

Teaching Survival Skills. Survival skills must be taught to all patients with newly diagnosed type 1 or type 2 diabetes and all patients receiving insulin for the first time. This basic information is literally what patients must know to survive (eg, to avoid severe hypoglycemic or acute hyperglycemic complications after discharge). An outline of survival information includes the following:

1. Simple pathophysiology
   a. Basic definition of diabetes (having a high blood glucose level)
   b. Normal blood glucose ranges and target blood glucose levels
   c. Effect of insulin and exercise (decrease glucose)
   d. Effect of food and stress, including illness and infections (increase glucose)
   e. Basic treatment approaches

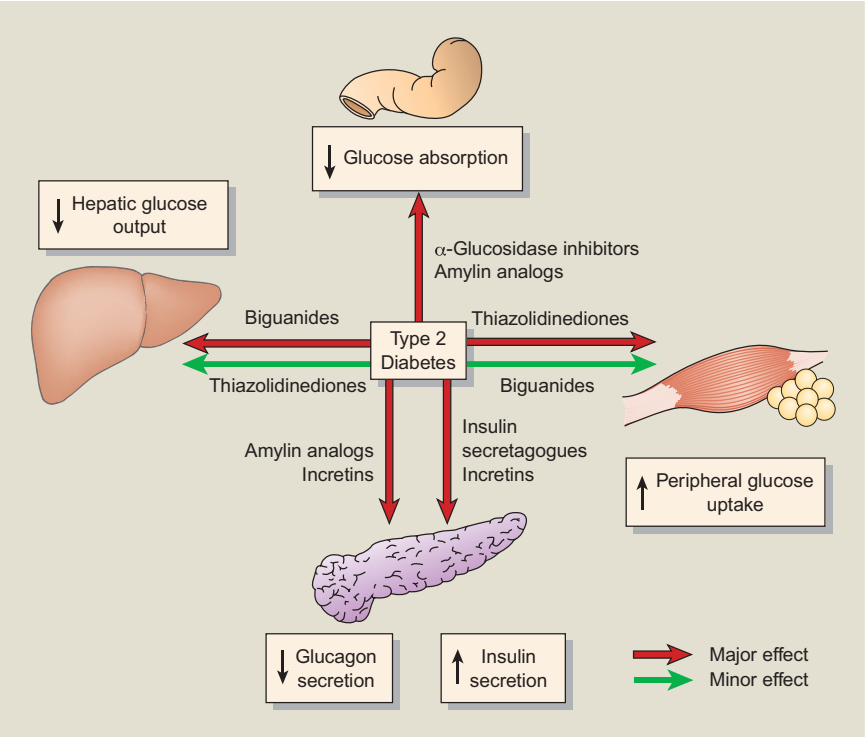


Figure 41-5 Action sites of hypoglycemic agents and mechanisms of lowering blood glucose in type 2 diabetes. The incretins are the dipeptidyl peptidase-4 (DPP-4) inhibitors and glucagon-like peptide-1 (GLP-1) agonists.

2. Treatment modalities
   a. Administration of insulin and oral antidiabetes medications
   b. Meal planning (food groups, timing of meals)
   c. Monitoring of blood glucose and urine ketones
3. Recognition, treatment, and prevention of acute complications
   a. Hypoglycemia
   b. Hyperglycemia
4. Pragmatic information
   a. Where to buy and store insulin, syringes, and glucose monitoring supplies
   b. When and how to contact the physician

For patients with newly diagnosed type 2 diabetes, emphasis is initially placed on meal planning and exercise. Those who are starting to take oral sulfonylureas or insulin secretagogues need to know about detecting, preventing, and treating hypoglycemia. If diabetes has gone undetected for many years, the patient may already be experiencing some chronic diabetic complications. Therefore, for some patients with newly diagnosed type 2 diabetes, basic diabetes teaching must include information on preventive skills, such as foot care and eye care (eg, planning yearly or more frequent complete [dilated eye] examinations by an ophthalmologist, understanding that retinopathy is largely asymptomatic until advanced stages).

Patients also need to realize that once they master the basic skills and information, further diabetes education must be pursued. Acquiring in-depth and advanced diabetes knowledge occurs throughout the patient's lifetime, both formally through programs of continuing education and informally through experience and sharing of information with other people with diabetes.

Planning In-Depth and Continuing Education. This education involves teaching more detailed information related to survival skills (eg, learning to vary food choices and insulin, preparing for travel) as well as learning preventive measures for avoiding long-term diabetic complications. Preventive measures include foot care, eye care, general hygiene (eg, skin care and oral hygiene), and risk factor management (eg, blood pressure control and blood glucose normalization).

More advanced continuing education may include alternative methods for insulin delivery, such as the insulin pump, and algorithms or rules for evaluating and adjusting insulin doses. The degree of advanced diabetes education to be provided depends on the patient's interest and ability. However, learning preventive measures (especially foot care and eye care) is mandatory for early detection and treatment to reduce the occurrence of amputations and blindness in patients with diabetes.

Assessing Readiness to Learn

Before initiating diabetes education, the nurse assesses the patient's (and family's) readiness to learn. When patients are first diagnosed with diabetes (or first told of their need for insulin), they often go through various stages of the grieving process. These stages may include shock and denial, anger, depression, negotiation, and acceptance. The amount of time it takes for the patient and family members to work through the grieving process varies from patient to patient. They may experience helplessness, guilt, altered body image, loss of self-esteem, and concern about the future. The nurse must assess the patient's coping strategies and reassure the patient and family that feelings of depression and shock are normal (Chart 41-6).

Asking the patient and family about their major concerns or fears is an important way to learn about any

CHART 41-6 NURSING RESEARCH PROFILE

Psychological Impact of Diabetes in Women

Penckofer, S., Ferrans, C. & Velsor-Friedrich, B. (2007). The psychological impact of living with diabetes: Women's day-to-day experiences. *The Diabetes Educator, 33*(4), 680–690.

Purpose

Diabetes is a leading cause of heart disease and cardiovascular-related deaths among women; further, depression is often associated with diabetes. Depression may serve as a barrier for women with diabetes to obtain treatment to ensure control of diabetes or to follow the recommended treatment regimen. In an effort to gain an understanding of how psychological issues influence diabetes management, this study was undertaken. Its specific purpose was to identify the feelings experienced by women with diabetes and the impact these feeling have on the quality of life.

Design

A descriptive, exploratory design using a focus groups approach for data collection was undertaken. Four focus groups were conducted by professional moderators. The sessions were audiotaped and transcriptions were analyzed. Forty-one women with type 2 diabetes participated; their mean age was 55.6 years. Guidelines for focus groups published by the American Diabetes Association were used. A moderator guide was used to explore depression, anger, and anxiety.

Findings

Five major themes were identified: (1) struggling with health challenges, (2) challenges in relationships, (3) worrying about the present and the future, (4) multiple responsibilities for self and others, and (5) choosing to take a break from caring for their diabetes. Struggling with health situations was the most prevalent theme. The difficulty of carrying on a normal life in the context of experiencing hypoglycemia and complications promoted angry feelings.

Nursing Implications

The incidence of depression is high in people with diabetes and more so in women than in men. The burden to take on self-care responsibilities while being responsible for others may result in anger. The findings support the need to address the psychological impact of having diabetes on treatment. Tending to the psychological component of diabetes and its effect on patients and families is important because of the potential negative effect of unresolved psychological issues on overall health and prevention of long-term complications.

misinformation that may be contributing to anxiety. Some common misconceptions regarding diabetes and its treatment are listed in Table 41-7. Simple, direct information should be provided to dispel misconceptions. More information can be provided once the patient masters survival skills.

Nurses whose patients are in the hospital rarely have the luxury of waiting until the patient feels ready to learn; short hospital stays necessitate initiation of survival skill education as early as possible. This gives the patient the opportunity to practice skills with supervision by the nurse before discharge. Follow-up by home health nurses is often necessary for reinforcement of survival skills.

The nurse evaluates the patient's social situation for factors that may influence the diabetes treatment and education plan, such as

- Low literacy level (may be evaluated while assessing for visual deficits by having the patient read from teaching materials)
- Limited financial resources or lack of health insurance
- Presence or absence of family support
- Typical daily schedule (the patient is asked about timing and number of usual daily meals, work and exercise schedule, plans for travel)
- Neurologic deficits caused by stroke, other neurologic disorders, or other disabling conditions, obtained from the patient's health history and physical assessment (the patient is assessed for aphasia or decreased ability to follow simple commands)

Teaching Experienced Patients

Nurses should continue to assess the skills and self-care behaviors of patients who have had diabetes for many years, because it is estimated that as many as 50% of patients make errors in self-care. Assessment of these patients must include direct observation of skills, not just the patient's self-report of self-care behaviors. In addition, these patients must be fully aware of preventive measures related to foot care, eye care, and risk factor management. Those experiencing long-term diabetic complications for the first time may go through the grieving process again. Some patients may have a renewed interest in diabetes self-care in the hope of delaying further complications. Others may be overwhelmed by feelings of guilt and depression. The patient is encouraged to discuss feelings and fears related to complications. Meanwhile, the nurse provides appropriate information regarding diabetic complications.

Determining Teaching Methods

Maintaining flexibility with regard to teaching approaches is important. Teaching skills and information in a logical sequence is not always the most helpful method for patients. For example, many patients fear self-injection. Before they learn how to prepare, purchase, store, and mix insulins, they should be taught to insert the needle and inject insulin (or practice with saline solution).

Various tools can be used to complement teaching. Many of the companies that manufacture products for diabetes self-care also provide booklets and videotapes to assist in patient teaching. Teaching/educational materials are also available from the AADE and the ADA. It is important to use a variety of written handouts that match the patient's learning needs (including different languages, low-literacy information, large print) and reading level and to ensure that these materials are technically accurate. Patients can continue learning about diabetes care by participating in

Table 41-7 MISCONCEPTIONS RELATED TO INSULIN TREATMENT

Misconception	Response
Once insulin injections are started (for treatment of type 2 diabetes), they can never be discontinued	During periods of acute stress (eg, illness, infection, surgery) or when receiving certain medications that cause elevations in blood glucose, some patients with type 2 diabetes require insulin. If the diabetes had previously been well controlled with diet alone or diet with oral antidiabetic agents, the patient should be able to resume previous methods for control of diabetes after the stress is resolved. In addition, insulin is sometimes used to control blood glucose levels in obese type 2 diabetic patients who have been unsuccessful at weight loss. If the patient can lose weight after insulin therapy is initiated, the insulin doses may be tapered and the patient may be able to switch to diet and exercise alone or with oral antidiabetic agents for control of blood glucose. (For patients with type 1 diabetes, insulin is needed on an ongoing basis. For thin patients with type 2 diabetes, once insulin has to be started, it is usually required permanently.)
If increasing doses of insulin are needed to control the blood glucose, the diabetes must be getting "worse"	Explain to the patient that unlike other medications that are given in standard doses, there is not a standard dose of insulin that is effective for all patients. Rather, the dose must be adjusted according to blood glucose test results. If the initial insulin dose prescribed for the patient does not adequately decrease the glucose level, the patient may assume that he or she has a "bad" case of diabetes or that the diabetes is getting worse. It is important to instruct patients that many different factors may affect the ability of insulin to lower the glucose, including obesity, puberty, pregnancy, illness, and certain medications. In addition, to avoid hypoglycemia, physicians frequently initiate insulin therapy with smaller dosages than will eventually be needed. The doses are then increased in small increments until blood glucose levels are in the desired range.
Insulin causes blindness (or other diabetic complications)	If the patient has a diabetic acquaintance in whom the initiation of insulin therapy happened to coincide with the onset of diabetic complications, the patient may view insulin as the cause of complications such as blindness or amputation. In these situations, the acquaintance probably had type 2 diabetes that was no longer controllable with diet and oral hypoglycemic agents. It must be explained to the patient that factors such as elevated blood glucose (and not insulin therapy) contribute to some of the diabetic complications. Further, emphasize that insulin is a natural hormone that is present in every person's body, helps control blood glucose levels, and definitely does *not* cause long-term complications of diabetes.
Insulin must be injected directly into the vein	When patients first learn that one area used for insulin injections is on the arm, they may envision inserting the needle directly into a vein in the antecubital area, as in blood withdrawal. The patient must be reassured that insulin is injected into the fat tissue on the *back* of the arm (or on the abdomen, thigh, or hip) and that the needle is much shorter than that used for venipuncture.
There is extreme danger in injecting insulin if there are any air bubbles in the syringe	Patients may have a fear of dying if air bubbles are injected with a syringe. (This may be related to the misconception that insulin is injected directly into the vein.) Reassure patients that the main danger in having air bubbles in the insulin syringe is that the amount of insulin being injected is less than the required dose. It is often difficult to remove every small "champagne" bubble from the syringe. Thus, patients should be reassured that injection of insulin when these bubbles are present does not cause any harm.
Insulin always causes people to have bad (hypoglycemic) reactions	First, make sure that patients are aware that low blood sugar reactions are often related to an imbalance with the insulin, food, and activity and can often be avoided. Thus, before starting on insulin, patients should discuss their usual schedule of meals and activities as well as the content of meals with the health care team. Make sure that patients are aware that various different insulins and insulin schedules can be used to try to allow patients to maintain some of their usual lifestyle habits. Reassure patients that avoiding hypoglycemic reactions is a high priority for the diabetes team. In addition, tell patients of the importance of reporting any hypoglycemic reactions to the health care team immediately so that early adjustments can be made in the insulin dosage. Focus early insulin education on treatment and prevention of hypoglycemia.
People who take insulin must travel only where there is a refrigerator to store the insulin	Insulin bottles in use may be kept at room temperature. Therefore, for most business trips or vacations, keeping the insulin in a purse or briefcase (or special diabetes supply case) is acceptable. If a prolonged trip is planned (more than 2 to 3 months), patients may want to consult the pharmacist or insulin manufacturer for suggestions. Most importantly, emphasize with patients that taking insulin should never deter them from pursuing activities they enjoy.

Pearce, M. A., Rosenberg, C. S. & Davidson, M. B. (2003). Patient education. In Davidson, M. B. (Ed.). *Diabetes mellitus: Diagnosis and treatment* (4th ed.). New York: Churchill Livingstone. Reprinted with permission from Elsevier Science.

activities sponsored by local hospitals and diabetes organizations. In addition, magazines and Web sites with information on diabetes management are available (see Resources).

Teaching Patients to Self-Administer Insulin

Insulin injections are self-administered into the subcutaneous tissue with the use of special insulin syringes. Basic information includes explanations of the equipment, insulins, and syringes and how to mix insulin.

Storing Insulin. Whether insulin is the short-acting or the long-acting preparation, vials not in use, including spare vials, should be refrigerated. Extremes of temperature should be avoided; insulin should not be allowed to freeze and should not be kept in direct sunlight or in a hot car. The insulin vial in use should be kept at room temperature to reduce local irritation at the injection site, which may occur if cold insulin is injected. If a vial of insulin will be used up within 1 month, it may be kept at room temperature. The patient should be instructed to always have a spare vial of the type or types of insulin he or she uses (ADA, 2004). Cloudy insulins should be thoroughly mixed by gently inverting the vial or rolling it between the hands before drawing the solution into a syringe or a pen.

Bottles of intermediate-acting insulin should also be inspected for flocculation, which is a frosted, whitish coating

inside the bottle. This occurs most commonly with human insulins that are exposed to extremes of temperature. If a frosted, adherent coating is present, some of the insulin is bound, and it should not be used.

Selecting Syringes. Syringes must be matched with the insulin concentration (eg, U-100). Currently, three sizes of U-100 insulin syringes are available:

- 1-mL syringes that hold 100 units
- 0.5-mL syringes that hold 50 units
- 0.3-mL syringes that hold 30 units

The concentration of insulin used in the United States is U-100; that is, there are 100 units per milliliter (or cubic centimeter). Small syringes allow patients who require small amounts of insulin to measure and draw up the amount of insulin accurately. Patients who require large amounts of insulin use larger syringes. There is a U-500 (500 units/mL) concentration of insulin available by special order for patients who have severe insulin resistance and require massive doses of insulin.

Most insulin syringes have a disposable 27- to 29-gauge needle that is approximately 0.5 inch long. The smaller syringes are marked in 1-unit increments and may be easier to use for patients with visual deficits and those taking very small doses of insulin. The 1-mL syringes are marked in 1- and 2-unit increments. A small disposable insulin needle (31 gauge, 8 mm long) is available for very thin patients and children.

Mixing Insulins. When rapid-acting or short-acting insulins are to be given simultaneously with longer-acting insulins, they are usually mixed together in the same syringe; the longer-acting insulins must be mixed thoroughly before drawing into the syringe. The most important issue is that patients be consistent in how they prepare their insulin injections from day to day.

There are varying opinions regarding which type of insulin (short-acting or longer-acting) should be drawn up into the syringe first when they are going to be mixed, but the ADA recommends that the regular insulin be drawn up first. Again, the most important issues are (1) that patients be consistent in technique, so as not to draw up the wrong dose in error or the wrong type of insulin, and (2) that patients not inject one type of insulin into the bottle containing a different type of insulin (ADA, 2004). Injecting cloudy insulin into a vial of clear insulin contaminates the entire vial of clear insulin and alters its action.

For patients who have difficulty mixing insulins, several options are available: they may use a premixed insulin, they may have prefilled syringes prepared (see Fig. 41-3), or they may take 2 injections. Premixed insulins are available in several different ratios of NPH insulin to regular insulin. The ratio of 70/30 (70% NPH and 30% regular insulin in one bottle) is most common; this combination is available as Novolin 70/30 (Novo-Nordisk) and Humulin 70/30 (Lilly). Combinations with a ratio of 75% NPL (neutral protamine lispro) and 25% insulin lispro are also available (ADA, 2004). NPL is used only in the mix with Humalog; its action is the same as NPH. The appropriate initial dosage of premixed insulin must be calculated so that the ratio of NPH to regular insulin most closely approximates the separate doses needed.

For patients who can inject insulin but who have difficulty drawing up a single or mixed dose, syringes may be prefilled with the help of home care nurses or family and friends. A 3-week supply of insulin syringes may be prepared and kept in the refrigerator. The prefilled syringes should be stored with the needle in an upright position to avoid clogging of the needle (ADA, 2004); they should be mixed thoroughly before the insulin is injected.

Withdrawing Insulin. Most (if not all) of the printed materials available on insulin dose preparation instruct patients to inject air into the bottle of insulin equivalent to the number of units of insulin to be withdrawn. The rationale for this is to prevent the formation of a vacuum inside the bottle, which would make it difficult to withdraw the proper amount of insulin.

Selecting and Rotating the Injection Site. The four main areas for injection are the abdomen, upper arms (posterior surface), thighs (anterior surface), and hips (Fig. 41-6). Insulin is absorbed faster in some areas of the body than others. The speed of absorption is greatest in the abdomen and decreases progressively in the arm, thigh, and hip, respectively.

Systematic rotation of injection sites within an anatomic area is recommended to prevent localized changes in fatty tissue (lipodystrophy). In addition, to promote consistency in insulin absorption, the patient should be encouraged to use all available injection sites within one area rather than randomly rotating sites from area to area (ADA, 2004). For example, some patients almost exclusively use the abdominal area, administering each injection 0.5 to 1 inch away from the previous injection. Another approach to rotation is always to use the same area at the same time of day. For example, patients may inject morning doses into the abdomen and evening doses into the arms or legs.

A few general principles apply to all rotation patterns. First, the patient should try not to use the same site more than once in 2 to 3 weeks. In addition, if the patient is planning to exercise, insulin should not be injected into

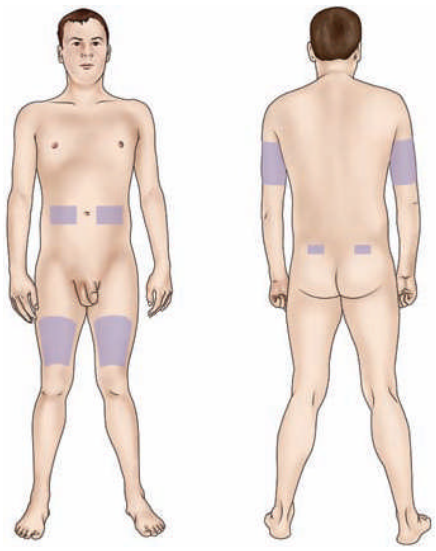


Figure 41-6 Suggested areas for insulin injection.

the limb that will be exercised because this will cause the drug to be absorbed faster, which may result in hypoglycemia.

In the past, patients were taught to rotate injections from one area to the next (eg, injecting once in the right arm, then once in the right abdomen, then once in the right thigh). Patients who still use this system must be taught to avoid repeated injections into the same site within an area. However, as previously stated, it is preferable for patients to use the same anatomic area at the same time of day consistently; this reduces day-to-day variation in blood glucose levels caused by different absorption rates.

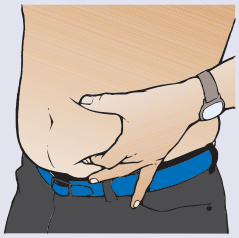
Preparing the Skin. Use of alcohol to cleanse the skin is not recommended, but patients who have learned this technique often continue to use it. They should be cautioned to allow the skin to dry after cleansing with alcohol. If the skin is not allowed to dry before the injection, the alcohol may be carried into the tissues, resulting in a localized reddened area and a burning sensation.

Inserting the Needle. There are varying approaches to inserting the needle for insulin injections. The correct technique is based on the need for the insulin to be injected into the subcutaneous tissue (Chart 41-7). Injection that is too deep (eg, intramuscular) or too shallow may affect the rate of absorption of the insulin. For a normal or overweight person, a 90-degree angle is the best insertion angle. Aspiration (inserting the needle and then pulling back on the plunger to assess for blood being drawn into the syringe) is generally not recommended with self-injection of insulin. Many patients who have been using insulin for an extended

CHART 41-7 PATIENT EDUCATION

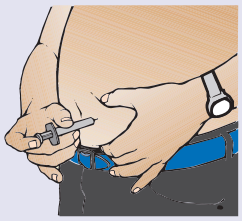
Self-Injection of Insulin

1. With one hand, stabilize the skin by spreading it or pinching up a large area.



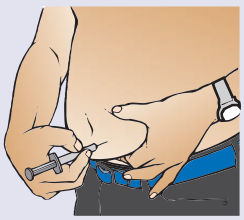
Pinching the skin

2. Pick up syringe with the other hand and hold it as you would a pencil. Insert needle straight into the skin.*



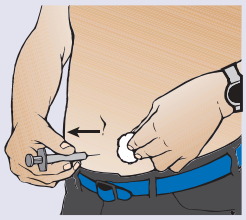
Inserting the needle into the skin

3. To inject the insulin, push the plunger all the way in.



Injecting the insulin

4. Pull needle straight out of skin. Press cotton ball over injection site for several seconds.



Removing the needle and holding cotton ball over site

5. Use disposable syringe *only once* and discard into hard plastic container (with a tight-fitting top) such as an empty bleach or detergent container.† Follow state regulations for disposal of syringes and needles.

Disposing of syringe

*Some patients may be taught to insert the needle at a 45-degree angle.

†Although some studies suggest that reusing disposable syringes may be safe, it is recommended that this be done only in the absence of poor personal hygiene, an acute concurrent illness, open wounds on the hands, or decreased resistance to infection.

period have eliminated this step from their insulin injection routine with no apparent adverse effects.

Disposing of Syringes and Needles. Insulin syringes and pens, needles, and lancets should be disposed of according to local regulations. If community disposal programs are unavailable, used sharps should be placed in a puncture-resistant container. The patient should contact local trash authorities for instructions about proper disposal of filled containers, which should not be mixed with containers to be recycled.

Promoting Home and Community-Based Care

Promoting Self-Care

If problems exist with glucose control or with the development of preventable complications, it is the nurse's responsibility to assess the reasons for the patient's ineffective management of the treatment regimen. It should not be assumed that problems with diabetes management are related to the patient's willful decision to ignore self-management. The patient may have forgotten or never learned certain information. The problem may be correctable simply through providing complete information and ensuring that the patient understands the information. The focus of diabetes education should be patient empowerment. Patient education should address behavior change, self-efficacy, and health beliefs. Chart 41-8 details how to evaluate the effectiveness of self-injection of insulin education.

If knowledge deficit is not the problem, certain physical or emotional factors may be impairing the patient's ability to perform self-care skills. For example, decreased visual acuity may impair the patient's ability to administer insulin accurately, measure the blood glucose level, or inspect the skin and feet. In addition, decreased joint mobility (especially in the elderly) or preexisting disability may impair the patient's ability to inspect the bottom of the feet. Denial of the diagnosis or depression may impair the patient's ability to carry out multiple daily self-care measures. The patient whose family, personal, or work problems may be of higher priority may benefit from assistance in establishing priorities. It is also important to assess the patient for infection or emotional stress, which may lead to elevated blood glucose levels despite adherence to the treatment regimen.

The following approaches are helpful for promoting self-care management skills:

- Address any underlying factors (eg, knowledge deficit, self-care deficit, illness) that may affect diabetic control.
- Simplify the treatment regimen if it is too difficult for the patient to follow.
- Adjust the treatment regimen to meet patient requests (eg, adjust diet or insulin schedule to allow increased flexibility in meal content or timing).
- Establish a specific plan or contract with each patient with simple, measurable goals.
- Provide positive reinforcement of self-care behaviors performed instead of focusing on behaviors that were neglected (eg, positively reinforce blood glucose tests that were performed instead of focusing on the number of missed tests).
- Help the patient identify personal motivating factors rather than focusing on wanting to please physicians or nurses.
- Encourage the patient to pursue life goals and interests, and discourage an undue focus on diabetes.

Continuing Care

The degree to which patients interact with health care providers to obtain ongoing care depends on many factors. Age, socioeconomic level, existing complications, type of diabetes, and comorbid conditions all may dictate the frequency of follow-up visits. Many patients with diabetes are seen by home health nurses for diabetes education, wound care, insulin preparation, or assistance with glucose monitoring. Even patients who achieve excellent glucose control and have no complications can expect to see their primary health care provider at least twice a year for ongoing evaluation and should receive routine nutrition updates. In addition, the nurse should remind the patient to participate in recommended health promotion activities (eg, immunizations) and age-appropriate health screenings (eg, pelvic examinations, mammograms).

In addition, participation in support groups is encouraged for patients who have had diabetes for many years as well as for those who are newly diagnosed. Such participation may help the patient and family cope with changes in lifestyle that occur with the onset of diabetes and its complications. People who participate in support groups often share valuable information and experiences and learn from others. Support groups provide an opportunity for discussion of strategies to deal with diabetes and its management and to clarify and verify information with nurses or other health care professionals. Participation in support groups may also promote healthy activities.

ACUTE COMPLICATIONS OF DIABETES

There are three major acute complications of diabetes related to short-term imbalances in blood glucose levels: hypoglycemia, DKA, and hyperglycemic hyperosmolar nonketotic syndrome, which is also called hyperglycemic hyperosmolar syndrome or state.

Hypoglycemia (Insulin Reactions)

Hypoglycemia occurs when the blood glucose falls to less than 50 to 60 mg/dL (2.7 to 3.3 mmol/L), because of too much insulin or oral hypoglycemic agents, too little food, or excessive physical activity. Hypoglycemia may occur at any time of the day or night. It often occurs before meals, especially if meals are delayed or snacks are omitted. For example, midmorning hypoglycemia may occur when the morning regular insulin is peaking, whereas hypoglycemia that occurs in the late afternoon coincides with the peak of the morning NPH or Lente insulin. Middle-of-the-night hypoglycemia may occur because of peaking evening or predinner NPH or Lente insulins, especially in patients who have not eaten a bedtime snack.

Chart 41-8 • *Outcome Criteria for Determining Effectiveness of Self-Injection of Insulin Education*

Equipment

Insulin

1. Identifies information on label of insulin bottle:
   - Type (eg, NPH, regular, 70/30)
   - Species (human, biosynthetic, pork)
   - Manufacturer (Lilly, Novo Nordisk)
   - Concentration (eg, U-100)
   - Expiration date
2. Checks appearance of insulin:
   - Clear or milky white
   - Checks for flocculation (clumping, frosted appearance)
3. Identifies where to purchase and store insulin:
   - Indicates approximately how long bottle will last (1,000 units per bottle U-100 insulin)
   - Indicates how long opened bottles can be used

Syringes

1. Identifies concentration (U-100) marking on syringe
2. Identifies size of syringe (eg, 100-unit, 50-unit, 30-unit)
3. Describes appropriate disposal of used syringe

Preparation and Administration of Insulin Injection

1. Draws up correct amount and type of insulin
2. Properly mixes two insulins if necessary
3. Inserts needle and injects insulin
4. Describes site rotation:
   - Demonstrates injection with all anatomic areas to be used
   - Describes pattern for rotation, such as using abdomen only or using certain areas at the same time of day
   - Describes system for remembering site locations, such as horizontal pattern across the abdomen as if drawing a dotted line

Knowledge of Insulin Action

1. Lists prescription:
   - Type and dosage of insulin
   - Timing of insulin injections
2. Describes approximate time course of insulin action:
   - Identifies longing and short-acting insulins by name
   - States approximate time delay until onset of insulin action
   - Identifies need to delay food until 5 to 15 min after injection of rapid-acting insulin (lispro, aspart, glulisine [Apidra])
   - Knows that longer time delays are safe when blood glucose level is high, and time delays may need to be shortened when blood glucose level is low

Incorporation of Insulin Injections Into Daily Schedule

1. Recites proper order of premeal diabetes activities:
   - May use mnemonic device such as the word "tie," which helps the patient remember the order of activities ("t" = test [blood glucose], "i" = insulin injection, "e" = eat)
   - Describes daily schedule, such as test, insulin, eat before breakfast and dinner; test and eat, before lunch and bedtime
2. Describes information regarding hypoglycemia:
   - Symptoms: shakiness, sweating, nervousness, hunger, weakness
   - Causes: too much insulin, too much exercise, not enough food
   - Treatment: 15 g concentrated carbohydrate, such as two or three glucose tablets, 1 tube glucose gel, 0.5 cup juice
   - After initial treatment, follow with snack including starch and protein, such as cheese and crackers, milk and crackers, half sandwich
3. Describes information regarding prevention of hypoglycemia:
   - Avoids delays in meal timing
   - Eats a meal or snack approximately every 4 to 5 h (while awake)
   - Does not skip meals
   - Increases food intake before exercise if blood glucose level is <100 mg/dL
   - Checks blood glucose regularly
   - Identifies safe modification of insulin doses consistent with management plan
   - Carries a form of fast-acting sugar at all times
   - Wears a medical identification bracelet
   - Teaches family, friends, coworkers about signs and treatment of hypoglycemia
   - Has family, roommates, traveling companions learn to use injectable glucagon for severe hypoglycemic reactions
4. Maintains regular follow-up for evaluation of diabetes control:
   - Keeps written record of blood glucose, insulin doses, hypoglycemic reactions, variations in diet
   - Keeps all appointments with health professionals
   - Sees health care provider regularly (usually two to four times per year)
   - States how to contact health care provider in case of emergency
   - States when to call health care provider to report variations in blood glucose levels

Gerontologic Considerations

In elderly patients with diabetes, hypoglycemia is a particular concern for many reasons:

- Elderly people frequently live alone and may not recognize the symptoms of hypoglycemia.
- With decreasing renal function, it takes longer for oral hypoglycemic agents to be excreted by the kidneys.
- Skipping meals may occur because of decreased appetite or financial limitations.
- Decreased visual acuity may lead to errors in insulin administration.

Clinical Manifestations

The clinical manifestations of hypoglycemia may be grouped into two categories: adrenergic symptoms and central nervous system (CNS) symptoms.

In mild hypoglycemia, as the blood glucose level falls, the sympathetic nervous system is stimulated, resulting in a surge of epinephrine and norepinephrine. This causes symptoms

such as sweating, tremor, tachycardia, palpitation, nervousness, and hunger.

In moderate hypoglycemia, the drop in blood glucose level deprives the brain cells of needed fuel for functioning. Signs of impaired function of the CNS may include inability to concentrate, headache, lightheadedness, confusion, memory lapses, numbness of the lips and tongue, slurred speech, impaired coordination, emotional changes, irrational or combative behavior, double vision, and drowsiness. Any combination of these symptoms (in addition to adrenergic symptoms) may occur with moderate hypoglycemia.

In severe hypoglycemia, CNS function is so impaired that the patient needs the assistance of another person for treatment of hypoglycemia. Symptoms may include disoriented behavior, seizures, difficulty arousing from sleep, or loss of consciousness.

Assessment and Diagnostic Findings

Symptoms of hypoglycemia may occur suddenly and vary considerably from person to person. To some degree, this may be related to the actual level to which the blood glucose falls or to the rate at which it falls. For example, patients who usually have a blood glucose level in the hyperglycemic range (eg, 200 mg/dL or greater) may feel hypoglycemic (adrenergic) symptoms when their blood glucose falls rapidly to 120 mg/dL (6.6 mmol/L) or less. Conversely, patients who frequently have a glucose level in the low range of normal (eg, 80 to 100 mg/dL) may be asymptomatic when the blood glucose falls slowly to less than 50 mg/dL (2.7 mmol/L).

Decreased hormonal (adrenergic) response to hypoglycemia may also contribute to lack of symptoms of hypoglycemia. This occurs in some patients who have had diabetes for many years. It may be related to autonomic neuropathy, a chronic diabetic complication (see later discussion). As the blood glucose level falls, the normal surge in adrenalin does not occur, and the usual adrenergic symptoms, such as sweating and shakiness, do not take place. The hypoglycemia may not be detected until moderate or severe CNS impairment occurs. Affected patients must perform SMBG on a frequent regular basis, especially before driving or engaging in other potentially dangerous activities.

Management

Treating with Carbohydrates

Immediate treatment must be given when hypoglycemia occurs. The usual recommendation is for 15 g of a fast-acting concentrated source of carbohydrate such as the following, given orally:

- Three or four commercially prepared glucose tablets
- 4 to 6 oz of fruit juice or regular soda
- 6 to 10 hard candies
- 2 to 3 teaspoons of sugar or honey

It is not necessary to add sugar to juice, even if it is labeled as unsweetened juice: the fruit sugar in juice contains enough carbohydrate to raise the blood glucose level. Adding table sugar to juice may cause a sharp increase in the blood glucose level, and patients may experience hyperglycemia for hours after treatment.

The blood glucose level should be retested in 15 minutes and retreated if it is less than 70 to 75 mg/dL (3.8 to 4 mmol/L). If the symptoms persist for longer than 10 to 15 minutes after initial treatment, the treatment is repeated even if blood glucose testing is not possible. Once the symptoms resolve, a snack containing protein and starch (eg, milk or cheese and crackers) is recommended unless the patient plans to eat a regular meal or snack within 30 to 60 minutes.

Initiating Emergency Measures

In emergency situations, for adults who are unconscious and cannot swallow, an injection of glucagon 1 mg can be administered either subcutaneously or intramuscularly. Glucagon is a hormone produced by the alpha cells of the pancreas that stimulates the liver to breakdown glycogen, the stored glucose. Injectable glucagon is packaged as a powder in 1-mg vials and must be mixed with a diluent immediately before being injected. After injection of glucagon, the patient may take as long as 20 minutes to regain consciousness. A concentrated source of carbohydrate followed by a snack should be given to the patient on awakening to prevent recurrence of hypoglycemia (because the duration of the action of 1 mg of glucagon is brief—its onset is 8 to 10 minutes, and its action lasts 12 to 27 minutes) and to replenish liver stores of glucose. Some patients experience nausea after the administration of glucagon. If this occurs, the patient should be turned to the side to prevent aspiration in case the patient vomits.

Glucagon is sold by prescription only and should be part of the emergency supplies available to patients with diabetes who require insulin. Family members, friends, neighbors, and coworkers should be instructed in the use of glucagon, especially for patients who have little or no warning of hypoglycemic episodes. Patients should be instructed to notify their physician after severe hypoglycemia has occurred and been treated.

In hospitals and emergency departments, for patients who are unconscious or cannot swallow, 25 to 50 mL of 50% dextrose in water ($D_{50}W$) may be administered IV. The effect is usually seen within minutes. The patient may complain of a headache and of pain at the injection site. Assuring patency of the IV line used for injection of 50% dextrose is essential because hypertonic solutions such as 50% dextrose are very irritating to veins.

Providing Patient Education

Hypoglycemia is prevented by a consistent pattern of eating, administering insulin, and exercising. Between-meal and bedtime snacks may be needed to counteract the maximum insulin effect. In general, the patient should cover the time of peak activity of insulin by eating a snack and by taking additional food when physical activity is increased. Routine blood glucose tests are performed so that changing insulin requirements may be anticipated and the dosage adjusted. Because unexpected hypoglycemia can occur, all patients treated with insulin should wear an identification bracelet or tag stating that they have diabetes.

Patients and family members must be instructed to recognize the symptoms of hypoglycemia. Family members in particular must be made aware that any subtle (but unusual)

change in behavior may be an indication of hypoglycemia. They should be taught to encourage and even insist that the person with diabetes assess blood glucose levels if hypoglycemia is suspected. When some patients are hypoglycemic, they become very resistant to testing or eating and become angry at family members who are trying to treat the hypoglycemia. Family members must be taught to persevere and to understand that the hypoglycemia can cause irrational behavior.

Autonomic neuropathy or beta-blockers such as propranolol (Inderal) to treat hypertension or cardiac dysrhythmias may mask the typical symptoms of hypoglycemia. It is very important that these patients perform blood glucose tests on a frequent and regular basis. Patients who have type 2 diabetes and who take oral sulfonylurea agents may also develop hypoglycemia, which can be prolonged and severe; this is a particular risk for elderly patients.

It is important that patients with diabetes, especially those receiving insulin, learn to carry some form of simple sugar with them at all times (ADA, 2008b). There are many different commercially prepared glucose tablets and gels that the patient may find convenient to carry. If the patient has a hypoglycemic reaction and does not have any of the recommended emergency foods available, he or she should eat any available food (preferably a carbohydrate food).

Patients are advised to refrain from eating high-calorie, high-fat dessert foods (eg, cookies, cakes, doughnuts, ice cream) to treat hypoglycemia because their high fat content may slow the absorption of the glucose and resolution of the hypoglycemic symptoms. The patient may subsequently eat more of the foods when symptoms do not resolve rapidly, which may cause very high blood glucose levels for several hours and may contribute to weight gain.

Patients who feel unduly restricted by their meal plan may view hypoglycemic episodes as a time to reward themselves with desserts. It may be more prudent to teach these patients to incorporate occasional desserts into the meal plan. This may make it easier for them to limit their treatment of hypoglycemic episodes to simple (low-calorie) carbohydrates such as juice or glucose tablets.

Diabetic Ketoacidosis

DKA is caused by an absence or markedly inadequate amount of insulin. This deficit in available insulin results in disorders in the metabolism of carbohydrate, protein, and fat. The three main clinical features of DKA are

- Hyperglycemia
- Dehydration and electrolyte loss
- Acidosis

Pathophysiology

Without insulin, the amount of glucose entering the cells is reduced, and production and release of glucose by the liver (gluconeogenesis) is increased, leading to hyperglycemia (Fig. 41-7). In an attempt to rid the body of the excess glucose, the kidneys excrete the glucose along with water and electrolytes (eg, sodium, potassium). This osmotic diuresis, which is characterized by excessive urination (polyuria),

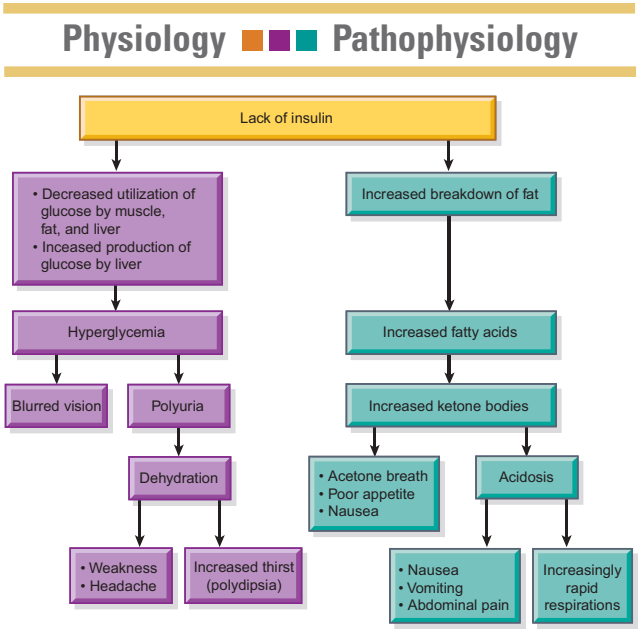


Figure 41-7 Abnormal metabolism that causes signs and symptoms of diabetic ketoacidosis. (Redrawn from Pearce, M. A., Rosenberg, C. S. & Davidson, M. D. (2003). Patient education. In Davidson, M. B. (Ed.). *Diabetes mellitus: Diagnosis and treatment.* New York: Churchill Livingstone.)

leads to dehydration and marked electrolyte loss. Patients with severe DKA may lose up to 6.5 L of water and up to 400 to 500 mEq each of sodium, potassium, and chloride over a 24-hour period.

Another effect of insulin deficiency or deficit is the breakdown of fat (lipolysis) into free fatty acids and glycerol. The free fatty acids are converted into ketone bodies by the liver. Ketone bodies are acids; their accumulation in the circulation due to lack of insulin leads to metabolic acidosis.

Three main causes of DKA are decreased or missed dose of insulin, illness or infection, and undiagnosed and untreated diabetes (DKA may be the initial manifestation of diabetes). An insulin deficit may result from an insufficient dosage of insulin prescribed or from insufficient insulin being administered by the patient. Errors in insulin dosage may be made by patients who are ill and who assume that if they are eating less or if they are vomiting, they must decrease their insulin doses. (Because illness, especially infections, can cause increased blood glucose levels, the patient does not need to decrease the insulin dose to compensate for decreased food intake when ill and may even need to increase the insulin dose.)

Other potential causes of decreased insulin include patient error in drawing up or injecting insulin (especially in patients with visual impairments), intentional skipping of insulin doses (especially in adolescents with diabetes who are having difficulty coping with diabetes or other aspects of their lives), or equipment problems (eg, occlusion of insulin pump tubing). Illness and infections are associated with insulin resistance. In response to physical (and emotional) stressors, there is an increase in the level of "stress"

CHART 41-9 PATIENT EDUCATION

Guidelines to Follow During Periods of Illness ("Sick Day Rules")

- Take insulin or oral antidiabetic agents as usual.
- Test blood glucose and test urine ketones every 3 to 4 h.
- Report elevated glucose levels (>300 mg/dL [16.6 mmol/L] or as otherwise specified) or urine ketones to your health care provider.
- If you take insulin, you may need supplemental doses of regular insulin every 3 to 4 h.
- If you cannot follow your usual meal plan, substitute soft foods (eg, ⅓ cup regular gelatin, 1 cup cream soup, ½ cup custard, 3 squares graham crackers) six to eight times per day.
- If vomiting, diarrhea, or fever persists, take liquids (eg, ½ cup regular cola or orange juice, ½ cup broth, 1 cup Gatorade) every ½ to 1 hour to prevent dehydration and to provide calories.
- Report nausea, vomiting, and diarrhea to your health care provider, because extreme fluid loss may be dangerous.
- If you are unable to retain oral fluids, you may require hospitalization to avoid diabetic ketoacidosis and possibly coma.

hormones—glucagon, epinephrine, norepinephrine, cortisol, and growth hormone. These hormones promote glucose production by the liver and interfere with glucose utilization by muscle and fat tissue, counteracting the effect of insulin. If insulin levels are not increased during times of illness and infection, hyperglycemia may progress to DKA (ADA, 2006c).

Prevention

For prevention of DKA related to illness, "sick day" rules for managing their diabetes when ill (Chart 41-9) should be reviewed. The most important concept in this is to never eliminate insulin doses when nausea and vomiting occur. Instead, the patient should take the usual insulin dose (or previously prescribed special "sick day" doses) and then attempt to consume frequent small portions of carbohydrates (including foods usually avoided, such as juices, regular sodas, and gelatin). Drinking fluids every hour is important to prevent dehydration. Blood glucose and urine ketones must be assessed every 3 to 4 hours.

If the patient cannot take fluids without vomiting, or if elevated glucose or ketone levels persist, the physician must be contacted. Patients are taught to have foods available for use on sick days. In addition, a supply of urine test strips (for ketone testing) and blood glucose test strips should be available. The patient must know how to contact his or her physician 24 hours a day. These materials should be assembled in a "sick day" kit.

After the acute phase of DKA has resolved, the nurse should assess for underlying causes of DKA. If there are psychological reasons for the patient's deliberately missing insulin doses, the patient and family may be referred for evaluation and counseling or therapy.

Clinical Manifestations

The hyperglycemia of DKA leads to polyuria and polydipsia (increased thirst). In addition, the patient may experience blurred vision, weakness, and headache. Patients with marked intravascular volume depletion may have orthostatic hypotension (drop in systolic blood pressure of 20 mm Hg or more on changing from a reclining to a standing position). Volume depletion may also lead to frank hypotension with a weak, rapid pulse.

The ketosis and acidosis of DKA lead to gastrointestinal symptoms such as anorexia, nausea, vomiting, and abdominal pain. The abdominal pain and physical findings on examination can be so severe that they resemble an acute abdominal disorder that requires surgery. The patient may have acetone breath (a fruity odor), which occurs with elevated ketone levels. In addition, hyperventilation (with very deep, but not labored, respirations) may occur. These Kussmaul respirations represent the body's attempt to decrease the acidosis, counteracting the effect of the ketone buildup. In addition, mental status in DKA varies widely. The patient may be alert, lethargic, or comatose.

Assessment and Diagnostic Findings

Blood glucose levels may vary between 300 and 800 mg/dL (16.6 to 44.4 mmol/L). Some patients have lower glucose values, and others have values of 1000 mg/dL (55.5 mmol/L) or higher (usually depending on the degree of dehydration). The severity of DKA is not necessarily related to the blood glucose level. Evidence of ketoacidosis is reflected in low serum bicarbonate (0 to 15 mEq/L) and low pH (6.8 to 7.3) values. A low partial pressure of carbon dioxide (PCO_2; 10 to 30 mm Hg) reflects respiratory compensation (Kussmaul respirations) for the metabolic acidosis. Accumulation of ketone bodies (which precipitates the acidosis) is reflected in blood and urine ketone measurements.

Sodium and potassium concentrations may be low, normal, or high, depending on the amount of water loss (dehydration). Despite the plasma concentration, there has been a marked total body depletion of these (and other) electrolytes and they will need to be replaced.

Increased levels of creatinine, blood urea nitrogen (BUN), and hematocrit may also be seen with dehydration. After rehydration, continued elevation in the serum creatinine and BUN levels suggests underlying renal insufficiency.

Management

In addition to treating hyperglycemia, management of DKA is aimed at correcting dehydration, electrolyte loss, and acidosis.

Rehydration

In dehydrated patients, rehydration is important for maintaining tissue perfusion. In addition, fluid replacement enhances the excretion of excessive glucose by the kidneys. The patient may need as much as 6 to 10 L of IV fluid to replace fluid losses caused by polyuria, hyperventilation, diarrhea, and vomiting.

Initially, 0.9% sodium chloride (normal saline) solution is administered at a rapid rate, usually 0.5 to 1 L/h for 2 to 3 hours. Half-strength normal saline (0.45%) solution (also known as hypotonic saline solution) may be used for patients with hypertension or hypernatremia and those at risk for heart failure. After the first few hours, half-strength normal saline solution is the fluid of choice for continued rehydration, provided the blood pressure is stable and the sodium level is not low. Moderate to high rates of infusion (200 to 500 mL/h) may be needed for several more hours. When the blood glucose level reaches 300 mg/dL (16.6 mmol/L) or less, the IV solution may be changed to dextrose 5% in water (D_5W) to prevent a precipitous decline in the blood glucose level (Fowler, 2009).

Monitoring of fluid volume status involves frequent measurements of vital signs (including monitoring for orthostatic changes in blood pressure and heart rate), lung assessment, and monitoring of intake and output. Initial urine output lags behind IV fluid intake as dehydration is corrected. Plasma expanders may be necessary to correct severe hypotension that does not respond to IV fluid treatment. Monitoring for signs of fluid overload is especially important for patients who are older, have renal impairment, or are at risk for heart failure.

Restoring Electrolytes

The major electrolyte of concern during treatment of DKA is potassium. Although the initial plasma concentration of potassium may be low, normal, or even high, there is a major loss of potassium from body stores and an intracellular-to-extracellular shift of potassium. Furthermore, the serum level of potassium decreases as potassium reenters the cells during the course of treatment of DKA; therefore, the serum potassium level must be monitored frequently. Some of the factors related to treating DKA that reduce the serum potassium concentration include rehydration, which leads to increased plasma volume and subsequent decreases in the concentration of serum potassium. Rehydration also leads to increased urinary excretion of potassium. Insulin administration enhances the movement of potassium from the extracellular fluid into the cells.

Cautious but timely potassium replacement is vital to avoid dysrhythmias that may occur with hypokalemia. As much as 40 mEq/h may be needed for several hours. Because extracellular potassium levels decrease during DKA treatment, potassium must be infused even if the plasma potassium level is normal.

Frequent (every 2 to 4 hours initially) ECGs and laboratory measurements of potassium are necessary during the first 8 hours of treatment. Potassium replacement is withheld only if hyperkalemia is present or if the patient is not urinating.

NURSING ALERT

Because a patient's serum potassium level may drop quickly as a result of rehydration and insulin treatment, potassium replacement must begin once potassium levels drop to normal.

Reversing Acidosis

Ketone bodies (acids) accumulate as a result of fat breakdown. The acidosis that occurs in DKA is reversed with insulin, which inhibits fat breakdown, thereby stopping acid buildup. Insulin is usually infused intravenously at a slow, continuous rate (eg, 5 units/h). Hourly blood glucose values must be measured. IV fluid solutions with higher concentrations of glucose, such as normal saline (NS) solution (eg, D_5NS, $D_5{\cdot}45NS$), are administered when blood glucose levels reach 250 to 300 mg/dL (13.8 to 16.6 mmol/L) to avoid too rapid a drop in the blood glucose level (ie, hypoglycemia) during treatment.

Regular insulin, the only type of insulin approved for IV use, may be added to IV solutions. The nurse must convert hourly rates of insulin infusion (frequently prescribed as units per hour) to IV drip rates. For example, if 100 units of regular insulin are mixed into 500 mL of 0.9% NS, then 1 unit of insulin equals 5 mL; therefore, an initial insulin infusion rate of 5 units/h would equal 25 mL/h. The insulin is often infused separately from the rehydration solutions to allow frequent changes in the rate and content of the latter.

Insulin must be infused continuously until subcutaneous administration of insulin can be resumed. Any interruption in administration may result in the reaccumulation of ketone bodies and worsening acidosis. Even if blood glucose levels are decreasing and returning to normal, the insulin drip must not be stopped until subcutaneous insulin therapy has been started. Rather, the rate or concentration of the dextrose infusion should be increased. Blood glucose levels are usually corrected before the acidosis is corrected. Therefore, IV insulin may be continued for 12 to 24 hours, until the serum bicarbonate level increases (to at least 15 to 18 mEq/L) and until the patient can eat. In general, bicarbonate infusion to correct severe acidosis is avoided during treatment of DKA because it precipitates further, sudden (and potentially fatal) decreases in serum potassium levels. Continuous insulin infusion is usually sufficient for reversal of DKA (ADA, 2006c).

NURSING ALERT

When mixing the insulin drip, it is important to flush the insulin solution through the entire IV infusion set and to discard the first 50 mL of fluid. Insulin molecules adhere to the inner surface of IV infusion sets; therefore, the initial fluid may contain a decreased concentration of insulin.

Hyperglycemic Hyperosmolar Nonketotic Syndrome

Hyperglycemic hyperosmolar nonketotic syndrome (HHNS) is a serious condition in which hyperosmolarity and hyperglycemia predominate, with alterations of the sensorium (sense of awareness). At the same time, ketosis is usually minimal or absent. The basic biochemical defect is lack of effective insulin (ie, insulin resistance). Persistent hyperglycemia causes osmotic diuresis, which results in losses of water and electrolytes. To maintain osmotic equilibrium, water shifts from the intracellular fluid space to the

Table 41-8 COMPARISON OF DIABETIC KETOACIDOSIS (DKA) AND HYPERGLYCEMIC HYPEROSMOLAR NONKETOTIC SYNDROME (HHNS)

Characteristics	DKA	HHNS
Patients most commonly affected	Can occur in type 1 or type 2 diabetes; more common in type 1 diabetes	Can occur in type 1 or type 2 diabetes; more common in type 2 diabetes, especially elderly patients with type 2 diabetes
Precipitating event	Omission of insulin; physiologic stress (infection, surgery, CVA, MI)	Physiologic stress (infection, surgery, CVA, MI)
Onset	Rapid (<24 h)	Slower (over several days)
Blood glucose levels	Usually >250 mg/dL (>13.9 mmol/L)	Usually >600 mg/dL (>33.3 mmol/L)
Arterial pH level	<7.3	Normal
Serum and urine ketones	Present	Absent
Serum osmolality	300–350 mOsm/L	>350 mOsm/L
Plasma bicarbonate level	<15 mEq/L	Normal
BUN and creatinine levels	Elevated	Elevated
Mortality rate	<5%	10–40%

BUN, blood urea nitrogen; CVA, cerebrovascular accident; MI, myocardial infarction.

extracellular fluid space. With glycosuria and dehydration, hypernatremia and increased osmolarity occur. Table 41-8 compares DKA and HHNS.

HHNS occurs most often in older people (50 to 70 years of age) who have no known history of diabetes or who have type 2 diabetes. HHNS often can be traced to a precipitating event such as an acute illness (eg, pneumonia, cerebrovascular accident [CVA]), medications that exacerbate hyperglycemia (eg, thiazides), or treatments such as dialysis. The history includes days to weeks of polyuria with adequate fluid intake. What distinguishes HHNS from DKA is that ketosis and acidosis generally do not occur in HHNS, partly because of differences in insulin levels. In DKA, no insulin is present, and this promotes the breakdown of stored glucose, protein, and fat, which leads to the production of ketone bodies and ketoacidosis. In HHNS, the insulin level is too low to prevent hyperglycemia (and subsequent osmotic diuresis), but it is high enough to prevent fat breakdown. Patients with HHNS do not have the ketosis-related gastrointestinal symptoms that lead them to seek medical attention. Instead, they may tolerate polyuria and polydipsia until neurologic changes or an underlying illness (or family members or others) prompts them to seek treatment. Because of possible delays in therapy, hyperglycemia, dehydration, and hyperosmolarity may be more severe in HHNS.

Clinical Manifestations

The clinical picture of HHNS is one of hypotension, profound dehydration (dry mucous membranes, poor skin turgor), tachycardia, and variable neurologic signs (eg, alteration of sensorium, seizures, hemiparesis). The mortality rate ranges from 10% to 40%, usually related to an underlying illness, the vulnerability of the elderly patient, and the severity of HHNS.

Assessment and Diagnostic Findings

Diagnostic assessment includes a range of laboratory tests, including blood glucose, electrolytes, BUN, complete blood count, serum osmolality, and arterial blood gas analysis. The blood glucose level is usually 600 to 1200 mg/dL, and the osmolality exceeds 350 mOsm/kg. Electrolyte and BUN levels are consistent with the clinical picture of severe dehydration. Mental status changes, focal neurologic deficits, and hallucinations are common secondary to the cerebral dehydration that results from extreme hyperosmolality. Postural hypotension accompanies the dehydration.

Management

The overall approach to the treatment of HHNS is similar to that of DKA: fluid replacement, correction of electrolyte imbalances, and insulin administration. Because patients with HHNS are typically older, close monitoring of volume and electrolyte status is important for prevention of fluid overload, heart failure, and cardiac dysrhythmias. Fluid treatment is started with 0.9% or 0.45% NS, depending on the patient's sodium level and the severity of volume depletion. Central venous or hemodynamic pressure monitoring guides fluid replacement. Potassium is added to IV fluids when urinary output is adequate and is guided by continuous ECG monitoring and frequent laboratory determinations of potassium.

Extremely elevated blood glucose concentrations decrease as the patient is rehydrated. Insulin plays a less important role in the treatment of HHNS because it is not needed for reversal of acidosis, as in DKA. Nevertheless, insulin is usually administered at a continuous low rate to treat hyperglycemia, and replacement IV fluids with dextrose are administered (as in DKA) after the glucose level has decreased to the range of 250 to 300 mg/dL (13.8 to 16.6 mmol/L) (ADA, 2008d; Fowler, 2009).

Other therapeutic modalities are determined by the underlying illness and the results of continuing clinical and laboratory evaluation. It may take 3 to 5 days for neurologic symptoms to clear, and treatment of HHNS usually continues well after metabolic abnormalities have resolved. After recovery from HHNS, many patients can control their diabetes with MNT alone or with MNT and oral antidiabetic medications. Insulin may not be needed once the acute hyperglycemic complication is resolved. Frequent SBGM is important in prevention of recurrence of HHNS.

NURSING PROCESS

The Patient With Diabetic Ketoacidosis or Hyperglycemic Hyperosmolar Nonketotic Syndrome

Assessment

For the patient with DKA, the nurse monitors the ECG for dysrhythmias indicating abnormal potassium levels. Vital signs (especially blood pressure and pulse), arterial blood gases, breath sounds, and mental status are assessed every hour and recorded on a flow sheet. Neurologic status checks are included as part of the hourly assessment as cerebral edema can be a severe and sometimes fatal outcome.

For the patient with HHNS, the nurse assesses vital signs, fluid status, and laboratory values. Fluid status and urine output are closely monitored because of the high risk of renal failure secondary to severe dehydration. Because HHNS tends to occur in older patients, the physiologic changes that occur with aging should be considered. Careful assessment of cardiovascular, pulmonary, and renal function throughout the acute and recovery phases of HHNS is important.

Diagnosis

Nursing Diagnoses

Based on the assessment data, major nursing diagnoses may include the following:

- Risk for fluid volume deficit related to polyuria and dehydration
- Fluid and electrolyte imbalance related to fluid loss or shifts
- Deficient knowledge about diabetes self-care skills or information
- Anxiety related to loss of control, fear of inability to manage diabetes, misinformation related to diabetes, fear of diabetes complications

Collaborative Problems/Potential Complications

Based on assessment data, potential complications may include the following:

- Fluid overload, pulmonary edema, and heart failure
- Hypokalemia
- Hyperglycemia and ketoacidosis
- Hypoglycemia
- Cerebral edema

Planning and Goals

The major goals for the patient may include maintenance of fluid and electrolyte balance, optimal control of blood glucose levels, ability to perform diabetes survival skills and self-care activities, and absence of complications.

Nursing Interventions

Maintaining Fluid and Electrolyte Balance

Intake and output are measured. IV fluids and electrolytes are administered as prescribed, and oral fluid intake is encouraged when it is permitted. Laboratory values of serum electrolytes (especially sodium and potassium) are monitored. Vital signs are monitored hourly for signs of dehydration (tachycardia, orthostatic hypotension) along with assessment of breath sounds, level of consciousness, presence of edema, and cardiac status (ECG rhythm strips).

Increasing Knowledge About Diabetes Management

The development of DKA or HHNS suggests the need for the nurse to carefully assess the patient's understanding of and adherence to the diabetes management plan. Further, factors that may have led to the development of DKA or HHNS are explored with the patient and family. If the patient's blood glucose monitoring, dietary intake, use of antidiabetes (insulin or oral agents) medications, and exercise patterns differ from those identified in the diabetes management plan, their relationship to the development of DKS or HHNS is discussed, along with early manifestations of DKA or HHNS. If other factors, such as trauma, illness, surgery, or stress, are implicated, appropriate strategies to respond to these and similar situations in the future are described so that the patient can respond in the future without developing life-threatening complications. It may be necessary to reteach survival skills to patients who may not be able to recall them. If the patient has omitted insulin or oral antidiabetes agents that have been prescribed, it is important to explore the reasons for doing so and address those issues to prevent future recurrence and readmissions for treatment of these complications.

If the patient has not previously been diagnosed with diabetes, the opportunity is used to teach the patient about the need for maintaining blood glucose at a normal level and learning about diabetes management and survival skills.

Monitoring and Managing Potential Complications

FLUID OVERLOAD. Fluid overload can occur because of the administration of a large volume of fluid at a rapid rate, which is often required to treat patients with DKA or HHNS. This risk is increased in elderly patients and in those with preexisting cardiac or renal disease. To avoid fluid overload and resulting heart failure and pulmonary edema, the nurse monitors the patient closely during treatment by measuring vital signs and intake and output at frequent intervals. Central venous pressure monitoring and hemodynamic monitoring may be initiated to provide additional measures of fluid status. Physical examination focuses on assessment of cardiac rate and rhythm, breath sounds, venous distention, skin turgor, and urine output. The nurse monitors fluid intake and keeps careful records of IV and other fluid intake, along with urine output measurements.

HYPOKALEMIA. As previously described, hypokalemia is a potential complication during the treatment of DKA as potassium is lost from body stores. Low serum potassium levels may result from rehydration, increased urinary excretion of potassium, and movement of potassium from the extracellular fluid into the cells with insulin administration.

Prevention of hypokalemia includes cautious replacement of potassium; however, before its administration, it is important to ensure that a patient's kidneys are functioning. Because of the adverse effects of hypokalemia on cardiac function, monitoring of the cardiac rate, cardiac rhythm, ECG, and serum potassium levels is essential.

CEREBRAL EDEMA. Although the cause of cerebral edema is unknown, rapid correction of hyperglycemia, resulting in fluid shifts, is thought to be the cause. Cerebral edema, which occurs more often in children than in adults, can be prevented by gradual reduction in the blood glucose level (ADA, 2006c). An hourly flow sheet is used to enable close monitoring of the blood glucose level, serum electrolyte levels, urine output, mental status, and neurologic signs. Precautions are taken to minimize activities that could increase intracranial pressure.

Teaching Patients Self-Care

The patient is taught survival skills, including treatment modalities (diet, insulin administration, monitoring of blood glucose, and, for type 1 diabetes, monitoring of urine ketones); recognition, treatment, and prevention of DKA and HHNS. Teaching addresses those factors leading to DKA or HHNS. Follow-up education is arranged with a home care nurse and dietitian or an outpatient diabetes education center. This is particularly important for patients who have experienced DKA or HHNS because of the need to address factors that led to its occurrence. The importance of self-monitoring and of monitoring and follow-up by primary health care providers is reinforced, and the patient is reminded about the importance of keeping follow-up appointments.

Evaluation

Expected Outcomes

1. Achieves fluid and electrolyte balance
   a. Demonstrates intake and output balance
   b. Exhibits electrolyte values within normal limits
   c. Exhibits vital signs that remain stable, with resolution of orthostatic hypotension and tachycardia
2. Demonstrates knowledge about DKA and HHNS
   a. Identifies factors leading to DKA and HHNS
   b. Describes signs and symptoms of DKA and HHNS
   c. Describes short-term and long-term consequences of DKA and HHNS
   d. Identifies strategies to prevent the development of DKA and HHNS
   e. States when contact with health care provider is needed to treat early signs of DKS and HHNS
3. Absence of complications
   a. Exhibits normal cardiac rate and rhythm and normal breath sounds
   b. Exhibits no jugular venous distention
   c. Exhibits blood glucose and urine ketone levels within target range
   d. Exhibits no manifestations of hypoglycemia or hyperglycemia
   e. Shows improved mental status without signs of cerebral edema

LONG-TERM COMPLICATIONS OF DIABETES

There has been a steady decline in the number of deaths attributable to ketoacidosis and infection in patients with diabetes but an alarming increase in the number of deaths from cardiovascular and renal complications. Long-term complications, which are becoming more common as more people live longer with diabetes, can affect almost every organ system of the body and are a major cause of disability. The general categories of long-term diabetic complications are macrovascular disease, microvascular disease, and neuropathy.

The specific causes and pathogenesis of each type of complication are still being investigated. However, it appears that increased levels of blood glucose may play a role in neuropathic disease, microvascular complications, and risk factors contributing to macrovascular complications. Hypertension may also be a major contributing factor, especially in macrovascular and microvascular diseases.

Long-term complications are seen in both type 1 and type 2 diabetes but usually do not occur within the first 5 to 10 years after diagnosis. However, evidence of these complications may be present at the time of diagnosis of type 2 diabetes, because patients may have had undiagnosed diabetes for many years. Renal (microvascular) disease is more prevalent in patients with type 1 diabetes, and cardiovascular (macrovascular) complications are more prevalent in older patients with type 2 diabetes.

Macrovascular Complications

Diabetic macrovascular complications result from changes in the medium to large blood vessels. Blood vessel walls thicken, sclerose, and become occluded by plaque that adheres to the vessel walls. Eventually, blood flow is blocked. These atherosclerotic changes tend to occur more often and at an earlier age in patients with diabetes. Coronary artery disease, cerebrovascular disease, and peripheral vascular disease are the three main types of macrovascular complications that occur frequently in the diabetic population.

Myocardial infarction (MI) is twice as common in men with diabetes and three times as common in women with diabetes, compared to people without diabetes. There is also an increased risk for complications resulting from MI and an increased likelihood of a second MI. Coronary artery disease may account for 50% to 60% of all deaths among patients with diabetes. The typical ischemic symptoms may be absent in patients with diabetes. Therefore, the patient may not experience the early warning signs of decreased coronary blood flow and may have "silent" MIs, which may be discovered only as changes on the ECG. However, ECG changes may not be apparent. This lack of ischemic symptoms may be secondary to autonomic neuropathy (see later discussion). Cardiac disease is discussed in detail in Chapter 28.

Cerebral blood vessels are similarly affected by accelerated atherosclerosis. Occlusive changes or the formation of an embolus elsewhere in the vasculature that lodges in a

cerebral blood vessel can lead to transient ischemic attacks and strokes. People with diabetes have twice the risk of developing cerebrovascular disease, and an increased risk of death from CVA. In addition, recovery from a stroke may be impaired in patients who have elevated blood glucose levels at the time of and immediately after a stroke. Because symptoms of CVA may be similar to symptoms of acute diabetic complications (HHNS or hypoglycemia), it is very important to assess the blood glucose level (and treat abnormal levels) rapidly in patients with these symptoms, so that testing and treatment of CVA (stroke) can be initiated promptly if indicated.

Atherosclerotic changes in the large blood vessels of the lower extremities are responsible for the increased incidence (two to three times higher than in nondiabetic people) of occlusive peripheral arterial disease in patients with diabetes. Signs and symptoms of peripheral vascular disease include diminished peripheral pulses and intermittent claudication (pain in the buttock, thigh, or calf during walking). The severe form of arterial occlusive disease in the lower extremities is largely responsible for the increased incidence of gangrene and subsequent amputation in patients with diabetes. Neuropathy and impairments in wound healing also play a role in diabetic foot disease (see later discussion).

Role of Diabetes in Macrovascular Diseases

Researchers continue to investigate the relationship between diabetes and macrovascular diseases. The main feature unique to diabetes is elevated blood glucose; however, a direct link has not been found between hyperglycemia and atherosclerosis. Although it may be tempting to attribute the increased prevalence of macrovascular diseases to the increased prevalence of certain risk factors (eg, obesity, increased triglyceride levels, hypertension) in patients with diabetes, there is a higher-than-expected rate of macrovascular diseases among patients with diabetes compared with patients without diabetes who have the same risk factors (ADA, 2009b). Therefore, diabetes itself is seen as an independent risk factor for accelerated atherosclerosis. Other potential factors that may play a role in diabetes-related atherosclerosis include platelet and clotting factor abnormalities, decreased flexibility of red blood cells, decreased oxygen release, changes in the arterial wall related to hyperglycemia, and possibly hyperinsulinemia.

Management

The focus of management is aggressive modification and reduction of risk factors. This involves prevention and treatment of the commonly accepted risk factors for atherosclerosis. MNT and exercise are important in managing obesity, hypertension, and hyperlipidemia. In addition, the use of medications to control hypertension and hyperlipidemia is indicated. Smoking cessation is essential. Control of blood glucose levels may reduce triglyceride concentrations and can significantly reduce the incidence of complications.

When macrovascular complications do occur, patients may require increased amounts of insulin or may need to switch from oral antidiabetic agents to insulin during illnesses.

Microvascular Complications

Diabetic microvascular disease (or microangiopathy) is characterized by capillary basement membrane thickening. The basement membrane surrounds the endothelial cells of the capillary. Researchers believe that increased blood glucose levels react through a series of biochemical responses to thicken the basement membrane to several times its normal thickness. Two areas affected by these changes are the retina and the kidneys.

DIABETIC RETINOPATHY

Diabetic retinopathy is the leading cause of blindness among people between 20 and 74 years of age in the United States; it occurs in both type 1 and type 2 diabetes (ADA, 2009b; CDC, 2008).

People with diabetes are subject to many visual complications (Table 41-9). The eye pathology referred to as diabetic retinopathy is caused by changes in the small blood vessels in the retina, the area of the eye that receives images and sends information about the images to the brain (Fig. 41-8). The retina is richly supplied with blood vessels of all

Table 41-9 OCULAR COMPLICATIONS OF DIABETES

Eye Disorder	Characteristics
Retinopathy	Deterioration of the small blood vessels that nourish the retina.
Background	Early stage, asymptomatic retinopathy. Blood vessels within the retina develop microaneurysms that leak fluid, causing swelling and forming deposits (exudates). In some cases, macular edema causes distorted vision.
Preproliferative	Represents increased destruction of retinal blood vessels.
Proliferative	Abnormal growth of new blood vessels on the retina. New vessels rupture, bleeding into the vitreous and blocking light. Ruptured blood vessels in the vitreous form scar tissue, which can pull on and detach the retina.
Cataracts	Opacity of the lens of the eye; cataracts occur at an earlier age in patients with diabetes.
Lens Changes	The lens of the eye can swell when blood glucose levels are elevated. For some patients, visual changes related to lens swelling may be the first symptoms of diabetes. It may take up to 2 months of improved blood glucose control before hyperglycemic swelling subsides and vision stabilizes. Therefore, patients are advised not to change eyeglass prescriptions during the 2 months after discovery of hyperglycemia.
Extraocular Muscle Palsy	This may occur as a result of diabetic neuropathy. The involvement of various cranial nerves responsible for ocular movements may lead to double vision. This usually resolves spontaneously.
Glaucoma	Results from occlusion of the outflow channels by new blood vessels. Glaucoma may occur with slightly higher frequency in the diabetic population.

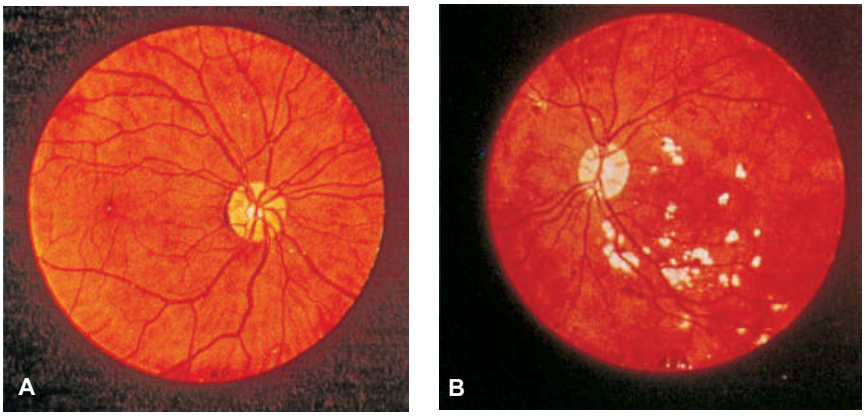


Figure 41-8 Diabetic retinopathy. **A,** In the fundus photograph of a normal eye, the light circular area over which a number of blood vessels converge is the optic disk, where the optic nerve meets the back of the eye. **B,** The fundus photograph of a patient with diabetic retinopathy shows characteristic waxy-looking retinal lesions, microaneurysms of the vessels, and hemorrhages. (Courtesy of American Optometric Association.)

kinds: small arteries and veins, arterioles, venules, and capillaries. Retinopathy has three main stages: nonproliferative (background), preproliferative, and proliferative.

Almost all patients with type 1 diabetes and more than 60% of patients with type 2 diabetes have some degree of retinopathy after 20 years (ADA, 2009b). Changes in the microvasculature include microaneurysms, intraretinal hemorrhage, hard exudates, and focal capillary closure. Although most patients do not develop visual impairment, it can be devastating if it occurs. A complication of nonproliferative retinopathy, macular edema, occurs in approximately 10% of people with type 1 or type 2 diabetes and may lead to visual distortion and loss of central vision (ADA, 2008e).

An advanced form of background retinopathy, preproliferative retinopathy, is considered to be a precursor to the more serious proliferative retinopathy. In preproliferative retinopathy, there are more widespread vascular changes and loss of nerve fibers. Epidemiologic evidence suggests that 10% to 50% of patients with preproliferative retinopathy will develop proliferative retinopathy within a short time (possibly as little as 1 year). As with background retinopathy, if visual changes occur during the preproliferative stage, they are usually caused by macular edema.

Proliferative retinopathy represents the greatest threat to vision and is characterized by the proliferation of new blood vessels growing from the retina into the vitreous. These new vessels are prone to bleeding. The visual loss associated with proliferative retinopathy is caused by this vitreous hemorrhage, retinal detachment, or both. The vitreous is normally clear, allowing light to be transmitted to the retina. When there is a hemorrhage, the vitreous becomes clouded and cannot transmit light, resulting in loss of vision. Another consequence of vitreous hemorrhage is that resorption of the blood in the vitreous leads to the formation of fibrous scar tissue. This scar tissue may place traction on the retina, resulting in retinal detachment and subsequent visual loss.

Clinical Manifestations

Retinopathy is a painless process. In nonproliferative and preproliferative retinopathy, blurry vision secondary to macular edema occurs in some patients, although many patients are asymptomatic. Even patients with a significant degree of proliferative retinopathy and some hemorrhaging may not experience major visual changes. However, symptoms indicative of hemorrhaging include floaters or cobwebs in the visual field, sudden visual changes including spotty or hazy vision, or complete loss of vision.

Assessment and Diagnostic Findings

Diagnosis is by direct visualization of the retina through dilated pupils with an ophthalmoscope or with a technique known as fluorescein angiography. Fluorescein angiography can document the type and activity of the retinopathy. Dye is injected into an arm vein and is carried to various parts of the body through the blood, but especially through the vessels of the retina of the eye. This technique allows an ophthalmologist, using special instruments, to see the retinal vessels in bright detail and gives useful information that cannot be obtained with just an ophthalmoscope.

Side effects of this diagnostic procedure may include nausea during the dye injection; yellowish, fluorescent discoloration of the skin and urine lasting 12 to 24 hours; and occasionally allergic reactions, usually manifested by hives or itching. However, the diagnostic procedure is generally safe.

Medical Management

The first focus of management of retinopathy is on primary and secondary prevention. The DCCT study (1993) demonstrated that in patients without preexisting retinopathy, maintenance of blood glucose to a normal or near-normal level in type 1 diabetes through intensive insulin therapy and patient education decreased the risk of retinopathy by 76%, compared with conventional therapy. The progression of retinopathy was decreased by 54% in patients with very mild to moderate nonproliferative retinopathy at the time of initiation of treatment. Similarly, the UKPDS study (1998) demonstrated that better control of blood glucose levels in patients with type 2 diabetes led to reduced risk of retinopathy.

Other strategies that may slow the progression of diabetic retinopathy include control of hypertension, control of blood glucose, and cessation of smoking.

For advanced cases of diabetic retinopathy, the main treatment is argon laser photocoagulation. The laser treatment

destroys leaking blood vessels and areas of neovascularization. For patients who are at increased risk for hemorrhage, panretinal photocoagulation may significantly reduce the rate of progression to blindness. Panretinal photocoagulation involves the systematic application of multiple (more than 1000) laser burns throughout the retina (except in the macular region). This stops the widespread growth of new vessels and hemorrhaging of damaged vessels. The role of "mild" panretinal photocoagulation (with only one-third to one-half as many laser burns) in the early stages of proliferative retinopathy or in patients with preproliferative changes is being investigated. For patients with macular edema, focal photocoagulation is used to apply smaller laser burns to specific areas of microaneurysms in the macular region. This may reduce the rate of visual loss from macular edema by 50% (ADA, 2008e).

Photocoagulation treatments are usually performed on an outpatient basis, and most patients can return to their usual activities by the next day. Limitations may be placed on activities involving weight bearing or bearing down. In most cases, the treatment does not cause intense pain, although patients may report varying degrees of discomfort. Usually an anesthetic eye drop is all that is needed during the treatment. A few patients may experience slight visual loss, loss of peripheral vision, or impairments in adaptation to the dark. However, the risk of slight visual changes from the laser treatment itself is much less than the potential for loss of vision from progression of retinopathy.

A major hemorrhage into the vitreous may occur, with the vitreous fluid becoming mixed with blood, preventing light from passing through the eye; this can cause blindness. A vitrectomy is a surgical procedure in which vitreous humor filled with blood or fibrous tissue is removed with a special drill-like instrument and replaced with saline or another liquid. A vitrectomy is performed for patients who already have visual loss and in whom the vitreous hemorrhage has not cleared on its own after 6 months. The purpose is to restore useful vision; recovery to near-normal vision is not usually expected.

Nursing Management

Nursing management of patients with diabetic retinopathy or other eye disorders involves implementing the individual plan of care and providing patient education. Education focuses on prevention through regular ophthalmologic examinations and blood glucose control and self-management of eye care regimens. The effectiveness of early diagnosis and prompt treatment is emphasized in teaching the patient and family. If vision loss occurs, nursing care must also address the patient's adjustment to impaired vision and use of adaptive devices for diabetes self-care as well as activities of daily living. Nursing care for patients with low vision or loss of vision is discussed in detail in Chapter 58.

Promoting Home and Community-Based Care

Teaching Patients Self-Care

Because the course of the retinopathy may be long and stressful, patient teaching is essential. In teaching and counseling patients, it is important to stress the following:

- Retinopathy may appear after many years of diabetes, and its appearance does not necessarily mean that the diabetes is on a downhill course.
- The odds for maintaining vision are in the patient's favor, especially with adequate control of glucose levels and blood pressure.
- Frequent eye examinations allow for the detection and prompt treatment of retinopathy.

A patient's response to vision loss depends on personality, self-concept, and coping mechanisms. Acceptance of blindness occurs in stages; some patients may learn to accept blindness in a rather short period, and others may never do so. An important issue in teaching patients is that several complications of diabetes occur simultaneously. For example, a patient who is blind due to diabetic retinopathy may also have peripheral neuropathy and may experience impairment of manual dexterity and tactile sensation. To prevent further losses, glycemic control remains a priority.

Continuing Care

The importance of careful diabetes management is emphasized as one means of slowing the progression of visual changes. The patient is reminded of the need to see an ophthalmologist regularly. If eye changes are progressive and unrelenting, the patient should be prepared for inevitable blindness. Therefore, consideration is given to making referrals for teaching the patient Braille and for training him or her with guide (ie, service) dogs. Referral to state agencies should be made to ensure that the patient receives services for the blind. Family members are also taught how to assist the patient to remain as independent as possible despite decreasing visual acuity.

Referral for home care may be indicated for some patients, particularly those who live alone, those who are not coping well, and those who have other health problems or complications of diabetes that may interfere with their ability to perform self-care. During home visits, the nurse can assess the patient's home environment and his or her ability to manage diabetes despite visual impairments. Medical management and nursing care of patients with visual disturbances are discussed in detail in Chapter 58.

NEPHROPATHY

Nephropathy, or renal disease secondary to diabetic microvascular changes in the kidney, is a common complication of diabetes (ADA, 2008f, 2009b). In the United States each year, people with diabetes account for almost 50% of new cases of end-stage renal disease (ESRD) and about 25% of those require dialysis or transplantation. About 20% to 30% of people with type 1 or type 2 diabetes develop nephropathy, but fewer of those with type 2 diabetes progress to ESRD. Native American, Latino, African American, Asian American, and Pacific Island people with type 2 diabetes are at greater risk for ESRD than non-Latino whites (ADA, 2009b).

Patients with type 1 diabetes frequently show initial signs of renal disease after 10 to 15 years; patients with type 2 diabetes develop renal disease within 10 years after the diagnosis of diabetes. Many patients with type 2 diabetes have

had diabetes for many years before the diabetes is diagnosed and treated. Therefore, they may have evidence of nephropathy at the time of diagnosis (ADA, 2008f). If blood glucose levels are elevated consistently for a significant period of time, the kidney's filtration mechanism is stressed, allowing blood proteins to leak into the urine. As a result, the pressure in the blood vessels of the kidney increases. It is thought that this elevated pressure serves as the stimulus for the development of nephropathy. Various medications and diets are being tested to prevent these complications.

The DCCT (1993) results showed that intensive treatment of type 1 diabetes with a goal of achieving a hemoglobin A_{1C} level as close to the nondiabetic range as possible reduced the occurrence of early signs of nephropathy. Similarly, the UKPDS study (1998) demonstrated a reduced incidence of overt nephropathy in patients with type 2 diabetes who controlled their blood glucose levels.

Clinical Manifestations

Most of the signs and symptoms of renal dysfunction in patients with diabetes are similar to those seen in patients without diabetes (see Chapter 44). In addition, as renal failure progresses, the catabolism (breakdown) of both exogenous and endogenous insulin decreases, and frequent hypoglycemic episodes may result. Insulin needs change as a result of changes in the catabolism of insulin, changes in diet related to the treatment of nephropathy, and changes in insulin clearance that occur with decreased renal function. The stress of renal disease affects self-esteem, family relationships, marital relations, and virtually all aspects of daily life. As renal function decreases, patients commonly have multiple-system failure (eg, declining visual acuity, impotence, foot ulcerations, heart failure, nocturnal diarrhea).

Assessment and Diagnostic Findings

Albumin is one of the most important blood proteins that leaks into the urine. Although small amounts may leak undetected for years, its leakage into the urine is among the earliest signs that can be detected. Clinical nephropathy eventually develops in more than 85% of people with microalbuminuria but in fewer than 5% of people without microalbuminuria. The urine should be checked annually for the presence of microalbumin. If the microalbuminuria exceeds 30 mg/24 hours on two consecutive random urine tests, a 24-hour urine sample should be obtained and tested. If results are positive, treatment is indicated (see later discussion).

In addition, tests for serum creatinine and BUN levels should be conducted annually. Diagnostic testing for cardiac or other systemic disorders may also be required with progression of other complications, and caution is indicated if contrast agents are used with these tests. Contrast agents and dyes used for some diagnostic tests may not be easily cleared by the damaged kidney, and the potential benefits of these diagnostic tests must be weighed against their potential risks.

Hypertension often develops in patients (with and without diabetes) who are in the early stages of renal disease. However, hypertension occurs in as many as 50% of all people with diabetes (for unknown reasons). Therefore, this symptom may or may not be due to renal disease; other diagnostic criteria must also be present.

Management

In addition to achieving and maintaining near-normal blood glucose levels, management for all patients with diabetes should include careful attention to the following:

- Control of hypertension (the use of angiotensin-converting enzyme [ACE] inhibitors, such as captopril [Capoten]), because control of hypertension may also decrease or delay the onset of early proteinuria
- Prevention or vigorous treatment of urinary tract infections
- Avoidance of nephrotoxic substances (eg, antibiotics, other selected medications)
- Adjustment of medications as renal function changes
- Low-sodium diet
- Low-protein diet

If the patient has already developed microalbuminuria and its level exceeds 30 mg/24 hours on two consecutive tests, an ACE inhibitor should be prescribed. ACE inhibitors lower blood pressure and reduce microalbuminuria, thereby protecting the kidney. Alternatively, angiotensin-receptor blocking (ARB) agents may be prescribed. This preventive strategy should be part of the standard of care for all people with diabetes. Carefully designed low-protein diets also appear to reverse early leakage of small amounts of protein from the kidney.

In chronic or end-stage renal failure, two types of treatment are available: dialysis (hemodialysis or peritoneal dialysis) and transplantation from a relative or a cadaver. Hemodialysis for patients with diabetes is similar to that for patients without the disease (see Chapter 44). Because hemodialysis creates additional stress on patients with cardiovascular disease, it may not be appropriate for some patients.

Continuous ambulatory peritoneal dialysis is being used by an increasing number of patients with diabetes, mainly because of the independence it allows. In addition, insulin can be mixed into the dialysate, which may result in better blood glucose control and end the need for insulin injections. In some cases, they may require higher doses of insulin because the dialysate contains glucose. Major risks of peritoneal dialysis are infection and peritonitis. The mortality rate for patients with diabetes undergoing dialysis is higher than that for patients without diabetes undergoing dialysis and is closely related to the severity of cardiovascular problems.

Renal disease is frequently accompanied by advancing retinopathy that may require laser treatments and surgery. Severe hypertension also worsens eye disease because of the additional stress it places on the blood vessels. Patients being treated with hemodialysis who require eye surgery may be changed to peritoneal dialysis and have their hypertension aggressively controlled for several weeks before surgery to prevent bleeding and damage to the retina. The rationale for this change is that hemodialysis requires anticoagulants that can increase the risk of bleeding after the surgery, and peritoneal dialysis minimizes pressure changes in the eyes.

The success rate for kidney transplantation in patients with diabetes has improved. In medical centers performing large numbers of transplantations, the chances are 75% to 80% that the transplanted kidney will continue to function in patients with diabetes for at least 5 years. Like the

original kidneys, transplanted kidneys can eventually be damaged if blood glucose levels are consistently high after the transplantation. Therefore, monitoring blood glucose levels frequently and adjusting insulin levels in patients with diabetes are essential for long-term success of kidney transplantation.

Diabetic Neuropathies

Diabetic neuropathy refers to a group of diseases that affect all types of nerves, including peripheral (sensorimotor), autonomic, and spinal nerves. The disorders appear to be clinically diverse and depend on the location of the affected nerve cells. The prevalence increases with the age of the patient and the duration of the disease and may be as high as 50% in patients who have had diabetes for 25 years (NIDDK, 2008a).

The etiology of neuropathy may involve elevated blood glucose levels over a period of years. The DCCT results (1993) showed that control of blood glucose levels to normal or near-normal levels decreased the incidence of neuropathy by 60%. The pathogenesis of neuropathy may be attributed to either a vascular or metabolic mechanism or both. Capillary basement membrane thickening and capillary closure may be present. In addition, there may be demyelinization of the nerves, which is thought to be related to hyperglycemia. Nerve conduction is disrupted when there are aberrations of the myelin sheaths.

The two most common types of diabetic neuropathy are sensorimotor polyneuropathy and autonomic neuropathy. Sensorimotor polyneuropathy is also called peripheral neuropathy. Cranial mononeuropathies—those affecting the oculomotor nerve—also occur in diabetes, especially in the elderly.

Peripheral Neuropathy

Peripheral neuropathy most commonly affects the distal portions of the nerves, especially the nerves of the lower extremities; it affects both sides of the body symmetrically and may spread in a proximal direction.

Clinical Manifestations

Although approximately half of patients with diabetic neuropathy do not have symptoms, initial symptoms may include paresthesias (prickling, tingling, or heightened sensation) and burning sensations (especially at night). As the neuropathy progresses, the feet become numb. In addition, a decrease in proprioception (awareness of posture and movement of the body and of position and weight of objects in relation to the body) and a decreased sensation of light touch may lead to an unsteady gait. Decreased sensations of pain and temperature place patients with neuropathy at increased risk for injury and undetected foot infections. Deformities of the foot may also occur; neuropathy-related joint changes produce Charcot joints. These joint deformities result from the abnormal weight distribution on joints resulting from lack of proprioception.

On physical examination, a decrease in deep tendon reflexes and vibratory sensation is found. For patients who have few or no symptoms of neuropathy, these physical findings may be the only indication of neuropathic changes. For patients with signs or symptoms of neuropathy, it is important to rule out other possible causes, including alcohol-induced and vitamin-deficiency neuropathies.

Management

The results of the DCCT study (1993) demonstrated that intensive insulin therapy and control of blood glucose levels delay the onset and slow the progression of neuropathy. Pain, particularly of the lower extremities, is a disturbing symptom in some people with neuropathy secondary to diabetes. In some cases, neuropathic pain spontaneously resolves within 6 months; for others, pain persists for many years. Various approaches to pain management can be tried. These include analgesics (preferably nonopioid); tricyclic antidepressants; antiseizure medications (phenytoin [Dilantin], carbamazepine [Tegretol], or gabapentin [Neurontin]); mexiletine (Mexitil, an antiarrhythmic); and transcutaneous electrical nerve stimulation (TENS).

Duloxetine (Cymbalta), an antidepressant medication, has been approved for treatment of peripheral diabetic neuropathy.

Autonomic Neuropathies

Neuropathy of the autonomic nervous system results in a broad range of dysfunctions affecting almost every organ system of the body (NIDDK, 2008a).

Clinical Manifestations

Three manifestations of autonomic neuropathy are related to the cardiac, gastrointestinal, and renal systems. Cardiovascular symptoms range from a fixed, slightly tachycardic heart rate and orthostatic hypotension to silent, or painless, myocardial ischemia and infarction. Delayed gastric emptying may occur with the typical gastrointestinal symptoms of early satiety, bloating, nausea, and vomiting. "Diabetic" constipation or diarrhea (especially nocturnal diarrhea) may occur as a result. In addition, there may be unexplained wide swings in blood glucose levels related to inconsistent absorption of the glucose from ingested foods secondary to the inconsistent gastric emptying.

Urinary retention, a decreased sensation of bladder fullness, and other urinary symptoms of neurogenic bladder result from autonomic neuropathy. The patient with a neurogenic bladder is predisposed to development of urinary tract infections because of the inability to empty the bladder completely. This is especially true of patients with poorly controlled diabetes because hyperglycemia impairs resistance to infection.

Hypoglycemic Unawareness

Autonomic neuropathy that affects the adrenal medulla is responsible for diminished or absent adrenergic symptoms of hypoglycemia. Patients may report that they no longer feel the typical shakiness, sweating, nervousness, and palpitations associated with hypoglycemia. Frequent blood glucose monitoring is recommended for these patients. Their inability to detect and treat these warning signs of hypoglycemia puts them at risk for development of dangerously low blood glucose levels. Therefore, their goals for blood glucose levels may need to be adjusted to reduce the risk for hypoglycemia. Patients and families need to be taught to recognize subtle

and atypical symptoms of hypoglycemia, such as numbness around the mouth and impaired ability to concentrate.

Sudomotor Neuropathy

The neuropathic condition called sudomotor neuropathy refers to a decrease or absence of sweating (anhidrosis) of the extremities, with a compensatory increase in upper body sweating. Dryness of the feet increases the risk for the development of foot ulcers.

Sexual Dysfunction

Sexual dysfunction, especially erectile dysfunction in men, is a complication of diabetes. The effects of autonomic neuropathy on female sexual functioning are not well documented. Reduced vaginal lubrication has been mentioned as a possible neuropathic effect. Other possible changes in sexual function in women with diabetes include decreased libido and lack of orgasm. Vaginal infection, which increases in incidence in women with diabetes, may be associated with decreased lubrication and vaginal pruritus (itching) and tenderness. Urinary tract infections and vaginitis may also affect sexual function.

Impotence (inability of the penis to become rigid and sustain an erection adequate for penetration) occurs with greater frequency in men with diabetes than in other men of the same age. Some men with autonomic neuropathy have normal erectile function and can experience orgasm but do not ejaculate normally. Retrograde ejaculation occurs; seminal fluid is propelled backward through the posterior urethra and into the urinary bladder. Examination of the urine confirms the diagnosis because of the large number of active sperm present. Fertility counseling may be necessary for couples attempting conception.

Diabetic neuropathy is not the only cause of impotence in men with diabetes. Medications such as antihypertensive agents, psychological factors, and other medical conditions (eg, vascular insufficiency) that may affect other men also play a role in impotence in men with diabetes (see Chapter 49).

Management

Management strategies for autonomic neuropathy focus on alleviating symptoms and on modification and management of risk factors. The prognosis for painless cardiac ischemia is poor. However, detection is important so that education about avoiding strenuous exercise can be provided. Orthostatic hypotension may respond to a diet high in sodium, discontinuation of medications that impede autonomic nervous system responses, use of sympathomimetics and other agents (eg, caffeine) that stimulate an autonomic response, mineralocorticoid therapy, and use of lower-body elastic garments that maximize venous return and prevent pooling of blood in the extremities.

Treatment of delayed gastric emptying includes a low-fat diet, frequent small meals, frequent blood glucose monitoring, and use of agents that increase gastric motility (eg, metoclopramide [Reglan], bethanechol [Myotonachol]). Treatment of diabetic diarrhea may include bulk-forming laxatives or antidiarrheal agents. Constipation is treated with a high-fiber diet and adequate hydration; medications, laxatives, and enemas may be necessary if constipation is severe. Management of sexual dysfunction in women and men is discussed in Chapters 47 and 49, respectively.

Treatment of sudomotor dysfunction focuses on education about skin care and heat intolerance.

Foot and Leg Problems

Between 50% and 75% of lower extremity amputations are performed on people with diabetes. More than 50% of these amputations are thought to be preventable, provided patients are taught foot care measures and practice them on a daily basis (ADA, 2009b). Complications of diabetes that contribute to the increased risk of foot problems and infections include the following:

- Neuropathy: Sensory neuropathy leads to loss of pain and pressure sensation, and autonomic neuropathy leads to increased dryness and fissuring of the skin (secondary to decreased sweating). Motor neuropathy results in muscular atrophy, which may lead to changes in the shape of the foot.
- Peripheral vascular disease: Poor circulation of the lower extremities contributes to poor wound healing and the development of gangrene.
- Immunocompromise: Hyperglycemia impairs the ability of specialized leukocytes to destroy bacteria. Therefore, in poorly controlled diabetes, there is a lowered resistance to certain infections.

The typical sequence of events in the development of a diabetic foot ulcer begins with a soft tissue injury of the foot, formation of a fissure between the toes or in an area of dry skin, or formation of a callus (Fig. 41-9). Patients with an insensitive foot do not feel injuries, which may be thermal (eg, from using heating pads, walking barefoot on hot concrete, testing bath water with the foot), chemical (eg, burning the foot while using caustic agents on calluses, corns, or bunions), or traumatic (eg, injuring skin while cutting nails, walking with an undetected foreign object in the shoe, or wearing ill-fitting shoes and socks).

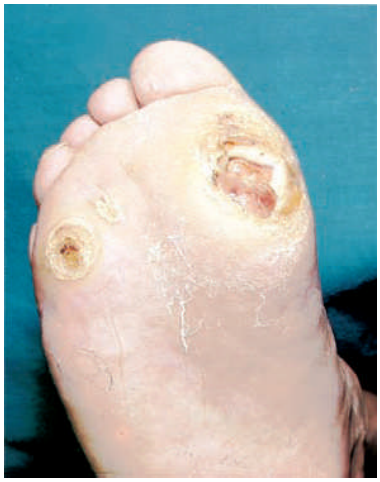


Figure 41-9 Neuropathic ulcers occur on pressure points in areas with diminished sensation in diabetic polyneuropathy. Because pain is absent, the ulcer may go unnoticed.

If the patient is not in the habit of thoroughly inspecting both feet on a daily basis, the injury or fissure may go unnoticed until a serious infection has developed. Drainage, swelling, redness of the leg (from cellulitis), or gangrene may be the first sign of foot problems that the patient notices. Treatment of foot ulcers involves bed rest, antibiotics, and débridement. In addition, controlling glucose levels, which tend to increase when infections occur, is important for promoting wound healing. When peripheral vascular disease is present, foot ulcers may not heal because of the decreased ability of oxygen, nutrients, and antibiotics to reach the injured tissue. Amputation may be necessary to prevent the spread of infection, particularly if it involves the bone (osteomyelitis).

Foot assessment and foot care instructions are most important when caring for patients who are at high risk for foot infections. Some of the high-risk characteristics include:

- Duration of diabetes more than 10 years
- Age older than 40 years
- History of smoking
- Decreased peripheral pulses
- Decreased sensation
- Anatomic deformities or pressure areas (eg, bunions, calluses, hammer toes)
- History of previous foot ulcers or amputation

Management

Teaching proper foot care is a nursing intervention that can prevent costly and painful complications that result in disability (Chart 41-10). Preventive foot care begins with careful daily assessment of the feet, which should be inspected on a daily basis for any redness, blisters, fissures, calluses, ulcerations, changes in skin temperature, or development of foot deformities (hammer toes, bunions). Visual impairment or decreased joint mobility (especially in the elderly) requires use of a mirror to inspect the bottoms of both feet or the help of a family member in foot inspection. The interior surfaces of shoes should also be inspected for any rough spots or foreign objects (NIDDK, 2008b).

In addition to the daily visual and manual inspection of the feet, the feet should be examined during every health care visit or at least once per year (more often if there is an increase in risk) by a podiatrist, physician, or nurse (ADA, 2009b). All patients should be assessed for neuropathy and undergo evaluation of neurologic status by an experienced examiner using a monofilament device (Fig. 41-10). Pressure areas, such as calluses, or thick toenails should be treated by a podiatrist in addition to routine trimming of nails.

Additional aspects of preventive foot care that are taught to patients and families include the following:

- Properly bathing, drying, and lubricating the feet, taking care not to allow moisture (water or lotion) to accumulate between the toes
- Wearing closed-toed shoes that fit well. A podiatrist can provide the patient with inserts (orthotics) to remove pressure from pressure points on the foot. New shoes should be broken in slowly (ie, worn for 1 to 2 hours initially, with gradual increases in the length of time worn) to avoid blister formation. Patients with bony deformities may need custom-made shoes with extra width or depth. High-risk behaviors, such as walking barefoot, using heating pads on the feet, wearing open-toed shoes, soaking the feet, and shaving calluses, should be avoided.
- Trimming toenails straight across and filing sharp corners to follow the contour of the toe. If the patient has

CHART 41-10 PATIENT EDUCATION
Foot Care Tips

1. Take care of your diabetes.
   - Work with your health care team to keep your blood glucose level within a normal range.
2. Inspect your feet every day.
   - Look at your bare feet every day for cuts, blisters, red spots, and swelling.
   - Use a mirror to check the bottoms of your feet or ask a family member for help if you have trouble seeing.
   - Check for changes in temperature.
3. Wash your feet every day.
   - Wash your feet in warm, not hot, water.
   - Dry your feet well. Be sure to dry between the toes.
   - Do not soak your feet.
   - Do not check water temperature with your feet; use a thermometer or elbow.
4. Keep the skin soft and smooth.
   - Rub a thin coat of skin lotion over the tops and bottoms of your feet, but not between your toes.
5. Smooth corns and calluses gently.
   - Use a pumice stone to smooth corns and calluses.
6. Trim your toenails each week or when needed.
   - Trim your toenails straight across and file the edges with an emery board or nail file.
7. Wear shoes and socks at all times.
   - Never walk barefoot.
   - Wear comfortable shoes that fit well and protect your feet.
   - Feel inside your shoes before putting them on each time to make sure the lining is smooth and there are no objects inside.
8. Protect your feet from hot and cold.
   - Wear shoes at the beach or on hot pavement.
   - Wear socks at night if your feet get cold.
9. Keep the blood flowing to your feet.
   - Put your feet up when sitting.
   - Wiggle your toes and move your ankles up and down for 5 minutes, 2 or 3 times a day.
   - Do not cross your legs for long periods of time.
   - Do not smoke.
10. Check with your health care provider.
    - Have your health care provider check your bare feet and find out whether you are likely to have serious foot problems. Remember that you may not feel the pain of an injury.
    - Call your health care provider right away if a cut, sore, blister, or bruise on your foot does not begin to heal after one day.
    - Follow your health care provider's advice about foot care.
    - Do not self-medicate or use home remedies or over-the-counter agents to treat foot problems.

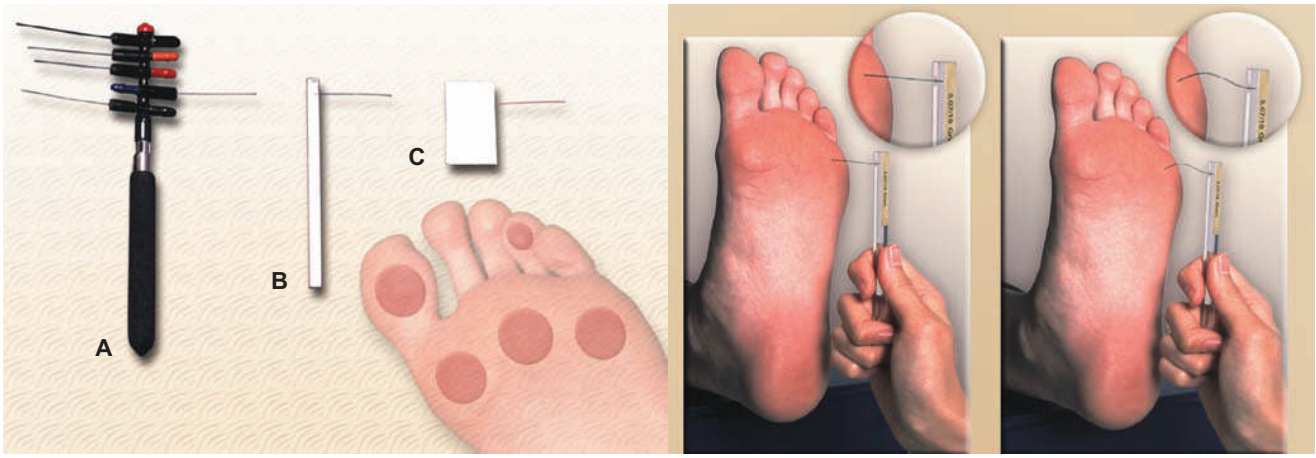


Figure 41-10 The monofilament test is used to assess the sensory threshold in patients with diabetes. The test instrument—a monofilament—is gently applied to about five pressure points on the foot (as shown in image on *left*). **A,** Example of a monofilament used for advanced quantitative assessment. **B,** Semmes-Weinstein monofilament used by clinicians. **C,** Disposable monofilament used by patients. The examiner applies the monofilament to the test area to determine whether the patient feels the device. (Adapted with permission from Cameron, B. L. (2002). Making diabetes management routine. *American Journal of Nursing, 102*(2), 26–32.)

visual deficits, is unable to reach the feet because of disability, or has thickened toenails, a podiatrist should cut the nails.
- Reducing risk factors, such as smoking and elevated blood lipids, that contribute to peripheral vascular disease.
- Avoiding home remedies, over-the-counter agents, and self-medicating to treat foot problems.

Blood glucose control is important for avoiding decreased resistance to infections and for preventing diabetic neuropathy.

SPECIAL ISSUES IN DIABETES CARE

Patients With Diabetes Who Are Undergoing Surgery

During periods of physiologic stress, such as surgery, blood glucose levels tend to increase, because levels of stress hormones (epinephrine, norepinephrine, glucagon, cortisol, and growth hormone) increase. If hyperglycemia is not controlled during surgery, the resulting osmotic diuresis may lead to excessive loss of fluids and electrolytes. Patients with type 1 diabetes also risk developing ketoacidosis during periods of stress.

Hypoglycemia is also a concern in patients with diabetes who are undergoing surgery. For example, this is a special concern during the preoperative period if surgery is delayed beyond the morning in a patient who received a morning injection of intermediate-acting insulin.

There are various approaches to managing glucose control during the perioperative period. Frequent blood glucose monitoring is essential throughout the preoperative and postoperative periods, regardless of the method used for glucose control. Examples of these approaches are described in Chart 41-11. The use of IV insulin and dextrose has become widespread with the increased availability of meters for intraoperative glucose monitoring.

During the postoperative period, patients with diabetes must also be closely monitored for cardiovascular complications because of the increased prevalence of atherosclerosis, wound infections, and skin breakdown (especially in patients with decreased sensation in the extremities due to neuropathy). Maintaining adequate nutrition and blood glucose control promote wound healing.

Management of Hospitalized Patients With Diabetes

At any one time, 10% to 20% of hospitalized general medical-surgical patients have diabetes. This number may increase as elderly patients make up an increasing proportion of the hospitalized population. Often diabetes is not the primary medical diagnosis, yet problems with control of diabetes frequently result from changes in the patient's normal routine or from surgery or illness. During the course of treatment for the primary medical diagnosis, blood glucose control may worsen. In addition, the only opportunity for some patients with diabetes to update their knowledge about diabetes self-care and prevention of complications may be during hospitalization. It is important for nurses caring for patients with diabetes to focus attention on the diabetes as well as the primary health issue. Control of blood glucose levels is important because hyperglycemia impairs resistance to certain infections and impedes wound healing.

Self-Care Issues

For patients who are actively involved in diabetes self-management (especially insulin dose adjustment), relinquishing control over meal timing, insulin timing, and insulin dosage can be particularly difficult and anxiety provoking. The patient may fear hypoglycemia and express much concern over possible delays in receiving attention from the nurse if hypoglycemic symptoms occur or may disagree with a planned dose of insulin.

Chart 41-11 • *Approaches to Management of Glucose Control During the Perioperative Period*

- Monitor blood glucose levels frequently (every 1 to 2 h).
- For patients taking insulin
  1. The morning of surgery, all subcutaneous insulin doses are withheld, unless the blood glucose level is elevated (eg, >200 mg/dL [11.1 mmol/L]), in which case a small dose of subcutaneous regular insulin may be prescribed. The blood glucose level is controlled during surgery with the IV infusion of regular insulin, which is balanced by an infusion of dextrose. The insulin and dextrose infusion rates are adjusted according to frequent (hourly) capillary glucose determinations. After surgery, the insulin infusion may be continued until the patient can eat. If IV insulin is discontinued, subcutaneous regular insulin may be administered at set intervals (every 4–6 h), or intermediate-acting insulin may be administered every 12 h with supplemental regular insulin as necessary until the patient is eating and the usual pattern of insulin dosing is resumed.
     - Carefully monitor the insulin infusion rate and blood glucose levels in a patient with diabetes who is receiving IV insulin. IV insulin has a much shorter duration of action than subcutaneous insulin. If the infusion is interrupted or discontinued, hyperglycemia will develop rapidly (within 1 h in type 1 diabetes and within a few hours in type 2 diabetes).
     - Ensure that subcutaneous insulin is administered 30 min before the IV insulin infusion is discontinued.
  2. One half to two thirds of the patient's usual morning dose of insulin (either intermediate-acting insulin alone or both short-act and intermediate-acting insulins) is administered subcutaneously in the morning before surgery. The remainder is then administered after surgery.
  3. The patient's usual daily dose of subcutaneous insulin is divided into four equal doses of regular insulin. These are then administered at 6-h intervals. The last two approaches do not provide the control achieved by IV administration of insulin and dextrose.
- Patients with type 2 diabetes who do not usually take insulin may require insulin during the perioperative period to control blood glucose elevations. Patients who are taking metformin may be instructed to discontinue the oral agent 24 to 48 h before surgery, if possible. Some of these patients may resume their usual regimen of diet and oral agent during the recovery period. Other patients (whose diabetes is probably not well controlled with diet and an oral antidiabetic agent before surgery) need to continue with insulin injections after discharge.
- For patients with type 2 diabetes who are undergoing minor surgery but who do not normally take insulin, glucose levels may remain stable provided no dextrose is infused during the surgery. After surgery, these patients may require small doses of regular insulin until the usual diet and oral agent are resumed.

It is important for the nurse to acknowledge the patient's concerns and involve the patient in the plan of care as much as possible. If the patient disagrees with certain aspects of the nursing or medical care related to diabetes, the nurse must communicate this to other members of the health care team. Nurses and other health care providers must pay particular attention to patients who are successful in managing self-care; they should assess these patients' self-care management skills and encourage them to continue if their performance is correct and effective.

Hospitalization of a patient with diabetes should be considered an opportunity to evaluate the patient's self-care skills and to reinforce teaching that might be needed. The nurse observes the patient preparing and injecting the insulin, monitoring blood glucose, and performing foot care. (Simply questioning the patient about these skills without actually observing performance of the skills is not sufficient.) The patient's knowledge about diet can be assessed with the help of a dietitian through direct questioning and review of the patient's menu choices. The patient's understanding about signs and symptoms, treatment, and prevention of hypoglycemia and hyperglycemia is assessed, along with knowledge of risk factors for macrovascular disease, including hypertension, increased lipids, and smoking. In addition, the patient is asked the date of his or her last eye examination (including dilation of the pupils). Teaching about these issues is critical.

Hyperglycemia During Hospitalization

Hyperglycemia may occur in hospitalized patients as a result of the original illness that led to the need for hospitalization. A number of other factors may contribute to hyperglycemia; examples include:

- Changes in the usual treatment regimen (eg, increased food, decreased insulin, decreased activity)
- Medications (eg, corticosteroids such as prednisone, which are used in the treatment of a variety of inflammatory disorders)
- IV dextrose, which may be part of the maintenance fluids or may be used for the administration of antibiotics and other medications, without adequate insulin therapy
- Overly vigorous treatment of hypoglycemia
- Inappropriate withholding of insulin or inappropriate use of "sliding scales"
- Mismatched timing of meals and insulin (eg, postmeal hyperglycemia may occur if short-acting insulin is administered immediately before or even after a meal)

Nursing actions to correct some of these factors are important for avoiding hyperglycemia. Assessment of the patient's usual home routine is important. The nurse should try to approximate as much as possible the home schedule of insulin, meals, and activities. Monitoring blood glucose levels has been identified by the ADA as an additional "vital sign" essential in assessment of patients (ADA, 2009b). The results of blood glucose monitoring provide information needed to obtain orders for extra doses of insulin (at times when insulin is usually taken), an important nursing function. Insulin doses must not be withheld when blood glucose levels are normal.

Short-acting insulin is usually needed to avoid postprandial hyperglycemia (even in patients with normal premeal glucose levels), and NPH insulin does not peak until many hours after the dose is given. IV antibiotics should be mixed

CHART 41-12 NURSING RESEARCH PROFILE

Hypoglycemia in Hospitalized Patients With Diabetes

Anthony, M. (2007). Treatment of hypoglycemia in hospitalized adults, A descriptive study. *The Diabetes Educator, 33*(4), 709–715.

Purpose

Diabetes mellitus is a primary or secondary reason for over 24 million days spent in the hospital. The goal of achieving normal blood glucose levels is a challenge in hospitalized patients but has the potential to improve patient outcomes. This goal may, however, result in increased risk of hypoglycemia in patients with diabetes who are hospitalized. The purpose of this study was to describe the treatment of hypoglycemia in two Midwestern hospitals to determine patterns and adherence to policies and procedures.

Design

A retrospective chart audit was conducted on 210 patients who had experienced hypoglycemia while hospitalized. The purpose of the audit was to examine adherence to five steps identified in hospital policies and procedures for treatment of hypoglycemia. The five steps were: (1) administer 15 g of carbohydrates, (2) retest blood glucose in 15 minutes, (3) retest in 1 hour, (4) notify the physician, and (5) document the event in the patient's record. A checklist of expected behaviors was derived from the practice manual and used as the evaluation tool.

Findings

A total of 484 episodes of hypoglycemia in 105 patients at two hospitals were analyzed. Hypoglycemia was defined as a blood glucose level of less than 70 mg/dL. Adherence to practice guidelines for treatment of hypoglycemia was low. There was not one case where all five steps were followed. The adherence ranged from 2.1% for the 1-hour retest to a high of 70.9% for documentation.

Nursing Implications

Nursing policy and procedures are put in place to ensure that a standard of care is followed for patient safety. The treatment of hypoglycemia is an important patient safety issue because severe hypoglycemia can threaten the patient's well-being and lead to seizures, coma, or a fall. Following guidelines provides for the treatment of the condition and a return of blood glucose levels to normal without causing hyperglycemia. Although it is possible that all five steps of the guidelines were taken but not documented, if actions are not documented it is assumed that the actions were not taken. The findings indicate that staff education is needed to increase the likelihood that important guidelines are followed to improve patient safety and prevent catastrophic outcomes.

in normal saline (if possible) to avoid excess infusion of dextrose (especially in patients who are eating). It is important to avoid overly vigorous treatment of hypoglycemia, which may lead to hyperglycemia.

Hypoglycemia During Hospitalization

Hypoglycemia in hospitalized patients is usually the result of too much insulin or delays in eating (Chart 41-12). Specific examples include:

- Overuse of "sliding scale" regular insulin, particularly as a supplement to regularly scheduled, twice-daily short-acting and intermediate-acting insulins
- Lack of change in insulin dosage when dietary intake is changed (eg, in the patient taking nothing by mouth [NPO])
- Overly vigorous treatment of hyperglycemia (eg, giving too-frequent successive doses of regular insulin before the time of peak insulin activity is reached), resulting in a cumulative effect
- Delayed meal after administration of lispro, aspart, or glulisine [Apidra] insulin (the patient should eat within 5 to 15 minutes after insulin administration)

Treatment of hypoglycemia should be based on the established hospital protocol. If the initial treatment does not increase the glucose level adequately, the same treatment may be repeated after 15 minutes. The nurse must assess the pattern of glucose values and avoid giving doses of insulin that repeatedly lead to hypoglycemia. Successive doses of subcutaneous regular insulin should be administered no more frequently than every 3 to 4 hours. For patients receiving NPH or Lente insulin before breakfast and dinner, the nurse must use caution in administering supplemental doses of regular insulin at lunch and bedtime. Hypoglycemia may occur when two insulins peak at similar times (eg, morning NPH peaks with lunchtime regular insulin and may lead to late-afternoon hypoglycemia; dinnertime NPH peaks with bedtime regular insulin and may lead to nocturnal hypoglycemia). To avoid hypoglycemic reactions caused by delayed food intake, the nurse should arrange for snacks to be given to the patient if meals are going to be delayed because of procedures, physical therapy, or other activities.

Common Alterations in Diet

Dietary modifications commonly prescribed during hospitalization require special consideration for patients who have diabetes (ADA, 2008b).

Nothing by Mouth

For patients who must be NPO in preparation for diagnostic or surgical procedures, the nurse must ensure that the usual insulin dosage has been changed. These changes may include eliminating the rapid-acting insulin and giving a decreased amount (eg, half the usual dose) of intermediate-acting NPH or Lente insulin. Another approach is to use frequent (every 3 to 4 hours) dosing of rapid-acting insulin only. IV dextrose may be administered to provide calories and to avoid hypoglycemia.

Even without food, glucose levels may increase as a result of hepatic glucose production, especially in patients with type 1 diabetes and lean patients with type 2 diabetes. Furthermore, in type 1 diabetes, elimination of the insulin dose may lead to the development of DKA. Therefore, administration of insulin to patients with type 1 diabetes who are NPO is an important nursing action.

For patients with type 2 diabetes who are taking insulin, DKA does not usually develop when insulin doses are eliminated because the patient's pancreas produces some insulin. Therefore, skipping the insulin dose altogether (when the patient is receiving IV dextrose) may be safe; however, close monitoring of blood glucose levels is essential.

For patients who are NPO for extended periods (24 hours), glucose testing and insulin administration should be performed at regular intervals, usually four times per day. Insulin regimens for the patient who is NPO for an extended period may include NPH insulin every 12 hours (with rapid-acting insulin added to the NPH, depending on the results of glucose testing) or rapid-acting insulin only every 4 to 6 hours. These patients should receive dextrose infusions to provide some calories and limit ketosis.

To prevent the problems that result from the need to withhold food, diagnostic tests and procedures and surgery should be scheduled early in the morning if possible.

Clear Liquid Diet

When the diet is advanced to include clear liquids, patients with diabetes receive more simple carbohydrate foods, such as juice and gelatin desserts, than are usually included in the diabetic diet. It is important for hospitalized patients to maintain their nutritional status as much as possible to promote healing. Therefore, the use of reduced-calorie substitutes such as diet soda or diet gelatin desserts would not be appropriate when the only source of calories is clear liquids. Simple carbohydrates, if eaten alone, cause a rapid rise in blood glucose levels; therefore, it is important to try to match peak times of insulin effect with peaks in the blood glucose concentration. If the patient receives insulin at regular intervals while NPO, the scheduled times for glucose tests and insulin injections must match meal times.

Enteral Tube Feedings

Tube feeding formulas contain more simple carbohydrates and less protein and fat than the typical meal plan for diabetes. This results in increased levels of glucose in patients with diabetes who are receiving tube feedings. It is important that insulin doses be administered at regular intervals (eg, NPH every 12 hours or regular insulin every 4 to 6 hours) when continuous tube feedings are administered. If insulin is administered at routine (prebreakfast and predinner) times, hypoglycemia during the day may result (because the patient receives more insulin without more calories); and hyperglycemia may occur during the night if feedings continue but insulin action decreases.

A common cause of hypoglycemia in patients receiving both continuous tube feedings and insulin is inadvertent or purposeful discontinuation of the feeding. The nurse must discuss with the medical team any plans for temporarily discontinuing the tube feeding (eg, when the patient is away from the unit). Planning ahead may allow for alterations to be made in the insulin dose or for administration of IV dextrose. In addition, if problems with the tube feeding develop unexpectedly (eg, the patient pulls out the tube, the tube clogs, the feeding is discontinued when residual gastric contents are found), the nurse must notify the physician, assess blood glucose levels more frequently, and administer IV dextrose if indicated.

Parenteral Nutrition

Patients receiving parenteral nutrition may receive both IV insulin (added to the parenteral nutrition container) and subcutaneous intermediate-acting or short-acting insulins. If the patient is receiving continuous parenteral nutrition, the blood glucose level should be monitored and insulin administered at regular intervals. If the parenteral nutrition is infused over a limited number of hours, subcutaneous insulin should be administered so that peak times of insulin action coincide with times of parenteral nutrition infusion.

Hygiene

Nurses caring for hospitalized patients with diabetes must focus attention on oral hygiene and skin care. Because these patients are at increased risk for periodontal disease, it is important for the nurse to assist the patient with daily dental care. The patient may also require assistance in keeping the skin clean and dry, especially in areas of contact between two skin surfaces (eg, groin, axilla, under the breasts), where chafing and fungal infections tend to occur.

Careful assessment of the skin, especially at pressure points and on the lower extremities, is important. The skin is assessed for dryness, cracks, skin breakdown, and redness. The patient is asked about symptoms of neuropathy, such as tingling and pain or numbness of the feet. Deep tendon reflexes are assessed.

As with any patient confined to bed, nursing care must emphasize the prevention of skin breakdown at pressure points. The heels are particularly susceptible to breakdown because of loss of sensation of pain and pressure associated with sensory neuropathy.

Feet should be cleaned, dried, lubricated with lotion (but not between the toes), and inspected frequently. If the patient is in the supine position, pressure on the heels can be alleviated by elevating the lower legs on a pillow, with the heels positioned over the edge of the pillow. When the patient is seated in a chair, the feet should be positioned so that pressure is not placed on the heels. If the patient has an ulcer on one foot, it is important to provide preventive care to the unaffected foot as well as special care of the affected foot.

As always, every opportunity should be taken to teach the patient about diabetes self-management, including daily oral, skin, and foot care. Female patients should also be instructed about measures for the avoidance of vaginal infections, which occur more frequently when blood glucose levels are elevated. Patients often take their cues from nurses and realize the importance of daily personal hygiene if this is emphasized during their hospitalization.

Stress

Physiologic stress, such as infections and surgery, contributes to hyperglycemia and may precipitate DKA or HHNS. Emotional stress related to hospitalization for any reason can also have a negative impact on diabetic control. An increase in stress hormones leads to an increase in glucose levels, especially if intake of food and insulin remains unchanged. In addition, during periods of emotional stress,

people with diabetes may alter their usual pattern of meals, exercise, and medication. This can contribute to hyperglycemia or even hypoglycemia (eg, in the patient taking insulin or oral antidiabetic agents who stops eating in response to stress).

People with diabetes must be made aware of the potential deterioration in diabetic control that can accompany emotional stress. They must be encouraged to follow the diabetes treatment plan as much as possible during times of stress. In addition, learning strategies for minimizing stress and coping with stress when it does occur are important aspects of diabetes education. Healthy coping is one of the seven steps to managing diabetes identified by the AADE (2007).

Gerontologic Considerations

Because people with diabetes are living longer, both type 1 and type 2 diabetes are being seen more frequently in elderly patients hospitalized for various reasons. Regardless of the type or duration of diabetes, the goals of diabetes treatment may need to be altered when caring for hospitalized elderly patients. The focus is on quality-of-life issues, such as maintaining independent functioning and promoting general well-being.

Some of the barriers to learning and self-care during hospital stays and in preparing patients for discharge include decreased vision, hearing loss, memory deficits, decreased mobility and fine motor coordination, increased tremors, depression and isolation, decreased financial resources, and limitations related to disabilities and other medical disorders. Assessing these barriers is important in planning diabetes treatment and educational activities. Presenting brief, simplified instructions with ample opportunity for practice of skills is important. The use of special devices such as a magnifier for the insulin syringe, an insulin pen, or a mirror for foot inspection is helpful. Frequent evaluation of self-care skills (insulin administration, blood glucose monitoring, foot care, diet planning) is essential, especially in patients with deteriorating vision and memory.

If appropriate, family members may be called on to assist with diabetes survival skills, and referral to community resources may be made. It is preferable to teach the patient or family members to test blood glucose at home; the choice of meter should be tailored to the patient's visual and cognitive status and dexterity.

NURSING ALERT

Careful monitoring for diabetes complications must not be neglected in elderly patients. Hypoglycemia is especially dangerous, because it may go undetected and result in falls. Dehydration is a concern in patients who have chronically elevated blood glucose levels. Assessment for long-term complications, especially eye and foot problems, is important. Avoiding blindness and amputation through early detection and treatment of retinopathy and foot ulcers may mean the difference between placement in a long-term care facility and continued independent living for the elderly person with diabetes.

Monitoring and Managing Potential Complications

Assessment for hypoglycemia and hyperglycemia involves frequent blood glucose monitoring (usually prescribed before meals and at bedtime) and monitoring for signs and symptoms of hypoglycemia or prolonged hyperglycemia (including DKA or HHNS), as described previously. Inadequate control of blood glucose levels may hinder recovery from the primary health problem. Blood glucose levels are monitored, and insulin is administered as prescribed. It is important for the nurse to ensure that prescribed insulin dosage is modified as needed to compensate for changes in the patient's schedule or eating pattern. Treatment is given for hypoglycemia (with oral glucose) or hyperglycemia (with supplemental regular insulin no more often than every 3 to 4 hours). Blood glucose records are assessed for patterns of hypoglycemia and hyperglycemia at the same time of day, and findings are reported to the physician for modification in insulin orders. In the patient with prolonged elevations in blood glucose, laboratory values and the patient's physical condition are monitored for signs and symptoms of DKA or HHNS.

Promoting Home and Community-Based Case

Teaching Patients Self-Care

Even if the patient has had diabetes for many years, it is important to assess his or her knowledge and adherence to the plan of care. A new plan of care may need to be devised using concepts mentioned earlier. The nurse also reminds the patient and family about the importance of health promotion activities and recommended health screening.

Continuing Care

A patient who is hospitalized may require referral for home care. The home care nurse can use this opportunity to assess the patient's knowledge about diabetes management and the patient's and family's ability to carry out that management. The nurse reinforces the teaching provided in the hospital, clinic, office, or diabetes education center and assesses the home care environment to determine its adequacy for self-care and safety.

CRITICAL THINKING EXERCISES

1 A 35-year-old pregnant woman with type 2 diabetes is admitted to the hospital for an elective Caesarean section. What modifications in her diabetes care are needed before, during, and after her Caesarean section? How would her care differ if she had type 1 diabetes?

EBP **2** A 25-year-old patient is newly diagnosed with type 1 diabetes. His physician has discussed intensive insulin therapy with him, although the patient indicates that he needs more information about the advantages and disadvantages of intensive therapy before he can make a decision about therapy. What is the evidence base for intensive insulin therapy and the strength of that evidence?

How would you present information to him about the advantages and disadvantages of intensive therapy?

3 You are the nurse in a diabetes education center with many patients with type 1 and type 2 diabetes. Identify strategies you would use to provide diabetes education for the following patients: (1) a patient who has managed her diabetes well for 20 years, but has recently been prescribed insulin for the first time, (2) a patient who has cerebral palsy that affects her lower extremities and one hand, (3) a patient who speaks very little English, and (4) a patient who is angry and resentful that he has developed diabetes.

4 You are providing discharge instructions for a 55-year-old executive being discharged from the hospital after carotid endarterectomy and cardiac artery bypass surgery. He indicates that although he has had diabetes for 10 years, he has only "a little diabetes" because until recently he has not required insulin. He is a smoker and travels extensively. Develop a teaching plan for this patient and identify the priorities of teaching for him.

5 A 68-year-old woman who was found at home unresponsive by her neighbors is admitted to the emergency department with possible diabetic ketoacidosis. She is well known to the emergency department personnel because of repeated episodes of hypoglycemia in the past. Compare the pathophysiology and signs and symptoms of diabetic ketoacidosis and hypoglycemia. How would assessment, medical management, and nursing care for these two disorders compare? What would be your priorities for patient teaching once she has recovered from the acute medical problem?

The Smeltzer suite offers these additional resources to enhance learning and facilitate understanding of this chapter:

- thePoint online resource, thepoint.lww.com/Smeltzer12E
- Student CD-ROM included with the book
- *Study Guide to Accompany Brunner & Suddarth's Textbook of Medical-Surgical Nursing*
- *Handbook for Brunner & Suddarth's Textbook of Medical-Surgical Nursing*

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**Asterisk indicates nursing research.*
***Double asterisk indicates classic reference.*

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the patient usually has catheters and drains in place to maintain a patent urinary tract, to remove drainage, and to permit accurate measurement of urine output. Because of the location of the surgical incision, the patient's position during surgery, and the nature of the surgical procedure, pain and muscle soreness are common. Pharmacologic management often includes immunosuppressant agents; therefore, patients are monitored for infection (Aschenbrenner, 2007).

The patient requires frequent analgesia during the postoperative period and assistance with turning, coughing, use of incentive spirometry, and deep breathing to prevent atelectasis and other pulmonary complications. The patient and family require assistance and support to cope with the diagnosis and uncertain prognosis. (See this chapter for discussion of postoperative care of the patient undergoing kidney surgery and Chapter 16 for discussion of care of the patient with cancer.)

Promoting Home and Community-Based Care

Teaching Patients Self-Care

The nurse teaches the patient to inspect and care for the incision and perform other general postoperative care including activity and lifting restrictions, driving, and pain management. Instructions are provided about when to notify the physician about problems (eg, fever, breathing difficulty, wound drainage, blood in the urine, pain or swelling of the legs).

The nurse encourages the patient to eat a healthy diet and to drink adequate liquids to avoid constipation and to maintain an adequate urine volume. Education and emotional support are provided related to the diagnosis, treatment, and continuing care because many patients are concerned about the loss of the other kidney, the possible need for dialysis, or the recurrence of cancer.

Continuing Care

Follow-up care is essential to detect signs of metastases and to reassure the patient and family about the patient's status and well-being. The patient who has had surgery for renal carcinoma should have a yearly physical examination and chest x-ray, because late metastases are not uncommon. All subsequent symptoms should be evaluated with possible metastases in mind.

If follow-up chemotherapy is necessary, the patient and family are informed about the treatment plan or chemotherapy protocol, what to expect with each visit, and when to notify the physician. Evaluation of remaining renal function (creatinine clearance, BUN and serum creatinine levels) may also be carried out periodically. A home care nurse may monitor the patient's physical status and psychological well-being and coordinate other indicated services and resources.

RENAL FAILURE

Renal failure results when the kidneys cannot remove the body's metabolic wastes or perform their regulatory functions. The substances normally eliminated in the urine accumulate in the body fluids as a result of impaired renal excretion, affecting endocrine and metabolic functions as well as fluid, electrolyte, and acid–base disturbances. Renal failure is a systemic disease and is a final common pathway of many different kidney and urinary tract diseases. Each year, the number of deaths from irreversible renal failure increases (USRDS, 2007).

Acute Renal Failure

Acute renal failure (ARF) is a rapid loss of renal function due to damage to the kidneys. Depending on the duration and severity of ARF, a wide range of potentially life-threatening metabolic complications can occur, including metabolic acidosis as well as fluid and electrolyte imbalances. Treatment is aimed at replacing renal function temporarily to minimize potentially lethal complications and reduce potential causes of increased renal injury with the goal of minimizing long-term loss of renal function. ARF is a problem seen in hospitalized patients and those in outpatient settings. A widely accepted criterion for ARF is a 50% or greater increase in serum creatinine above baseline (normal creatinine is less than 1.0 mg/dL) (Best & Counselman, 2008). Urine volume may be normal, or changes may occur. Possible changes include oliguria (less than 500 mL/day), nonoliguria (greater than 800 mL/day), or **anuria** (less than 50 mL/day) (Counts, 2008).

Pathophysiology

Although the pathogenesis of ARF and oliguria is not always known, many times there is a specific underlying problem. Some of the factors may be reversible if identified and treated promptly, before kidney function is impaired. This is true of the following conditions that reduce blood flow to the kidney and impair kidney function: (1) hypovolemia; (2) hypotension; (3) reduced cardiac output and heart failure; (4) obstruction of the kidney or lower urinary tract by tumor, blood clot, or kidney stone; and (5) bilateral obstruction of the renal arteries or veins. If these conditions are treated and corrected before the kidneys are permanently damaged, the increased BUN and creatinine levels, oliguria, and other signs may be reversed.

Although renal stones are not a common cause of ARF, some types may increase the risk for ARF. Some hereditary stone diseases (see Chapter 45), primary struvite stones, and infection-related urolithiasis associated with anatomic and functional urinary tract anomalies and spinal cord injury may cause recurrent bouts of obstruction as well as crystal-specific damage to tubular epithelial cells and interstitial renal cells.

Categories of Acute Renal Failure

The major categories of ARF are prerenal (hypoperfusion of kidney), intrarenal (actual damage to kidney tissue), and postrenal (obstruction to urine flow). Prerenal ARF, which occurs in 60% to 70% of cases, is the result of impaired blood flow that leads to hypoperfusion of the kidney and a decrease in the GFR. Intrarenal ARF is the result of actual

parenchymal damage to the glomeruli or kidney tubules. **Acute tubular necrosis (ATN)** is the most common type of intrinsic ARF. Characteristics of ATN are intratubular obstruction, tubular back leak (abnormal reabsorption of filtrate and decreased urine flow through the tubule), vasoconstriction, and changes in glomerular permeability. These processes result in a decrease of GFR, progressive azotemia, and fluid and electrolyte imbalances. CKD, diabetes, heart failure, hypertension, and cirrhosis can lead to ATN (Bednarski, Castner & Douglas, 2008). Postrenal ARF usually results from obstruction distal to the kidney. Pressure rises in the kidney tubules and eventually, the GFR decreases. Common causes of each type of ARF are summarized in Chart 44-4.

Chart 44-4 • *Causes of Acute Renal Failure*

Prerenal Failure

- Volume depletion resulting from:
  Hemorrhage
  Renal losses (diuretics, osmotic diuresis)
  Gastrointestinal losses (vomiting, diarrhea, nasogastric suction)
- Impaired cardiac efficiency resulting from:
  Myocardial infarction
  Heart failure
  Dysrhythmias
  Cardiogenic shock
- Vasodilation resulting from:
  Sepsis
  Anaphylaxis
  Antihypertensive medications or other medications that cause vasodilation

Intrarenal Failure

- Prolonged renal ischemia resulting from:
  Pigment nephropathy (associated with the breakdown of blood cells containing pigments that in turn occlude kidney structures)
  Myoglobinuria (trauma, crush injuries, burns)
  Hemoglobinuria (transfusion reaction, hemolytic anemia)
- Nephrotoxic agents such as:
  Aminoglycoside antibiotics (gentamicin, tobramycin)
  Radiopaque contrast agents
  Heavy metals (lead, mercury)
  Solvents and chemicals (ethylene glycol, carbon tetrachloride, arsenic)
  Nonsteroidal anti-inflammatory drugs (NSAIDs)
  Angiotensin-converting enzyme inhibitors (ACE inhibitors)
- Infectious processes such as:
  Acute pyelonephritis
  Acute glomerulonephritis

Postrenal Failure

- Urinary tract obstruction, including:
  Calculi (stones)
  Tumors
  Benign prostatic hyperplasia
  Strictures
  Blood clots

Phases of Acute Renal Failure

There are four phases of ARF: initiation, oliguria, diuresis, and recovery.

- The initiation period begins with the initial insult and ends when oliguria develops.
- The oliguria period is accompanied by an increase in the serum concentration of substances usually excreted by the kidneys (urea, creatinine, uric acid, organic acids, and the intracellular cations [potassium and magnesium]). The minimum amount of urine needed to rid the body of normal metabolic waste products is 400 mL. In this phase uremic symptoms first appear and life-threatening conditions such as hyperkalemia develop.

Some patients have decreased renal function with increasing nitrogen retention, yet actually excrete normal amounts of urine (2 L/day or more). This is the nonoliguric form of renal failure and occurs predominantly after exposure of the patient to nephrotoxic agents, burns, traumatic injury, and the use of halogenated anesthetic agents.

- The diuresis period is marked by a gradual increase in urine output, which signals that glomerular filtration has started to recover. Laboratory values stabilize and eventually decrease. Although the volume of urinary output may reach normal or elevated levels, renal function may still be markedly abnormal. Because uremic symptoms may still be present, the need for expert medical and nursing management continues. The patient must be observed closely for dehydration during this phase; if dehydration occurs, the uremic symptoms are likely to increase.
- The recovery period signals the improvement of renal function and may take 3 to 12 months. Laboratory values return to the patient's normal level. Although a permanent 1% to 3% reduction in the GFR is common, it is not clinically significant.

Clinical Manifestations

Almost every system of the body is affected with failure of the normal renal regulatory mechanisms. The patient may appear critically ill and lethargic. The skin and mucous membranes are dry from dehydration. Central nervous system signs and symptoms include drowsiness, headache, muscle twitching, and seizures. Table 44-2 summarizes common clinical characteristics in all three categories of ARF.

Assessment and Diagnostic Findings

Assessment of the patient with ARF includes evaluation for changes in the urine, diagnostic tests that evaluate the kidney contour, and a variety of laboratory values. See Chapter 43 for information about the normal characteristics of urine, diagnostic findings, and laboratory values in the renal system.

In ARF, urine output varies from scanty to a normal volume, hematuria may be present, and the urine has a low specific gravity (compared with a normal value of 1.010 to 1.025). One of the earliest manifestations of tubular damage is the inability to concentrate the urine (Porth & Matfin, 2009). Patients with prerenal azotemia have a decreased amount of sodium in the urine (less than 20 mEq/L) and

Table 44-2 COMPARING CLINICAL CHARACTERISTICS OF ACUTE RENAL FAILURE

Characteristics	Categories		
	Prerenal	Intrarenal	Postrenal
Etiology	Hypoperfusion	Parenchymal damage	Obstruction
Blood urea nitrogen value	Increased (out of normal 20:1 proportion to creatinine)	Increased	Increased
Creatinine	Increased	Increased	Increased
Urine output	Decreased	Varies, often decreased	Varies, may be decreased, or sudden anuria
Urine sodium	Decreased to <20 mEq/L	Increased to >40 mEq/L	Varies, often decreased to 20 mEq/L or less
Urinary sediment	Normal, few hyaline casts	Abnormal casts and debris	Usually normal
Urine osmolality	Increased to 500 mOsm	About 350 mOsm similar to serum	Varies, increased or equal to serum
Urine specific gravity	Increased	Low normal	Varies

normal urinary sediment. Patients with intrarenal azotemia usually have urinary sodium levels greater than 40 mEq/L with urinary casts and other cellular debris.

Ultrasonography is a critical component of the evaluation of patients with renal failure. A renal sonogram or a CT or MRI scan may show evidence of anatomic changes.

The BUN level increases steadily at a rate dependent on the degree of catabolism (breakdown of protein), renal perfusion, and protein intake. Serum creatinine levels are useful in monitoring kidney function and disease progression and increase with glomerular damage.

With a decline in the GFR, oliguria, and anuria, patients are at high risk for hyperkalemia. Protein catabolism results in the release of cellular potassium into the body fluids, causing severe hyperkalemia (high serum potassium levels). Hyperkalemia may lead to dysrhythmias, such as ventricular tachycardia and cardiac arrest. Sources of potassium include normal tissue catabolism, dietary intake, blood in the GI tract, or blood transfusion and other sources (eg, IV infusions, potassium penicillin, and extracellular shift in response to metabolic acidosis).

Progressive metabolic acidosis occurs in renal failure because patients cannot eliminate the daily metabolic load of acid-type substances produced by the normal metabolic processes. In addition, normal renal buffering mechanisms fail. This is reflected by a decrease in the serum CO_2-combining power and blood pH.

There may be an increase in blood phosphate concentrations; calcium levels may be low due to decreased absorption of calcium from the intestine and as a compensatory mechanism for the elevated blood phosphate levels. Anemia is another common laboratory finding in ARF, as a result of reduced erythropoietin production, uremic GI lesions, reduced RBC lifespan, and blood loss from the GI tract.

Prevention

ARF has a high mortality rate that ranges from 25% to 90%. Factors that influence mortality include increased age, comorbid conditions, and preexisting renal and vascular diseases (Dirkes & Hodge, 2007). Therefore, prevention of ARF is essential (Chart 44-5).

A careful history is obtained to identify exposure to nephrotoxic agents or environmental toxins. The kidneys are susceptible to the adverse effects of medications because the kidneys are repeatedly exposed to substances in the blood. Patients taking nephrotoxic medications (eg, aminoglycosides, gentamicin, tobramycin, colistimethate, polymyxin B, amphotericin B, vancomycin, amikacin, cyclosporine) should be monitored closely for changes in renal function. BUN and serum creatinine levels should be obtained at baseline within 24 hours after initiation of these medications and at least twice a week while the patient is receiving them.

Chart 44-5 • *Preventing Acute Renal Failure*

1. Provide adequate hydration to patients at risk for dehydration including:
   - Before, during, and after surgery
   - Patients undergoing intensive diagnostic studies requiring fluid restriction and contrast agents (eg, barium enema, intravenous pyelograms), especially elderly patients who may have marginal renal reserve
   - Patients with neoplastic disorders or disorders of metabolism (eg, gout) and those receiving chemotherapy
2. Prevent and treat shock promptly with blood and fluid replacement.
3. Monitor central venous and arterial pressures and hourly urine output of critically ill patients to detect the onset of renal failure as early as possible.
4. Treat hypotension promptly.
5. Continually assess renal function (urine output, laboratory values) when appropriate.
6. Take precautions to ensure that the appropriate blood is administered to the correct patient in order to avoid severe transfusion reactions, which can precipitate renal failure.
7. Prevent and treat infections promptly. Infections can produce progressive renal damage.
8. Pay special attention to wounds, burns, and other precursors of sepsis.
9. To prevent infections from ascending in the urinary tract, give meticulous care to patients with indwelling catheters. Remove catheters as soon as possible.
10. To prevent toxic drug effects, closely monitor dosage, duration of use, and blood levels of all medications metabolized or excreted by the kidneys.

Any agent that reduces renal blood flow (eg, long-term analgesic use) may cause renal insufficiency. Chronic use of analgesic agents, particularly nonsteroidal anti-inflammatory drugs (NSAIDs), may cause **interstitial nephritis** (inflammation within the renal tissue) and papillary necrosis. Patients with heart failure or cirrhosis with ascites are at particular risk for NSAID-induced renal failure. Increased age, preexisting renal disease, and the simultaneous administration of several nephrotoxic agents increase the risk for kidney damage.

Radiocontrast-induced nephropathy (CIN) is a major cause of hospital-acquired ARF. Patients undergo more than 1 million radiocontrast studies in the United States annually; of these approximately 150,000 will experience CIN, and at least 1% of these will require dialysis and experience a prolonged hospital stay (Barreto, 2007). This is a potentially preventable condition. Baseline levels of creatinine greater than 2 mg/dL identify patients at high risk. Limiting the patient's exposure to contrast agents and nephrotoxic medications will reduce the risk of CIN (Steward, 2007). Administration of *N*-acetylcysteine and sodium bicarbonate before and during procedures reduces risk, but prehydration with saline is considered the most effective method to prevent CIN (Barreto, 2007).

Gerontologic Considerations

About half of all patients who develop ARF during hospitalization are older than 60 years. The etiology of ARF in older adults includes prerenal causes such as dehydration, intrarenal causes such as **nephrotoxic** agents (eg, medications, contrast agents), and complications of major surgery (Steward, 2007). Suppression of thirst, enforced bed rest, lack of access to drinking water, and confusion all contribute to the older patient's failure to consume adequate fluids and may lead to dehydration, further compromising already decreased renal function.

ARF in the elderly is also often seen in the community setting. Nurses in the ambulatory setting need to be aware of the risk. All medications need to be monitored for potential side effects that could result in damage to the kidney either through reduced circulation or nephrotoxicity. Outpatient procedures that require fasting or a bowel preparation may cause dehydration and therefore require careful monitoring.

Medical Management

The kidneys have a remarkable ability to recover from insult. The objectives of treatment of ARF are to restore normal chemical balance and prevent complications until repair of renal tissue and restoration of renal function can occur. Management includes eliminating the underlying cause; maintaining fluid balance; avoiding fluid excesses; and, when indicated, providing renal replacement therapy. Prerenal azotemia is treated by optimizing renal perfusion, whereas postrenal failure is treated by relieving the obstruction. Intrarenal azotemia is treated with supportive therapy, with removal of causative agents, aggressive management of prerenal and postrenal failure, and avoidance of associated risk factors. Shock and infection, if present, are treated promptly (see Chapter 15).

Maintenance of fluid balance is based on daily body weight, serial measurements of central venous pressure, serum and urine concentrations, fluid losses, blood pressure, and the clinical status of the patient. The parenteral and oral intake and the output of urine, gastric drainage, stools, wound drainage, and perspiration are calculated and are used as the basis for fluid replacement. The insensible fluid produced through the normal metabolic processes and lost through the skin and lungs is also considered in fluid management.

Fluid excesses can be detected by the clinical findings of dyspnea, tachycardia, and distended neck veins. The patient's lungs are auscultated for moist crackles. Because pulmonary edema may be caused by excessive administration of parenteral fluids, extreme caution must be used to prevent fluid overload. The development of generalized edema is assessed by examining the presacral and pretibial areas several times daily. Mannitol (Osmitrol), furosemide (Lasix), or ethacrynic acid (Edecrin) may be prescribed to initiate diuresis.

Adequate renal blood flow in patients with prerenal causes of ARF may be restored by IV fluids or transfusions of blood products. If ARF is caused by hypovolemia secondary to hypoproteinemia, an infusion of albumin may be prescribed. Dialysis may be initiated to prevent complications of ARF, such as hyperkalemia, metabolic acidosis, pericarditis, and pulmonary edema. Dialysis corrects many biochemical abnormalities; allows for liberalization of fluid, protein, and sodium intake; diminishes bleeding tendencies; and promotes wound healing. **Hemodialysis** (a procedure that circulates the patient's blood through a dialyzer to remove waste products and excess fluid), **peritoneal dialysis** (PD) (a procedure that uses the patient's peritoneal membrane [the lining of the peritoneal cavity] as the semipermeable membrane to exchange fluid and solutes), or a variety of **continuous renal replacement therapies** (CRRTs) (methods used to replace normal kidney function by circulating the patient's blood through a hemofilter) may be performed. These and other treatment modalities for patients with renal dysfunction are discussed later in this chapter.

Pharmacologic Therapy

Hyperkalemia is the most life-threatening of the fluid and electrolyte changes that occur in patients with renal disturbances. Therefore, the patient is monitored for hyperkalemia through serial serum electrolyte levels (potassium value greater than 5.0 mEq/L [5 mmol/L]), ECG changes (tall, tented, or peaked T waves), and changes in clinical status (see Chapter 14). Other symptoms of hyperkalemia include irritability, abdominal cramping, diarrhea, paresthesia, and generalized muscle weakness. Muscle weakness may present as slurred speech, difficulty breathing, paresthesia, and paralysis. As the potassium level increases, both cardiac and other muscular function declines, making this a true medical emergency (Counts, 2008).

The elevated potassium levels may be reduced by administering cation-exchange resins (sodium polystyrene sulfonate [Kayexalate]) orally or by retention enema. Kayexalate works by exchanging sodium ions for potassium ions in the intestinal tract. Sorbitol may be administered in combination with Kayexalate to induce a diarrhea-type effect

(it induces water loss in the GI tract). If a Kayexalate retention enema is administered (the colon is the major site of potassium exchange), a rectal catheter with a balloon may be used to facilitate retention if necessary. The patient should retain the Kayexalate for 30 to 45 minutes to promote potassium removal. Afterward, a cleansing enema may be prescribed to remove remaining medication as a precaution against fecal impaction.

If the patient is hemodynamically unstable (low blood pressure, changes in mental status, dysrhythmia), IV dextrose 50%, insulin, and calcium replacement may be administered to shift potassium back into the cells. Albuterol sulfate (Ventolin HFA) by nebulizer can lower plasma potassium concentration by 0.5 to 1.5 mEq/L (Best & Counselman, 2008). The shift of potassium into the intracellular space is temporary, so arrangements for dialysis need to be made on an emergent basis.

Since many medications are eliminated through the kidneys, dosages must be reduced when a patient has ARF. Examples of commonly used agents that require adjustment are antibiotic medications (especially aminoglycosides), digoxin, ACE inhibitors, and magnesium-containing agents.

In addition, many medications have been used in patients with ARF in an attempt to improve patient outcomes. Diuretic agents are often used to control fluid volume, but they have not been shown to improve recovery from ARF (Dirkes & Hodge, 2007).

In patients with severe acidosis, the arterial blood gases and serum bicarbonate levels (CO_2-combining power) must be monitored because the patient may require sodium bicarbonate therapy or dialysis. If respiratory problems develop, appropriate ventilatory measures must be instituted. The elevated serum phosphate level may be controlled with phosphate-binding agents (eg, calcium or lanthanum carbonate) that help prevent a continuing rise in serum phosphate levels by decreasing the absorption of phosphate from the intestinal tract.

Nutritional Therapy

ARF causes severe nutritional imbalances (because nausea and vomiting contribute to inadequate dietary intake), impaired glucose use and protein synthesis, and increased tissue catabolism. The patient is weighed daily and loses 0.2 to 0.5 kg (0.5 to 1 lb) daily if the nitrogen balance is negative (ie, caloric intake falls below caloric requirements). If the patient gains or does not lose weight or develops hypertension, fluid retention should be suspected.

Nutritional support is based on the underlying cause of ARF, the catabolic response, the type and frequency of renal replacement therapy, comorbidities, and nutritional status. Replacement of dietary proteins is individualized to provide the maximum benefit and minimize uremic symptoms. Caloric requirements are met with high-carbohydrate meals, because carbohydrates have a protein-sparing effect (ie, in a high-carbohydrate diet, protein is not used for meeting energy requirements but is "spared" for growth and tissue healing). Foods and fluids containing potassium or phosphorus (eg, bananas, citrus fruits and juices, coffee) are restricted.

The oliguric phase of ARF may last 10 to 20 days and is followed by the diuretic phase, at which time urine output begins to increase, signaling that kidney function is returning. Results of blood chemistry tests are used to determine the amounts of sodium, potassium, and water needed for replacement, along with assessment for overhydration or underhydration. Following the diuretic phase, the patient is placed on a high-protein, high-calorie diet and is encouraged to resume activities gradually.

Nursing Management

The nurse has an important role in caring for the patient with ARF. The nurse monitors for complications, participates in emergency treatment of fluid and electrolyte imbalances, assesses the patient's progress and response to treatment, and provides physical and emotional support. Additionally, the nurse keeps family members informed about the patient's condition, helps them understand the treatments, and provides psychological support. Although the development of ARF may be the most serious problem, the nurse continues to provide nursing care indicated for the primary disorder (eg, burns, shock, trauma, obstruction of the urinary tract).

Monitoring Fluid and Electrolyte Balance

Because of the serious fluid and electrolyte imbalances that can occur with ARF, the nurse monitors the patient's serum electrolyte levels and physical indicators of these complications during all phases of the disorder. Hyperkalemia is the most immediate life-threatening imbalance seen in ARF. Parenteral fluids, all oral intake, and all medications are screened carefully to ensure that hidden sources of potassium are not inadvertently administered or consumed. IV solutions must be carefully selected based on the patient's fluid and electrolyte status. The patient's cardiac function and musculoskeletal status are monitored closely for signs of hyperkalemia.

The nurse monitors fluid status by paying careful attention to fluid intake (IV medications should be administered in the smallest volume possible), urine output, apparent edema, distention of the jugular veins, alterations in heart sounds and breath sounds, and increasing difficulty in breathing. Accurate daily weights, as well as I&O records, are essential. Indicators of deteriorating fluid and electrolyte status are reported immediately to the physician, and preparation is made for emergency treatment. Severe fluid and electrolyte disturbances may be treated with hemodialysis, PD, or CRRT.

Reducing Metabolic Rate

The nurse takes steps to reduce the patient's metabolic rate. Bed rest may be indicated to reduce exertion and the metabolic rate during the most acute stage of the disorder. Fever and infection, both of which increase the metabolic rate and catabolism, are prevented or treated promptly.

Promoting Pulmonary Function

Attention is given to pulmonary function, and the patient is assisted to turn, cough, and take deep breaths frequently to prevent atelectasis and respiratory tract infection. Drowsiness and lethargy may prevent the patient from moving and turning without encouragement and assistance.

Preventing Infection

Asepsis is essential with invasive lines and catheters to minimize the risk of infection and increased metabolism. An indwelling urinary catheter is avoided whenever possible due to the high risk of UTI associated with its use but may be required to provide ongoing data required to monitor fluid I&O.

Providing Skin Care

The skin may be dry or susceptible to breakdown as a result of edema; therefore, meticulous skin care is important. Additionally, excoriation and itching of the skin may result from the deposit of irritating toxins in the patient's tissues. Bathing the patient with cool water, frequent turning, and keeping the skin clean and well moisturized and the fingernails trimmed to avoid excoriation are often comforting and prevent skin breakdown.

Providing Psychosocial Support

The patient with ARF may require treatment with hemodialysis, PD, or CRRT. The length of time that these treatments are necessary varies with the cause and extent of damage to the kidneys. The patient and family need assistance, explanation, and support during this period. The purpose of the treatment is explained to the patient and family by the physician. However, high levels of anxiety and fear may necessitate repeated explanation and clarification by the nurse. The family members may initially be afraid to touch and talk to the patient during these procedures but should be encouraged and assisted to do so.

In an intensive care setting, many of the nurse's functions are devoted to the technical aspects of patient care; however, it is essential that the psychological needs and other concerns of the patient and family be addressed. Continued assessment of the patient for complications of ARF and precipitating causes is essential.

Chronic Renal Failure (End-Stage Renal Disease)

When a patient has sustained enough kidney damage to require renal replacement therapy on a permanent basis, the patient has moved into the fifth or final stage of CKD, also referred to as chronic renal failure (CRF) or ESRD.

Pathophysiology

As renal function declines, the end products of protein metabolism (normally excreted in urine) accumulate in the blood. Uremia develops and adversely affects every system in the body. The greater the buildup of waste products, the more pronounced the symptoms are.

The rate of decline in renal function and progression of ESRD is related to the underlying disorder, the urinary excretion of protein, and the presence of hypertension. The disease tends to progress more rapidly in patients who excrete significant amounts of protein or have elevated blood pressure than in those without these conditions.

Clinical Manifestations

Because virtually every body system is affected in ESRD, patients exhibit a number of signs and symptoms (Broscious & Castagnola, 2006). The severity of these signs and symptoms depends in part on the degree of renal impairment, other underlying conditions, and the patient's age. Cardiovascular disease is the predominant cause of death in patients with ESRD (Burrows & Muller, 2007). Peripheral neuropathy, a disorder of the peripheral nervous system, is present in some patients. Patients complain of severe pain and discomfort. Restless leg syndrome and burning feet can occur in the early stage of uremic peripheral neuropathy (Phillips & Ryr, 2005; Slack & Landis, 2006). The precise mechanisms for many of these systemic signs and symptoms have not been identified. However, it is generally thought that the accumulation of uremic waste products is the probable cause. Chart 44-6 summarizes the systemic signs and symptoms.

Assessment and Diagnostic Findings

Glomerular Filtration Rate

As the GFR decreases (due to nonfunctioning glomeruli), the creatinine clearance decreases, while the serum creatinine and BUN levels increase. Serum creatinine is a more sensitive indicator of renal function than BUN. The BUN is affected not only by renal disease but also by protein intake in the diet, catabolism (tissue and RBC breakdown), parenteral nutrition, and medications such as corticosteroids.

Sodium and Water Retention

The kidney cannot concentrate or dilute the urine normally in ESRD. Appropriate responses by the kidney to changes in the daily intake of water and electrolytes, therefore, do not occur. Some patients retain sodium and water, increasing the risk for edema, heart failure, and hypertension. Hypertension may also result from activation of the renin–angiotensin–aldosterone axis and the concomitant increased aldosterone secretion. Other patients have a tendency to lose sodium and run the risk of developing hypotension and hypovolemia. Vomiting and diarrhea may cause sodium and water depletion, which worsens the uremic state.

Acidosis

Metabolic acidosis occurs in ESRD because the kidneys are unable to excrete increased loads of acid. Decreased acid secretion results from the inability of the kidney tubules to excrete ammonia (NH_3^-) and to reabsorb sodium bicarbonate (HCO_3^-). There is also decreased excretion of phosphates and other organic acids.

Anemia

Anemia develops as a result of inadequate erythropoietin production, the shortened lifespan of RBCs, nutritional deficiencies, and the patient's tendency to bleed, particularly from the GI tract. Erythropoietin, a substance normally produced by the kidneys, stimulates bone marrow to produce RBCs (Brattich, 2007). In ESRD, erythropoietin production decreases and profound anemia results, producing fatigue, angina, and shortness of breath.

Chart 44-6 • *Assessing for End-stage Renal Disease*

Be alert for the following signs and symptoms:

Neurologic

- Weakness and fatigue
- Confusion
- Inability to concentrate
- Disorientation
- Tremors
- Seizures
- Asterixis
- Restlessness of legs
- Burning of soles of feet
- Behavior changes

Integumentary

- Gray-bronze skin color
- Dry, flaky skin
- Pruritus
- Ecchymosis
- Purpura
- Thin, brittle nails
- Coarse, thinning hair

Cardiovascular

- Hypertension
- Pitting edema (feet, hands, sacrum)
- Periorbital edema
- Pericardial friction rub
- Engorged neck veins
- Pericarditis
- Pericardial effusion
- Pericardial tamponade
- Hyperkalemia
- Hyperlipidemia

Pulmonary

- Crackles
- Thick, tenacious sputum
- Depressed cough reflex
- Pleuritic pain
- Shortness of breath
- Tachypnea
- Kussmaul-type respirations
- Uremic pneumonitis

Gastrointestinal

- Ammonia odor to breath ("uremic fetor")
- Metallic taste
- Mouth ulcerations and bleeding
- Anorexia, nausea, and vomiting
- Hiccups
- Constipation or diarrhea
- Bleeding from gastrointestinal tract

Hematologic

- Anemia
- Thrombocytopenia

Reproductive

- Amenorrhea
- Testicular atrophy
- Infertility
- Decreased libido

Musculoskeletal

- Muscle cramps
- Loss of muscle strength
- Renal osteodystrophy
- Bone pain
- Bone fractures
- Foot drop

Calcium and Phosphorus Imbalance

Another abnormality seen in ESRD is a disorder in calcium and phosphorus metabolism (McCarley & Arjomand, 2008). Serum calcium and phosphate levels have a reciprocal relationship in the body: As one increases, the other decreases. With a decrease in filtration through the glomerulus of the kidney, there is an increase in the serum phosphate level and a reciprocal or corresponding decrease in the serum calcium level. The decreased serum calcium level causes increased secretion of parathormone from the parathyroid glands. However, in renal failure, the body does not respond normally to the increased secretion of parathormone; as a result, calcium leaves the bone, often producing bone changes and bone disease as well as calcification of major blood vessels in the body. In addition, the active metabolite of vitamin D (1,25-dihydroxycholecalciferol) normally manufactured by the kidney decreases as renal failure progresses (Gesek & Desmond, 2008). Uremic bone disease, often called renal osteodystrophy, develops from the complex changes in calcium, phosphate, and parathormone balance. There is also evidence of calcification of blood vessels.

Complications

Potential complications of chronic renal failure that concern the nurse and necessitate a collaborative approach to care include the following:

- Hyperkalemia due to decreased excretion, metabolic acidosis, catabolism, and excessive intake (diet, medications, fluids)
- Pericarditis, pericardial effusion, and pericardial tamponade due to retention of uremic waste products and inadequate dialysis
- Hypertension due to sodium and water retention and malfunction of the renin–angiotensin–aldosterone system
- Anemia due to decreased erythropoietin production, decreased RBC lifespan, bleeding in the GI tract from irritating toxins and ulcer formation, and blood loss during hemodialysis
- Bone disease and metastatic and vascular calcifications due to retention of phosphorus, low serum calcium levels, abnormal vitamin D metabolism, and elevated aluminum levels

Medical Management

The goal of management is to maintain kidney function and homeostasis for as long as possible. All factors that contribute to ESRD and all factors that are reversible (eg, obstruction) are identified and treated. Management is accomplished primarily with medications and diet therapy, although dialysis may also be needed to decrease the level of uremic waste products in the blood and to control electrolyte balance.

Pharmacologic Therapy

Complications can be prevented or delayed by administering prescribed phosphate-binding agents, calcium supplements, antihypertensive and cardiac medications, antiseizure medications, and erythropoietin (Epogen).

Calcium and Phosphorus Binders

Hyperphosphatemia and hypocalcemia are treated with medications that bind dietary phosphorus in the GI tract. Binders such as calcium carbonate (Os-Cal) or calcium acetate (PhosLo) are prescribed, but there is a risk of hypercalcemia. If calcium is high or the calcium–phosphorus product exceeds 55 mg/dL, a polymeric phosphate binder such as sevelamer hydrochloride (Renagel) may be prescribed (Zonderman & Doyle, 2006). These medications bind dietary phosphorus in the intestinal tract. All binding agents must be administered with food to be effective. Magnesium-based antacids are avoided to prevent magnesium toxicity.

Antihypertensive and Cardiovascular Agents

Hypertension is managed by intravascular volume control and a variety of antihypertensive agents. Heart failure and pulmonary edema may also require treatment with fluid restriction, low-sodium diets, diuretic agents, inotropic agents such as digoxin (Lanoxin) or dobutamine (Dobutrex), and dialysis. The metabolic acidosis of ESRD usually produces no symptoms and requires no treatment; however, sodium bicarbonate supplements or dialysis may be needed to correct the acidosis if it causes symptoms (Molzahn & Butera, 2006).

Antiseizure Agents

Neurologic abnormalities may occur, so the patient must be observed for early evidence of slight twitching, headache, delirium, or seizure activity. If seizures occur, the onset of the seizure is recorded along with the type, duration, and general effect on the patient. The physician is notified immediately. IV diazepam (Valium) or phenytoin (Dilantin) is usually administered to control seizures. The side rails of the bed should be raised and padded to protect the patient. The nursing management of the patient with seizures is discussed in Chapter 61.

Erythropoietin

Anemia associated with ESRD is treated with recombinant human erythropoietin (Epogen). Patients with anemia (hematocrit less than 30%) present with nonspecific symptoms, such as malaise, general fatigability, and decreased activity tolerance. Erythropoietin therapy is initiated to achieve a hematocrit of 33% to 38% and a target hemoglobin of 12 g/dL, which generally alleviates the symptoms of anemia (Brattich, 2007).

Erythropoietin is administered intravenously or subcutaneously three times a week in ESRD. It may take 2 to 6 weeks for the hematocrit to increase; therefore, the medication is not indicated for patients who need immediate correction of severe anemia. Adverse effects seen with erythropoietin therapy include hypertension (especially during early stages of treatment), increased clotting of vascular access sites, seizures, and depletion of body iron stores (Zonderman & Doyle, 2006).

Management involves adjustment of heparin to prevent clotting of the lines during hemodialysis treatments, frequent monitoring of hemoglobin and hematocrit, and periodic assessment of serum iron and transferrin levels. Because adequate stores of iron are necessary for an adequate response to erythropoietin, supplementary iron may be prescribed. Common iron supplements include iron sucrose (Venofer) and ferric gluconate (Ferrlecit). In addition, the patient's blood pressure and serum potassium level are monitored to detect hypertension and increasing serum potassium levels, which may occur with therapy and the increasing RBC mass. The occurrence of hypertension requires initiation or adjustment of the patient's antihypertensive therapy. Hypertension that cannot be controlled is a contraindication to recombinant erythropoietin therapy.

Patients who have received erythropoietin therapy have reported decreased levels of fatigue, increased feelings of well-being, better tolerance of dialysis, higher energy levels, and improved exercise tolerance. Additionally, this therapy has decreased the need for transfusion and its associated risks, including bloodborne infectious disease, antibody formation, and iron overload.

Nutritional Therapy

Dietary intervention is necessary with deterioration of renal function and includes careful regulation of protein intake, fluid intake to balance fluid losses, sodium intake to balance sodium losses, and some restriction of potassium. At the same time, adequate caloric intake and vitamin supplementation must be ensured. Protein is restricted because urea, uric acid, and organic acids—the breakdown products of dietary and tissue proteins—accumulate rapidly in the blood when there is impaired renal clearance. The allowed protein must be of high biologic value (dairy products, eggs, meats). High-biologic-value proteins are those that are complete proteins and supply the essential amino acids necessary for growth and cell repair.

Usually, the fluid allowance per day is 500 mL to 600 mL more than the previous day's 24-hour urine output. Calories are supplied by carbohydrates and fat to prevent wasting. Vitamin supplementation is necessary because a protein-restricted diet does not provide the necessary complement of vitamins. Additionally, the patient on dialysis may lose water-soluble vitamins during the dialysis treatment.

Hyperkalemia is usually prevented by ensuring adequate dialysis treatments with potassium removal and careful monitoring of diet, medications, and fluids for their potassium content. Sodium polystyrene sulfonate (Kayexalate), a cation-exchange resin, may be needed for acute hyperkalemia.

Dialysis

The patient with increasing symptoms of renal failure is referred to a dialysis and transplantation center early in the course of progressive renal disease. Dialysis is usually initiated when the patient cannot maintain a reasonable lifestyle with conservative treatment.

Nursing Management

The patient with ESRD requires astute nursing care to avoid the complications of reduced renal function and the stresses and anxieties of dealing with a life-threatening illness.

Nursing care is directed toward assessing fluid status and identifying potential sources of imbalance, implementing a dietary program to ensure proper nutritional intake within the limits of the treatment regimen, and promoting positive feelings by encouraging increased self-care and greater independence. It is extremely important to provide explanations and information to the patient and family concerning ESRD, treatment options, and potential complications. A great deal of emotional support is needed by the patient and family because of the numerous changes experienced. Specific interventions, along with rationale and evaluation criteria, are presented in more detail in the plan of nursing care for the patient with chronic renal failure (Chart 44-7).

Promoting Home and Community-Based Care

Teaching Patients Self-Care

The nurse plays an important role in teaching the patient with ESRD. Because of the extensive teaching needed, the home care nurse, dialysis nurse, and nurses in the hospital and outpatient settings all provide ongoing education and reinforcement while monitoring the patient's progress and compliance with the treatment regimen.

A referral to a nutritionist is made because of the dietary changes required. The patient is taught how to check the vascular access device for patency and appropriate precautions, such as avoiding venipuncture and blood pressure measurements on the arm with the access device.

Additionally, the patient and family need to know what problems to report to the health care provider. These include the following:

- Worsening signs and symptoms of renal failure (nausea, vomiting, change in usual urine output [if any], ammonia odor on breath)
- Signs and symptoms of hyperkalemia (muscle weakness, diarrhea, abdominal cramps)
- Signs and symptoms of access problems (clotted fistula or graft, infection)

These signs and symptoms of decreasing renal function, in addition to increasing BUN and serum creatinine levels, may indicate a need to alter the dialysis prescription. The dialysis nurses also provide ongoing education and support at each treatment visit.

Continuing Care

The importance of follow-up examinations and treatment is stressed to the patient and family because of changing physical status, renal function, and dialysis requirements. Referral for home care provides the home care nurse with the opportunity to assess the patient's environment and emotional status and the coping strategies used by the patient and family to deal with the changes in family roles often associated with chronic illness.

The home care nurse also assesses the patient for further deterioration of renal function and signs and symptoms of complications resulting from the primary renal disorder, the resulting renal failure, and effects of treatment strategies (eg, dialysis, medications, dietary restrictions). Patients need education and reinforcement of the dietary restrictions required, including fluid, sodium, potassium, and protein restriction. Reminders about the need for health promotion activities and health screening are an important part of nursing care for the patient with renal failure.

Gerontologic Considerations

Diabetes, hypertension, chronic glomerulonephritis, interstitial nephritis, and urinary tract obstruction are the causes of ESRD in the elderly. The signs and symptoms of renal disease in the elderly are often nonspecific. The occurrence of symptoms of other disorders (heart failure, dementia) can mask the symptoms of renal disease and delay or prevent diagnosis and treatment. Patients often develop signs and symptoms of nephrotic syndrome, such as edema and proteinuria.

Hemodialysis and PD are used effectively in treating elderly patients. The number of elderly patients initiating dialysis has dramatically increased in the past decade (Kurella, Covinsky, Collins, et al., 2007). Although there is no specific age limitation for renal transplantation, concomitant disorders (eg, coronary artery disease, peripheral vascular disease) have made it a less common treatment for the elderly. However, the outcome is comparable to that of younger patients. Some elderly patients elect not to undergo dialysis or transplantation. Conservative management, including nutritional therapy, fluid control, and medications such as phosphate binders, may be considered in patients who are not suitable for or elect not to have dialysis or transplantation.

RENAL REPLACEMENT THERAPIES

The use of renal replacement therapies becomes necessary when the kidneys can no longer remove wastes, maintain electrolytes, and regulate fluid balance. This can occur rapidly or over a long period of time and the need for replacement therapy can be acute (short term) or chronic (long term). The main renal replacement therapies include the various types of dialysis and kidney transplantation.

Dialysis

Types of dialysis include hemodialysis, CRRT, and PD. Acute dialysis is indicated when there is a high and increasing level of serum potassium, fluid overload, or impending pulmonary edema, increasing acidosis, pericarditis, and severe confusion. It may also be used to remove medications or toxins (poisoning or medication overdose) from

CHART 44-7

PLAN OF NURSING CARE
The Patient With Chronic Renal Failure

NURSING DIAGNOSIS: Excess fluid volume related to decreased urine output, dietary excesses, and retention of sodium and water
GOAL: Maintenance of ideal body weight without excess fluid

Nursing Interventions	Rationale	Expected Outcomes
1. Assess fluid status: a. Daily weight b. Intake and output balance c. Skin turgor and presence of edema d. Distention of neck veins e. Blood pressure, pulse rate, and rhythm f. Respiratory rate and effort	1. Assessment provides baseline and ongoing database for monitoring changes and evaluating interventions.	• Demonstrates no rapid weight changes • Maintains dietary and fluid restrictions • Exhibits normal skin turgor without edema • Exhibits normal vital signs • Exhibits no neck vein distention • Reports no difficulty breathing or shortness of breath
2. Limit fluid intake to prescribed volume.	2. Fluid restriction will be determined on basis of weight, urine output, and response to therapy.	• Performs oral hygiene frequently • Reports decreased thirst • Reports decreased dryness of oral mucous membranes
3. Identify potential sources of fluid: a. Medications and fluids used to take or administer medications: oral and intravenous b. Foods	3. Unrecognized sources of excess fluids may be identified.	
4. Explain to patient and family rationale for fluid restriction.	4. Understanding promotes patient and family cooperation with fluid restriction.	
5. Assist patient to cope with the discomforts resulting from fluid restriction.	5. Increasing patient comfort promotes compliance with dietary restrictions.	
6. Provide or encourage frequent oral hygiene.	6. Oral hygiene minimizes dryness of oral mucous membranes.	

NURSING DIAGNOSIS: Imbalanced nutrition: less than body requirements related to anorexia, nausea, vomiting, dietary restrictions, and altered oral mucous membranes
GOAL: Maintenance of adequate nutritional intake

Nursing Interventions	Rationale	Expected Outcomes
1. Assess nutritional status: a. Weight changes b. Laboratory values (serum electrolyte, BUN, creatinine, protein, transferrin, and iron levels)	1. Baseline data allow for monitoring of changes and evaluating effectiveness of interventions.	• Consumes protein of high biologic value • Chooses foods within dietary restrictions that are appealing • Consumes high-calorie foods within dietary restrictions
2. Assess patient's nutritional dietary patterns: a. Diet history b. Food preferences c. Calorie counts	2. Past and present dietary patterns are considered in planning meals.	• Explains in own words rationale for dietary restrictions and relationship to urea and creatinine levels • Takes medications on schedule that does not produce anorexia or feeling of fullness
3. Assess for factors contributing to altered nutritional intake: a. Anorexia, nausea, or vomiting b. Diet unpalatable to patient c. Depression d. Lack of understanding of dietary restrictions e. Stomatitis	3. Information about other factors that may be altered or eliminated to promote adequate dietary intake is provided.	• Consults written lists of acceptable foods • Reports increased appetite at meals • Exhibits no rapid increases or decreases in weight • Demonstrates normal skin turgor without edema; wound healing and acceptable plasma albumin levels
4. Provide patient's food preferences within dietary restrictions.	4. Increased dietary intake is encouraged.	
5. Promote intake of high-biologic-value protein foods: eggs, dairy products, meats.	5. Complete proteins are provided for positive nitrogen balance needed for growth and healing.	

Continued on following page

CHART 44-7 PLAN OF NURSING CARE

The Patient With Chronic Renal Failure (Continued)

Nursing Interventions	Rationale	Expected Outcomes
6. Encourage high-calorie, low-protein, low-sodium, and low-potassium snacks between meals.	6. Reduces source of restricted foods and proteins and provides calories for energy, sparing protein for tissue growth and healing.	
7. Alter schedule of medications so that they are not given immediately before meals.	7. Ingestion of medications just before meals may produce anorexia and feeling of fullness.	
8. Explain rationale for dietary restrictions and relationship to kidney disease and increased urea and creatinine levels.	8. Promotes patient understanding of relationships between diet and urea and creatinine levels to renal disease	
9. Provide written lists of foods allowed and suggestions for improving their taste without use of sodium or potassium.	9. Lists provide a positive approach to dietary restrictions and a reference for patient and family to use when at home.	
10. Provide pleasant surroundings at meal-times.	10. Unpleasant factors that contribute to patient's anorexia are eliminated.	
11. Weigh patient daily.	11. Allows monitoring of fluid and nutritional status	
12. Assess for evidence of inadequate protein intake: a. Edema formation b. Delayed wound healing c. Decreased serum albumin levels	12. Inadequate protein intake can lead to decreased albumin and other proteins, edema formation, and delay in wound healing.	

NURSING DIAGNOSIS: Deficient knowledge regarding condition and treatment
GOAL: Increased knowledge about condition and related treatment

Nursing Interventions	Rationale	Expected Outcomes
1. Assess understanding of cause of renal failure, consequences of renal failure, and its treatment: a. Cause of patient's renal failure b. Meaning of renal failure c. Understanding of renal function d. Relationship of fluid and dietary restrictions to renal failure e. Rationale for treatment (hemodialysis, peritoneal dialysis, transplantation)	1. Provides baseline for further explanations and teaching	• Verbalizes relationship of cause of renal failure to consequences • Explains fluid and dietary restrictions as they relate to failure of kidney's regulatory functions • States in own words relationship of renal failure and need for treatment • Asks questions about treatment options, indicating readiness to learn • Verbalizes plans to continue as normal a life as possible • Uses written information and instructions to clarify questions and seek additional information
2. Provide explanation of renal function and consequences of renal failure at patient's level of understanding and guided by patient's readiness to learn.	2. Patient can learn about renal failure and treatment as he or she becomes ready to understand and accept the diagnosis and consequences.	
3. Assist patient to identify ways to incorporate changes related to illness and its treatment into lifestyle.	3. Patient can see that his or her life does not have to revolve around the disease.	
4. Provide oral and written information as appropriate about: a. Renal function and failure b. Fluid and dietary restrictions c. Medications d. Reportable problems, signs, and symptoms e. Follow-up schedule f. Community resources g. Treatment options	4. Provides patient with information that can be used for further clarification at home	

Continued

CHART 44-7

PLAN OF NURSING CARE
The Patient With Chronic Renal Failure (Continued)

NURSING DIAGNOSIS: Activity intolerance related to fatigue, anemia, retention of waste products, and dialysis procedure
GOAL: Participation in activity within tolerance

Nursing Interventions	Rationale	Expected Outcomes
1. Assess factors contributing to activity intolerance: a. Fatigue b. Anemia c. Fluid and electrolyte imbalances d. Retention of waste products e. Depression	1. Indicates factors contributing to severity of fatigue	• Participates in increasing levels of activity and exercise • Reports increased sense of well-being • Alternates rest and activity • Participates in selected self-care activities
2. Promote independence in self-care activities as tolerated; assist if fatigued.	2. Promotes improved self-esteem	
3. Encourage alternating activity with rest.	3. Promotes activity and exercise within limits and adequate rest	
4. Encourage patient to rest after dialysis treatments.	4. Adequate rest is encouraged after dialysis treatments, which are exhausting to many patients.	

NURSING DIAGNOSIS: Risk for situational low self-esteem related to dependency, role changes, change in body image, and change in sexual function
GOAL: Improved self-esteem

Nursing Interventions	Rationale	Expected Outcomes
1. Assess patient's and family's responses and reactions to illness and treatment.	1. Provides data about problems encountered by patient and family in coping with changes in life	• Identifies previously used coping styles that have been effective and those no longer possible due to disease and treatment (alcohol or drug use; extreme physical exertion)
2. Assess relationship of patient and significant family members.	2. Identifies strengths and supports of patient and family	
3. Assess usual coping patterns of patient and family members.	3. Coping patterns that may have been effective in past may be harmful in view of restrictions imposed by disease and treatment.	• Patient and family identify and verbalize feelings and reactions to disease and necessary changes in their lives
4. Encourage open discussion of concerns about changes produced by disease and treatment: a. Role changes b. Changes in lifestyle c. Changes in occupation d. Sexual changes e. Dependence on health care team	4. Encourages patient to identify concerns and steps necessary to deal with them	• Seeks professional counseling, if necessary, to cope with changes resulting from renal failure • Reports satisfaction with method of sexual expression
5. Explore alternate ways of sexual expression other than sexual intercourse.	5. Alternative forms of sexual expression may be acceptable.	
6. Discuss role of giving and receiving love, warmth, and affection.	6. Sexuality means different things to different people, depending on stage of maturity.	

COLLABORATIVE PROBLEMS: Hyperkalemia; pericarditis, pericardial effusion, and pericardial tamponade; hypertension; anemia; bone disease and metastatic calcifications
GOAL: Absence of complications

Nursing Interventions	Rationale	Expected Outcomes
Hyperkalemia		
1. Monitor serum potassium levels. Notify physician if level greater than 5.5 mEq/L, and prepare to treat hyperkalemia.	1. Hyperkalemia causes potentially life-threatening changes in the body.	• Patient has normal potassium level • Experiences no muscle weakness or diarrhea • Exhibits normal ECG pattern
2. Assess patient for muscle weakness, diarrhea, ECG changes (tall-tented T waves and widened QRS).	2. Cardiovascular signs and symptoms are characteristic of hyperkalemia.	• Vital signs are within normal limits

Continued on following page

CHART 44-7 PLAN OF NURSING CARE

The Patient With Chronic Renal Failure (Continued)

Nursing Interventions	Rationale	Expected Outcomes
Pericarditis, Pericardial Effusion, and Pericardial Tamponade		
1. Assess patient for fever, chest pain, and a pericardial friction rub (signs of pericarditis) and, if present, notify physician.	1. About 30%–50% of patients with chronic renal failure develop pericarditis due to uremia; fever, chest pain, and a pericardial friction rub are classic signs.	• Has strong and equal peripheral pulses • Absence of a paradoxical pulse • Absence of pericardial effusion or tamponade on cardiac ultrasound • Patient has normal heart sounds
2. If patient has pericarditis, assess for the following every 4 hours: a. Paradoxical pulse >10 mm Hg b. Extreme hypotension c. Weak or absent peripheral pulses d. Altered level of consciousness e. Bulging neck veins	2. Pericardial effusion is a common fatal sequela of pericarditis. Signs of an effusion include a paradoxical pulse (>10 mm Hg drop in blood pressure during inspiration) and signs of shock due to compression of the heart by a large effusion. Cardiac tamponade exists when the patient is severely compromised hemodynamically.	
3. Prepare patient for cardiac ultrasound to aid in diagnosis of pericardial effusion and cardiac tamponade.	3. Cardiac ultrasound is useful in visualizing pericardial effusions and cardiac tamponade.	
4. If cardiac tamponade develops, prepare patient for emergency pericardiocentesis.	4. Cardiac tamponade is a life-threatening condition, with a high mortality rate. Immediate aspiration of fluid from the pericardial space is essential.	
Hypertension		
1. Monitor and record blood pressure as indicated.	1. Provides objective data for monitoring. Elevated levels may indicate nonadherence to the treatment regimen.	• Blood pressure within normal limits • Reports no headaches, visual problems, or seizures • Edema is absent • Demonstrates compliance with dietary and fluid restrictions
2. Administer antihypertensive medications as prescribed.	2. Antihypertensive medications play a key role in treatment of hypertension associated with chronic renal failure.	
3. Encourage compliance with dietary and fluid restriction therapy.	3. Adherence to diet and fluid restrictions and dialysis schedule prevents excess fluid and sodium accumulation.	
4. Teach patient to report signs of fluid overload, vision changes, headaches, edema, or seizures.	4. These are indications of inadequate control of hypertension and the need to alter therapy.	
Anemia		
1. Monitor RBC count, hemoglobin, and hematocrit levels as indicated.	1. Provides assessment of degree of anemia	• Patient has a normal skin color without pallor • Exhibits hematology values within acceptable limits • Experiences no bleeding from any site
2. Administer medications as prescribed, including iron and folic acid supplements, Epogen, and multivitamins.	2. RBCs need iron, folic acid, and vitamins to be produced. Epogen stimulates the bone marrow to produce RBC.	
3. Avoid drawing unnecessary blood specimens.	3. Anemia is worsened by drawing numerous specimens.	
4. Teach patient to prevent bleeding: avoid vigorous nose blowing and contact sports, and use a soft toothbrush.	4. Bleeding from anywhere in the body worsens anemia.	
5. Administer blood component therapy as indicated.	5. Blood component therapy may be needed if the patient has symptoms.	

Continued

CHART 44-7 PLAN OF NURSING CARE
The Patient With Chronic Renal Failure (Continued)

Nursing Interventions	Rationale	Expected Outcomes
Bone Disease and Metastatic Calcifications		
1. Administer the following medications as prescribed: phosphate binders, calcium supplements, vitamin D supplements.	1. Chronic renal failure causes numerous physiologic changes affecting calcium, phosphorus, and vitamin D metabolism.	• Exhibits serum calcium, phosphorus, and aluminum levels within acceptable ranges
2. Monitor serum lab values as indicated (calcium, phosphorus, aluminum levels) and report abnormal findings to physician.	2. Hyperphosphatemia, hypocalcemia, and excess aluminum accumulation are common in chronic renal failure.	• Exhibits no symptoms of hypocalcemia • Has no bone demineralization on bone scan
3. Assist patient with an exercise program.	3. Bone demineralization increases with immobility.	• Discusses importance of maintaining activity level and exercise program

the blood or for edema that does not respond to other treatment, hepatic coma, hyperkalemia, hypercalcemia, hypertension, and uremia (Mosenkis, Kirk & Berns, 2006).

Chronic or maintenance dialysis is indicated in advanced CKD and ESRD in the following instances: the presence of uremic signs and symptoms affecting all body systems (nausea and vomiting, severe anorexia, increasing lethargy, mental confusion), hyperkalemia, fluid overload not responsive to diuretics and fluid restriction, and a general lack of well-being. An urgent indication for dialysis in patients with renal failure is pericardial friction rub.

The decision to initiate dialysis should be reached only after thoughtful discussion among the patient, family, physician, and others as appropriate. Many potentially life-threatening issues are associated with the need for dialysis. The nurse can assist the patient and family by answering their questions, clarifying the information provided, and supporting their decision.

Successful kidney transplantation eliminates the need for dialysis. Not only is the quality of life much improved in patients with ESRD who undergo transplantation, but physiologic function is improved as well. Patients who undergo renal transplantation from living donors before dialysis is initiated generally have longer survival of the transplanted kidney than patients who receive transplantation after dialysis treatment is initiated.

HEMODIALYSIS

Hemodialysis is used for patients who are acutely ill and require short-term dialysis (days to weeks) and for patients with advanced CKD and ESRD who require long-term or permanent renal replacement therapy. Hemodialysis prevents death but does not cure renal disease and does not compensate for the loss of endocrine or metabolic activities of the kidneys. More than 90% of patients requiring long-term renal replacement therapy are on chronic hemodialysis (USRDS, 2007). Most patients receive intermittent hemodialysis that involves treatments three times a week with the average treatment duration of 3 to 4 hours in an outpatient setting. Hemodialysis can also be performed at home by the patient and a caregiver. With home dialysis, treatment time and frequency can be adjusted to meet optimal patient needs.

The objectives of hemodialysis are to extract toxic nitrogenous substances from the blood and to remove excess water. A **dialyzer** (also referred to as an artificial kidney) serves as a synthetic semipermeable membrane, replacing the renal glomeruli and tubules as the filter for the impaired kidneys. In hemodialysis, the blood, laden with toxins and nitrogenous wastes, is diverted from the patient to a machine, a dialyzer, where toxins are filtered out and removed and the blood is returned to the patient.

Diffusion, osmosis, and ultrafiltration are the principles on which hemodialysis is based (see Chapter 14). The toxins and wastes in the blood are removed by **diffusion**—that is, they move from an area of higher concentration in the blood to an area of lower concentration in the **dialysate.** The dialysate is a solution made up of all the important electrolytes in their ideal extracellular concentrations. The electrolyte level in the patient's blood can be brought under control by properly adjusting the dialysate bath. The semipermeable membrane impedes the diffusion of large molecules, such as RBCs and proteins.

Excess water is removed from the blood by **osmosis,** in which water moves from an area of low concentration potential (the blood) to an area of high concentration potential (the dialysate bath). In **ultrafiltration,** water moves under high pressure to an area of lower pressure. This process is much more efficient than osmosis at water removal and is accomplished by applying negative pressure or a suctioning force to the dialysis membrane. Because patients with renal disease usually cannot excrete water, this force is necessary to remove fluid to achieve fluid balance.

The body's buffer system is maintained using a dialysate bath made up of bicarbonate (most common) or acetate, which is metabolized to form bicarbonate. The anticoagulant heparin is administered to keep blood from clotting in the dialysis circuit. Cleansed blood is returned to the body. By the end of the dialysis treatment, many waste products have been removed, the electrolyte balance has been restored to normal, and the buffer system has been replenished.

Dialyzers

Dialyzers are hollow-fiber devices containing thousands of tiny strawlike tubes that carry the blood through the dialyzer. The tubes are porous and act as a semipermeable membrane

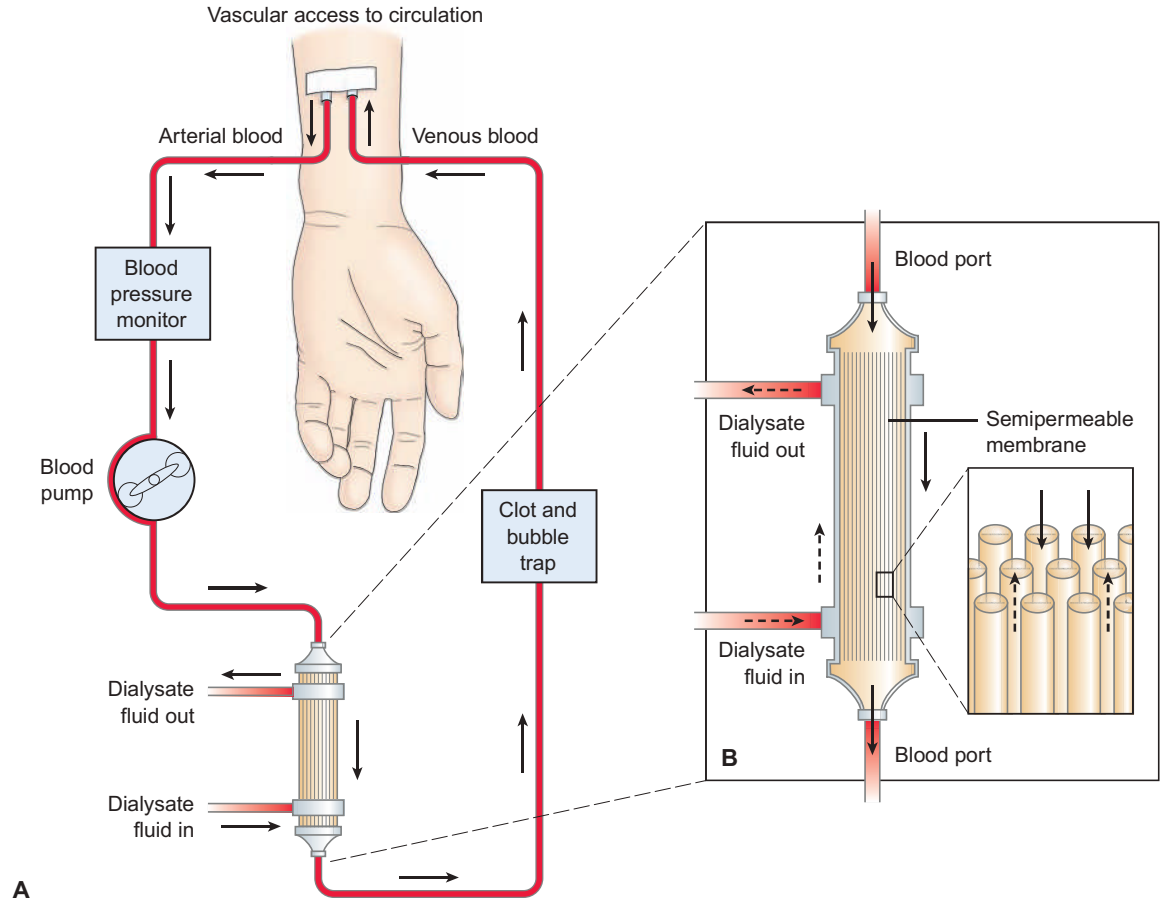

Figure 44-3 Hemodialysis system. **A,** Blood from an artery is pumped into **(B)** a dialyzer where it flows through the cellophane tubes, which act as the semipermeable membrane (*inset*). The dialysate, which has the same chemical composition as the blood except for urea and waste products, flows in around the tubules. The waste products in the blood diffuse through the semipermeable membrane into the dialysate.

allowing toxins, fluid, and electrolytes to pass through. The constant flow of the solution maintains the concentration gradient to facilitate the exchange of wastes from the blood through the semipermeable membrane into the dialysate solution, where they are removed and discarded (Fig. 44-3).

Dialyzers have undergone many technologic changes in performance and biocompatibility. High-flux dialysis uses highly permeable membranes to increase the clearance of low- and mid-molecular-weight molecules. These special membranes are used with higher than traditional rates of flow for the blood entering and exiting the dialyzer (500 to 550 mL/min). High-flux dialysis increases the efficiency of treatments while shortening their duration and reducing the need for heparin.

Vascular Access

Access to the patient's vascular system must be established to allow blood to be removed, cleansed, and returned to the patient's vascular system at rates between 300 and 800 mL/min. Several types of access are available.

Vascular Access Devices

Immediate access to the patient's circulation for acute hemodialysis is achieved by inserting a double-lumen, noncuffed, large-bore catheter into the subclavian, internal jugular, or femoral vein by the physician (Fig. 44-4). This method of vascular access involves some risk (eg, hematoma, pneumothorax, infection, thrombosis of the subclavian vein, inadequate flow). The catheter is removed when no longer needed (eg, because the patient's condition has improved or another type of access has been established). Double-lumen, cuffed catheters may also be inserted, usually by either a surgeon or interventional radiologist, into the internal jugular vein of the patient. Since these catheters have cuffs under the skin, the insertion site heals, sealing the wound and reducing the risk for ascending infection. This feature makes these catheters safe for longer-term use. Infection rates, however, remain high and septicemia continues to be a common cause for hospital admission.

Arteriovenous Fistula

The preferred method of permanent access is an **arteriovenous fistula (AVF)** that is created surgically (usually in the forearm) by joining (anastomosing) an artery to a vein, either side to side or end to side (Fig. 44-5A). Needles are inserted into the vessel to obtain blood flow adequate to pass through the dialyzer. The arterial segment of the fistula is used for arterial flow to the dialyzer and the venous segment for reinfusion of the dialyzed blood. This

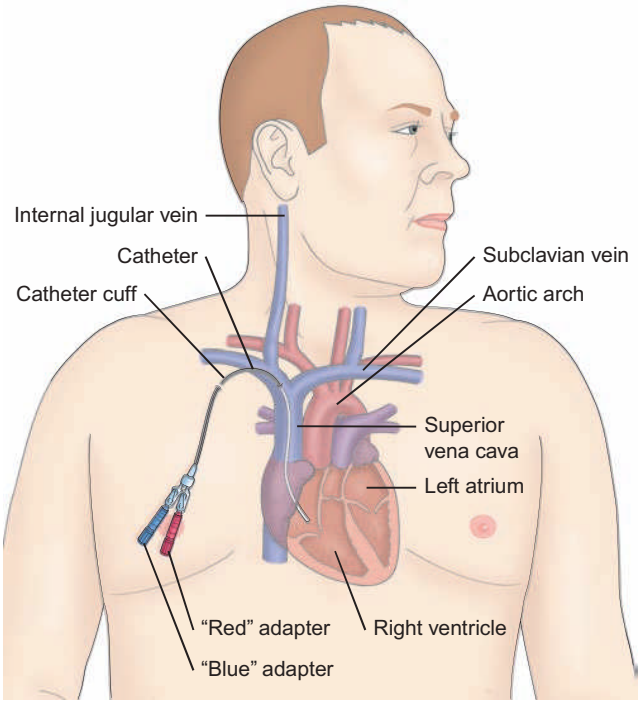

Figure 44-4 Double-lumen, cuffed hemodialysis catheter used in acute hemodialysis. The red adapter is attached to a blood line through which blood is pumped from the patient to the dialyzer. After the blood passes through the dialyzer (artificial kidney), it returns to the patient through the blue adapter.

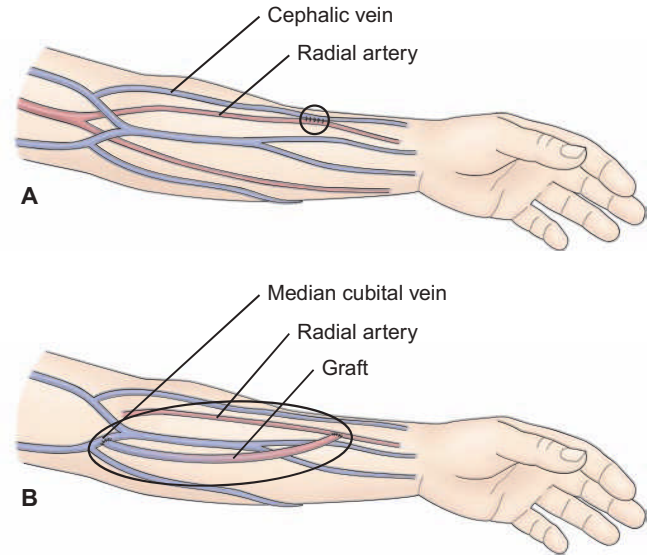

Figure 44-5 **A,** Arteriovenous fistulas are created by anastomosing a patient's vein to an artery. This illustrates a side-to-side anastomosis. **B,** Arteriovenous grafts are established by placing synthetic tubing between the artery and vein.

access will need time, (2 to 3 months) to "mature" before it can be used. As the AVF matures, the venous segment dilates due to the increased blood flow coming directly from the artery. Once sufficiently dilated it will then accommodate two large-bore (14-, 15-, or 16-gauge) needles that are inserted for each dialysis treatment. The patient is encouraged to perform hand exercises to increase the size of these vessels (ie, squeezing a rubber ball for forearm fistulas) to accommodate the large-bore needles. Once established, this access has the longest useful life and thus is the best option for vascular access for the chronic hemodialysis patient.

Arteriovenous Graft

An **arteriovenous graft** can be created by subcutaneously interposing a biologic, semibiologic, or synthetic graft material between an artery and vein (Fig. 44-5B). Usually a graft is created when the patient's vessels are not suitable for creation of an AV fistula. Patients with compromised vascular systems (eg, from diabetes) will require a graft because their native vessels are not suitable for creation of an AV Fistula. Grafts are usually placed in the arm but may be placed in the thigh or chest area. Stenosis, infection, and thrombosis are the most common complications that result in loss of this access. It is not at all uncommon to see a dialysis patient with numerous "old" or "nonfunctioning" accesses present on their arms. The patient is asked to identify which is the current access in use and it is checked carefully for the presence of a bruit and thrill.

NURSING ALERT

Failure of the permanent dialysis access (fistula or graft) accounts for most hospital admissions of patients undergoing chronic hemodialysis. Thus, protection of the access is of high priority.

Complications

While hemodialysis can prolong life indefinitely, it does not alter the natural course of the underlying CKD, nor does it completely replace kidney function. The CKD complications previously discussed will continue to worsen and require more aggressive treatment. With the initiation of dialysis, disturbances of lipid metabolism (hypertriglyceridemia) are accentuated and contribute to cardiovascular complications. Heart failure, coronary heart disease, angina, stroke, and peripheral vascular insufficiency may occur and can incapacitate the patient. Cardiovascular disease remains the leading cause of death in patients receiving dialysis (Burrows & Muller, 2007).

Anemia is compounded by blood lost during hemodialysis. Gastric ulcers may result from the physiologic stress of chronic illness, medication, and preexisting medical conditions (eg, diabetes). Patients with uremia report a metallic taste and nausea when they require dialysis. Vomiting may occur during the hemodialysis treatment when rapid fluid shifts and hypotension occur. These contribute to the malnutrition seen in patients on dialysis. Worsening calcium metabolism and renal osteodystrophy can result in bone pain and fractures, interfering with mobility. As time on dialysis continues, calcification of major blood vessels has been reported and linked to hypertension and other vascular complications. Phosphorus deposits in the skin can occur and cause itching.

Up to 85% of people undergoing hemodialysis experience major sleep problems that further complicate their overall health status (Phillips & Ryr, 2005). Early-morning or late-afternoon dialysis may be a risk factor for developing sleep disturbances.

Other complications of dialysis treatment may include the following:

- Episodes of shortness of breath often occur as fluid accumulates between dialysis treatments.
- Hypotension may occur during the treatment as fluid is removed. Nausea and vomiting, diaphoresis, tachycardia, and dizziness are common signs of hypotension.
- Painful muscle cramping may occur, usually late in dialysis as fluid and electrolytes rapidly leave the extracellular space.
- Exsanguination may occur if blood lines separate or dialysis needles become dislodged.
- Dysrhythmias may result from electrolyte and pH changes or from removal of antiarrhythmic medications during dialysis.
- Air embolism is rare but can occur if air enters the vascular system.
- Chest pain may occur in patients with anemia or arteriosclerotic heart disease.
- Dialysis disequilibrium results from cerebral fluid shifts. Signs and symptoms include headache, nausea and vomiting, restlessness, decreased level of consciousness, and seizures. It is more likely to occur in acute renal failure or when blood urea nitrogen levels are very high (exceeding 150 mg/dL).

Nursing Management

The nurse in the dialysis unit has an important role in monitoring, supporting, assessing, and educating the patient. During dialysis, the patient, the dialyzer, and the dialysate bath require constant monitoring because numerous complications are possible, including clotting of the circuit, air embolism, inadequate or excessive ultrafiltration hypotension, cramping, vomiting, blood leaks, contamination, and access complications. Nursing care of the patient and maintenance of the vascular access device are especially important and are discussed later in this chapter in the section titled Special Considerations: Nursing Management of the Hospitalized Patient on Dialysis.

Promoting Pharmacologic Therapy

Many medications are removed from the blood during hemodialysis; therefore, dosage or timing of the medication administration may require adjustment. Medications that are water soluble are readily removed during hemodialysis treatment and those that are fat soluble or adhere to other substances (like albumin) are not dialyzed out very well. This is the reason some drug overdoses are treated with emergency hemodialysis and others are not.

Patients undergoing hemodialysis who require medications (eg, cardiac glycosides, antibiotic agents, antiarrhythmic medications, antihypertensive agents) are monitored closely to ensure that blood and tissue levels of these medications are maintained without toxic accumulation. Antihypertensive therapy, often part of the regimen of patients on dialysis, is one example when communication, teaching, and evaluation can make a difference in patient outcomes. The patient must know when and when not to take the medication. For example, if an antihypertensive agent is taken on a dialysis day, hypotension may occur during dialysis, causing dangerously low blood pressure. Many medications that are taken once daily can be held until after the dialysis treatment.

Promoting Nutritional and Fluid Therapy

Diet is important for patients on hemodialysis because of the effects of uremia. Goals of nutritional therapy are to minimize uremic symptoms and fluid and electrolyte imbalances; to maintain good nutritional status through adequate protein, calorie, vitamin, and mineral intake; and to enable the patient to eat a palatable and enjoyable diet. Restricting dietary protein decreases the accumulation of nitrogenous wastes, reduces uremic symptoms, and may even postpone the initiation of dialysis for a few months. Restriction of fluid is also part of the dietary prescription because fluid accumulation may occur, leading to weight gain, heart failure, and pulmonary edema.

With the initiation of hemodialysis, the patient usually requires some restriction of dietary protein, sodium, potassium, and fluid intake. Protein intake is restricted to about 1.2 to 1.3 g/kg ideal body weight per day; therefore, protein must be of high biologic quality. Sodium is usually restricted to 2 to 3 g/day; fluids are restricted to an amount equal to the daily urine output plus 500 mL/day. The goal for patients on hemodialysis is to keep their interdialytic (between dialysis treatments) weight gain under 1.5 kg (Welch & Perkins, 2006). Potassium restriction depends on the amount of residual renal function and the frequency of dialysis. Dietary restriction is an unwelcome change in lifestyle for many patients with chronic renal failure. Patients can feel stigmatized in social situations because there may be few food choices available for their diet. If the restrictions are ignored, life-threatening complications, such as hyperkalemia and pulmonary edema, may result. Thus, the patient may feel punished for responding to basic human drives to eat and drink. The nurse who cares for a patient with symptoms or complications resulting from dietary indiscretion must avoid harsh, judgmental, or punitive tones when communicating with him or her. Regular education with reinforcement is needed to achieve this difficult change in life style (Welch & Perkins, 2006) (Chart 44-8).

Meeting Psychosocial Needs

Patients requiring long-term hemodialysis are often concerned about the unpredictability of the illness and the disruption of their lives. They often have financial problems, difficulty holding a job, waning sexual desire and impotence, depression from being chronically ill, and fear of dying. Younger patients worry about marriage, having children, and the burden that they bring to their families. The regimented lifestyle that frequent dialysis treatments and restrictions in food and fluid intake impose is often demoralizing to the patient and family.

Dialysis alters the lifestyle of the patient and family. The amount of time required for dialysis and physician visits and being chronically ill can create conflict, frustration, guilt,

CHART 44-8 NURSING RESEARCH PROFILE
Hemodialysis and Nonadherence

Belguzar, K., Kayser, C. & Kilic, S. (2007). Nonadherence with diet and fluid restrictions and perceived social support in patients receiving hemodialysis. *Journal of Nursing Scholarship, 39*(3), 243–248.

Purpose

Nonadherence to diet and fluid restrictions has adverse consequences for patients receiving hemodialysis. The purpose of this study was to describe nonadherence with diet and fluid restrictions and the level of perceived social support in hemodialysis patients. Nonadherence often occurs when a person's behavior conflicts with medical advice regarding taking medications, following diets, or other lifestyle changes.

Design

This descriptive study surveyed 160 patients on hemodialysis in three centers in Turkey. Participants were asked about personal characteristics, and data were collected using the Dialysis Diet and Fluid Nonadherence Questionnaire (DDFQ) and Multidimensional Scale of Perceived Social Support (MSP). Data were collected during the patient's regularly scheduled dialysis session. The DDFQ is a four-item self-report questionnaire that assesses the frequency of nonadherence to diet and fluid restrictions for the previous 14 days. The MSP is a 12-item scale used to assess emotional support and the degree of satisfaction with perceived social support from family, friends, and significant others.

Findings

Adherence to fluid restriction is a difficult and stressful aspect of hemodialysis treatment, and most patients in this study showed some degree of nonadherence to fluid restrictions (68%) and diet (58%). Participants perceived total social support as low. Nonadherence was most common with younger patients, those who were married, and those with lower levels of perceived social support.

Nursing Implications

Nurses working in hemodialysis centers need to consider social support and how it affects adherence in patients receiving hemodialysis. The results of this study suggest that younger, married patients may require assistance to develop the levels of social support needed to adhere to fluid and diet restrictions between hemodialysis sessions.

and depression. It may be difficult for the patient, spouse, and family to express anger and negative feelings.

The nurse needs to give the patient and family the opportunity to express feelings of anger and concern about the limitations that the disease and treatment impose, possible financial problems, and job insecurity. If anger is not expressed, it may be directed inward and lead to depression, despair, and attempts at suicide (suicide is more prevalent in patients on dialysis); however, if anger is projected outward to other people, it may destroy already threatened family relationships.

Although these feelings are normal in this situation, they are often profound and overwhelming. Counseling and psychotherapy may be necessary. Depression may require treatment with antidepressant agents. Referring the patient and family to a mental health provider with expertise in the care of patients receiving dialysis may also be helpful. Clinical nurse specialists, psychologists, and social workers may be helpful in assisting the patient and family to cope with the changes brought about by renal failure and its treatment.

The "sense of loss" that the patient experiences cannot be underestimated because every aspect of a "normal life" is disrupted. Some patients use denial to deal with the overwhelming array of medical problems (eg, infections, hypertension, anemia, neuropathy). Staff who are tempted to label the patient as noncompliant must consider the impact of renal failure and its treatment on the patient and family and the coping strategies that they may use.

Palliative care principles that focus on symptom control are becoming increasingly important as greater attention is focused on quality-of-life issues (Cohen, Moss, Weisord, et al., 2006). Patients and their families should be encouraged to discuss end-of-life options and have developed advanced directives or living wills.

Promoting Home and Community-Based Care

Teaching Patients Self-Care

Preparing a patient for hemodialysis is challenging. Often the patient does not fully comprehend the impact of dialysis, and learning needs may go unrecognized. Good communication between dialysis staff and home care nurses is essential.

Assessment helps identify the learning needs of the patient and family members. In many cases, the patient is discharged home before learning needs and readiness to learn can be thoroughly evaluated; therefore, hospital-based nurses, dialysis staff, and home care nurses must work together to provide appropriate teaching that meets the patient's and family's changing needs and readiness to learn.

The diagnosis of chronic renal failure and the need for dialysis often overwhelm the patient and family. In addition, many patients with ESRD have depressed mentation, a shortened attention span, a decreased level of concentration, and altered perception. Therefore, teaching must occur in brief, 10- to 15-minute sessions, with time added for clarification, repetition, reinforcement, and questions from the patient and family. The nurse needs to convey a nonjudgmental attitude to enable the patient and family to discuss options and their feelings about those options. Team conferences are helpful for sharing information and providing every team member the opportunity to discuss the needs of the patient and family.

Home Hemodialysis

Most patients who undergo hemodialysis do so in an outpatient setting, but home hemodialysis is an option for some. Home hemodialysis requires a highly motivated patient who is willing to take responsibility for the procedure and is able to adjust each treatment to meet the body's changing

needs. It also requires the commitment and cooperation of a caregiver to assist the patient. However, many patients are not comfortable imposing on others this way and do not wish to subject family members to the feeling that their home is being turned into a clinic. The health care team never forces a patient to use home hemodialysis because this treatment requires significant changes in the home and family. Home hemodialysis must be the patient's and family's decision (American Nephrology Nurses Association, 2007a).

The patient undergoing home hemodialysis and the caregiver assisting that patient must be trained to prepare, operate, and disassemble the dialysis machine; maintain and clean the equipment; administer medications (eg, heparin) into the machine lines; and handle emergency problems (hemodialysis dialyzer rupture, electrical or mechanical problems, hypotension, shock, and seizures). Because home hemodialysis places primary responsibility for the treatment on the patient and the family member, they must understand and be capable of performing all aspects of the hemodialysis procedure (Chart 44-9).

Before home hemodialysis is initiated, the home environment, household and community resources, and ability and willingness of the patient and family to carry out this treatment are assessed. The home is surveyed to see if electrical outlets, plumbing facilities, and storage space are adequate. Modifications may be needed to enable the patient and assistant to perform dialysis safely and to deal with emergencies.

Once home hemodialysis is initiated, the home care nurse must visit periodically to evaluate compliance with the recommended techniques, to assess the patient for complications, to reinforce previous teaching, and to provide reassurance.

Continuing Care

The health care team's goal in treating patients with chronic renal failure is to maximize their vocational potential, functional status, and quality of life. To facilitate renal rehabilitation, appropriate follow-up and monitoring by members of the health care team (physicians, dialysis nurses, social worker, psychologist, home care nurses, and others as appropriate) are essential to identify and resolve problems early on. Many patients with chronic renal failure can resume relatively normal lives, doing the things that are important to them: traveling, exercising, working, or actively participating in family activities. If appropriate interventions are available early in the course of dialysis, the potential for better health improves, and the patient can remain active in family and community life. Outcome goals for renal rehabilitation include employment for those able to work, improved physical functioning of all patients, improved understanding about adaptation and options for living well, increased control over the effects of kidney disease and dialysis, and resumption of activities enjoyed before dialysis.

CONTINUOUS RENAL REPLACEMENT THERAPIES

Continuous renal replacement therapies (CRRTs) may be indicated for patients with acute or chronic renal failure who are too clinically unstable for traditional hemodialysis,

CHART 44-9 HOME CARE CHECKLIST
Hemodialysis

At the completion of the home care instruction, the patient or caregiver will be able to:	PATIENT	CAREGIVER
• Discuss renal failure and its effects on the body.	✔	✔
• Describe the cause of renal failure and why hemodialysis is necessary.	✔	✔
• Describe the basic principles of hemodialysis.	✔	✔
• Discuss common problems that may occur during hemodialysis and their prevention and management.	✔	✔
• Demonstrate knowledge about prescribed medications and the reason for their use, potential side effects, guidelines on when to notify physician, and the schedule of medications on dialysis and nondialysis days.	✔	✔
• Acknowledge dietary and fluid restrictions, rationale, and consequences of noncompliance.	✔	✔
• Describe commonly measured laboratory values, results, and implications.	✔	✔
• List guidelines for prevention and detection of fluid overload, meaning of "dry" weight, and how to weigh self.	✔	✔
• Demonstrate vascular access care, how to check patency, signs and symptoms of infection, and prevention of complications.	✔	✔
• Discuss strategies for detection, management, and relief of pruritus, neuropathy, and other complications of renal failure.	✔	✔
• Develop strategies to manage or reduce anxiety and maintain independence.	✔	✔
• Coordinate financial arrangements for dialysis and strategies to identify and obtain resources.	✔	✔

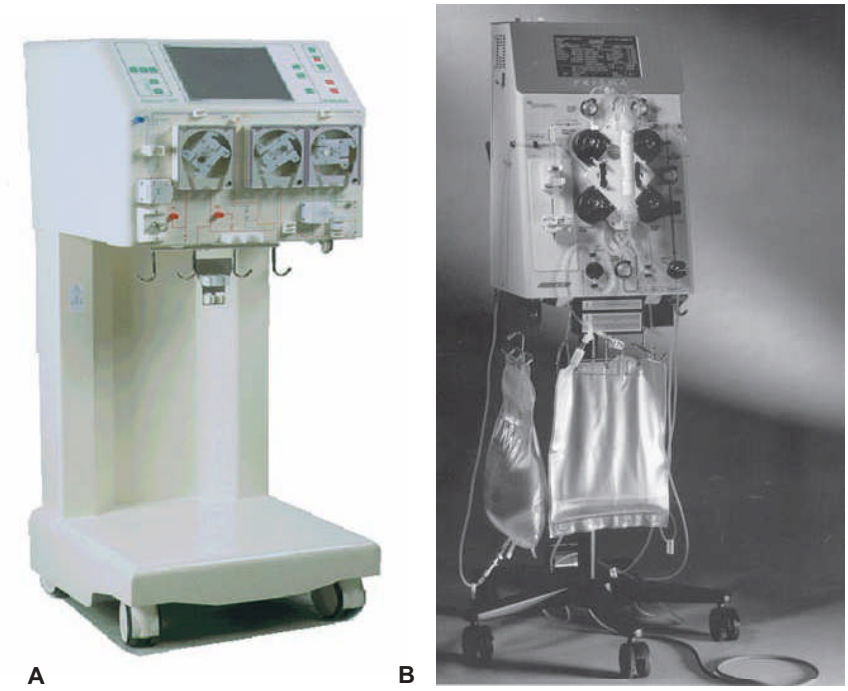

Figure 44-6 Devices for administering continuous renal replacement therapy (CRRT) offer an integrated fluid warmer for the heating of infusion and dialysate fluids, a weighing system to reduce the possibility of error in assessing fluid balance, and a battery backup that allows treatments to continue when the patient is moved. **A,** Diapact CRRT System, B-Braun Medical, Inc., Bethlehem, PA. **B,** PRISMA, Gambro Corporation, Lakewood, CO.

for patients with fluid overload secondary to oliguric (low urine output) renal failure, and for patients whose kidneys cannot handle their acutely high metabolic or nutritional needs. CRRT does not produce rapid fluid shifts, does not require dialysis machines or dialysis personnel to carry out the procedures, and can be initiated quickly. Several types of CRRT are available and widely used in critical care units (Fig. 44-6). The methods are similar as they require access to the circulation and blood to pass through an artificial filter. A hemofilter (an extremely porous blood filter containing a semipermeable membrane) is used in all types.

Continuous Venovenous Hemofiltration

Continuous venovenous hemofiltration (CVVH) is used to manage acute renal failure. Blood from a double-lumen venous catheter is pumped (using a small blood pump) through a hemofilter and then returned to the patient through the same catheter. CVVH provides continuous slow fluid removal (ultrafiltration); therefore, hemodynamic effects are mild and better tolerated by patients with unstable conditions. CVVH does not require arterial access, and critical care nurses can set up, initiate, maintain, and terminate the system.

Continuous Venovenous Hemodialysis

Continuous venovenous hemodialysis (CVVHD) is similar to CVVH. Blood is pumped from a double-lumen venous catheter through a hemofilter and returned to the patient through the same catheter. In addition to the benefits of ultrafiltration, CVVHD uses a concentration gradient to facilitate the removal of uremic toxins and fluid. No arterial access is required, hemodynamic effects are usually mild, and critical care nurses can set up, initiate, maintain, and terminate the system (Martin & Jurschak, 2007).

Examples of less frequently used CRRT include slow continuous ultrafiltration (SCUF), continuous arteriovenous hemofiltration (CAVH), and continuous arteriovenous hemodialysis (CAVHD) (Martin & Jurschak, 2007).

PERITONEAL DIALYSIS

The goals of PD are to remove toxic substances and metabolic wastes and to reestablish normal fluid and electrolyte balance. PD may be the treatment of choice for patients with renal failure who are unable or unwilling to undergo hemodialysis or renal transplantation. Patients who are susceptible to the rapid fluid, electrolyte, and metabolic changes that occur during hemodialysis experience fewer of these problems with the slower rate of PD. Therefore, patients with diabetes or cardiovascular disease, many older patients, and those who may be at risk for adverse effects of systemic heparin are likely candidates for PD. Additionally, severe hypertension, heart failure, and pulmonary edema not responsive to usual treatment regimens have been successfully treated with PD. Less than 8% of patients with ESRD receive PD as their treatment modality (USRDS, 2007).

In PD, the peritoneal membrane that covers the abdominal organs and lines the abdominal wall serves as the semipermeable membrane. Sterile dialysate fluid is introduced into the peritoneal cavity through an abdominal catheter at intervals (Fig. 44-7). Once the sterile solution is in the peritoneal cavity, uremic toxins such as urea and creatinine begin to be cleared from the blood. Diffusion and osmosis occur as waste products move from an area of higher concentration (the blood stream) to an area of lesser

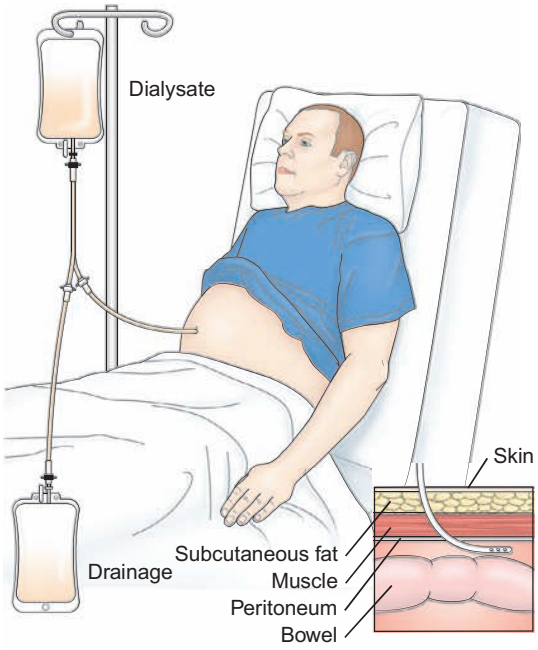

Figure 44-7 In peritoneal dialysis and in acute intermittent peritoneal dialysis, dialysate is infused into the peritoneal cavity by gravity, after which the clamp on the infusion line is closed. After a dwell time (when the dialysate is in the peritoneal cavity), the drainage tube is unclamped and the fluid drains from the peritoneal cavity, again by gravity. A new container of dialysate is infused as soon as drainage is complete. The duration of the dwell time depends on the type of peritoneal dialysis.

concentration (the dialysate fluid) through a semipermeable membrane (the peritoneum). This movement of solute from the blood into the dialysate fluid is called clearance. Since substances cross the peritoneal membrane at different rates, adjustments in dwell time and amount of fluid used are made to facilitate the process. Ultrafiltration (water removal) occurs in PD through an osmotic gradient created by using a dialysate fluid with a higher glucose concentration. PD usually takes 36 to 48 hours to achieve what hemodialysis accomplishes in 6 to 8 hours.

Procedure

As with other forms of treatment, the decision to begin PD is made by the patient and family in consultation with the physician. The patient may be acutely ill, thus requiring short-term treatment to correct severe disturbances in fluid and electrolyte status, or may have ESRD and need to receive ongoing treatments.

Preparing the Patient

The nurse's preparation of the patient and family for PD depends on the patient's physical and psychological status, level of alertness, previous experience with dialysis, and understanding of and familiarity with the procedure.

The nurse explains the procedure to the patient and assists in obtaining signed consent. Baseline vital signs, weight, and serum electrolyte levels are recorded. Evaluation of the abdomen for placement of the catheter is done to facilitate self-care. Typically the catheter is placed on the nondominant side to allow the patient easier access to the catheter connection site when exchanges are done. The patient is encouraged to empty the bladder and bowel to reduce the risk of puncture of internal organs during the insertion procedure. Broad-spectrum antibiotic agents may be administered to prevent infection. The peritoneal catheter can be inserted in interventional radiology, in the operating room, or at the bedside. Depending on the situation, this will need to be explained to the patient and family.

Preparing the Equipment

In addition to assembling the equipment for PD, the nurse consults with the physician to determine the concentration of dialysate to be used and the medications to be added to it. Heparin may be added to prevent fibrin formation and resultant occlusion of the peritoneal catheter. Potassium chloride may be prescribed to prevent hypokalemia. Antibiotics may be added to treat **peritonitis** (inflammation of the peritoneal membrane) caused by infection. Regular insulin may be added for patients with diabetes. Aseptic technique is imperative whenever medications are added.

Before medications are added, the dialysate is warmed to body temperature to prevent patient discomfort and abdominal pain and to dilate the vessels of the peritoneum to increase urea clearance. Solutions that are too cold cause pain, cramping, and vasoconstriction and reduce clearance. Dry heating (heating cabinet, incubator, or heating pad) is recommended. Methods not recommended include soaking the bags of solution in warm water (can introduce bacteria to the exterior of the bags of solution and increase the chance of peritonitis) and use of a microwave to heat the fluid (increases the danger of burning the peritoneum).

Immediately before initiating dialysis, using aseptic technique, the nurse assembles the administration set and tubing. The tubing is filled with the prepared dialysate to reduce the amount of air entering the catheter and peritoneal cavity, which could increase abdominal discomfort and interfere with instillation and drainage of the fluid.

Inserting the Catheter

Ideally, the peritoneal catheter is inserted in the operating room or radiology suite to maintain surgical asepsis and minimize the risk of contamination. However, in some circumstances, the physician may insert the rigid stylet catheter at the bedside using strict asepsis. Whenever a rigid catheter is used, careful securing and close observation for bowel perforation is essential to minimize complications.

Catheters for long-term use (eg, Tenckhoff, Swan, or Cruz) are usually soft and flexible and made of silicone with a radiopaque strip to permit visualization on x-ray. These catheters have three sections: (1) an intraperitoneal section, with numerous openings and an open tip to let dialysate flow freely; (2) a subcutaneous section that passes from the peritoneal membrane and tunnels through muscle and subcutaneous fat to the skin; and (3) an external section for connection to the dialysate system. Most of these catheters have two cuffs, which are made of Dacron polyester. The cuffs stabilize the catheter, limit movement, prevent leaks, and provide a barrier against microorganisms. One cuff is placed just distal to the peritoneum, and the other cuff is placed subcutaneously. The subcutaneous tunnel (5 to 10 cm long) further protects against bacterial infection (Fig. 44-8).

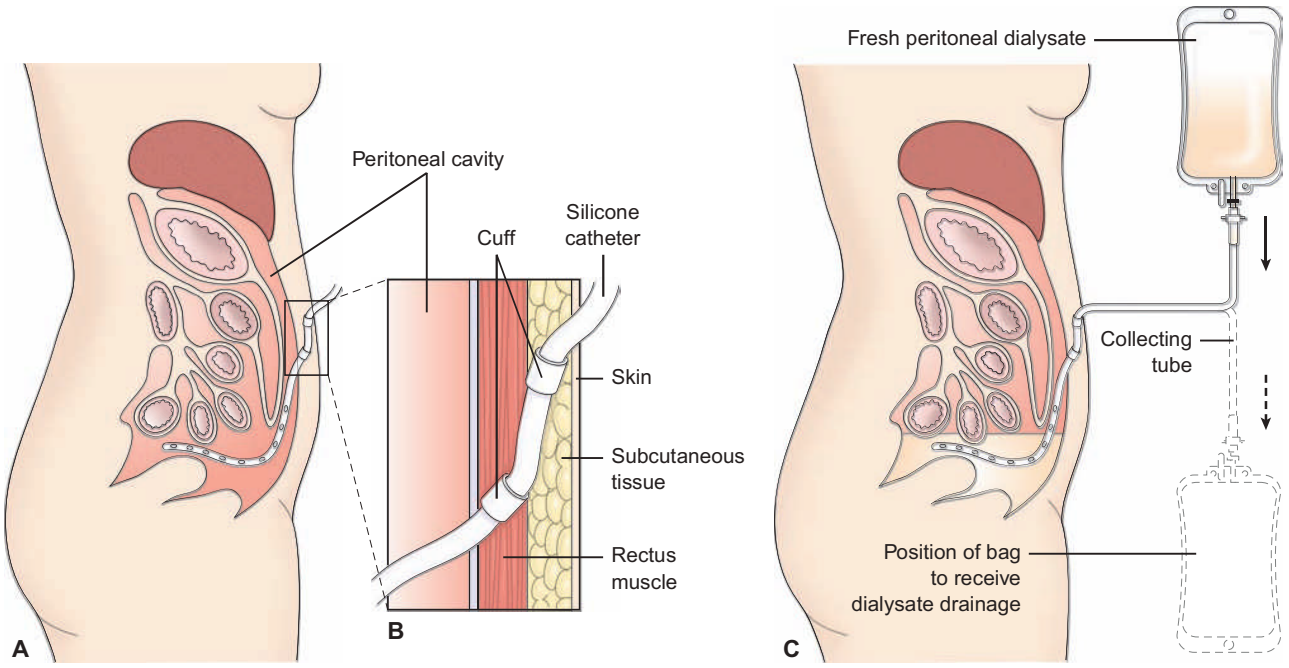

Figure 44-8 Continuous ambulatory peritoneal dialysis. **A,** The peritoneal catheter is implanted through the abdominal wall. **B,** Dacron cuffs and a subcutaneous tunnel provide protection against bacterial infection. **C,** Dialysate flows by gravity through the peritoneal catheter into the peritoneal cavity. After a prescribed period of time, the fluid is drained by gravity and discarded. New solution is then infused into the peritoneal cavity until the next drainage period. Dialysis thus continues on a 24-hour-a-day basis, during which the patient is free to move around and engage in his or her usual activities.

Performing the Exchange

PD involves a series of exchanges or cycles. An **exchange** is defined as the infusion (fill), dwell, and drainage of the dialysate. This cycle is repeated throughout the course of the dialysis. The dialysate is infused by gravity into the peritoneal cavity. A period of about 5 to 10 minutes is usually required to infuse 2 to 3 L of fluid. The prescribed dwell, or equilibration, time allows diffusion and osmosis to occur. At the end of the dwell time, the drainage portion of the exchange begins. The tube is unclamped and the solution drains from the peritoneal cavity by gravity through a closed system. Drainage is usually completed in 10 to 20 minutes. The drainage fluid is normally colorless or straw-colored and should not be cloudy. Bloody drainage may be seen in the first few exchanges after insertion of a new catheter but should not occur after that time. The number of cycles or exchanges and their frequency are prescribed based on monthly laboratory values and presence of uremic symptoms.

The removal of excess water during PD occurs because dialysate has a high dextrose concentration, making it hypertonic. An osmotic gradient is created between the blood and the dialysate solution. Dextrose solutions of 1.5%, 2.5%, and 4.25% are available in several volumes, from 1000 mL to 3000 mL. The higher the dextrose concentration, the greater the osmotic gradient and the more water will be removed. Selection of the appropriate solution is based on the patient's fluid status.

Complications

Most complications of PD are minor, but several, if unattended, can have serious consequences.

Acute Complications

Peritonitis

Peritonitis is the most common and serious complication of PD. The first sign of peritonitis is cloudy dialysate drainage fluid. Diffuse abdominal pain and rebound tenderness occur much later. Hypotension and other signs of shock may also occur with advancing infection. The patient with peritonitis may be treated as an inpatient or outpatient (most common), depending on the severity of the infection and the patient's clinical status. Drainage fluid is examined for cell count; Gram stain and culture are used to identify the organism and guide treatment. Antibiotic agents (aminoglycosides or cephalosporins) are usually added to subsequent exchanges until Gram stain or culture results are available for appropriate antibiotic determination. Intraperitoneal administration of antibiotics is as effective as IV administration and therefore most often used. Antibiotic therapy continues for 10 to 14 days. Careful selection and calculation of the antibiotic dosage are needed to prevent nephrotoxicity and further compromise of residual renal function.

Regardless of which organism causes peritonitis, the patient with peritonitis loses large amounts of protein through the peritoneum. Acute malnutrition and delayed healing may result. Therefore, attention must be given to detecting and promptly treating peritonitis.

Leakage

Leakage of dialysate through the catheter site may occur immediately after the catheter is inserted. Usually, the leak stops spontaneously if dialysis is withheld for several days,

giving the tissue surrounding the cuffs located on the abdominal catheter a chance to infiltrate the Dacron and seal the insertion tunnel. It also allows the exit site time to heal. During this time, it is important to reduce factors that might delay healing, such as undue abdominal muscle activity and straining during bowel movement. In many cases, leakage can be avoided by using small volumes (500 mL) of dialysate, gradually increasing the volume up to 2000 to 3000 mL.

Bleeding

A bloody **effluent** (drainage) may be observed occasionally, especially in young, menstruating women. (The hypertonic fluid pulls blood from the uterus, through the opening in the fallopian tubes, and into the peritoneal cavity.) Bleeding is also common during the first few exchanges after a new catheter insertion because some blood enters the abdominal cavity following insertion. In many cases, no cause can be found for the bleeding, although catheter displacement from the pelvis has occasionally been associated with bleeding. Some patients have had bloody effluent after an enema or from minor trauma. Invariably, bleeding stops in 1 to 2 days and requires no specific intervention. More frequent exchanges and the addition of heparin to the dialysate during this time may be necessary to prevent blood clots from obstructing the catheter.

Long-Term Complications

Hypertriglyceridemia is common in patients undergoing long-term PD, suggesting that the therapy may accelerate atherogenesis. Despite this, the use of cardioprotective medications is relatively uncommon, and many patients have suboptimal blood pressure control. Given the high burden of disease in these patients, beta-blockers and ACE inhibitors should be used to control hypertension or protect the heart, and the use of aspirin and statins should be considered.

Other complications that may occur with long-term PD include abdominal hernias (incisional, inguinal, diaphragmatic, and umbilical), probably resulting from continuously increased intra-abdominal pressure. The persistently elevated intra-abdominal pressure also aggravates symptoms of hiatal hernia and hemorrhoids. Low back pain and anorexia from fluid in the abdomen and a constant sweet taste related to glucose absorption may also occur.

Mechanical problems occasionally occur and may interfere with instillation or drainage of the dialysate. Formation of clots in the peritoneal catheter and constipation are factors that may contribute to these problems.

Approaches

PD can be performed using several different approaches: acute intermittent peritoneal dialysis, **continuous ambulatory peritoneal dialysis (CAPD),** and **continuous cyclic peritoneal dialysis (CCPD).**

Acute Intermittent Peritoneal Dialysis

Indications for acute intermittent PD, a variation of PD, include uremic signs and symptoms (nausea, vomiting, fatigue, altered mental status), fluid overload, acidosis, and hyperkalemia. Although PD is not as efficient as hemodialysis in removing solute and fluid, it permits a more gradual change in the patient's fluid volume status and in waste product removal. Therefore, it may be the treatment of choice for the hemodynamically unstable patient. It can be carried out manually (the nurse warms, spikes, and hangs each container of dialysate) or by a cycler machine. Exchange times range from 30 minutes to 2 hours. A common routine is hourly exchanges consisting of a 10-minute infusion, a 30-minute dwell time, and a 20-minute drain time.

Maintaining the PD cycle is a nursing responsibility. Strict aseptic technique is maintained when changing solution containers and emptying drainage containers. Vital signs, weight, I&O, laboratory values, and patient status are frequently monitored. The nurse uses a flow sheet to document each exchange and records vital signs, dialysate concentration, medications added, exchange volume, dwell time, dialysate fluid balance for each exchange (fluid lost or gained), and cumulative fluid balance. The nurse also carefully assesses skin turgor and mucous membranes to evaluate fluid status and monitor the patient for edema.

If the peritoneal fluid does not drain properly, the nurse can facilitate drainage by turning the patient from side to side or raising the head of the bed. The catheter should never be pushed further into the peritoneal cavity. Other measures to promote drainage include checking the patency of the catheter by inspecting for kinks, closed clamps, or an air lock. The nurse monitors for complications, including peritonitis, bleeding, respiratory difficulty, and leakage of peritoneal fluid. Abdominal girth may be measured periodically to determine if the patient is retaining large amounts of dialysis solution. Additionally, the nurse must ensure that the PD catheter remains secure and that the dressing remains dry. Physical comfort measures, frequent turning, and skin care are provided. The patient and family are educated about the procedure and are kept informed about progress (fluid loss, weight loss, laboratory values). Emotional support and encouragement are given to the patient and family during this stressful and uncertain time.

Continuous Ambulatory Peritoneal Dialysis

CAPD is the second most common form of dialysis for patients with ESRD to be started on (USRDS, 2007). CAPD is performed at home by the patient or a trained caregiver who is usually a family member. The procedure allows the patient reasonable freedom and control of daily activities but requires a serious commitment to be successful. Chart 44-10 discusses suitability for CAPD.

CAPD works on the same principles as other forms of PD: diffusion and osmosis. Less extreme fluctuations in the patient's laboratory values occur with CAPD than with intermittent PD or hemodialysis because the dialysis is constantly in progress. The serum electrolyte levels usually remain in the normal range.

Procedure

The patient performs exchanges four or five times a day, 24 hours a day, 7 days a week, at intervals scheduled throughout the day. Different manufacturers supply different equipment. Most commonly used is a Y-shaped system, in which a bag containing dialysate solution comes connected to one branch of the "Y" and a sterile empty

Chart 44-10 • *Considerations in CAPD*

Although CAPD is not suitable for all patients with end-stage renal disease (ESRD), it is a viable therapy for those who can perform self-care and exchanges and who can fit therapy into their own routines. Often, patients report having more energy and feeling healthier once they begin CAPD. Nurses can be instrumental in helping patients with ESRD find the dialysis therapy that best suits their lifestyle. Those considering CAPD need to understand the advantages and disadvantages along with the indications and contraindications for this form of therapy.

Advantages

- Freedom from a dialysis machine
- Control over daily activities
- Opportunities to avoid dietary restrictions, increase fluid intake, raise serum hematocrit values, improve blood pressure control, avoid venipuncture, and gain a sense of well-being

Disadvantages

- Continuous dialysis 24 hours a day, 7 days a week

Indications

- Patient's willingness, motivation, and ability to perform dialysis at home
- Strong family or community support system (essential for success), particularly if the patient is an older adult
- Special problems with long-term hemodialysis, such as dysfunctional or failing vascular access devices, excessive thirst, severe hypertension, postdialysis headaches, and severe anemia requiring frequent transfusion
- Interim therapy while awaiting kidney transplantation
- ESRD secondary to diabetes because hypertension, uremia, and hyperglycemia are easier to manage with CAPD than with hemodialysis

Contraindications

- Adhesions from previous surgery (adhesions reduce clearance of solutes) or systemic inflammatory disease
- Chronic backache and preexisting disk disease, which could be aggravated by the continuous pressure of dialysis fluid in the abdomen
- Risk of complications, for example, in patients receiving immunosuppressive medications, which impede healing of the catheter site, and in patients with a colostomy, ileostomy, nephrostomy, or ileal conduit because of the risk of peritonitis. The risk for complications is not an absolute contraindication for CAPD therapy.
- Diverticulitis because CAPD has been associated with rupture of the diverticulum
- Severe arthritis or poor hand strength necessitating assistance in performing the exchange. However, blind or partially blind patients and those with other physical limitations can learn to perform CAPD.

bag is connected to the second branch. This leaves the third part of the "Y" open and available for connection to the transfer set on the PD catheter. To perform an exchange, the patient (or person doing the exchange) washes his or her hands, dons a mask, and then removes the cap from the transfer set while maintaining sterility. The open end of the "Y" set is connected to the end of the transfer set and the dialysate infused where it will dwell. After the dialysate is infused, the patient clamps off the transfer set and the tubing set, disconnects the tubing set, and applies a new cap to the transfer set, making it a closed system. The patient drains the fluid (effluent) from the peritoneal cavity through the catheter (over about 20 to 30 minutes) into an empty bag. Once the effluent has been fully drained, fresh fluid is instilled into the peritoneal cavity.

The longer the dwell time, the better the clearance of uremic toxins is. If dwell time is excessive, the patient will absorb some of the effluent back into the body simply because the osmotic gradient is lost. Once equilibrium is reached, the movement of fluid and toxins stops.

Complications

To reduce the risk of peritonitis, the patient (and all caregivers) must use meticulous care to avoid contaminating the catheter, fluid, or tubing and to avoid accidentally disconnecting the catheter from the tubing. Whenever a connection/disconnection is made, hands must be washed and a mask worn by anyone within 6 feet of the area to avoid contamination with airborne bacteria. Excess manipulation should be avoided and meticulous care of the catheter entry site is provided using a standardized protocol.

Continuous Cyclic Peritoneal Dialysis

CCPD uses a machine called a cycler to provide the exchanges. It is programmed as to how much fluid to use and how long and how many exchanges need to be done. Since it is programmed, it also keeps track of the total amounts removed and will sound an alarm if limits are not met. It requires that a person set up and break down the system for use, which typically takes about 15 minutes.

CCPD combines overnight intermittent PD with a prolonged dwell time during the day. The peritoneal catheter is connected to a cycler machine every evening, usually just before the patient goes to sleep for the night. Because the machine is very quiet, the patient can sleep, and the extra-long tubing allows the patient to move and turn normally during sleep.

In the morning, the patient disconnects from the cycler. Sometimes dialysate is left in the abdominal cavity for a longer day dwell cycle. This day exchange is drained during the day either by using a "Y" set or reattaching to the cycler. This process is done every day to achieve the effects of dialysis required.

CCPD has a lower infection rate than other forms of PD because there are fewer opportunities for contamination with bag changes and tubing disconnections. It also allows the patient to be free from exchanges throughout the day, making it possible to engage in work and activities of daily living more freely.

Nursing Management

Meeting Psychosocial Needs

In addition to the complications of PD previously described, patients who elect to do PD may experience altered body image because of the presence of the abdominal catheter, bag, tubing, and cycler. Waist size increases from 1 to 2 inches (or more) with fluid in the abdomen. This affects clothing selection and may make the patient feel "fat." Body image may be so altered that patients do not want to look at or care for the catheter for days or weeks. The nurse may arrange for the patient to talk with other patients who have adapted well to PD. Although some patients have no psychological problems with the catheter—they think of it as their lifeline and as a life-sustaining device—other patients feel they are doing exchanges all day long and have no free time, particularly in the beginning. They may experience depression because they feel overwhelmed with the responsibility of self-care.

Patients undergoing PD may also experience altered sexuality patterns and sexual dysfunction. The patient and partner may be reluctant to engage in sexual activities, partly because of the catheter being psychologically "in the way" of sexual performance. The peritoneal catheter, drainage bag, and about 2 L of dialysate may interfere with the patient's sexual function and body image as well. In patients on CCPD, the presence of the dialysis cycler in the bedroom and the continual connection during the sleeping hours can also cause interference with intimacy. Although these problems may resolve with time, some problems may warrant special counseling. Questions by the nurse about concerns related to sexuality and sexual function often provide the patient with a welcome opportunity to discuss these issues and a first step toward their resolution.

Promoting Home and Community-Based Care

Teaching Patients Self-Care

Patients are taught as inpatients or outpatients to perform PD once their condition is medically stable. Training usually takes 5 days to 2 weeks. Patients are taught according to their own learning ability and knowledge level and only as much at one time as they can handle without feeling uncomfortable or becoming overwhelmed. Education topics for the patient and family who will be performing PD at home are described in Chart 44-11. The use of an adult learning theory–based curriculum may decrease peritonitis and exit site infection rates.

CHART 44-11 HOME CARE CHECKLIST

Peritoneal Dialysis (CAPD or CCPD)

At the completion of the home care instruction, the patient or caregiver will be able to:	PATIENT	CAREGIVER
• Discuss basic information about normal kidney function.	✔	✔
• Discuss basic information about the disease process.	✔	✔
• Discuss the basic principles of peritoneal dialysis.	✔	✔
• Demonstrate catheter and exit site care.	✔	✔
• Demonstrate measurement of vital signs and weight measurement.	✔	✔
• Discuss monitoring and management of fluid balance.	✔	✔
• Discuss basic principles of aseptic technique.	✔	✔
• Demonstrate the CAPD exchange procedure using aseptic technique (CCPD patients should also demonstrate exchange procedure in case of failure or unavailability of cycling machine).	✔	✔
• Demonstrate cycler set-up procedure and maintenance if on CCPD.	✔	✔
• Discuss complications of peritoneal dialysis; prevention, recognition, and management of complications.	✔	✔
• Demonstrate procedure for adding medications to the dialysis solution.	✔	✔
• Demonstrate procedure for obtaining sterile dialysis fluid samples.	✔	✔
• Discuss routine laboratory tests needed and implications of results.	✔	✔
• Discuss dietary restrictions.		
• Discuss medications: name of medications, their actions, potential side effects, and when to contact physician.	✔	✔
• Discuss ordering, storage, and inventory of dialysis supplies.	✔	✔
• Describe plan for follow-up care.	✔	✔
• Demonstrate maintenance of home dialysis records.	✔	✔
• Describe actions in case of emergency.	✔	✔

Because of protein loss with continuous PD, the patient is instructed to eat a high-protein, well-balanced diet. The patient is also encouraged to increase his or her daily fiber intake to help prevent constipation, which can impede the flow of dialysate into or out of the peritoneal cavity. Many patients gain 3 to 5 lb within a month of initiating PD, so they may be asked to limit their carbohydrate intake to avoid excessive weight gain. Potassium, sodium, and fluid restrictions are not usually needed. Patients commonly lose about 2 to 3 L of fluid over and above the volume of dialysate infused into the abdomen during a 24-hour period, permitting a normal fluid intake even in an anephric patient (a patient without kidneys).

Continuing Care

Follow-up care through phone calls, visits to the outpatient department, and continuing home care assists patients in the transition to home and promotes their active participation in their own health care. Patients often depend on checking with the nurse to see if they are making the correct choices about dialysate or control of blood pressure, or simply to discuss a problem.

Patients may be seen by the PD team as outpatients once a month or more often if needed. The exchange procedure is evaluated at that time to see that strict aseptic technique is being used. Blood chemistry values are followed closely to make certain the therapy is adequate for the patient.

If a referral is made for home care, the home care nurse assesses the home environment and suggests modifications to accommodate the equipment and facilities needed to carry out PD. In addition, the nurse assesses the patient's and family's understanding of PD and evaluates their technique in performing PD. Assessments include checking for changes related to renal disease, complications such as peritonitis, and treatment-related problems such as heart failure, inadequate drainage, and weight gain or loss. The nurse continues to reinforce and clarify teaching about PD and renal disease and assesses the patient's and family's progress in coping with the procedure. This is also an opportunity to remind patients about the need to participate in appropriate health promotion activities and health screening (eg, gynecologic examinations, colonoscopy).

Because of the projected high numbers of elderly patients who will develop ESRD, the nursing home or extended care facility is likely to become an increasingly important site for both rehabilitation and long-term management of patients with renal failure.

SPECIAL CONSIDERATIONS: NURSING MANAGEMENT OF THE HOSPITALIZED PATIENT ON DIALYSIS

Whether undergoing hemodialysis or PD, the patient may be hospitalized for treatment of complications related to the dialysis treatment, the underlying renal disorder, or health problems not related to renal dysfunction or its treatment.

Protecting Vascular Access

When the patient undergoing hemodialysis is hospitalized for any reason, care must be taken to protect the vascular access. The nurse assesses the vascular access for patency and takes precautions to ensure that the extremity with the vascular access is not used for measuring blood pressure or for obtaining blood specimens; tight dressings, restraints, or jewelry over the vascular access must be avoided as well.

The bruit, or "thrill," over the venous access site must be evaluated at least every 8 hours. Absence of a palpable thrill or audible bruit may indicate blockage or clotting in the vascular access. Clotting can occur if the patient has an infection anywhere in the body (serum viscosity increases) or if the blood pressure has dropped. When blood flow is reduced through the access for any reason (hypotension, application of blood pressure cuff or tourniquet), the access can clot. If a patient has a hemodialysis catheter or implanted hemodialysis access device, the nurse must observe for signs and symptoms of infection such as redness, swelling, drainage from the exit site, fever, and chills. The nurse must assess the integrity of the dressing and change it as needed. Patients with renal disease are more prone to infection; therefore, infection control measures must be used for all procedures.

Taking Precautions During Intravenous Therapy

When the patient needs IV therapy, the rate of administration must be as slow as possible and should be strictly controlled by a volumetric infusion pump. Because patients on dialysis cannot excrete water, rapid or excessive administration of IV fluid can result in pulmonary edema. Accurate intake and output records are essential.

Monitoring Symptoms of Uremia

As metabolic end products accumulate, symptoms of uremia worsen. Patients whose metabolic rate accelerates (those receiving corticosteroid medications or parenteral nutrition, those with infections or bleeding disorders, those undergoing surgery) accumulate waste products more quickly and may require daily dialysis. These same patients are more likely than other patients receiving dialysis to experience complications.

Detecting Cardiac and Respiratory Complications

Cardiac and respiratory assessment must be conducted frequently. As fluid builds up, fluid overload, heart failure, and pulmonary edema develop. Crackles in the bases of the lungs may indicate pulmonary edema.

Pericarditis may result from the accumulation of uremic toxins. If not detected and treated promptly, this serious complication may progress to pericardial effusion and cardiac tamponade. Pericarditis is detected by the patient's report of substernal chest pain (if the patient can communicate), low-grade fever (often overlooked), and pericardial friction rub. A pulsus paradoxus (a decrease in blood pressure of more than 10 mm Hg during inspiration) is often present. When pericarditis progresses to effusion, the friction rub disappears, heart sounds become distant and muffled, ECG waves show very low voltage, and the pulsus paradoxus worsens.

The effusion may progress to life-threatening cardiac tamponade, noted by narrowing of the pulse pressure in

addition to muffled or inaudible heart sounds, crushing chest pain, dyspnea, and hypotension. Although pericarditis, pericardial effusion, and cardiac tamponade can be detected by chest x-ray, they should also be detected through astute nursing assessment. Because of their clinical significance, assessment of the patient for these complications is a priority.

Controlling Electrolyte Levels and Diet

Electrolyte alterations are common, and potassium changes can be life-threatening. All IV solutions and medications to be administered are evaluated for their electrolyte content. Serum laboratory values are assessed daily. If blood transfusions are required, they may be administered during hemodialysis, if possible, so that excess potassium can be removed. Dietary intake must also be monitored. The patient's frustrations related to dietary restrictions typically increase if the hospital food is unappetizing. The nurse needs to recognize that this may lead to dietary indiscretion and hyperkalemia.

Hypoalbuminemia is an indicator of malnutrition in patients undergoing long-term or maintenance dialysis. Although some patients can be treated with adequate nutrition alone, some patients remain hypoalbuminemic for reasons that are poorly understood.

Managing Discomfort and Pain

Complications such as pruritus and pain secondary to neuropathy must be managed. Antihistamine agents, such as diphenhydramine hydrochloride (Benadryl), are commonly used, and analgesic medications may be prescribed. However, because elimination of the metabolites of medications occurs through dialysis rather than through renal excretion, medication dosages may need to be adjusted. Keeping the skin clean and well moisturized using bath oils, superfatted soap, and creams or lotions helps promote comfort and reduce itching. Teaching the patient to keep the nails trimmed to avoid scratching and excoriation also promotes comfort.

Monitoring Blood Pressure

Hypertension in renal failure is common. It is usually the result of fluid overload and, in part, oversecretion of renin. Many patients undergoing dialysis receive some form of antihypertensive therapy and require ongoing teaching about its purpose and adverse effects. The trial-and-error approach that may be necessary to identify the most effective antihypertensive agent and dosage may confuse the patient if no explanation is provided. Antihypertensive agents must be withheld before dialysis to avoid hypotension due to the combined effect of the dialysis and the medication.

Typically these patients require single or multiple antihypertensive agents to achieve normal blood pressure, thus adding to the total number of medications needed on an ongoing basis.

Preventing Infection

Patients with ESRD commonly have low WBC counts (and decreased phagocytic ability), low RBC counts (anemia), and impaired platelet function. Together, these pose a high risk for infection and potential for bleeding after even minor trauma. Preventing and controlling infection are essential because the incidence of infection is high. Infection of the vascular access site and pneumonia are common.

Caring for the Catheter Site

Patients receiving CAPD usually know how to care for the catheter site; however, the hospital stay is an opportunity to assess catheter care technique and correct misperceptions or deviations from recommended technique. Recommended daily or three-or-four-times-weekly routine catheter site care is typically performed during showering or bathing. The exit site should not be submerged in bath water. The most common cleaning method is soap and water; liquid soap is recommended. During care, the nurse and patient need to make sure that the catheter remains secure to avoid tension and trauma. The patient may wear a gauze or semitransparent dressing over the exit site.

Administering Medications

All medications and the dosage prescribed for any patient on dialysis must be closely monitored to avoid those that are toxic to the kidneys and may threaten remaining renal function. Medications are also scrutinized for potassium and magnesium content, because medications containing potassium or magnesium must be avoided. Care must be taken to evaluate all problems and symptoms that the patient reports without automatically attributing them to renal failure or to dialysis therapy.

Providing Psychological Support

Patients undergoing dialysis for a while may begin to reevaluate their status, the treatment modality, their satisfaction with life, and the impact of these factors on their families and support systems. Nurses must provide opportunities for these patients to express their feelings and reactions and to explore options. The decision to begin dialysis does not require that dialysis be continued indefinitely, and it is not uncommon for patients to consider discontinuing treatment. These feelings and reactions must be taken seriously, and the patient should have the opportunity to discuss them with the dialysis team as well as with a psychologist, psychiatrist, psychiatric nurse, trusted friend, or spiritual advisor. The patient's informed decision about discontinuing treatment, after thoughtful deliberation, should be respected.

KIDNEY SURGERY

A patient may undergo surgery to remove obstructions that affect the kidney (tumors or calculi), to insert a tube for draining the kidney (nephrostomy, ureterostomy), or to remove the kidney involved in unilateral kidney disease, renal carcinoma, or kidney transplantation.

Management of Patients Undergoing Kidney Surgery

Preoperative Considerations

Surgery is performed only after a thorough evaluation of renal function. Patient preparation to ensure that optimal renal function is maintained is essential. Fluids are encouraged to promote increased excretion of waste products

before surgery unless contraindicated because of preexisting renal or cardiac dysfunction. If kidney infection is present preoperatively, broad-spectrum antimicrobial agents may be prescribed to prevent bacteremia. Antibiotic agents must be given with extreme care because many are toxic to the kidneys. Coagulation studies (prothrombin time, partial thromboplastin time, platelet count) may be indicated if the patient has a history of bruising and bleeding. The preoperative preparation is similar to that described in Chapter 18.

Because many patients facing kidney surgery are apprehensive, the nurse encourages the patient to recognize and verbalize concerns. Confidence is reinforced by establishing a relationship of trust and by providing expert care. Patients faced with the prospect of losing a kidney may think that they will be dependent on dialysis for the rest of their lives. It is important to teach the patient and family that normal function may be maintained by a single healthy kidney.

Perioperative Concerns

Renal surgery requires various patient positions to expose the surgical site adequately. Three surgical approaches are common: flank, lumbar, and thoracoabdominal (Fig. 44-9). During surgery, plans are carried out for managing altered urinary drainage. These may include inserting a nephrostomy or other drainage tube.

Postoperative Management

Because the kidney is a highly vascular organ, hemorrhage and shock are the chief complications of renal surgery. Fluid and blood component replacement is frequently necessary in the immediate postoperative period to treat intraoperative blood loss.

Abdominal distention and paralytic ileus are fairly common after renal and ureteral surgery and are thought to be due to a reflex paralysis of intestinal peristalsis and manipulation of the colon or duodenum during surgery. Abdominal distention is relieved by decompression through a nasogastric tube (see Chapter 38 for treatment of paralytic ileus). Oral fluids are permitted when the passage of flatus is noted.

If infection occurs, antibiotics are prescribed after a culture reveals the causative organism. The toxic effects that antibiotic agents have on the kidneys (nephrotoxicity) must be kept in mind when assessing the patient. Low-dose heparin therapy may be initiated postoperatively to prevent thromboembolism in patients who had any type of urologic surgery.

Nursing Management

In addition to those interventions listed in this section, Chart 44-12 provides a plan of nursing care for the patient undergoing kidney surgery.

Providing Immediate Postoperative Care

Immediate postoperative care of the patient who has undergone surgery of the kidney includes assessment of all body systems. Respiratory and circulatory status, pain level, fluid and electrolyte status, and patency and adequacy of urinary drainage systems are assessed.

Respiratory Status

As with any surgery, the use of anesthesia increases the risk for respiratory complications. Noting the location of the surgical incision assists the nurse in anticipating respiratory problems and pain. Respiratory status is assessed by monitoring the rate, depth, and pattern of respirations. The location of the incision frequently causes pain on inspiration and coughing; therefore, the patient tends to splint the chest wall and take shallow respirations. Auscultation is performed to assess normal and adventitious breath sounds.

Circulatory Status and Blood Loss

The patient's vital signs and arterial or central venous pressure are monitored. Skin color and temperature and urine output provide information about circulatory status. The surgical incision and drainage tubes are observed frequently to help detect unexpected blood loss and hemorrhage.

Pain

Postoperative pain is a major problem for the patient because of the location of the surgical incision and patient's

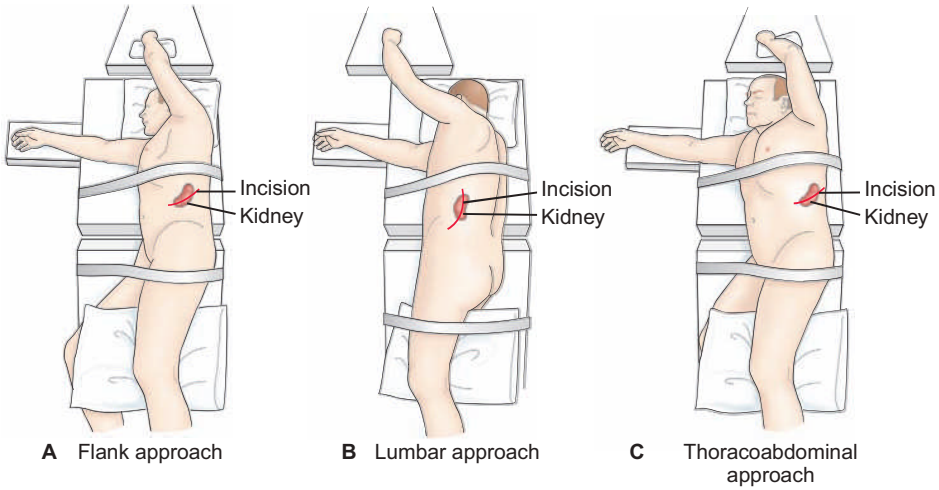

Figure 44-9 Patient positioning and incisional approaches (**A,** flank; **B,** lumbar; **C,** thoracoabdominal) for kidney surgery are associated with significant postoperative discomfort.

CHART 44-12

PLAN OF NURSING CARE
Care of Patient Undergoing Kidney Surgery

NURSING DIAGNOSIS: Ineffective airway clearance related to pain of high abdominal or flank incision, abdominal discomfort, and immobility; risk for ineffective breathing pattern related to high abdominal incision
GOAL: Improved airway clearance

Nursing Interventions	Rationale	Expected Outcomes
1. Administer analgesic agent as prescribed.	1. Enables patient to take deep breaths and cough	• Takes deep breaths and coughs adequately when encouraged and assisted
2. Splint incision with hands or pillow to assist patient in coughing.	2. Splints incision and promotes adequate cough and prevention of atelectasis	• Exhibits respiratory rate of 12–18 breaths/min
3. Assist patient to change positions frequently.	3. Promotes drainage and inflation of all lobes of the lungs	• Exhibits normal breath sounds without adventitious sounds
4. Encourage use of incentive spirometer if indicated or prescribed.	4. Encourages adequate deep breaths	• Exhibits full thoracic excursion without shallow respirations
5. Assist with and encourage early ambulation.	5. Mobilizes pulmonary secretions	• Uses incentive spirometer with encouragement • Splints incision while taking deep breaths and coughing • Reports progressively less pain and discomfort with coughing and deep breaths • Exhibits normal blood gas levels and chest x-ray • Exhibits normal body temperature with no signs of atelectasis or pneumonia on assessment

NURSING DIAGNOSIS: Acute pain and discomfort related to surgical incision, positioning, and stretching of muscles during kidney surgery
GOAL: Relief of pain and discomfort

Nursing Interventions	Rationale	Expected Outcomes
1. Assess level of pain.	1. Provides baseline for later evaluation of pain relief strategies	• Reports relief of severe pain and discomfort
2. Administer analgesic agents as prescribed.	2. Promotes pain relief	• Takes analgesia as prescribed
3. Splint incision with hands or pillow during movement or deep breathing and coughing exercises.	3. Minimizes sensation of pulling or tension on incision and provides sense of support to the patient	• Exercises aching muscles within recommendations
4. Assist and encourage early ambulation.	4. Promotes resumption of muscle activity exercise	• Uses distraction, relaxation exercises, and imagery to relieve pain • Exhibits no behavioral manifestations of pain and discomfort (eg, restlessness, perspiration, verbal expressions of pain) • Participates in deep-breathing and coughing exercises • Gradually increases physical activity and exercise

NURSING DIAGNOSIS: Fear and anxiety related to diagnosis, outcome of surgery, and alteration in urinary function
GOAL: Reduction of fear and anxiety

Nursing Interventions	Rationale	Expected Outcomes
1. Assess patient's anxiety and fear before surgery if possible.	1. Provides a baseline for postoperative assessment	• Verbalizes reactions and feelings to staff
2. Assess patient's knowledge about procedure and expected surgical outcome preoperatively.	2. Provides a basis for further teaching	• Shares reactions and feelings with family or partner
3. Evaluate the meaning of alterations resulting from surgical procedure for the patient and family or partner.	3. Enables understanding of patient's reactions and responses to expected and unexpected results of surgery	• Grieves appropriately for self and for changes in role and function • Identifies information needed to promote own adaptation and coping
4. Encourage patient to verbalize reactions, feelings, and fears.	4. Affirms patient's understanding of and ultimate resolution of feelings and fears	• Participates in activities and events in immediate environment

Continued

CHART 44-12 PLAN OF NURSING CARE

Care of Patient Undergoing Kidney Surgery (Continued)

Nursing Interventions	Rationale	Expected Outcomes
5. Encourage patient to share feelings with spouse or partner.	5. Enables patient and partner to receive mutual support and reduces sense of isolation from each other	• Accepts visit from ostomy group if indicated • Identifies support person or support group
6. Offer and arrange for visit from member of support group (eg, ostomy group, if indicated).	6. Provides support from another person who has encountered the same or a similar surgical procedure and an example of how others have coped with the alteration	
NURSING DIAGNOSIS: Impaired urinary elimination related to urinary drainage; risk for infection related to altered urinary drainage **GOAL:** Maintenance of urinary elimination; infection-free urinary tract		
Nursing Interventions	**Rationale**	**Expected Outcomes**
1. Assess urinary drainage system immediately.	1. Provides basis for further assessment and action	• Exhibits adequate urinary output and patent drainage system
2. Assess adequacy of urinary output and patency of drainage system.	2. Provides baseline	• Exhibits urinary output consistent with fluid intake
3. Use asepsis and hand hygiene when providing care and manipulating drainage system.	3. Prevents or reduces risk of contamination of urinary drainage system	• Demonstrates normal laboratory values: BUN, serum creatinine levels, urine specific gravity, and osmolality
4. Maintain closed urinary drainage system.	4. Reduces risk of bacterial contamination and infection	• Exhibits sterile urine on urine culture
5. If irrigation of the drainage system is necessary, use sterile gloves and sterile irrigating solution and a closed drainage and irrigation system.	5. Permits irrigation when necessary while maintaining closed drainage system, minimizing risk of infection	• Exhibits clear, dilute urine without debris or encrustation in the drainage system
6. If irrigation is necessary and prescribed, perform it gently with sterile saline and the prescribed amount of irrigating fluid.	6. Maintains patency of the catheter or drainage system and prevents sudden increases in pressure in the urinary tract that may cause trauma, pressure on sutures or urinary tract structures, and pain	• States rationale for avoiding manipulation of catheter, drainage, or irrigation system • Exhibits normal placement of urinary stent or ureteral catheters until removed by physician
7. Assist patient in turning and moving in bed and when ambulating to prevent displacement or inadvertent removal of urinary stent or ureteral catheters if in place.	7. Prevents trauma from accidental displacement of urinary stent or ureteral catheter necessitating repeated instrumentation of the urinary tract (eg, cystoscopy) to replace them	• Maintains closed urinary drainage system • Exhibits normal body temperature without signs or symptoms of urinary tract infection
8. Observe urine color, volume, odor, and components.	8. Provides information about adequacy of urine output, condition and patency of drainage system, and debris in urine	• Cleans catheter with soap and water • Consumes adequate fluid intake (6 to 8 glasses of water or more per day, unless contraindicated)
9. Minimize trauma and manipulation of catheter, drainage system, and urethra.	9. Reduces risk of contamination of drainage system and eliminates site of bacterial invasion	• Urinary drainage system remains in place until physician removes or discontinues it
10. Clean catheter gently with soap during bath, avoiding any to-and-fro movement of catheter.	10. Removes debris and encrustations without causing trauma to or contamination of urethra	• Maintains urinary drainage system without infection or obstruction
11. Anchor drainage tube.	11. Prevents movement or slipping of drainage tube, minimizing trauma to and contamination of urethra or catheter	• Maintains urinary diversion as instructed • Maintains self-care so that environment is odor-free
12. Maintain adequate fluid intake.	12. Promotes adequate urine output and prevents urinary stasis	• States rationale for close follow-up and maintains recommended schedule of appointments with health care providers
13. Assist with and encourage early ambulation while ensuring placement of urinary drainage system.	13. Minimizes cardiovascular and pulmonary complications while preventing loss, dislodging, or disruption of drainage system	
14. If patient is to be discharged with urinary drainage system (catheter) in place or a urinary diversion, instruct patient and family member in care.	14. Knowledge and understanding of the drainage system or urinary diversion are essential to prevent infection and other complications.	

Continued on following page

CHART 44-12 PLAN OF NURSING CARE

Care of Patient Undergoing Kidney Surgery (Continued)

NURSING DIAGNOSIS: Risk for imbalanced fluid volume related to surgical fluid loss, altered urinary output, parenteral fluid administration

GOAL: Normal fluid balance will be maintained

Nursing Interventions	Rationale	Expected Outcomes
1. Weigh patient daily.	1. Daily weight is the most sensitive indicator of fluid loss or gain.	• Patient's weight will be within 2–3 lb of patient's baseline.
2. Take accurate intake and output measurements.	2. Detects fluid retention due to poor cardiac or renal output	• Intake that exceeds output will be detected early.
3. Place all parenteral therapy on an infusion pump.	3. Ensures that the patient does not receive excess or insufficient intravenous fluids	• The exact amount of solution is infused with no adverse effects resulting from overinfusion or underinfusion.
4. Monitor amount and characteristics of urine.	4. Assists in early detection of possible complications of surgery or tube insertion	• Urine is clear and absent of blood, pus, or any foreign substances.
5. Monitor vital signs: temperature, pulse, respirations, and blood pressure.	5. When fluid volume or cardiac output is altered, vital signs are affected.	• Temperature, pulse, respiration, and blood pressure are normal.
6. Auscultate heart and lungs every shift.	6. When fluid volume is increased because of poor cardiac or renal output, fluid accumulates in the lungs. Also, heart sounds change as heart failure develops; frequent auscultation ensures early detection.	• Normal heart and lung sounds are present.

position on the operating table to permit access to the kidney. The location and severity of pain are assessed before and after analgesic medications are administered. Abdominal distention, which increases discomfort, is also noted.

Urinary Drainage

Urine output and drainage from tubes inserted during surgery are monitored for amount, color, and type or characteristics. Decreased or absent drainage is promptly reported to the physician because it may indicate obstruction that could cause pain, infection, and disruption of the suture lines.

Monitoring and Managing Potential Complications

Bleeding is a major complication of kidney surgery. If undetected and untreated it can result in hypovolemia and hemorrhagic shock. The nurse's role is to observe for these complications, to report their signs and symptoms, and to administer prescribed parenteral fluids and blood and blood components. Monitoring of vital signs, skin condition, the urinary drainage system, the surgical incision, and the level of consciousness is necessary to detect evidence of bleeding, decreased circulating blood, and fluid volume and cardiac output. Frequent monitoring of vital signs (initially monitored at least at hourly intervals) and urinary output is necessary for early detection of these complications.

If bleeding goes undetected or is not detected promptly, the patient may lose significant amounts of blood and may experience hypoxemia. In addition to hypovolemic shock due to hemorrhage, this type of blood loss may precipitate a myocardial infarction or transient ischemic attack. Bleeding may be suspected when the patient experiences fatigue and when urine output is less than 30 mL/h. As bleeding persists, late signs of hypovolemia occur, such as cool skin, flat neck veins, and change in level of consciousness or responsiveness. Transfusions of blood components are indicated, along with surgical repair of the bleeding vessel.

Pneumonia may be prevented through use of an incentive spirometer, adequate pain control, and early ambulation. Early signs of pneumonia include fever, increased heart and respiratory rates, and adventitious breath sounds.

Preventing infection is the rationale for using asepsis when changing dressings and handling and preparing catheters, other drainage tubes, central venous catheters, and IV catheters for administration of fluids. Insertion sites are monitored closely for signs and symptoms of inflammation: redness, drainage, heat, and pain. Special care must be taken to prevent urinary tract infection, which is associated with the use of indwelling urinary catheters. Catheters and other invasive tubes are removed as soon as they are no longer needed.

Antibiotics are commonly administered postoperatively to prevent infection. If antibiotic agents are prescribed, serum creatinine and BUN values must be monitored closely because many antibiotic agents are toxic to the kidney or can accumulate to toxic levels if renal function is decreased.

Preventing fluid imbalance is critical when caring for a patient undergoing kidney surgery, because both fluid loss and fluid excess are possible adverse effects of the surgery. Fluid loss may occur during surgery as a result of excessive urinary drainage when the obstruction is removed, or it may occur if diuretic agents are used. Such loss may also occur with GI losses, with diarrhea resulting from antibiotic use, or with nasogastric drainage. When postoperative IV therapy is inadequate to match the output or fluids lost, a fluid

deficit results. Fluid excess, or overload, may result from cardiac effects of anesthesia, administration of excessive amounts of fluids, or the patient's inability to excrete fluid because of changes in renal function. Decreased urine output may be an indication of fluid excess.

Astute assessment skills are needed to detect early signs of fluid excess (such as weight gain, pedal edema, urine output below 30 mL/h, and slightly elevated pulmonary wedge pressure, if available) before they become severe (appearance of adventitious breath sounds, shortness of breath).

Fluid excess may be treated with fluid restriction and administration of furosemide (Lasix) or other diuretic agents. If renal insufficiency is present, these medications may prove ineffective; therefore, dialysis may be necessary to prevent heart failure and pulmonary edema.

Deep venous thrombosis (DVT) may occur postoperatively because of surgical manipulation of the iliac vessels during surgery or prolonged immobility. Anti-embolism stockings are applied, and the patient is monitored closely for signs and symptoms of thrombosis and encouraged to exercise the legs. Heparin may be administered postoperatively to reduce the risk of thrombosis.

Promoting Home and Community-Based Care

Teaching Patients Self-Care

If the patient has a drainage system in place, measures are taken to ensure that both the patient and family understand the importance of maintaining the system correctly at home and preventing infection. Verbal and written instructions and guidelines are provided to the patient and family at the time of hospital discharge. The patient may be asked to demonstrate management of the drainage system to ensure understanding. The importance of strategies to prevent postoperative complications (urinary tract infection and obstruction, DVT, atelectasis, and pneumonia) is stressed to the patient and family. Those signs, symptoms, problems, and questions that should be referred to the physician or other primary health care provider are reviewed by the nurse with the patient and family.

Continuing Care

The need for postoperative assessment and care after renal surgery continues regardless of the setting: the home, subacute care unit, outpatient clinic or office, or rehabilitation facility. Referral for home care is indicated for the patient going home with a urinary drainage system in place. During the home visit, the home care nurse reviews the instructions and guidelines given to the patient at hospital discharge. The nurse assesses the patient's ability to carry out the instructions in the home and answers questions that the patient or family has about management of the drainage system and the surgical incision.

Additionally, the home care nurse obtains vital signs and assesses the patient for signs and symptoms of urinary tract infection and obstruction. The nurse also ensures that pain is adequately controlled and that the patient is complying with recommendations. The home care nurse encourages adequate fluid intake and increased levels of activity. Together the nurse, patient, and family review the signs, symptoms, problems, and questions that should be referred to the physician or other primary health care provider. If the patient has a drainage tube in place, the nurse assesses the site and the patency of the system and monitors the patient for complications, such as DVT, bleeding, or pneumonia.

Because it is easy for the patient, family, and health care team to focus on the patient's immediate disorder to the exclusion of other health issues, the nurse reminds the patient and family about the importance of participating in health promotion activities, including health screening.

Kidney Transplantation

Kidney transplantation has become the treatment of choice for most patients with ESRD. During the past 40 years, more than 400,000 kidney transplantations have been performed worldwide, and approximately 9000 are performed in the United States each year. In the United States, there are many more patients on the waiting list for kidney transplantation than there are organ donors (Danovitch, 2005). Patients choose kidney transplantation for various reasons, such as the desire to avoid dialysis or to improve their sense of well-being and the wish to lead a more normal life. Additionally, the cost of maintaining a successful transplantation is one-third the cost of dialysis treatment. Kidney transplantation is an elective procedure, not an emergency life-saving procedure. Therefore, patients should be in the best possible condition prior to transplantation.

Kidney transplantation involves transplanting a kidney from a living donor or deceased donor to a recipient who no longer has renal function (Chart 44-13). A living donor is a person who is alive at the time of donation and may or may not be related to the recipient. A deceased or cadaveric transplant comes from someone who has died and donated his or her organs. Transplantation from well-matched living donors who are related to the patient (those with compatible ABO and human leukocyte antigens) is slightly more successful than from cadaver donors. The success rate further increases if kidney transplantation from a living donor is performed before dialysis is initiated (Danovitch, 2005).

Prior to either receiving or donating an organ, an extensive medical evaluation is performed. Not everyone is suitable

Chart 44-13 • *Kidney Donation*

An inadequate number of available kidneys remains the greatest limitation to treating patients with end-stage renal disease successfully. For those interested in donating a kidney, the National Kidney Foundation provides written information describing the organ donation program and a card specifying the organs to be donated in the event of death.

The organ donation card is signed by the donor and two witnesses and should be carried by the donor at all times. Procurement of an adequate number of kidneys for potential recipients is still a major problem, despite national legislation that requires relatives of deceased patients or patients declared brain-dead to be asked if they would consider organ donation.

In some states in the United States, drivers can indicate their desire to be organ donors on their driver's license application or renewal.

for a kidney transplant. Contraindications include recent malignancy, active or chronic infection, severe irreversible extrarenal disease (eg, inoperable cardiac disease, chronic lung disease, severe peripheral vascular disease), active autoimmune disease (eg, HIV, hepatitis B and C), morbid obesity (body mass index greater than 35), current substance abuse, inability to give informed consent, and history of nonadherence to treatment regimens (Counts, 2008). Donors may be rejected for the same reasons or any condition that is determined to have an impact on the remaining kidney. Examples include hypertension and diabetes mellitus since both are known causes of renal disease. It is imperative when donors are evaluated that serious consideration be given to the overall long-term health of the donor. Every precaution must be taken to ensure that the remaining kidney in the donor will remain healthy. If these conditions are met, the donor should remain healthy after donation and have a normal lifespan. Since one kidney can easily handle the body's needs, no long-term adjustments will need to be made.

The patient's native kidneys are not usually removed. The transplanted kidney is placed in the patient's iliac fossa anterior to the iliac crest because it allows for easier access to the blood supply needed to perfuse the kidney. The ureter of the newly transplanted kidney is transplanted into the bladder or anastomosed to the ureter of the recipient (Fig. 44-10). Once the blood supply has been reestablished to the transplanted kidney in the operating room, urine should begin to flow. The production of urine at this stage is an important indicator of the overall success of the procedure and ultimate long-term outcome.

Preoperative Management

Preoperative management goals include bringing the patient's metabolic state to a level as close to normal as possible, making sure that the patient is free of infection, and preparing the patient for surgery and the postoperative course.

Medical Management

A complete physical examination is performed to detect and treat any conditions that could cause complications after transplantation. Tissue typing, blood typing, and antibody screening are performed to determine compatibility of the tissues and cells of the donor and recipient. Other diagnostic tests must be completed to identify conditions requiring treatment before transplantation. The lower urinary tract is studied to assess bladder neck function and to detect ureteral reflux.

The patient must be free of infection at the time of renal transplantation, because after surgery medications to prevent transplant rejection will be prescribed. These medications suppress the immune response, leaving the patient immunosuppressed and at risk for infection. Therefore, the patient is evaluated and treated for any infections, including gingival (gum) disease and dental caries.

A psychosocial evaluation is conducted to assess the patient's ability to adjust to the transplant, coping styles, social history, social support available, and financial resources. A history of psychiatric illness is important to obtain because psychiatric conditions are often aggravated by the corticosteroids needed for immunosuppression after transplantation. If a dialysis routine has been established, hemodialysis is often performed the day before the scheduled transplantation procedure to optimize the patient's physical status.

Nursing Management

The nursing aspects of preoperative care for the patient undergoing renal transplant are similar to those for patients undergoing other types of kidney or elective abdominal surgery. Preoperative teaching can be conducted in a variety of settings, including the outpatient preadmission area, the hospital, or the transplantation clinic during the preliminary workup phase. Patient teaching addresses postoperative pulmonary hygiene, pain management options, dietary

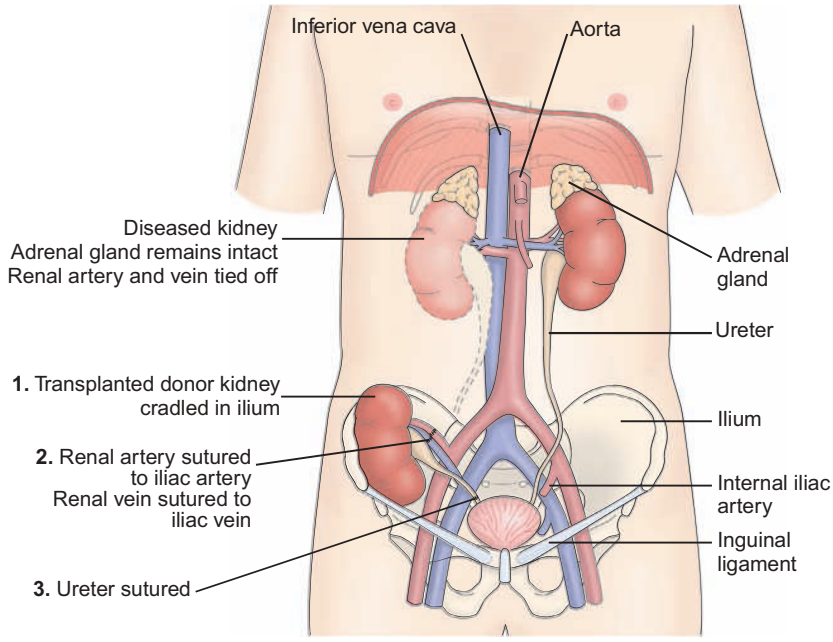

Figure 44-10 Renal transplantation: **1,** The transplanted kidney is placed in the iliac fossa. **2,** The renal artery of the donated kidney is sutured to the ileac artery, and the renal vein is sutured to the iliac vein. **3,** The ureter of the donated kidney is sutured to the bladder or to the patient's ureter.

restrictions, IV and arterial lines, tubes (indwelling catheter and possibly a nasogastric tube), and early ambulation. The patient who receives a kidney from a living related donor may be concerned about the donor and how the donor will tolerate the surgical procedure.

Most patients have been on dialysis for months or years before transplantation. Many have waited months to years for a kidney transplant and are anxious about the surgery, possible rejection, and the need to return to dialysis. Helping the patient to deal with these concerns is part of the nurse's role in preoperative management, as is teaching the patient about what to expect after surgery.

Postoperative Management

The goal of care is to maintain homeostasis until the transplanted kidney is functioning well. The patient whose kidney functions immediately has a more favorable prognosis than the patient whose kidney does not.

Medical Management

After a kidney transplant, rejection and failure can occur within 24 hours (hyperacute), within 3 to 14 days (acute), or after many years. The long-term survival of a transplanted kidney depends on how well it matches the recipient and how well the body's immune response is controlled. Since the body's immune system views the transplanted kidney as "foreign," it continually works to reject it. To overcome or minimize the body's defense mechanisms, immunosuppressive agents are administered. Optimally, medications modify the immune system enough to prevent rejection, but not enough to allow infections or malignancies to occur.

Combinations of glucocorticoids and medications specifically developed to affect the action of lymphocytes are used to minimize the body's reaction to the transplanted organ. Treatment with combinations of new agents has dramatically improved survival rates, and now 90% to 95% of transplanted kidneys still function after 1 year (American Nephrology Nurses Association, 2007b). Doses of immunosuppressive agents are often adjusted depending on the patient's immunologic response to the transplant. However, the patient will be required to take some form of immunosuppressive therapy for the entire time that he or she has the transplanted kidney.

The risks associated with taking these medications include nephrotoxicity, hypertension, hyperlipidemia, hirsutism, tremors, blood dyscrasias, cataracts, gingival hyperplasia, and several types of cancer (American Nephrology Nurses Association, 2006).

Nursing Management

Assessing the Patient for Transplant Rejection

After kidney transplantation, the nurse assesses the patient for signs and symptoms of transplant rejection: oliguria, edema, fever, increasing blood pressure, weight gain, and swelling or tenderness over the transplanted kidney or graft. Patients receiving cyclosporine may not exhibit the usual signs and symptoms of acute rejection. In these patients, the only sign may be an asymptomatic rise in the serum creatinine level (more than a 20% rise is considered acute rejection).

Preventing Infection

The results of blood chemistry tests and leukocyte and platelet counts are monitored closely because immunosuppression depresses the formation of leukocytes and platelets. The patient is closely monitored for infection because of susceptibility to impaired healing and infection related to immunosuppressive therapy and complications of renal failure. Clinical manifestations of infection include shaking chills, fever, rapid heartbeat (tachycardia), and respirations (tachypnea), as well as either an increase or a decrease in WBCs (leukocytosis or leukopenia).

Infection may be introduced through the urinary tract, the respiratory tract, the surgical site, or other sources. Urine cultures are performed frequently because of the high incidence of bacteriuria during early and late stages of transplantation. Any type of wound drainage should be viewed as a potential source of infection because drainage is an excellent culture medium for bacteria. Catheter and drain tips may be cultured when removed by cutting off the tip of the catheter or drain (using aseptic technique) and placing the tip in a sterile container to be taken to the laboratory for culture (Chart 44-14).

The nurse ensures that the patient is protected from exposure to infection by hospital staff, visitors, and other

Chart 44-14 • *Renal Transplant Rejection and Infection*

Renal graft rejection and failure may occur within 24 hours (hyperacute), within 3 to 14 days (acute), or after many years (chronic). It is not uncommon for rejection to occur during the first year after transplantation.

Detecting Rejection

Ultrasonography may be used to detect enlargement of the kidney; percutaneous renal biopsy (most reliable) and x-ray techniques are used to evaluate transplant rejection. If the body rejects the transplanted kidney, the patient needs to return to dialysis. The rejected kidney may or may not be removed, depending on when the rejection occurs (acute versus chronic) and the risk for infection if the kidney is left in place.

Potential Infection

About 75% of kidney transplant recipients have at least one episode of infection in the first year after transplantation because of immunosuppressant therapy. Immunosuppressants of the past made the transplant recipient more vulnerable to opportunistic infections (candidiasis, cytomegalovirus, *Pneumocystis* pneumonia) and infection with other relatively nonpathogenic viruses, fungi, and protozoa, which can be a major hazard. Cyclosporine therapy has reduced the incidence of opportunistic infections because it selectively exerts its effect, sparing T cells that protect the patient from life-threatening infections. In addition, combination immunosuppressant therapy and improved clinical care have produced 1-year patient survival rates approaching 100% and graft survival exceeding 90%. Infections, however, remain a major cause of death at all points in time for kidney transplant recipients (Danovitch, 2005).

patients with active infections. Attention to hand hygiene by all who come in contact with the patient is imperative.

Monitoring Urinary Function

A kidney from a living donor related to the patient usually begins to function immediately after surgery and may produce large quantities of dilute urine. A kidney from a cadaver donor may undergo acute tubular necrosis and therefore may not function for 2 or 3 weeks, during which time anuria, oliguria, or polyuria may be present. During this stage, the patient may experience significant changes in fluid and electrolyte status. Therefore, careful monitoring is indicated. The output from the urinary catheter (connected to a closed drainage system) is measured every hour. IV fluids are administered on the basis of urine volume and serum electrolyte levels and as prescribed by the physician. Hemodialysis may be necessary postoperatively to maintain homeostasis until the transplanted kidney is functioning well. It also may be required if fluid overload and hyperkalemia occur. After successful renal transplantation, the vascular access device may clot, possibly from improved coagulation with the return of renal function. The vascular access for hemodialysis is monitored to ensure patency and to evaluate for evidence of infection.

Addressing Psychological Concerns

The rejection of a transplanted kidney is of great concern to the patient, the family, and the health care team for many months. The fear of kidney rejection and the complications of immunosuppressive therapy (Cushing's syndrome, diabetes, capillary fragility, osteoporosis, glaucoma, cataracts, acne, nephrotoxicity) place tremendous psychological stress on the patient. Anxiety and uncertainty about the future and difficult posttransplantation adjustment are often sources of stress for the patient and family.

An important nursing function is the assessment of the patient's stress and coping. The nurse uses each visit with the patient to determine if the patient and family are coping effectively and the patient is adhering to the prescribed medication regimen. If indicated or requested, the nurse refers the patient for counseling.

Monitoring and Managing Potential Complications

The patient undergoing kidney transplantation is at risk for the postoperative complications that are associated with any surgical procedure. In addition, the patient's physical condition may be compromised because of the effects of long-standing renal failure and its treatment. Therefore, careful assessment for the complications related to renal failure and those associated with a major surgery are important aspects of nursing care. Breathing exercises, early ambulation, and care of the surgical incision are important aspects of postoperative care.

GI ulceration and corticosteroid-induced bleeding may occur. Fungal colonization of the GI tract (especially the mouth) and urinary bladder may occur secondary to corticosteroid and antibiotic therapy. Closely monitoring the patient and notifying the physician about the occurrence of these complications are important nursing interventions. In addition, the patient is monitored closely for signs and symptoms of adrenal insufficiency if the treatment has included use of corticosteroids.

Promoting Home and Community-Based Care

Teaching Patients Self-Care. The nurse works closely with the patient and family to be sure that they understand the need for continuing immunosuppressive therapy as prescribed. Additionally, the patient and family are instructed to assess for and report signs and symptoms of transplant rejection, infection, or significant adverse effects of the immunosuppressive regimen. These include decreased urine output; weight gain; malaise; fever; respiratory distress; tenderness over the transplanted kidney; anxiety; depression; changes in eating, drinking, or other habits; and changes in blood pressure. The patient is instructed to inform other health care providers (eg, dentist) about the kidney transplant and the use of immunosuppressive agents.

Continuing Care. The patient needs to know that follow-up care after transplantation is a lifelong necessity. Individual verbal and written instructions are provided concerning diet, medication, fluids, daily weight, daily measurement of urine, management of I&O, prevention of infection, resumption of activity, and avoidance of contact sports in which the transplanted kidney may be injured. Because of the risk for other potential complications, the patient is followed closely.

Cardiovascular disease is the major cause of morbidity and mortality after transplantation, due in part to the increasing age of patients with transplants. An additional problem is possible malignancy; patients receiving long-term immunosuppressive therapy are at higher risk for cancers than the general population. So the patient is reminded of the importance of health promotion and health screening.

The American Association of Kidney Patients (listed at the end of this chapter) is a nonprofit organization that serves the needs of those with kidney disease. It can provide many helpful suggestions for patients and family members learning to cope with dialysis and transplantation.

RENAL TRAUMA

The kidneys are protected by the rib cage and musculature of the back posteriorly and by a cushion of abdominal wall and viscera anteriorly. They are highly mobile and are fixed only at the renal pedicle (stem of renal blood vessels and the ureter). With traumatic injury, the kidneys can be thrust against the lower ribs, resulting in contusion and rupture. Rib fractures or fractures of the transverse process of the upper lumbar vertebrae may be associated with renal contusion or laceration. Failure to wear seat belts contributes to the incidence of renal trauma in motor vehicle crashes. Up to 80% of patients with renal trauma have associated injuries of other internal organs.

Injuries may be blunt (automobile and motorcycle crashes, falls, athletic injuries, assaults) or penetrating (gunshot wounds, stabbings). Blunt renal trauma accounts for 80% to 90% of all renal injuries; penetrating renal trauma accounts for the remaining 10% to 20%.

Blunt renal trauma is classified into one of four groups, as follows:

- Contusion: bruises or hemorrhages under the renal capsule; capsule and collecting system intact

- Minor laceration: superficial disruption of the cortex; renal medulla and collecting system are not involved
- Major laceration: parenchymal disruption extending into cortex and medulla, possibly involving the collecting system
- Vascular injury: tears of renal artery or vein

The most common renal injuries are contusions, lacerations, ruptures, and renal pedicle injuries or small internal lacerations of the kidney (Fig. 44-11). The kidneys receive half of the blood flow from the abdominal aorta; therefore, even a fairly small renal laceration can produce massive bleeding. About 70% of patients are in shock when admitted to the hospital. In some cases, there is an isolated renal artery thrombosis.

Clinical manifestations include pain, renal colic (due to blood clots or fragments obstructing the collecting system), hematuria, mass or swelling in the flank, ecchymoses, and lacerations or wounds of the lateral abdomen and flank. Hematuria is the most common manifestation of renal trauma; its presence after trauma suggests renal injury. There is no relationship between the degree of hematuria and the degree of injury. Hematuria may not occur, or it may be detectable only on microscopic examination. Signs and symptoms of hypovolemia and shock are likely with significant hemorrhage.

Medical Management

The goals of management in patients with renal trauma are to control hemorrhage, pain, and infection as well as to preserve and restore renal function. All urine is saved and sent to the laboratory for analysis to detect RBCs and to evaluate the course of bleeding. Hematocrit and hemoglobin levels are monitored closely; decreasing values indicate hemorrhage.

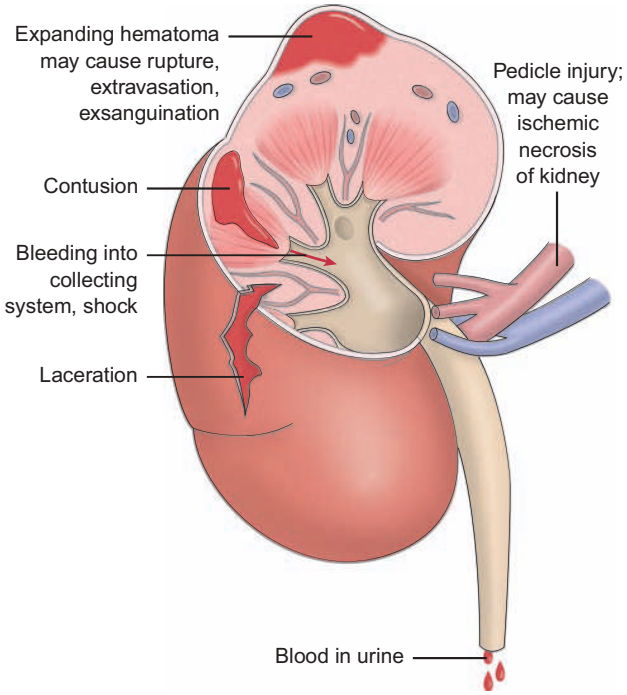

Figure 44-11 Types and pathophysiologic effects of renal injuries: contusions, lacerations, rupture, and pedicle injury.

The patient is monitored for oliguria and signs of hemorrhagic shock, because a pedicle injury or shattered kidney can lead to rapid exsanguination (lethal blood loss). An expanding hematoma may cause rupture of the kidney capsule. To detect hematoma, the area around the lower ribs, upper lumbar vertebrae, flank, and abdomen is palpated for tenderness. A palpable flank or abdominal mass with local tenderness, swelling, and ecchymosis suggests renal hemorrhage. The area of the original mass can be outlined with a marking pen so that the examiner can evaluate the area for change.

Renal trauma is often associated with other injuries to the abdominal organs (liver, colon, small intestines); therefore, the patient is assessed for skin abrasions, lacerations, and entry and exit wounds of the upper abdomen and lower thorax because these may be associated with renal injury.

With a contusion of the kidney, healing may take place with conservative measures. If the patient has microscopic hematuria and a normal IV urogram, outpatient management is possible. If gross hematuria or a minor laceration is present, the patient is hospitalized and kept on bed rest until the hematuria clears. Antimicrobial medications may be prescribed to prevent infection from perirenal hematoma or urinoma (a cyst containing urine). Patients with retroperitoneal hematomas may develop low-grade fever as absorption of the clot takes place.

Surgical Management

In renal trauma, any sudden change in the patient's condition suggests hemorrhage and requires rapid surgical intervention. Depending on the patient's condition and the nature of the injury, major lacerations may be treated through surgical intervention or conservatively (bed rest, no surgery). Vascular injuries require immediate exploratory surgery because of the high incidence of involvement of other organ systems and the serious complications that may result if these injuries are untreated. The patient is often in shock and requires aggressive fluid resuscitation. The damaged kidney may have to be removed (nephrectomy).

Early postoperative complications (within 6 months) include rebleeding, perinephritic abscess formation, sepsis, urine extravasation, and fistula formation. Other complications include stone formation, infection, cysts, vascular aneurysms, and loss of renal function. Hypertension can be a complication of any renal surgery but usually is a late complication of renal injury.

Nursing Management

The patient with renal trauma must be assessed frequently during the first few days after injury to detect flank and abdominal pain, muscle spasm, and swelling over the flank. During this time, the patient who has undergone surgery is instructed about care of the incision and the importance of an adequate fluid intake. In addition, instructions about changes that should be reported to the physician, such as fever, hematuria, flank pain, or any signs and symptoms of decreasing kidney function, are provided. Guidelines for gradually increasing activity, lifting, and driving are also provided in accordance with the physician's prescription.

Follow-up nursing care includes monitoring the blood pressure to detect hypertension and advising the patient to

restrict activities for about 1 month after trauma to minimize the incidence of delayed or secondary bleeding. The patient should be advised to schedule periodic follow-up assessments of renal function (creatinine clearance, BUN and serum creatinine analyses). If a nephrectomy was necessary, the patient is advised to wear medical identification.

CRITICAL THINKING EXERCISES

1 You are a staff nurse in an outpatient dialysis facility. A 50-year-old woman with ESRD is scheduled to be seen in the clinic; it is anticipated that she will need dialysis in the near future. The patient lives alone and will require teaching about the dialysis options. Develop a teaching plan to explain the different types of dialysis, goals, and level of involvement on the part of the patient.

EBP **2** A 45-year-old married man visits the nephrology department to discuss options for dealing with his ESRD. His brother has begun the workup to donate one of his kidneys and the preliminary reports show that a match is possible. The patient states that he does not want his brother to go through the process of kidney donation if dialysis is possible. Identify the evidence for and the criteria used to evaluate the strength of the evidence for dialysis compared to kidney transplantation.

3 You are caring for a 35-year-old woman who has been recently diagnosed with renal cancer. Identify possible treatment options. What nursing assessment and interventions should you make at this time? What explanations would you give the patient about renal cancer?

4 A 55-year-old man who is blind has just had a catheter placed for PD. His wife, his primary caretaker, is deaf. Develop a teaching plan to explain peritoneal dialysis, goals, and level of involvement to the patient and family.

The Smeltzer suite offers these additional resources to enhance learning and facilitate understanding of this chapter:

- thePoint online resource, thepoint.lww.com/Smeltzer12E
- Student CD-ROM included with the book
- *Study Guide to Accompany Brunner & Suddarth's Textbook of Medical-Surgical Nursing*
- *Handbook for Brunner & Suddarth's Textbook of Medical-Surgical Nursing*

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chapter 52

Management of Patients With HIV Infection and AIDS

LEARNING OBJECTIVES

On completion of this chapter, the learner will be able to:

1. Describe the modes of transmission of HIV infection and prevention strategies.
2. Describe the host/HIV interaction during primary infection.
3. Explain the pathophysiology associated with the clinical manifestations of HIV/AIDS.
4. Describe the clinical management of patients with HIV/AIDS.
5. Discuss the nursing interventions appropriate for patients with HIV/AIDS.
6. Use the nursing process as a framework for care of the patient with HIV/AIDS.

GLOSSARY

alpha-interferon: protein substance that the body produces in response to infection
B-cell lymphoma: common malignancy in patients with HIV/AIDS
candidiasis: yeast infection of skin or mucous membrane
CCR5: along with the CD4+ receptor, this cell surface molecule is used by HIV to fuse with the host's cell membranes
cytomegalovirus: a species-specific herpes virus that may cause retinitis in people with AIDS
EIA (enzyme immunoassay): a blood test that can determine the presence of antibodies to HIV in the blood or saliva; also referred to as **enzyme-linked immunosorbent assay (ELISA)**. Positive results must be validated, usually with Western blot test.
HIV-1: retrovirus isolated and recognized as the etiologic agent of AIDS
HIV-2: retrovirus identified in 1986 in AIDS patients in West Africa
HIV encephalopathy: degenerative neurologic condition characterized by a group of clinical presentations including loss of coordination, mood swings, loss of inhibitions, and widespread cognitive dysfunctions; formerly referred to as AIDS dementia complex (ADC)
human papillomavirus (HPV): viruses that cause various warts, including plantar and genital warts; some strains of HPV can also cause cervical cancer
immune reconstitution inflammatory syndrome: a syndrome that results from rapid restoration of pathogen-specific immune responses to opportunistic infections; most often occurs after starting antiretroviral therapy
Kaposi's sarcoma: malignancy that involves the epithelial layer of blood and lymphatic vessels
latent reservoir: the integrated HIV provirus within the CD4+ T cell during the resting memory state; does not express viral proteins and is invisible to the immune system and antiviral medications.

GLOSSARY *(Continued)*

macrophage: large immune cell that devours invading pathogens and other intruders; can harbor large quantities of HIV without being killed, acting as a reservoir of the virus
monocyte: large white blood cell that ingests microbes or other cells and foreign particles. When a monocyte enters tissues, it develops into a macrophage.
***Mycobacterium avium* complex:** opportunistic infection caused by mycobacterial organisms that commonly causes a respiratory illness but can also infect other body systems
opportunistic infection: illness caused by various organisms, some of which usually do not cause disease in people with normal immune systems
p24 antigen: blood test that measures viral core protein; accuracy of test is limited because the p24 antibody binds with the antigen and makes it undetectable
peripheral neuropathy: disorder characterized by sensory loss, pain, muscle weakness, and wasting of muscles in the hands or legs and feet
***Pneumocystis* pneumonia or *Pneumocystis jiroveci* pneumonia (PCP):** common opportunistic lung infection caused by an organism, believed to be a fungus based on its structure
polymerase chain reaction: a sensitive laboratory technique that can detect and quantify HIV in a person's blood or lymph nodes
primary infection: 4- to 7-week period of rapid viral replication immediately following infection; also known as acute HIV infection
progressive multifocal leukoencephalopathy: opportunistic infection that infects brain tissue and causes damage to the brain and spinal cord
protease inhibitor: medication that inhibits the function of protease, an enzyme needed for HIV replication
provirus: viral genetic material in the form of DNA that has been integrated into the host genome. When it is dormant in human cells, HIV is in a proviral form.
retrovirus: a virus that carries genetic material in RNA instead of DNA and contains reverse transcriptase
reverse transcriptase: enzyme that transforms single-stranded RNA into a double-stranded DNA
viral load test: measures the quantity of HIV RNA in the blood
viral set point: amount of virus present in the blood after the initial burst of viremia and the immune response that follows
wasting syndrome: involuntary weight loss of 10% of baseline body weight with chronic diarrhea or chronic weakness and documented fever
Western blot assay: a blood test that identifies antibodies to HIV and is used to confirm the results of an EIA (ELISA) test
window period: time from infection with HIV until seroconversion detected on HIV antibody test

Although advances have been made in treating human immunodeficiency virus (HIV) infection and acquired immunodeficiency syndrome (AIDS), the epidemic remains a critical public health issue in all communities across the country and around the world. Prevention, early detection, and ongoing treatment remain important aspects of care for people with HIV infection and AIDS. Nurses in all settings encounter people who are positive for HIV infection; therefore, nurses need an understanding of the pathophysiology, knowledge of the physical and psychological consequences associated with the diagnosis, and expert assessment and clinical management skills to provide optimal care for people with HIV infection and AIDS.

In 1987, just 6 years after the first cases of AIDS were reported, the U.S. Food and Drug Administration (FDA) approved the first antiretroviral agent. In 1988, the first randomized controlled trial of primary prophylaxis of *Pneumocystis jiroveci* pneumonia (PCP; formerly *Pneumocystis carinii* pneumonia) appeared in the literature. Currently, more than 25 antiviral agents are approved for use in the United States (Eron, 2008); these agents interfere with the life cycle of HIV in variety of ways. HIV/AIDS is a chronic condition that requires daily medication. Although damage to the immune system is significant, survival rates have increased dramatically.

HIV INFECTION AND AIDS

Since AIDS was first identified almost 30 years ago, remarkable progress has been made in improving the quality and duration of life for people living with HIV disease. During the first decade, this progress was associated with the recognition and treatment of opportunistic diseases and introduction of prophylaxis against common **opportunistic infections.** The second decade witnessed progress in the development of highly active antiretroviral therapies (HAARTs) as well as continuing progress in the treatment of opportunistic infections. The third decade has focused on issues of adherence to antiretroviral therapy, development of second-generation medications that affect different stages of the viral life cycle, and continued need for an effective vaccine. The HIV antibody test, an enzyme immunoassay (EIA; formerly enzyme-linked immunosorbent assay [ELISA]), became available in 1984, allowing early diagnosis of the infection before onset of symptoms. Since then, HIV infection has been best managed as a chronic disease, most appropriately in an outpatient care setting, whereas AIDS may involve acute conditions that require hospital treatment.

Epidemiology

In the fall of 1982, after the first 100 cases were reported, the Centers for Disease Control and Prevention (CDC) issued a case definition of AIDS. Since then, the CDC has revised the case definition a number of times (1985, 1987, and 1993). All 50 states, the District of Columbia, U.S. dependencies and possessions, and independent nations in free association with the United States report AIDS cases to the CDC using a uniform surveillance case definition and case report form (CDC, 2005). Starting in the late 1990s, many more states began to implement HIV case reporting in response to the changing epidemic and the need for information on the numbers and characteristics of people with HIV infection who had not yet developed AIDS. The demographic characteristics of people with HIV and AIDS are changing; increasing numbers of women, non-Hispanic blacks, residents of the southern United States, and adults older than 45 years of age are dying from AIDS (Coates, 2008).

The CDC now uses a new and precise method to identify cases of HIV. In 2008, the CDC reported that approximately 56,300 new HIV infections occurred in the United States in 2006. This figure was roughly 40% higher than their former estimate of 40,000 HIV infections per year, which was based on limited data and less precise methods (CDC, 2008).

Almost 7000 people around the world still contract HIV infection every day (McConnell, 2008). Although the global percentage of people living with HIV has stabilized since 2000, the level remains unacceptably high. An estimated 33 million people are living with HIV/AIDS; however, the number of new infections declined from 3 million in 2001 to 2.7 million in 2007. Globally, the percentage of women among people with HIV/AIDS remains stable at 50% but is increasing in several countries (Joint United Nations Programme on HIV/AIDS [UNAIDS], 2008). Although there has been considerable progress worldwide in providing treatment to people with HIV/AIDS, there are still significant gaps. Sub-Saharan Africa continues to be most heavily affected by HIV/AIDS, with 67% of all people living with the disease. In 2007, 72% of the deaths from HIV/AIDS occurred in this region (UNAIDS, 2008).

HIV Transmission

HIV-1 is transmitted in body fluids that contain free virions and infected CD4+ T cells. These fluids include blood, seminal fluid, vaginal secretions, amniotic fluid, and breast milk. Inflammation and breaks in the skin or mucosa result in the increased probability that an exposure to HIV will lead to infection. The amount of HIV and infected cells in the body fluid is associated with the probability that the exposure will result in infection. Mother-to-child transmission of HIV-1 may occur in utero, at the time of delivery, or through breast-feeding, but most perinatal infections are thought to occur after exposure during delivery. HIV is not transmitted through casual contact (Chart 52-1).

Blood and blood products can transmit HIV to recipients. However, the risk associated with transfusions has been virtually eliminated as a result of voluntary self-deferral, completion of a detailed health history, extensive testing, heat treatment of clotting factor concentrates, and more

CHART 52-1 *Risk Behaviors Associated With HIV Infection and AIDS*

- Sharing infected injection drug use equipment
- Having sexual relations with infected individuals (both male and female)

Also at risk are people who received HIV-infected blood or blood products (especially before blood screening was instituted in 1985) and infants born to mothers with HIV infection.

effective virus inactivation methods. Donated blood is tested for antibodies to HIV-1, **HIV-2,** and **p24 antigen;** in addition, since 1999, nucleic acid amplification testing (NAT) has been performed.

Gerontologic Considerations

The number of people between the ages of 55 and 64 years living with AIDS more than doubled between the years 1998 and 2003 (from 19,258 to 38,997) (CDC, 2005). HIV infection in middle-aged and older populations may be underreported and underdiagnosed because health care professionals erroneously believe that older adults are not at risk for HIV infection. Also, HIV-related dementia in the older adult may mimic Alzheimer's disease and may be misdiagnosed. The characteristics of older people living with HIV infection reflect those of others in their country of origin who have HIV infection (Nokes, Rivero-Mendez, Valencia, et al., 2006).

Several factors put older adults at risk for HIV infection:

- Many older adults are sexually active but do not use condoms, viewing them only as a means of unneeded birth control.
- Many older adults do not consider themselves at risk for HIV infection.
- Older gay men, who grew up and lived in an era when disclosure of their sexual orientation was not acceptable and who have lost long-time partners, may begin new relationships with younger men.
- Older adults may be intravenous (IV)/injection drug users.
- Older adults may have received HIV-infected blood through transfusions before 1985.
- Normal age-related changes include a reduction in immune system function, which puts the older adult at greater risk for infections, cancers, and autoimmune disorders. Many older adults also experience the loss of loved ones, resulting in depression and bereavement, factors that are associated with depressed immune function.

Prevention of HIV Infection

Until an effective vaccine is developed, nurses need to prevent HIV infection by teaching patients how to eliminate or reduce risky behaviors. Although HIV prevention strategies have focused on individual behaviors, political, economic, and social determinants of risk also need to be considered (Horton & Das, 2008). A combination of evidence-based prevention strategies that include behavioral, structural, and biomedical approaches tempered by the wisdom and ownership of communities offers the best hope for prevention (Merson, O'Malley, Serwadda, et al., 2008).

Preventive Education

Evidence-based programs have been used to educate the public regarding safer sexual practices to decrease the risk of transmitting HIV infection to sexual partners (Chart 52-2). The CDC, through the HIV/AIDS Prevention Research Synthesis Project, has identified evidence-based behavioral interventions that can be applied in a number of settings. Other than abstinence, consistent and correct use of condoms (Chart 52-3) is the only effective method to decrease the risk of sexual transmission of HIV infection. When male condoms are used consistently during vaginal or anal intercourse, their effectiveness can be as high as 95% (Padian, Buve, Balkus, et al., 2008). Nonlatex condoms made of natural materials such as lambskin are available for people with latex allergy but will not protect against HIV infection. A male condom should be used for oral contact with the penis, and a dental dam (a flat piece of latex used by dentists to isolate a tooth for treatment) should be used for oral contact with the vagina or rectum. The polyurethane female condom, which is an effective contraceptive, provides a physical barrier that also prevents exposure to genital secretions containing HIV such as semen and vaginal fluid (Padian, et al., 2008). The female condom is the only barrier method that can be controlled by the woman (see Chapter 46).

Microbicides are chemical products such as gels, creams, films, or suppositories that are inserted into the vagina or

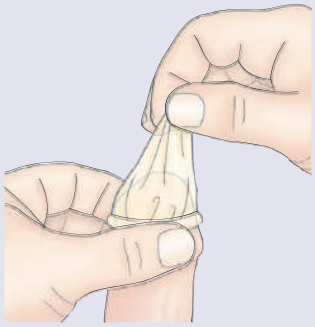
CHART 52-2 HEALTH PROMOTION
Safer Sexual Behaviors

- Advise patients to abstain from sharing sexual fluids.
- Advise patients to reduce the number of sexual partners to one.
- Advise patients to always use latex condoms. If the patient is allergic to latex, nonlatex condoms should be used.
- Advise patients to avoid reusing condoms.
- Advise patients to avoid using cervical caps or diaphragms without using a condom as well.
- Advise patients to always use dental dams for oral– genital or anal stimulation.
- Advise patients to avoid anal intercourse because this practice may injure tissues.
- Advise patients to avoid manual–anal intercourse ("fisting").
- Advise patients not to ingest urine or semen.
- Educate patients about nonpenetrative sexual activities, such as body massage, social kissing (dry), mutual masturbation, fantasy, and sex films.
- Advise patients to avoid sharing needles, razors, toothbrushes, sex toys, or blood-contaminated articles.
- Advise HIV-seropositive patients to inform previous, present, and prospective sexual and drug-using partners of their HIV-positive status. If the patient is concerned for his or her safety, advise the patient that many states have established mechanisms through the public health department in which professionals are available to notify exposed people.
- Advise HIV-seropositive patients to avoid having unprotected sex with another HIV-seropositive person. Cross-infection with that person's HIV can increase the severity of infection.
- Advise HIV-seropositive patients to avoid donating blood, plasma, body organs, or sperm.

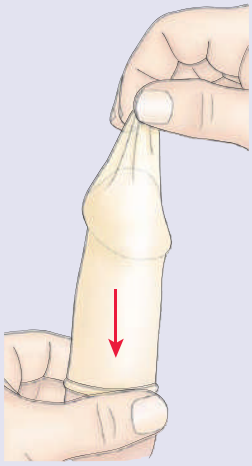
CHART 52-3 PATIENT EDUCATION

The Right Way to Use a Male Condom

1. Put on a new condom before any kind of sex.
2. Hold the condom by the tip to squeeze out the air.



3. Unroll the condom all the way over the erect penis



4. Have sex.
5. Hold the condom so it cannot come off the penis.
6. Pull out.
7. Use a new condom if you want to have sex again or if you want to have sex in a different place (eg, in the anus and then in the vagina).

   Keep condoms cool and dry. Never use skin lotions, baby oil, petroleum jelly, or cold cream with condoms. The oil in these products will cause the condom to break. Products made with water (such as K-Y jelly or glycerin) are safer to use.

rectum before sexual intercourse to prevent HIV transmission (Padian, et al., 2008). Nonoxynol-9 (N-9) was widely advocated to reduce the risk of HIV infection until a clinical trial conducted in almost 1000 female commercial sex workers in African countries revealed that those who used N-9 intravaginally along with condoms were 50% more likely to be infected with HIV than those who did not use the N-9 gel. Microbicide research is moving in three directions: (1) new microbicides that contain antiretroviral agents such as tenofovir gel or dapivirine gel and ring (International Partnership for Microbicides, 2009); (2) long-acting dispersal methods such as vaginal rings that remain in place or do not require frequent applications; and (3) combination products with different mechanisms of action (Padian, et al., 2008).

In March 2007, based on the results of three clinical trials, the World Health Organization (WHO) and UNAIDS recommended that circumcision be recognized as an effective strategy to reduce the risk of HIV acquisition in men because the presence of the foreskin, which harbors HIV target cells, might facilitate survival and entry of the virus (Padian, et al., 2008). Other topics important in preventive education include the importance of avoiding sexual practices that might cut or tear the lining of the rectum, penis, or vagina and avoiding sexual contact with multiple partners or people who are known to be HIV positive or IV/injection drug users. In addition, people who are HIV positive or who use injection drugs should be instructed not to donate blood or share drug equipment with others.

The Harm Reduction Model recognizes that total abstinence from addictive drugs might not be a realistic short-term goal. This model recommends working with drug users to assist them to increase their healthy behaviors. Needle exchange programs are available in some locations so that IV/injection drug users can obtain sterile drug equipment at no cost. Extensive research has demonstrated that needle exchange programs do not promote increased drug use; on the contrary, they have been found to decrease the incidence of bloodborne infections in people who use IV/injection drugs. Nurses should refer clients to needle exchange programs in their neighborhood whenever available. In the absence of needle exchange programs, IV/injection drug users should be instructed on methods to clean their syringes and advised to avoid sharing cotton and other drug use equipment. Drug users interested in treatment programs should be referred to those programs.

Related Reproductive Education

Because HIV infection in women often occurs during the childbearing years, family planning issues need to be addressed. Attempts to achieve pregnancy by couples in which only one partner has HIV expose the unaffected partner to the virus. Efforts at artificial insemination using processed semen from an HIV-infected partner continue. Studies are needed, because HIV has been found in the spermatozoa of patients with AIDS, and it is possible that HIV can replicate in the male germ cell. Women considering pregnancy need to have accurate information about the risks of transmitting HIV infection to themselves, their partner, and their future children, and about the benefits of taking antiretroviral agents to reduce perinatal HIV transmission. Women who are HIV positive should be instructed not to breast-feed their infants, because HIV is transmitted through breast milk.

Certain contraceptive methods may pose additional health risks for women. Estrogen in oral contraceptives may increase a woman's risk of HIV infection. In addition, HIV-infected women who use estrogen-containing oral contraceptives have shown increased shedding of HIV in vaginal and cervical secretions. The intrauterine contraceptive device (IUD) may also increase the risk of HIV transmission because the string of the IUD may serve as a means to transmit the virus.

Transmission to Health Care Providers

Standard Precautions

To reduce the risk of exposure of health care workers to HIV, the CDC developed standard precautions (Chart 52-4). Specifically, standard precautions are designed to reduce the risk of transmission of bloodborne pathogens and of pathogens from moist body substances. Standard precautions are used when working with all patients in all health care settings, regardless of their diagnosis or presumed infectious status (Siegel, Rhinehart, Jackson, et al., 2007).

Postexposure Prophylaxis for Health Care Providers

Postexposure prophylaxis in response to the exposure of health care personnel to blood or other body fluids reduces the risk of HIV infection. The CDC recommends that all health care providers who have sustained a significant exposure to HIV be counseled and offered anti-HIV postexposure prophylaxis, if appropriate (Chart 52-5). Some clinicians are considering the use of postexposure prophylaxis for patients exposed to HIV as a result of high-risk sexual behavior or IV/injection drug use. This use of postexposure prophylaxis is controversial because of concern that it may be substituted for safer sex practices and safer IV/injection drug use. However, it may be advocated in high-risk situations, especially when the uninfected person has few options because of economic constraints and cultural norms.

Guidelines for treatment of health care workers with possible occupational exposure to HIV are available from the CDC. Treatment should be started as quickly as possible after exposure. Those who choose postexposure prophylaxis must be prepared for the side effects of the medications, as well as the unknown long-term risks, because HIV often becomes resistant to the medications used to treat it. The cost may be of concern; the cost of a medication regimen ranges from $500 to more than $1000, in addition to the costs of testing and counseling. However, the health care institution where the provider is employed usually covers these costs.

Vaccination

Despite enormous international efforts to develop an effective vaccine, the complexity of the science and challenges of HIV are proving formidable obstacles. Hopes were dashed when a vaccine efficacy trial was halted in September 2007, after interim results showed that the vaccine being tested did not protect against HIV and did not reduce viral load after infection (Senior, 2008). Cooperation among all nations continues to grow, and resources are being allocated to develop a vaccine and to create and support the infrastructure needed to facilitate vaccine testing.

Pathophysiology

Because HIV infection is an infectious disease, it is important to understand how HIV-1 integrates itself into a person's immune system and how immunity plays a role in the course of HIV disease. This knowledge is also essential for understanding medication therapy and vaccine development.

Viruses are intracellular parasites. HIV belongs to a group of viruses known as **retroviruses,** which carry their genetic material in the form of ribonucleic acid (RNA) rather than deoxyribonucleic acid (DNA). As shown in Figure 52-1A, HIV consists of a viral core containing the viral RNA, surrounded by an envelope consisting of protruding glycoproteins.

Chart 52-4 • *Recommendations for Standard Precautions*

1. **Hand hygiene:** Use after touching blood, body fluids, secretions, excretions, or contaminated items; immediately after removing gloves; and between patient contacts.
2. **Personal protective equipment (PPE)**
   - Gloves: Use for touching blood, body fluids, secretions, excretions, and contaminated items, and for touching mucous membranes and nonintact skin.
   - Gown: Use during procedures and patient care activities when contact of clothing/exposed skin with blood or body fluids, secretions, and excretions is anticipated.
   - Mask, eye protection (goggles), face shield*: Use during procedures and patient care activities likely to generate splashes or sprays of blood, body fluids, and secretions, especially suctioning or endotracheal intubation.
3. **Soiled patient care equipment:** Handle in a manner that prevents transfer of microorganisms to others and to the environment; wear gloves if visibly contaminated; and perform hand hygiene.
4. **Environmental control:** Develop procedures for routine care, cleaning, and disinfection of environmental surfaces, especially frequently touched surfaces in patient care areas.
5. **Textiles and laundry:** Handle in a manner that prevents transfer of microorganisms to others and to the environment.

   Needles and other sharps: Do not recap, bend, break, or hand-manipulate used needles; if recapping is required, use a one-handed scoop technique only; use safety features when available; and place used sharps in a puncture-resistant container.
6. **Patient resuscitation:** Use mouthpiece, resuscitation bag, and other ventilation devices to prevent contact with mouth and oral secretions.
7. **Patient placement:** Prioritize for single-patient room if patient is at increased risk of transmission, is likely to contaminate the environment, does not maintain appropriate hygiene, or is at increased risk of acquiring infection or developing adverse outcome following infection.
8. **Respiratory hygiene/cough etiquette** (source containment of infectious respiratory secretions in symptomatic patients, beginning at initial point of encounter, such as triage and reception areas in emergency departments and physician offices): Instruct symptomatic people to cover mouth and nose when sneezing or coughing; use tissues and dispose in no-touch receptacle; observe hand hygiene after soiling of hands with respiratory secretions; and wear surgical mask if tolerated.

*During aerosol-generating procedures on patients with suspected or proven infections transmitted by respiratory aerosols (eg, severe acute respiratory syndrome [SARS]), wear a fit-tested N95 or higher respirator in addition to gloves, gown, and face/eye protection.
From the Centers for Disease Control and Prevention. www.cdc.gov/ncidod/dhqp/pdf/guidelines/Isolation2007.pdf

Chart 52-5 • *Postexposure Prophylaxis for Health Care Providers*

According to the Centers for Disease Control and Prevention (CDC; 2005), the average risk for HIV transmission to health care providers after a percutaneous exposure to HIV-infected blood is estimated to be approximately 0.3% and after a mucous membrane exposure, approximately 0.09%. If you sustain an occupational exposure to HIV, take the following actions immediately:

- Alert your supervisor/nursing faculty and initiate the injury-reporting system used in the setting.
- Identify the source patient, who may need to be tested for HIV, hepatitis B, and hepatitis C. State laws will determine whether written informed consent must be obtained from the source patient before his or her testing. OraQuick rapid testing should be used if possible if the HIV status of the source patient is unknown, because results can be available within 20 minutes.
- Report as quickly as possible to the employee health services, the emergency department, or other designated treatment facility. This visit should be documented in the health care worker's confidential medical record.
- Give consent for baseline testing for HIV, hepatitis B, and hepatitis C. Confidential HIV testing can be performed up to 72 hours after the exposure but should be performed as soon as the health care worker can give informed consent for baseline testing.
- Get postexposure prophylaxis for HIV in accordance with CDC guidelines. Start the prophylaxis medications within 2 hours after exposure. Make sure that you are being monitored for symptoms of toxicity. Practice safer sex until follow-up testing is complete. Continue the HIV medications for the full 4 weeks after exposure. The majority of HIV exposures will warrant a combination of antiretroviral agents. Combinations that may be prescribed for postexposure prophylaxis include zidovudine (ZDV) and lamivudine (3TC) or emtricitabine (FTC); stavudine (d4T) and 3TC or FTC; and tenofovir (TDF) and 3TC or FTC.
- Follow up with postexposure testing at 1 month, 3 months, and 6 months, and perhaps 1 year.
- Document the exposure in detail for your own records as well as for the employer.

Centers for Disease Control and Prevention. (2005). Updated U.S. Public Health Service guidelines for the management of occupational exposures to HIV and recommendations for postexposure prophylaxis. *MMWR–Morbidity and Mortality Weekly Report, 54*(RR-9), 1–17.

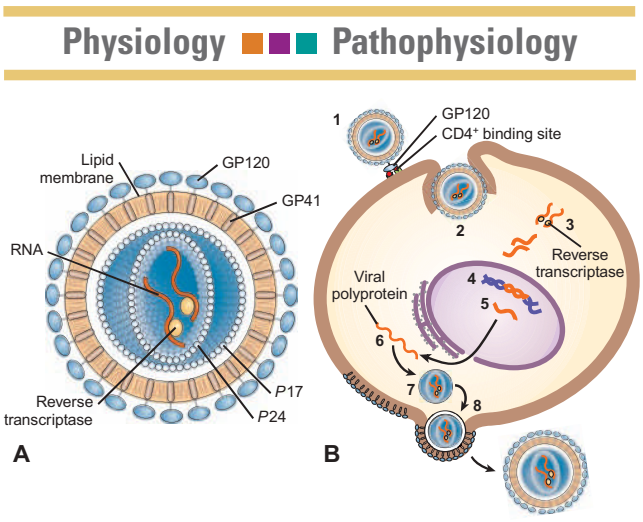


Figure 52-1 **A,** Structure of HIV-1. A glycoprotein envelope surrounds the virus, which carries its genetic material in RNA. Knobs, consisting of proteins GP120 and GP41, protrude from the envelope. These proteins are essential for binding the virus to the CD4+ T lymphocyte. **B,** Life cycle of HIV-1: (1) Attachment of the HIV virus to a CD4+ receptor; (2) internalization and uncoating of the virus with viral RNA and reverse transcriptase; (3) reverse transcription, which produces a mirror image of the viral RNA and double-stranded DNA molecule; (4) integration of viral DNA into host DNA using the integrase enzyme; (5) transcription of the inserted viral DNA to produce viral messenger RNA; (6) translation of viral messenger RNA to create viral polyprotein; (7) cleavage of viral polyprotein into individual viral proteins that make up the new virus; and (8) assembly and release of the new virus from the host cell. Redrawn from Porth, C. & Matfin, G. (2009). *Pathophysiology: Concepts of altered health states* (7th ed.). Philadelphia: Lippincott Williams & Wilkins.

All viruses target specific cells. HIV targets cells with CD4 receptors, which are expressed on the surface of T lymphocytes, **monocytes,** dendritic cells, and brain microglia. Mature T cells (T lymphocytes) are composed of two major subpopulations that are defined by cell surface receptors of CD4 or CD8. Approximately two thirds of peripheral blood T cells are CD4+, and approximately one third are CD8+. Most people have about 700 to 1000 CD4+ cells/mm^3, but a level as low as 500 cells/mm^3 can be considered within normal limits. During acute/recent infection, most varieties of HIV-1 use the chemokine receptor **CCR5** (R5 virus) for entry to T cells in addition to the CD4+ receptor, which suggests that the R5 variant is preferred to a different variant (CXCR4). Over the course of infection, viruses in the majority of untreated patients eventually exhibit a shift in coreceptor from CCR5 to either CXCR4 or both CCR5 and CXCR4 (dual- or mixed-tropic) receptors (1st International Reference Panel for HIV-1 RNA Genotypes Panel, 2004). The glycoproteins of HIV (GP120 and GP41) must attach to both the CD4+ and the CCR5 binding sites in order to bind to the CD4+ cell membrane, which results in fusion of HIV with the T cell.

Once HIV has attached to the host cell, the virus can replicate. The HIV life cycle is complex (see Fig. 52-1B) and consists of the following steps (Porth & Matfin, 2009):

1. Attachment. In this first step, the GP120 and GP41 glycoproteins of HIV bind with the host's uninfected CD4+ receptor and chemokine coreceptors, usually CCR5, which results in fusion of HIV with the CD4+ T-cell membrane.
2. Uncoating. The contents of HIV's viral core (two single strands of viral RNA and three viral enzymes: **reverse transcriptase,** integrase, and protease) are emptied into the CD4+ T cell.

3. DNA synthesis. HIV changes its genetic material from RNA to DNA through action of reverse transcriptase, resulting in double-stranded DNA that carries instruction for viral replication.
4. Integration. New viral DNA enters the nucleus of the CD4+ T cell and through action of integrase is blended with the DNA of the CD4+ T cell, resulting in permanent, lifelong infection. Prior to this step, the uninfected person has been only exposed to, not infected with, HIV. With this step, HIV infection is permanent.
5. Transcription. When the CD4+ T cell is activated, the double-stranded DNA forms single-stranded messenger RNA (mRNA), which builds new viruses.
6. Translation. The mRNA creates chains of new proteins and enzymes (polyproteins) that contain the components needed in the construction of new viruses.
7. Cleavage. The HIV enzyme protease cuts the polyprotein chain into the individual proteins that make up the new virus.
8. Budding. New proteins and viral RNA migrate to the membrane of the infected CD4+ T cell, exit from the cell, and start the process all over.

In resting (nondividing) CD4+ cells, HIV can survive in a latent state as an integrated **provirus** that produces few or no viral particles. These resting CD4+ T cells can be stimulated to produce new particles if something activates them. When a T cell that harbors this integrated DNA (also known as provirus) becomes activated against HIV or other microbes, the cell begins to produce new copies of both RNA and viral proteins. Activation of the infected cell may be achieved by antigens, mitogens, certain cytokines (tumor necrosis factor-alpha [TNF-α] or interleukin-1 [IL-1]), or virus gene products of such viruses as **cytomegalovirus (CMV)**, Epstein-Barr virus, herpes simplex virus, and hepatitis viruses. Consequently, whenever the infected CD4+ cell is activated, HIV replication and budding occur, which can destroy the host cell. Newly formed HIV released into the blood can infect other CD4+ cells (see Fig. 52-1B).

HIV-1 mutates quickly, at a relatively constant rate, with about 1% of the virus's genetic material changing annually. HIV-1 exhibits substantial genetic diversity and several different genotypes of HIV-1 exist. There is a major group (group M), which consists of subtypes A through L, and a more diverse collection of outliers, which has been referred to as groups N and O. Many of the early nucleic acid–based tests had a fairly narrow band of specificity targeted mainly at subtype B viruses, because these predominated in the western world (1st International Reference Panel for HIV-1 RNA Genotypes, 2004).

A mutation of CCR5 that is common in Caucasians, but not other ethnic groups, has been identified. About 1% of Caucasians lack functional CCR5 and are highly protected against HIV infection even if exposed (although protection is not absolute); about 18% are not markedly protected against infection but, if infected, demonstrate significantly slower rates of disease progression.

Stages of HIV Disease

The stage of HIV disease is based on clinical history, physical examination, laboratory evidence of immune dysfunction, signs and symptoms, and infections and malignancies. The CDC standard case definition of AIDS categorizes HIV infection and AIDS in adults and adolescents on the basis of clinical conditions associated with HIV infection and CD4+ T-cell counts. The classification system (Table 52-1) groups clinical conditions into one of three categories, denoted A, B, and C.

Primary Infection (Acute/Recent HIV Infection, Acute HIV Syndrome)

The period from infection with HIV to the development of HIV-specific antibodies is known as **primary infection.** Initially, there is a **window period** during which an HIV-positive person tests negative on the HIV antibody blood test, although he or she is infected and highly infectious, because his or her viral load is very high. After 2 to 3 weeks, antibodies to the glycoproteins of the HIV envelope can be detected in the sera of HIV-infected people, but most of these antibodies lack the ability to totally control the virus. By the time neutralizing antibodies can be detected, HIV-1 is firmly established in the host.

Primary infection is characterized by high levels of viral replication, widespread dissemination of HIV throughout the body, and destruction of CD4+ T cells. This leads to dramatic drops in CD4+ T-cell counts, which are normally 500 to 1500 cells/mm^3 of blood. The host is responding to the HIV infection through a CD4+ T-cell response that causes other immune cells, such as CD8+ lymphocytes, to increase their killing of infected, virus-producing cells. The body produces antibody molecules in an effort to contain the free HIV particles (outside cells) and assist in their removal. The remaining amount of virus in the body after this initial immune response is referred to as the **viral set point,** which results in a steady state of infection that lasts for years. The final level of the viral set point is inversely correlated with disease prognosis; that is, the higher the viral set point, the poorer the prognosis.

The primary infection stage is part of CDC category A and includes the acute symptomatic and early infection phases. During this stage, the virus is widely disseminated in lymphoid tissue, and a **latent reservoir** within resting memory CD4+ T cells is created (Zack & Park, 2008). An estimated 40% to 90% of patients who are acutely infected with HIV experience symptoms of acute retroviral syndrome characterized by fever, lymphadenopathy, pharyngitis, skin rash, myalgias/arthralgias, and other conditions (Panel on Antiretroviral Guidelines for Adults and Adolescents (Guidelines) (2008)).

HIV Asymptomatic (CDC Category A: More Than 500 CD4+ T Lymphocytes/mm^3)

After the viral set point is reached, HIV-positive people enter into a chronic stage in which the immune system cannot eliminate the virus despite its best efforts. This set point varies greatly from patient to patient and dictates the subsequent rate of disease progression; on average, 8 to 10 years pass before a major HIV-related complication develops. In

Table 52-1 CLASSIFICATION SYSTEM FOR HIV INFECTION AND EXPANDED AIDS SURVEILLANCE CASE DEFINITION FOR ADOLESCENTS AND ADULTS

Diagnostic Categories CD4+ T-Cell Category	Clinical Categories A Asymptomatic, Acute (Primary) HIV or PGL	B Symptomatic, Not (A) or (C) Conditions	C AIDS-Indicator Conditions
(1) ≥500/μL	A1	B1	C1
(2) 200–499/μL	A2	B2	C2
(3) <200/μL AIDS-indicator T-cell count	A3	B3	C3

People with AIDS-indicator conditions (clinical category C) and those in categories A3 or B3 are considered to have AIDS.

Clinical Category A

Includes one or more of the following in an adult or adolescent with confirmed HIV infection and without conditions in clinical categories B and C:

- Asymptomatic HIV infection
- Persistent generalized lymphadenopathy (PGL)
- Acute (primary) HIV infection with accompanying illness or history of acute HIV infection

Clinical Category B

Examples of conditions in clinical category B include, but are not limited to, the following:

- Bacillary angiomatosis
- Candidiasis, oropharyngeal (thrush) or vulvovaginal (persistent, frequent, or poorly responsive to therapy)
- Cervical dysplasia (moderate or severe)/cervical carcinoma in situ
- Constitutional symptoms, such as fever (38.5°C) or diarrhea exceeding 1 mo in duration
- Hairy leukoplakia, oral
- Herpes zoster (shingles), involving at least two distinct episodes or more than one dermatome
- Idiopathic thrombocytopenic purpura
- Listeriosis
- Pelvic inflammatory disease, particularly if complicated by tubo-ovarian abscess
- Peripheral neuropathy

Clinical Category C

Examples of conditions in adults and adolescents include the following:

- Candidiasis of bronchi, trachea, or lungs; esophagus
- Cervical cancer, invasive
- Coccidioidomycosis, disseminated or extrapulmonary
- Cryptococcosis, extrapulmonary
- Cryptosporidiosis, chronic intestinal (exceeding 1 mo's duration)
- Cytomegalovirus disease (other than liver, spleen, or lymph nodes)
- Cytomegalovirus retinitis (with loss of vision)
- Encephalopathy, HIV related
- Herpes simplex: chronic ulcer(s) (exceeding 1 mo's duration); or bronchitis, pneumonitis, or esophagitis
- Histoplasmosis, disseminated or extrapulmonary
- Isosporiasis, chronic intestinal (exceeding 1 mo's duration)
- Kaposi's sarcoma
- Lymphoma, Burkitt's (or equivalent term); immunoblastic (or equivalent term); primary, of brain
- *Mycobacterium avium* complex or *M. kansasii*, disseminated or extrapulmonary
- *Mycobacterium tuberculosis*, any site (pulmonary or extrapulmonary)
- *Mycobacterium*, other species or unidentified species, disseminated or extrapulmonary
- *Pneumocystis jiroveci* pneumonia
- Pneumonia, recurrent
- Progressive multifocal leukoencephalopathy
- *Salmonella* septicemia, recurrent
- Toxoplasmosis of brain
- Wasting syndrome due to HIV

Adapted from Centers for Disease Control, U.S. Department of Health and Human Services. (1992). 1993 revised classification system for HIV infection and expanded surveillance case definition for AIDS among adolescents and adults. *MMWR Morbidity and Mortality Weekly Report, 41*(RR-17), 1–19.

this prolonged, chronic stage, patients feel well and have few, if any, symptoms. Apparent good health continues because CD4+ T-cell levels remain high enough to preserve immune defensive responses.

HIV Symptomatic (CDC Category B: 200 to 499 CD4 + T Lymphocytes/mm^3)

Over time, the number of CD4+ T cells gradually falls. Category B consists of symptomatic conditions in HIV-infected patients that are not included in the conditions listed in category C. These conditions must also meet one of the following criteria: (1) the condition is caused by HIV infection or a defect in cellular immunity, or (2) the condition is considered to have a clinical course or to require management that is complicated by HIV infection. If a person was once treated for a category B condition and has not developed a category C disease but is now symptom-free, that person's stage of HIV disease is considered category B.

AIDS (CDC Category C: Fewer Than 200 CD4+ T Lymphocytes/mm^3)

When the CD4+ T-cell level drops below 200 cells/mm^3 of blood, the person is said to have AIDS. As levels decrease to fewer than 100 cells/mm^3, the immune system is significantly impaired. Once a patient has had a category C condition, he or she remains in category C even if CD4+ T cells rebound with treatment. This classification has implications for entitlements (ie, disability benefits, housing, and food stamps), because these programs are often linked to an AIDS diagnosis. Although the 1993 classification emphasizes CD4+ T-cell counts, it allows for CD4+ percentages (percentage of CD4+ T cells compared with total lymphocytes). The CD4+ percentage is less subject to variation on repeated measurements than is the absolute CD4+ T-cell count. A CD4+ percentage of less than 14% of the total lymphocytes is consistent with a diagnosis of AIDS. The percentage, as compared with the absolute number of CD4+ T cells, becomes particularly important when the patient has a heightened immune response to infections in addition to HIV.

Assessment and Diagnostic Findings in HIV Infection

During the first stage of HIV infection, the patient may be asymptomatic or may exhibit various signs and symptoms. The patient's health history should alert the health care

Table 52-2 SELECTED LABORATORY TESTS FOR DIAGNOSING AND TRACKING HIV AND ASSESSING IMMUNE STATUS

Test	Findings in HIV Infection
EIA (enzyme immunoassay)	Antibodies are detected, resulting in positive results and marking the end of the window period
Western blot	Also detects antibodies to HIV; used to confirm EIA
Viral load	Measures HIV RNA in the plasma
CD4/CD8	These are markers found on lymphocytes. HIV kills CD4+ cells, which results in a significantly impaired immune system.

provider about the need for HIV screening based on the patient's sexual practices, IV/injection drug use, and receipt of blood transfusions. Additionally, exposure to body fluids containing infected blood while providing care to others with HIV infection (eg, through needlesticks) should alert health care providers to possible HIV infection. Patients who are in later stages of HIV infection may have a variety of symptoms related to their immunosuppressed state. Several screening tests are used to diagnose HIV infection. Others are used to assess the stage and severity of the infection. Table 52-2 identifies common blood tests.

HIV Antibody Tests

In 2006, the CDC issued recommendations for HIV testing in public and private health care settings (CDC, 2006), including hospital emergency departments, inpatient facilities, urgent care clinics, primary care settings, public health clinics, community clinics, substance abuse treatment clinics, and correctional health care facilities. The objectives of those recommendations were to increase HIV screening of patients, including pregnant women, in health care settings; foster earlier detection of HIV infection; identify and counsel people with unrecognized HIV infection and refer them to clinical and preventive services; and further reduce perinatal transmission of HIV. The major changes from previously published guidelines are:

1. HIV screening is recommended for patients (18 to 64 years of age) in all health care settings after the patient is notified that testing will be performed unless he or she declines (opt-out screening).
2. People at high risk for HIV infection should be screened for the disease at least annually.
3. Separate written consent for HIV testing should not be required; general consent for medical care should be considered sufficient to encompass consent for HIV testing.
4. Prevention counseling should not be required with HIV diagnostic testing or as part of HIV screening programs in health care settings.

Although these guidelines are already in effect in some states, they have not been implemented in others because they violate the states' existing HIV confidentiality laws.

Before an HIV antibody test is performed, the meaning of the test and possible test results are explained, and informed consent for the test is obtained from the patient. When the result of the HIV antibody test is received, it is carefully explained to the patient in private (Chart 52-6). All test results are kept confidential. Education and counseling about the test result and about preventing transmission are essential. The patient's psychological response to a positive test result may include feelings of panic, depression, and hopelessness. The social and interpersonal consequences of a positive test result can be devastating. The patient may lose his or her sexual partner, housing, and health insurance because of disclosure. He or she may experience discrimination in employment and housing, as well as social ostracism. For these reasons and others, patients who test positive may need ongoing counseling as well as referrals for social, financial, medical, and psychological support services. Patients whose test results are seronegative may develop a false sense of security, possibly resulting in continued high-risk behaviors or feelings that they are immune to the virus. These patients may need ongoing counseling to help modify high-risk behaviors and to encourage returns for repeated testing. Other patients may experience anxiety regarding the uncertainty of their status.

Chart 52-6 • *HIV Test Results: Implications for Patients*

Interpretation of Positive Test Results

- Antibodies to HIV are present in the blood (the patient has been infected with the virus, and the body has produced antibodies).
- HIV is active in the body, and the patient can transmit the virus to others.
- Despite HIV infection, the patient does not necessarily have AIDS.
- The patient is not immune to HIV (the antibodies do not indicate immunity).

Interpretation of Negative Test Results

- Antibodies to HIV are not present in the blood at this time, which can mean that the patient has not been infected with HIV or, if infected, the body has not yet produced antibodies (window period—usually 3 weeks to 6 months).
- The patient should continue taking precautions. The test result does not mean that the patient is immune to the virus, nor does it mean the patient is not infected; it just means that the body may not have produced antibodies yet.

When a person is infected with HIV, the immune system responds by producing antibodies against the virus, usually within 3 to 12 weeks after infection. In 1985, the FDA licensed an HIV-1 antibody assay that uses approximately 5 to 7 mL of blood. Samples are tested using two different laboratory techniques to determine the presence of antibodies to HIV. The **EIA (enzyme immunoassay)** test, formerly referred to as the **ELISA (enzyme-linked immunosorbent assay)** test, identifies antibodies directed specifically against HIV. The **Western blot assay** is used to confirm seropositivity when the EIA result is positive. Adults whose blood contains antibodies for HIV are seropositive.

In addition to this HIV-1 antibody assay, two additional techniques are now available. The OraSure test uses saliva to perform an EIA antibody test. Using less than a drop of

blood, the OraQuick Rapid HIV-1 Antibody Test quickly (approximately 20 minutes) and reliably (99.6% accuracy) detects antibodies to HIV-1. The OraQuick test is becoming the standard method of testing in settings where a delay would seriously affect treatment, such as in labor and delivery rooms or in emergency departments when the HIV status of a sexual abuser is unknown.

Home-based testing for HIV antibodies using a small amount of blood was first proposed in 1985 and approved by the FDA in 1995. However, use of home testing kits raises concerns because of the lack of counseling and possible inaccurate results, including both false-positive and false-negative results.

Viral Load Tests

Target amplification methods quantify HIV RNA or DNA levels in the plasma and have replaced p24 antigen capture assays. Target amplification methods include reverse transcriptase–**polymerase chain reaction** (RT-PCR) and nucleic acid sequence–based amplification. A widely used **viral load test** measures plasma HIV RNA levels. Currently, these tests are used to track viral load and response to treatment of HIV infection. RT-PCR is also used to detect HIV in high-risk seronegative people before antibodies are measurable, to confirm a positive EIA result, and to screen neonates. HIV culture or quantitative plasma culture and plasma viremia are additional tests that measure viral burden, but they are used infrequently. Viral load is a better predictor of the risk of HIV disease progression than the CD4+ count. The lower the viral load, the longer the time to AIDS diagnosis and the longer the survival time.

Treatment of HIV Infection

Protocols on how and when to start treatment for HIV disease change relatively often. The U.S. Department of Health and Human Services Panel on Antiretroviral Guidelines for Adults and Adolescents (Guidelines, 2008) is composed of HIV specialists from across the country who meet periodically to review the latest scientific evidence. The CD4+ T-cell count is usually the most important consideration in deciding whether antiretroviral medications should be started. In general, antiretroviral medications should be offered to people with a T-cell count of less than 350 cells/mm^3 or plasma HIV RNA levels exceeding 100,000 copies/mL (Guidelines, 2008). Some clinicians and patients, however, are choosing not to start medications until the CD4+ cell count decreases to approximately 200 cells/mm^3. Monitoring the National Institutes of Health (NIH) Web site (see Resources at the end of the chapter) for frequent updates of the recommendations is essential before caring for patients with HIV/AIDS (Guidelines, 2008).

Clinicians, in partnership with patients, make treatment decisions based on a number of factors, including CD4+ T-cell count, viral load, severity of HIV/AIDS-related symptoms, and willingness of the patient to adhere to the lifelong treatment regimen. The increasing number of antiretroviral agents (Table 52-3) and the rapid evolution of new information have introduced extraordinary complexity into the treatment of HIV infection. More than 25 agents have been approved in the United States. Drugs from established classes (nucleoside/nucleotide reverse transcriptase inhibitors [NRTIs], non-nucleoside reverse transcriptase inhibitors [NNRTIs], and **protease inhibitors [PIs]**) continue to serve as the mainstays of antiretroviral therapy (Kuritzkes, 2008). In 2008, the integrate inhibitor (raltegravir [Isentress]) and the CCR5 antagonist (maraviroc [Selzentry]), a novel entry inhibitor, were approved and joined the other fusion inhibitors such as enfuvirtide (T-20), which inhibits entry of HIV into the CD4+ T cell (Moyle, Gatell, Perno, et al., 2008). In patients with resistant HIV disease, these new classes of antiretroviral agents offer considerable potential benefit because of the absence of cross-resistance (Cooper, Steigbiegel, Gatell, et al., 2008).

To achieve sustained viral suppression, patients must take more than one antiretroviral medication. Although HAART was defined originally as a regimen that included at least one PI, it has evolved to include any regimen with at least two to three different medications. Some pharmaceutical companies have combined two to three agents into one tablet or capsule, such as Kaletra (lopinavir and ritonavir) and Atripla (efavirenz, emtricitabine, and tenofovir) in a single tablet for once-a-day use. Simplifying treatment regimens and decreasing the number of medications that must be taken each day may increase patients' adherence to therapy.

Adherence to long-term treatment is required to manage HIV infection and many other chronic illnesses; however, overall adherence rates remain low (30% to 50%). Although antiretroviral regimens have become less complex, side effects create barriers to adherence, and this can lead to viral resistance. The goals of treatment include maximal and sustained suppression of viral load to a nondetectable level, restoration or preservation of immunologic function, improved quality of life, and reduction of HIV-related morbidity and mortality. Viral load testing is recommended at the time of diagnosis of HIV disease and every 3 to 4 months thereafter in the untreated person; T-cell counts should be measured at diagnosis and usually every 3 to 6 months thereafter (Guidelines, 2008). In the majority of patients, HAART leads to sustained reductions in HIV replication, a rise in CD4+ T-cell counts with reconstitution of immune function, and significant reductions in morbidity and mortality.

It is difficult to predict patients' adherence to medication regimens, but a positive relationship between the patient and health care provider is associated with better adherence. Individualized plans of care that take into consideration housing and social support issues, in addition to health indicators, are essential. Adherence to the antiretroviral treatment plan involves very complex behavior that can change over the duration of the medication regimen. Self-reported adherence measures can distinguish clinically meaningful patterns of medication-taking behaviors; therefore, nurses should ask patients if they are taking their medications as prescribed. Factors associated with nonadherence include active substance abuse, depression, and lack of social support. Gender, race, pregnancy, and history of past substance use have not been associated with nonadherence (Guidelines, 2008). Chart 52-7 summarizes various strategies that health care providers can encourage to promote

Table 52-3 ANTIRETROVIRAL AGENTS*

Generic Name (Abbreviation) and Trade Names	Food Interactions	Adverse Events
Nucleoside Reverse Transcriptase Inhibitors (NRTIs)		
Abacavir (ABC) *Ziagen* *Trizivir* (ABC + ZDV, + 3TC) *Epzicom* (ABC + 3TC)	Can be taken without regard to meals. Alcohol increases abacavir levels 41%. Abacavir has no effect on alcohol.	Hypersensitivity reaction, which can be fatal; symptoms may include fever, rash, nausea, vomiting, malaise or fatigue, loss of appetite, and respiratory symptoms such as sore throat, cough, shortness of breath.
Didanosine (ddI) *Videx* *Videx EC*	Levels decrease 55%; take half hour before or 2 h after meals.	Pancreatitis; peripheral neuropathy; nausea; diarrhea. Lactic acidosis with fatty degeneration of the liver (rare but potentially life-threatening toxicity associated with use of NRTIs)
Emtricitabine (FTC) *Emtriva* *Truvada* (FTC + TDF)	Can be taken without regard to meals	Minimal toxicity; lactic acidosis with hepatic steatosis (rare but potentially life-threatening toxicity with use of NRTIs)
Lamivudine (3TC) *Epivir* *Combivir* (3TC + ZDV) *Trizivir* (3TC + ZDV, + ABC)	Can be taken without regard to meals	Minimal toxicity; lactic acidosis with hepatic steatosis (rare but potentially life-threatening toxicity with use of NRTIs)
Stavudine (d4T) *Zerit*	Can be taken without regard to meals	Peripheral neuropathy; lipodystrophy; rapidly progressive ascending neuromuscular weakness (rare); pancreatitis; lactic acidosis with hepatic steatosis (higher incidence with d4T than with other NRTIs); hyperlipidemia
Tenofovir disoproxil fumarate (TDF) *Viread* *Truvada* (TDF + FTC)	Can be taken without regard to meals	Asthenia, headache, diarrhea, nausea, vomiting, and flatulence; renal insufficiency; lactic acidosis with hepatic steatosis (rare but potentially life-threatening toxicity with use of NRTIs)
Zalcitabine (ddC) *Hivid*	Can be taken without regard to meals	Peripheral neuropathy; stomatitis; lactic acidosis with hepatic steatosis (rare but potentially life-threatening toxicity with use of NRTIs); pancreatitis
Zidovudine (AZT or ZDV) *Retrovir* Combivir (AZT + 3TC) *Trizivir* (AZT + 3TC, + ABC)	Can be taken without regard to meals	Bone marrow suppression; macrocytic anemia or neutropenia; gastrointestinal intolerance, headache, insomnia, asthenia; lactic acidosis with hepatic steatosis (rare but potentially life-threatening toxicity with use of NRTIs)
Non-Nucleoside Reverse Transcriptase Inhibitors (NNRTIs)		
Delavirdine (DLV) *Rescriptor*	Can be taken without regard to meals	Rash (rare cases of Stevens-Johnson syndrome have been reported); increased transaminase levels; headaches
Efavirenz (EFV) *Sustiva*	High-fat/high-caloric meals increase peak plasma concentrations of capsules by 39% and tablets by 79%; take on an empty stomach.	Rash (rare cases of Stevens-Johnson syndrome have been reported); central nervous system symptoms (dizziness, somnolence, insomnia, abnormal dreams, confusion, abnormal thinking, impaired concentration, amnesia, agitation, depersonalization, hallucinations, and euphoria); increased transaminase levels; false-positive cannabinoid test; teratogenic in monkeys
Nevirapine (NVP) *Viramune*	Take without regard to meals.	Rash including Stevens-Johnson syndrome, symptomatic hepatitis including fatal hepatic necrosis has been reported. Single dose used in developing countries to prevent vertical transmission
Etravirine (TMC 125) *Intelence*	Take after a meal with water.	Serious side effects of this medication include severe skin rash Mild to moderate rash occurs in the second week of therapy and generally resolves within 1–2 weeks of continued therapy. Nausea, diarrhea, abdominal pain, vomiting, fatigue, peripheral neuropathy, headache, and high blood pressure
Protease Inhibitors (PIs)		
Amprenavir (APV) *Agenerase*	High-fat meal decreases blood concentration 21%; can be taken with or without food, but high-fat meal should be avoided	GI intolerance; nausea; vomiting; diarrhea; rash; oral paresthesias; hyperlipidemia; transaminase elevation; hyperglycemia; fat maldistribution; possible increased bleeding episodes in patients with hemophilia
Atazanavir (ATV) *Reyataz*	Administration with food increases bioavailability. Should be taken with food; avoid taking with antacids.	Indirect hyperbilirubinemia; prolonged PR interval—some patients experience asymptomatic first-degree AV block; use with caution in patients with underlying conduction defects or on concomitant medications that can cause PR prolongation; hyperglycemia; fat maldistribution; possible increased bleeding episodes in patients with hemophilia
Fosamprenavir (FOS-APV) *Lexiva*	Can be taken without regard to meals	Skin rash (19%); diarrhea; nausea; vomiting; headache; hyperlipidemia; transaminase elevation; hyperglycemia; fat maldistribution; possible increased bleeding episodes in patients with hemophilia
Indinavir (IDV) *Crixivan*	For unboosted IDV: Should be taken 1 h before or 2 h after meals; may take with skim or low-fat meal For RTV-boosted IDV: Can be taken with or without food	Nephrolithiasis; GI intolerance; nausea; indirect hyperbilirubinemia; hyperlipidemia; headache, asthenia; blurred vision; dizziness; rash; metallic taste; thrombocytopenia; alopecia; hemolytic anemia; hyperglycemia; fat maldistribution; possible increased bleeding episodes in patients with hemophilia

Continued on following page

Table 52-3 ANTIRETROVIRAL AGENTS* (Continued)

Generic Name (Abbreviation) and Trade Names	Food Interactions	Adverse Events
Lopinavir + ritonavir (LPV/RTV) *Kaletra*	Should be taken with food	GI intolerance; nausea; vomiting; diarrhea; asthenia; hyperlipidemia (especially hypertriglyceridemia); elevated serum transaminase; hyperglycemia; fat maldistribution; possible increased bleeding episodes in patients with hemophilia
Nelfinavir (NFV) *Viracept*	Should be taken with a meal or snack	Diarrhea; hyperlipidemia; hyperglycemia; fat maldistribution; possible increased bleeding episodes in patients with hemophilia; serum transaminase elevation
Ritonavir (RTV) *Norvir*	Should be taken with food if possible; may improve tolerability	GI intolerance; nausea; vomiting; diarrhea; paresthesias—circumoral and extremities; hyperlipidemia, especially hypertriglyceridemia; hepatitis; asthenia; taste perversion; hyperglycemia; fat maldistribution; possible increased bleeding in patients with hemophilia. Lower doses used as a booster
Saquinavir (SQV) *Invirase*		GI intolerance; nausea; diarrhea; abdominal pain and dyspepsia; headache; hyperlipidemia; elevated transaminase enzymes; hyperglycemia; fat maldistribution; possible increased bleeding episodes in patients with hemophilia. Take with RTV as booster, if prescribed.
Tipranavir (TPV) *Aptivus*	Take with food	Serious liver problems, bleeding on the brain, rash, increased cholesterol and triglyceride levels, and changes in body fat; women taking birth control pills that contain estrogen may be more likely to develop a rash. Individuals with hemophilia may have increased bleeding. Take with RTV, if prescribed.
Darunavir *Prezista*		Diarrhea; nausea; headache; and coldlike symptoms, including runny nose or sore throat; inflammation of the liver; abnormal liver function tests; severe skin rash; fever; and abnormally high cholesterol and triglyceride levels have been reported. Take with RTV.
Fusion Inhibitors		
Enfuvirtide (T-20) *Fuzeon*	Injected subcutaneously, so meals are not an issue	Local injection site reactions—almost 100% of patients (pain, erythema, induration, nodules and cysts, pruritus, ecchymosis); increased rate of bacterial pneumonia; hypersensitivity reaction—symptoms may include rash, fever, nausea, vomiting, chills, rigors, hypotension, or elevated serum transaminases; may recur on challenge
Maraviroc *Selzentry*	Taken with or without food; requires CCR5 tropism blood test before starting	Cough, fever, dizziness, headache, lowered blood pressure, nausea, and bladder irritation; possible liver problems and cardiac events; an increased risk for some infections; a slight increase in cholesterol levels
Integrase Strand Transfer Inhibitor		
Raltegravir *Isentress*	No food restrictions identified	Diarrhea, nausea, headache, and fever have been reported.
Multiclass Combination Products		
Efavirenz, emtricitabine, and tenofovir *Atripla*		

*This information changes often. Check the U.S. Food and Drug Administration Web site (www.fda.gov/oashi/aids/virals.html) and www.aidsinfo.nih.gov/DrugsNew/Default.aspx?MenuItem=Drugs for current information when caring for people with HIV/AIDS.

treatment regimen adherence. Every health care encounter should be used as an opportunity to briefly review the treatment regimen, identify any new issues, and reinforce successful behaviors.

Results of therapy are evaluated with viral load tests (Guidelines, 2008). Viral load levels should be measured immediately before initiation of antiretroviral therapy and again after 2 to 8 weeks, because in most patients adherence to a regimen of potent antiretroviral agents should result in a large decrease in the viral load by 2 to 8 weeks. The viral load should continue to decline over the following weeks, and in most individuals it will drop below detectable levels (currently defined as less than 50 RNA copies/mL) by 16 to 20 weeks. The rate of viral load decline toward undetectable levels is affected by the baseline T-cell count, the initial viral load, the potency of the medication, adherence to the medication regimen, prior exposure to antiretroviral agents, and the presence of any opportunistic infections. The confirmed absence of a viral load response should prompt the health care team to reevaluate the regimen. The CD4+ count should increase by 100 to 150 cells/mm^3 per year, with an accelerated response in the first 3 months of treatment (Guidelines, 2008).

Adverse effects associated with all HIV treatment regimens include hepatotoxicity, nephrotoxicity, and osteopenia, along with increased risk of cardiovascular disease and myocardial infarction (Moyle, et al., 2008) (see Table 52-3). Many of the antiretroviral agents that prolong life may

CHART 52-7 HOME CARE CHECKLIST

Adhering to Medication Therapy for HIV

At the completion of the home care instruction, the patient or caregiver will be able to:	PATIENT	CAREGIVER
• Verbalize knowledge of each medication name.	✔	✔
• State the action of each medication.	✔	✔
• State the correct times that medications are to be taken.	✔	✔
• Identify special guidelines to follow when taking medications (eg, with meals, on an empty stomach, medications that are not to be taken together).	✔	✔
• Demonstrate methods of keeping track of the medication regimen and storage of the prescribed medications and use reminders such as beepers and/or pill boxes.	✔	✔
• Identify specific laboratory tests such as viral load that are necessary to monitor the effectiveness of the prescribed medication regimen.	✔	✔
• List expected side effects of each medication.	✔	✔
• Identify side effects that should be reported to health care providers.	✔	✔
• Explain the importance of and necessity for adherence with prescribed medication regimen.	✔	✔
• Demonstrate correct administration of IM, SC, or IV medications.	✔	✔
• Demonstrate correct use and safe disposal of needles, syringes, and other IV equipment.	✔	✔
• Discuss with health care providers any problems that he or she is having with side effects and adherence.	✔	✔
• Discuss episodes of nonadherence to the medication regimen.	✔	✔

simultaneously cause fat redistribution syndrome and metabolic alterations such as dyslipidemia and insulin resistance, which put the patient at risk for early-onset heart disease and diabetes (Calza, Manfredi, Pocaterra, et al., 2008). The fat redistribution syndrome consists of lipoatrophy (localized subcutaneous fat loss in the face, arms, legs, and buttocks) and lipohypertrophy (central visceral fat [lipomata] accumulation in the abdomen, although possibly in the breasts, dorsocervical region [buffalo hump], and within the muscle and liver) (Calza, et al., 2008). These changes can be very disturbing to the body image of people living with HIV/AIDS and may be a reason that they decline treatment, especially with regimens that include a PI.

Facial wasting, characterized as a sinking of the cheeks, eyes, and temples caused by the loss of fat tissue under the skin, may be treated by injectable fillers such as poly-L-lactic acid (Sculptra) (Fig. 52-2). Hepatotoxicity associated with certain protease inhibitors may limit the use of these agents, especially in patients with underlying liver dysfunction (Guidelines, 2008).

Drug Resistance

Drug resistance can be broadly defined as the ability of pathogens to withstand the effects of medications that are intended to be toxic to them. There are two major components of antiretroviral drug resistance: (1) transmission of drug-resistant HIV at the time of initial infection and (2) selective drug resistance in patients who are receiving nonsuppressive regimens (Kuritzkes, 2008). Factors associated with the development of drug resistance include monotherapy (taking one medication), difficulty with adherence to complex and toxic regimens, and initiation of therapy late in the course of HIV/AIDS.

Resistance testing has a number of limitations and is more helpful in determining which antiretroviral agents should be eliminated rather than which ones should be used. Genotypic testing determines the sequence of viral RNA encoding relevant genes, which allows detection of amino acid mutations that are either proven or suspected to

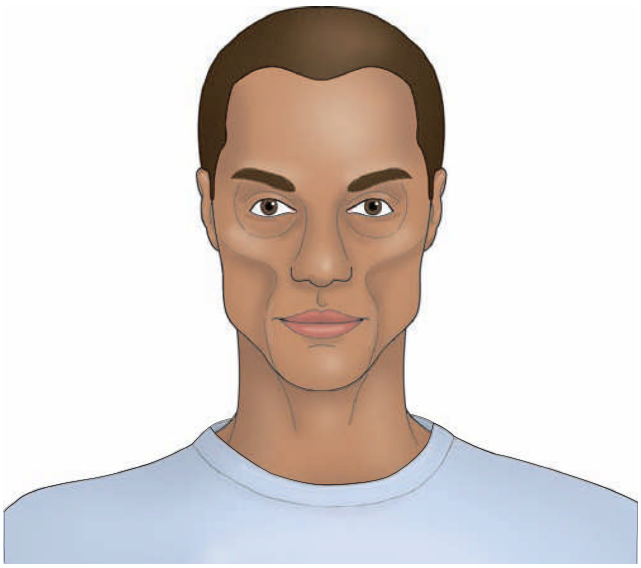


Figure 52-2 Facial lipoatrophy.

be associated with phenotypic resistance. Phenotypic testing determines the drug concentration needed to inhibit replication of a recombinant virus by 50% of a patient's isolate, when compared with a susceptible reference. Resistance testing is of greatest value when it is performed before drugs are discontinued or immediately afterward (within 4 weeks). Drug resistance testing is not advised for patients with a viral load of less than 1000 copies/mL, because the amount of the virus in the blood is too small to ensure reliable results (Guidelines, 2008).

In addition to resistance testing, several factors must be considered in choosing medications for a new regimen, once the prior regimen has failed. These factors include the patient's past treatment history, viral load, and medication tolerance; the likelihood of the patient's adhering to the medication regimen; and concomitant medical conditions or medications.

Treatment Interruption

Discontinuation of antiretroviral therapy may result in viral rebound, immune decompensation, and clinical progression. Unplanned interruption of antiretroviral therapy may become necessary because of severe drug toxicity, intervening illness, surgery that precludes oral therapy, pregnancy, or unavailability of antiretroviral medications. Planned treatment interruption, outside of clinical research trials, is not recommended (Guidelines, 2008).

Immune Reconstitution Inflammatory Syndrome

Immune reconstitution inflammatory syndrome (IRIS) results from rapid restoration of pathogen-specific immune responses to opportunistic infections that cause either the deterioration of a treated infection or new presentation of a subclinical infection. This syndrome typically occurs during the initial months after beginning antiretroviral treatment and is associated with a wide spectrum of pathogens, most commonly mycobacteria, herpes viruses, and deep fungal infections (Meintjes, Lawn, Scano, et al., 2008). IRIS is characterized by fever, respiratory and/or abdominal symptoms, and worsening of the clinical manifestations of an opportunistic infection or the appearance of new manifestations. The nurse should be alert to the possibility of IRIS, especially in the 3-month period after treatment with antiretroviral agents is initiated, because this syndrome is associated with significant morbidity and patients often require hospital admission. Rates of morbidity and mortality attributable to paradoxical tuberculosis-associated IRIS may be higher in settings with limited diagnostic and treatment options (eg, South Africa).

Clinical Manifestations

Patients with HIV/AIDS experience a number of symptoms related to the disease, side effects of treatment, and other illnesses such as hepatitis (Bova, Jaffarian, Himlan, et al., 2008). The clinical manifestations of HIV/AIDS are widespread and may involve virtually any organ system. Diseases associated with HIV infection and AIDS result from infections, malignancies, or the direct effect of HIV on body tissues. Nurses need to understand the causes, signs and symptoms, and interventions, including self-care strategies, that can enhance the quality of life for patients throughout the illness. Symptom assessment tools can be used to assess patients' symptom intensity and severity. People with HIV/AIDS use a variety of self-care strategies to minimize common symptoms, which can arise from HIV disease, comorbidities, or the effects of medications used to treat HIV and opportunistic infections.

Fatigue is frequently cited by people living with HIV/AIDS as one of the most bothersome symptoms. It has a multifactorial etiology. For more information about fatigue in HIV, see Chart 52-8.

Respiratory Manifestations

Shortness of breath, dyspnea (labored breathing), cough, chest pain, and fever are associated with various opportunistic infections, such as those caused by *P. jiroveci*, *Mycobacterium avium-intracellulare*, CMV, and *Legionella* species.

Pneumocystis Pneumonia

The most common infection in people with AIDS is ***Pneumocystis* pneumonia (PCP),** which is caused by *P. jiroveci*. It is the most common opportunistic infection associated with AIDS. Without prophylactic therapy (discussed later), 80% of all people infected with HIV will develop PCP.

The clinical presentation of PCP in HIV infection is generally less acute than in people who are immunosuppressed as a result of other conditions. The time between the onset of symptoms and the actual documentation of disease may be weeks to months. Patients with AIDS initially develop nonspecific signs and symptoms, such as nonproductive cough, fever, chills, shortness of breath, dyspnea, and occasionally chest pain. PCP may be present despite the absence of crackles. Arterial oxygen concentrations in patients who are breathing room air may be mildly decreased, indicating minimal hypoxemia.

If left untreated, PCP eventually progresses and causes significant pulmonary impairment and, ultimately, respiratory failure. A few patients have a dramatic onset and a fulminating course involving severe hypoxemia, cyanosis, tachypnea, and altered mental status. Respiratory failure can develop within 2 to 3 days after the initial appearance of symptoms.

PCP can be diagnosed definitively by identifying the organism in lung tissue or bronchial secretions. This is accomplished by such procedures as sputum induction, bronchial-alveolar lavage, and transbronchial biopsy (by fiberoptic bronchoscopy).

Mycobacterium avium Complex

***Mycobacterium avium* complex (MAC)** disease is a common opportunistic infection in people with AIDS. MAC comprises a group of acid-fast bacilli (mycobacteria) that includes *M. avium*, *M. intracellulare*, and *M. scrofulaceum*. MAC usually causes respiratory infection but is also commonly found in the gastrointestinal tract, lymph nodes, and bone marrow. Most patients with AIDS who have T-cell counts lower than 100 cells/mm^3 have widespread disease at diagnosis and are debilitated. MAC infections are associated with rising mortality rates.

Tuberculosis

In HIV-negative people with latent tuberculosis (TB) infection, the *lifetime* risk of developing active TB disease is

CHART 52-8 NURSING RESEARCH PROFILE

HIV-Related Fatigue

Barroso, J., Pence, B., Salahuddin, N., et al. (2008). Physiological correlates of HIV-related fatigue. *Clinical Nursing Research, 17*(1), 5–19.

Purpose

Symptom management in people with HIV infection is an increasingly pressing concern. The most frequent and debilitating symptom of HIV is fatigue, which has been defined as "awareness of a decreased capacity for physical and/or mental activity due to an imbalance in the availability, utilization, and/or restoration of resources needed to perform activity" (p. 6).

Design

This study used a longitudinal, repeated-measures design over a 3-year period with a total of seven study visits. The researchers investigated the cross-sectional relationship between fatigue and a wide range of physiologic characteristics in a sample of 128 HIV-positive individuals. Participants completed the HIV-related Fatigue Scale (HRFS), which consists of two subscales: (1) fatigue intensity and (2) impact of fatigue on daily functioning. After completion of the HRFS, blood was drawn to measure hepatic function, thyroid function, HIV viral load, immunologic function, gonadal function, hematologic function, and cellular injury.

Findings

None of the physiologic variables was significantly correlated with the HRFS scales in multivariate linear regression after controlling for income and years since HIV diagnosis. Income and years of HIV infection were more correlated with fatigue than any of the physiologic measures. Most of the participants had moderate fatigue.

Nursing Implications

Although fatigue is a common symptom that affects daily functioning and quality of life, and it was expected that physiologic variables such as viral load, liver function, and thyroid levels would be associated with fatigue, no statistically significant relationship was found. Fatigue affects the patient's ability to participate in activities of daily living such as grocery shopping and house cleaning and affects the patient's ability to maintain social relationships. Less income for people with HIV often means there are fewer choices and more energy is required to meet basic needs. The relationship between the length of time that patients have been HIV positive and have had fatigue might be a result of patients simply growing tired of living with the day-to-day challenges of HIV infection.

Until a better cause of fatigue is identified and treatments are developed, nurses need to help patients cope with this feeling of profound exhaustion. Nurses need to assist patients to create daily schedules around their periods of maximum energy and to assure them that fatigue is not necessarily associated with getting sicker. Nurses can support patients in accessing resources and also provide the emotional support needed to cope with an unpredictable chronic illness.

5% to 10%, whereas in HIV-positive people with latent TB, the *annual* risk is 10%. TB can develop in the lungs as well as in extrapulmonary sites such as the central nervous system (CNS), bone, pericardium, stomach, peritoneum, and scrotum. The CD4 T-cell count influences both the frequency and clinical picture of active TB disease. Important issues in the use of antiretroviral therapy in patients with active TB disease are (1) the sequencing of treatments, (2) the value of directly observed therapy, (3) risk of significant drug interactions with rifamycins, (4) the additive risk of hepatotoxicity and neuropathy associated with agents used to treat HIV and TB, (5) development of IRIS with TB after initiation of antiretroviral therapy, (6) the effect of antiretroviral therapy on results of tuberculin skin testing, and (7) the need for integration of therapy for HIV and TB (Guidelines, 2008).

Gastrointestinal Manifestations

The gastrointestinal manifestations of AIDS include loss of appetite, nausea, vomiting, oral and esophageal candidiasis, and chronic diarrhea. Diarrhea is a problem in 50% to 90% of all AIDS patients. Gastrointestinal symptoms may be related to the direct effect of HIV on the cells lining the intestines. Some of the enteric pathogens that occur most frequently, identified by stool cultures or intestinal biopsy, are *Cryptosporidium muris*, *Salmonella* species, *Isospora belli*, *Giardia lamblia*, CMV, *Clostridium difficile*, and *M. avium-intracellulare*. In patients with AIDS, the effects of diarrhea can be devastating in terms of profound weight loss (more than 10% of body weight), fluid and electrolyte imbalances, perianal skin excoriation, weakness, and inability to perform the usual activities of daily living.

Oral Candidiasis

Candidiasis, a fungal infection, occurs in almost all patients with AIDS and AIDS-related conditions. Commonly preceding other life-threatening infections, it is characterized by creamy-white patches in the oral cavity. If left untreated, oral candidiasis progresses to involve the esophagus and stomach. Associated signs and symptoms include difficult and painful swallowing and retrosternal pain. Some patients also develop ulcerating oral lesions and are particularly susceptible to dissemination of candidiasis to other body systems.

Wasting Syndrome

Wasting syndrome is part of the category C case definition for AIDS. Diagnostic criteria include profound involuntary weight loss exceeding 10% of baseline body weight and either chronic diarrhea for more than 30 days or chronic weakness and documented intermittent or constant fever in the absence of any concurrent illness that could explain these findings. This protein–energy malnutrition is multifactorial. In some AIDS-associated illnesses, patients experience a hypermetabolic state in which excessive calories are burned and lean body mass is lost. This state is similar to that seen in sepsis or trauma and can lead to organ failure. The distinction between cachexia (wasting) and

malnutrition, or between cachexia and simple weight loss, is important, because the metabolic derangement seen in wasting syndrome may not be modified by nutritional support alone.

Anorexia, diarrhea, gastrointestinal malabsorption, and lack of nutrition in chronic disease all contribute to wasting syndrome. Progressive tissue wasting, however, may occur with only modest gastrointestinal involvement and without diarrhea. TNF and IL-1 are cytokines that play important roles in AIDS-related wasting syndrome. Both act directly on the hypothalamus to cause anorexia. Cytokine-induced fever accelerates the body's metabolism by 14% for every 1°F increase in temperature. TNF causes inefficient use of lipids by reducing enzymes that are needed for fat metabolism, whereas IL-1 triggers the release of amino acids from muscle tissue. People with AIDS generally experience increased protein metabolism in relation to fat metabolism, which results in significant decreases in lean body mass due to muscle and protein breakdown.

Hypertriglyceridemia, seen in people with AIDS and attributed to chronically elevated cytokine levels, can persist for months without tissue wasting and loss of lean body mass. It is believed that infections and sepsis lead to transient increases in TNF, IL-1, and other cell mediators above the chronically elevated levels that are often seen with AIDS. These transient increases in TNF and IL-1 trigger muscle wasting.

Oncologic Manifestations

Certain types of cancer occur often in people with AIDS. As a result, these cancers are considered AIDS-defining conditions; that is, their presence in a person infected with HIV is a clear sign that AIDS has developed. These AIDS-related cancers include Kaposi's sarcoma, lymphoma (especially non-Hodgkin lymphoma and primary central nervous system lymphoma), and invasive cervical cancer. With HAART, the incidence of both Kaposi's sarcoma and non-Hodgkin lymphoma has decreased considerably (Levine, 2008). Kaposi's sarcoma and lymphoma are discussed below. Cervical carcinoma is described later in Gynecologic Manifestations.

Kaposi's Sarcoma

Kaposi's sarcoma (KS), the most common HIV-related malignancy, is a disease that involves the endothelial layer of blood and lymphatic vessels. In people with AIDS, epidemic KS is most often seen among male homosexuals and bisexuals. AIDS-related KS exhibits a variable and aggressive course, ranging from localized cutaneous lesions to disseminated disease involving multiple organ systems. Cutaneous signs may be the first manifestation of HIV, appearing in more than 90% of HIV-infected patients as immune functions deteriorate. These skin signs correlate to low CD4+ counts. They can appear anywhere on the body and are usually brownish pink to deep purple. They may be flat or raised and surrounded by ecchymoses (hemorrhagic patches) and edema (Fig. 52-3). Rapid development of lesions involving large areas of skin is associated with extensive disfigurement. The location and size of some lesions can lead to venous stasis, lymphedema, and pain. Ulcerative lesions disrupt skin integrity and increase discomfort and susceptibility to infection. The most common sites of visceral involvement are the lymph nodes, gastrointestinal tract, and lungs. Involvement of internal organs may eventually lead to organ failure, hemorrhage, infection, and death.

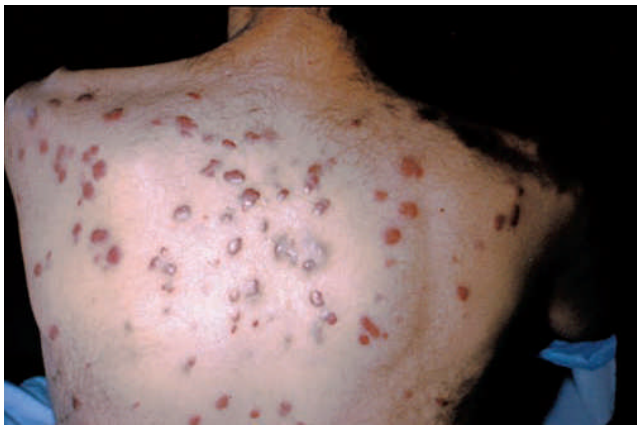


Figure 52-3 Lesions of the AIDS-related Kaposi's sarcoma. Whereas some patients may have lesions that remain flat, others experience extensively disseminated, raised lesions with edema. From DeVita Jr., V. T., Hellman, S. & Rosenberg, S. (Eds.). (1993). *AIDS: Etiology, diagnosis, treatment, and prevention* (4th ed.). Philadelphia: Lippincott Williams & Wilkins.

Diagnosis of KS is confirmed by biopsy of suspected lesions. Prognosis depends on the extent of the tumor, the presence of other symptoms of HIV infection, and the CD4+ count. Death may result from tumor progression. More often, however, it results from other complications of HIV infection.

B-Cell Lymphomas

B-cell lymphomas are the second most common malignancy occurring in people with AIDS. Lymphomas associated with AIDS usually differ from those occurring in the general population. Patients with AIDS are typically much younger than the usual population affected by non-Hodgkin lymphoma. In addition, AIDS-related lymphomas tend to develop outside the lymph nodes, most commonly in the brain, bone marrow, and gastrointestinal tract. These types of lymphomas are characteristically of a higher grade, indicating aggressive growth and resistance to treatment. The course of AIDS-related lymphomas includes multiple sites of organ involvement and complications related to opportunistic infections. Although aggressive combination chemotherapy is frequently successful in the treatment of non-Hodgkin lymphoma that is not associated with HIV infection, treatment is less successful in people with AIDS because of severe hematologic toxicity and complications of opportunistic infections that can occur from treatment.

Neurologic Manifestations

The advent of HAART greatly lowered the incidence of HIV dementia and increased the survival of people with HIV-associated neurocognitive disorders (McArthur, 2008). These disorders consist of cognitive impairment that is often accompanied by motor dysfunction and behavioral change. Neurologic dysfunction results from direct effects of

HIV on nervous system tissue, opportunistic infections, primary or metastatic neoplasms, cerebrovascular changes, metabolic encephalopathies, or complications secondary to therapy. Immune system response to HIV infection in the CNS includes inflammation, atrophy, demyelination, degeneration, and necrosis.

Peripheral Neuropathy

HIV-associated **peripheral neuropathy** is common across the trajectory of HIV disease and may occur in a variety of patterns, with distal sensory polyneuropathy (DSPN) or distal symmetric polyneuropathy the most frequently occurring type. DSPN occurs in advanced HIV disease as a result of immunosuppression, antiretroviral drug toxicity, and/or mitochondrial toxicity. It can lead to significant pain and decreased function (Nicholas, Voss, Corless, et al., 2007).

HIV Encephalopathy

HIV encephalopathy was formerly referred to as AIDS dementia complex (Chart 52-9). It is a clinical syndrome that is characterized by a progressive decline in cognitive, behavioral, and motor functions. Substantial evidence exists that HIV encephalopathy is a direct result of HIV infection. HIV has been found in the brain and cerebrospinal fluid (CSF) of patients with HIV encephalopathy. The brain cells infected by HIV are predominantly the CD4+ cells of monocyte-**macrophage** lineage. HIV infection is thought to trigger the release of toxins or lymphokines that result in cellular dysfunction or interference with neurotransmitter function rather than cellular damage.

Signs and symptoms may be subtle and difficult to distinguish from fatigue, depression, or the adverse effects of treatment for infections and malignancies. Early manifestations include memory deficits, headache, difficulty concentrating, progressive confusion, psychomotor slowing, apathy, and ataxia. Later stages include global cognitive impairments, delay in verbal responses, a vacant stare, spastic paraparesis, hyperreflexia, psychosis, hallucinations, tremor, incontinence, seizures, mutism, and death.

Confirming the diagnosis of HIV encephalopathy can be difficult. Extensive neurologic evaluation includes a computed tomography scan, which may indicate diffuse cerebral atrophy and ventricular enlargement. Other tests that may detect abnormalities include magnetic resonance imaging, analysis of CSF through lumbar puncture, and brain biopsy.

Cryptococcus neoformans

A fungal infection, *C. neoformans* is another common opportunistic infection among patients with AIDS, and it causes neurologic disease. Cryptococcal meningitis is characterized by symptoms such as fever, headache, malaise, stiff neck, nausea, vomiting, mental status changes, and seizures. Diagnosis is confirmed by CSF analysis.

Chart 52-9 • *Care of the Patient With HIV Encephalopathy*

Disturbed Thought Processes

- Assess mental status and neurologic functioning.
- Monitor for medication interactions, infections, electrolyte imbalance, and depression.
- Frequently orient the patient to time, place, person, reality, and the environment.
- Use simple explanations.
- Teach the patient to perform tasks in incremental steps.
- Provide memory aids (clocks and calendars).
- Provide memory aids for medication administration.
- Post activity schedule.
- Give positive feedback for appropriate behavior.
- Teach caretakers how to orient patient to time, place, person, reality, and the environment.
- Encourage the patient to designate a responsible person to assume power of attorney.

Disturbed Sensory Perception

- Assess sensory impairment.
- Decrease amount of stimuli in the patient's environment.
- Correct inaccurate perceptions.
- Provide reassurance and safety if the patient displays fear.
- Provide a secure and stable environment.
- Teach caregivers how to recognize inaccurate sensory perceptions.
- Teach caregivers techniques to correct inaccurate perceptions.
- Teach the patient and caregivers to report any changes in the patient's vision to the patient's health care provider.

Risk for Injury

- Assess the patient's level of anxiety, confusion, or disorientation.
- Assess the patient for delusions or hallucinations.
- Remove potentially dangerous objects from the patient's environment.
- Structure the environment for safety (ensure adequate lighting, avoid clutter, provide bed rails if needed).
- Supervise smoking.
- Do not let the patient drive a car if confusion is present.
- Instruct the patient and caregiver in home safety.
- Provide assistance as needed for ambulation and in getting in and out of bed.
- Pad headboard and side rails if the patient has seizures.

Self-Care Deficits

- Encourage activities of daily living within the patient's level of ability.
- Encourage independence but assist if the patient cannot perform an activity.
- Demonstrate any activity that the patient is having difficulty accomplishing.
- Monitor food and fluid intake.
- Weigh patient weekly.
- Encourage the patient to eat, and offer nutritious meals, snacks, and adequate fluids.
- If patient is incontinent, establish a routine toileting schedule.
- Teach caregivers how to meet the patient's self-care needs.

Progressive Multifocal Leukoencephalopathy

Progressive multifocal leukoencephalopathy (PML) is a demyelinating CNS disorder that affects the oligodendroglia. Clinical manifestations often begin with mental confusion and rapidly progress to include blindness, aphasia, muscle weakness, paresis (partial or complete paralysis), and death. Treatments have greatly reduced the threat of mortality associated with this disorder.

Other Neurologic Disorders

Other common infections involving the nervous system include *Toxoplasma gondii*, CMV, and *Mycobacterium tuberculosis* infections. Additional neurologic manifestations include both central and peripheral neuropathies. Vascular myelopathy is a degenerative disorder that affects the lateral and posterior columns of the spinal cord, resulting in progressive spastic paraparesis, ataxia, and incontinence.

Depressive Manifestations

The prevalence of depression among people with HIV infection is unknown. The causes of depression are multifactorial and may include a history of preexisting mental illness, neuropsychiatric disturbances, and psychosocial factors. Depression also occurs in people with HIV infection in response to the physical symptoms, including pain and weight loss, and the lack of someone to talk with about their concerns. People with HIV/AIDS who are depressed may experience irrational guilt and shame, loss of self-esteem, feelings of helplessness and worthlessness, and suicidal ideation.

Integumentary Manifestations

Cutaneous manifestations are associated with HIV infection and the accompanying opportunistic infections and malignancies. KS (described earlier) and opportunistic infections such as herpes zoster and herpes simplex are associated with painful vesicles that disrupt skin integrity. Molluscum contagiosum is a viral infection characterized by deforming plaque formation. Seborrheic dermatitis is associated with an indurated, diffuse, scaly rash involving the scalp and face. Patients with AIDS may also exhibit a generalized folliculitis associated with dry, flaking skin or atopic dermatitis, such as eczema or psoriasis. Up to 60% of patients treated with the antibacterial agent trimethoprim-sulfamethoxazole (TMP-SMZ) develop a drug-related rash that is pruritic with pinkish-red macules and papules. Patients with any of these rashes experience discomfort and are at increased risk for infection from disrupted skin integrity.

Endocrine Manifestations

The endocrine manifestations of HIV infection are not completely understood. At autopsy, endocrine glands show infiltration and destruction from opportunistic infections or neoplasms. Endocrine function may also be affected by therapeutic agents.

Gynecologic Manifestations

Persistent, recurrent vaginal candidiasis may be the first sign of HIV infection in women. Past or present genital ulcers are a risk factor for the transmission of HIV infection. Women with HIV infection are more susceptible to genital ulcers and venereal warts and have increased rates of incidence and recurrence of these conditions. Ulcerative sexually transmitted diseases (STDs) such as chancroid, syphilis, and herpes are more severe in women with HIV infection. **Human papillomavirus (HPV)** causes venereal warts and is a risk factor for cervical intraepithelial neoplasia, a cellular change that is frequently a precursor to cervical cancer. Women with HIV are 10 times more likely to develop cervical intraepithelial neoplasia than those not infected with HIV. There is a strong association between abnormal Papanicolaou (Pap) smears and HIV seropositivity. HIV-seropositive women with cervical carcinoma present at a more advanced stage of disease and have more persistent and recurrent disease and a shorter interval to recurrence and death than women without HIV infection.

A significant percentage of women who require hospitalization for pelvic inflammatory disease have HIV infection. Women with HIV are at increased risk for pelvic inflammatory disease, and the associated inflammation may potentiate the transmission of HIV infection. Moreover, women with HIV infection appear to have a higher incidence of menstrual abnormalities, including amenorrhea or bleeding between periods, than do women without HIV infection. The failure of health care providers to consider HIV infection in women may lead to a later diagnosis, thereby denying these patients appropriate treatment.

Medical Management

Treatment of Opportunistic Infections

Guidelines for the treatment of opportunistic infections should be consulted for the most current recommendations (Guidelines for Prevention and Treatment of Opportunistic Infections in HIV-Infected Adults and Adolescents [OI Guidelines], 2009). Despite the availability of antiretroviral medications, opportunistic infections (OIs) continue to cause considerable morbidity and mortality for three main reasons: (1) many patients are unaware of their HIV infection and present with an OI as the initial indicator of their disease, (2) some patients are aware of their HIV infection but do not take antiretroviral agents because of psychosocial or economic factors, and (3) others receive prescriptions for antiretroviral medications but fail to attain adequate virologic and immunologic response as a result of issues related to adherence, pharmacokinetics, or unexplained biologic factors (OI Guidelines, 2009).

Immune function should improve with initiation of HAART, resulting in faster resolution of the OI. This has been most clearly shown for OIs for which effective therapy does not exist, such as cryptosporidiosis, microsporidiosis, and PML. These conditions may resolve or at least stabilize after the institution of antiretroviral therapy as well as resolution of lesions of KS (OI Guidelines, 2009).

Pneumocystis Pneumonia

TMP-SMZ is the treatment of choice for PCP; it is as effective as parenteral pentamidine and more effective than other regimens. Oral outpatient therapy using TMP-SMZ is highly effective among patients with mild t-moderate PCP. Adjunctive corticosteroids should be started as early as possible and certainly within 72 hours after starting specific

PCP therapy. Alternative therapeutic regimens for patients with mild to moderate disease include (1) dapsone and TMP, (2) primaquine plus clindamycin, and (3) atovaquone suspension. Alternatives for patients with moderate to severe disease include (1) primaquine plus clindamycin or (2) IV pentamidine (generally the drug of second choice for severe disease; it can cause severe hypotension if it is administered too rapidly). Aerosolized pentamidine should not be used for the treatment of PCP because of limited efficacy and more frequent relapse. The recommended duration of therapy for PCP is 21 days (OI Guidelines, 2009). Adverse effects, in addition to hypotension, include impaired glucose metabolism leading to the development of diabetes mellitus from damage to the pancreas, renal damage, hepatic dysfunction, and neutropenia.

Mycobacterium Avium Complex

HIV-infected adults and adolescents should receive chemoprophylaxis against disseminated MAC disease if they have a CD4+ count less than 50 cells/μL. Azithromycin (Zithromax) or clarithromycin (Biaxin) are the preferred prophylactic agents. If azithromycin or clarithromycin cannot be tolerated, rifabutin is an alternative prophylactic agent for MAC disease, although drug interactions may make this agent difficult to use (OI Guidelines, 2009). Secondary prophylaxis for disseminated MAC may be discontinued in patients who have sustained increases in CD4 counts (greater than 100 cells/mm^3; longer than 3 months) in response to HAART, have completed 12 months of MAC therapy, and have no signs or symptoms attributable to MAC.

Cryptococcal Meningitis

Cryptococcosis among patients with HIV infection most commonly occurs as a subacute meningitis or meningoencephalitis with fever, malaise, and headache. Current primary therapy for cryptococcal meningitis is IV amphotericin B with or without oral flucytosine (5-FC, Ancobon) or fluconazole (Diflucan). Serious potential adverse effects of amphotericin B include anaphylaxis, renal and hepatic impairment, electrolyte imbalances, anemia, fever, and severe chills (OI Guidelines, 2009).

Cytomegalovirus Retinitis

Retinitis caused by CMV is a leading cause of blindness in patients with AIDS. Oral valganciclovir, IV ganciclovir, IV ganciclovir followed by oral valganciclovir, IV foscarnet, IV cidofovir, and the ganciclovir intraocular implant coupled with valganciclovir are all effective treatments for CMV retinitis (OI Guidelines, 2009). A common adverse reaction to ganciclovir is severe neutropenia, which limits the concomitant use of zidovudine (AZT, Compound S, Retrovir). Common adverse reactions to foscarnet are nephrotoxicity, including acute renal failure, and electrolyte imbalances, including hypocalcemia, hyperphosphatemia, and hypomagnesemia, which can be life-threatening. Other common adverse effects include seizures, gastrointestinal disturbances, anemia, phlebitis at the infusion site, and low back pain. Possible bone marrow suppression (producing a decrease in white blood cell and platelet counts), oral candidiasis, and liver and renal impairments require close monitoring.

Other Infections

Oral acyclovir, famciclovir, or valacyclovir may be used to treat infections caused by herpes simplex or herpes zoster. Esophageal or oral candidiasis is treated topically with clotrimazole (Mycelex) oral troches or nystatin suspension. Chronic refractory infection with candidiasis (thrush) or esophageal involvement is treated with ketoconazole (Nizoral) or fluconazole (Diflucan).

Prevention of Opportunistic Infections

TMP-SMZ (Bactrim, Cotrim, Septra) is an antibacterial agent used to treat various organisms causing infection. It also confers cross-protection against toxoplasmosis and some common respiratory bacterial infections. People with HIV infection who have a T-cell count of less than 200 cells/mm^3 should receive chemoprophylaxis with TMP-SMZ to prevent PCP. PCP prophylaxis can be safely discontinued in patients who are responding to HAART with a sustained increase in T lymphocytes.

Antidiarrheal Therapy

Although many forms of diarrhea respond to treatment, it is not unusual for this condition to recur and become a chronic problem for the patient with HIV infection. Therapy with octreotide acetate (Sandostatin), a synthetic analogue of somatostatin, has been shown to be effective in managing chronic severe diarrhea. High concentrations of somatostatin receptors have been found in the gastrointestinal tract and in other tissues. Somatostatin inhibits many physiologic functions, including gastrointestinal motility and intestinal secretion of water and electrolytes.

Chemotherapy

Kaposi's Sarcoma

Management of KS is usually difficult because of the variability of symptoms and the organ systems involved. KS is rarely life-threatening except when there is pulmonary or gastrointestinal involvement. The treatment goals are to reduce symptoms by decreasing the size of the skin lesions, to reduce discomfort associated with edema and ulcerations, and to control symptoms associated with mucosal or visceral involvement. No one treatment has been shown to increase survival. Radiation therapy is effective as a palliative measure to relieve localized pain due to tumor mass (especially in the legs) and for KS lesions that are in sites such as the oral mucosa, conjunctiva, face, and soles of the feet.

Interferon is known for its antiviral and antitumor effects. Patients with cutaneous KS treated with **alpha-interferon** have experienced tumor regression and improved immune system function. Alpha-interferon is administered by the IV, intramuscular, or subcutaneous route. Patients may self-administer interferon at home or receive interferon in an outpatient setting.

Lymphoma

Successful treatment of AIDS-related lymphomas has been limited because of the rapid progression of these malignancies. Combination chemotherapy and radiation therapy regimens may produce an initial response, but it is usually short-lived. Because standard regimens for non-AIDS

lymphomas have been ineffective, many clinicians suggest that AIDS-related lymphomas should be studied as a separate group in clinical trials.

Antidepressant Therapy

Treatment for depression in people with HIV infection involves psychotherapy integrated with pharmacotherapy. If depressive symptoms are severe and of sufficient duration, treatment with antidepressants may be initiated. Antidepressants such as imipramine (Tofranil), desipramine (Norpramin), and fluoxetine (Prozac) may be used, because these medications also alleviate the fatigue and lethargy that are associated with depression. A psychostimulant such as methylphenidate (Ritalin) may be used in low doses in patients with neuropsychiatric impairment. Electroconvulsive therapy may be an option for patients with severe depression who do not respond to pharmacologic interventions.

Nutrition Therapy

Malnutrition increases the risk of infection and the incidence of opportunistic infections. Nutrition therapy should be part of the overall management plan and should be tailored to meet the nutritional needs of the patient, whether by oral diet, enteral tube feedings, or parenteral nutritional support, if needed. As with all patients, a healthy diet is essential for the patient with HIV infection. For patients with diarrhea, a diet low in fat, lactose, insoluble fiber, and caffeine and high in soluble fiber is helpful (Anastasi, Capili, Kim, et al., 2006). For all patients with AIDS who experience unexplained weight loss, calorie counts should be obtained to evaluate nutritional status and initiate appropriate therapy. The goal is to maintain the ideal weight and, when necessary, to increase weight.

Appetite stimulants have been successfully used in patients with AIDS-related anorexia. Megestrol acetate (Megace), a synthetic oral progesterone preparation, promotes significant weight gain and inhibits cytokine IL-1 synthesis. In patients with HIV infection, it increases body weight primarily by increasing body fat stores. Dronabinol (Marinol), which is a synthetic tetrahydrocannabinol (THC), the active ingredient in marijuana, has been used to relieve nausea and vomiting associated with cancer chemotherapy. After beginning dronabinol therapy, almost all patients with HIV infection experience a modest weight gain. The effects on body composition are unknown.

Oral supplements may be used to supplement diets that are deficient in calories and protein. Ideally, oral supplements should be lactose-free (many people with HIV infection are intolerant to lactose), high in calories and easily digestible protein, low in fat with the fat easily digestible, palatable, inexpensive, and tolerated without causing diarrhea. Advera is a nutritional supplement that has been developed specifically for people with HIV infection and AIDS. Parenteral nutrition is the final option because of its prohibitive cost and associated risks, including risk of infections.

Complementary and Alternative Modalities

Traditional Western medicine focuses on the treatment of disease. These treatments or interventions are taught in medical schools and are used by physicians in the care of patients. Complementary and alternative medicine (CAM) is often viewed as consisting of unconventional and unorthodox treatments or interventions that are not traditionally taught in medical schools. These modalities and therapies stress the need to treat the whole person, recognizing the interaction of the body, mind, and spirit. What is considered to be CAM in one culture may actually be a traditional therapy in another. People with HIV infection report substantial use of CAM for symptom management (Andrade & Anderson, 2008). The use of CAM in HIV infection and AIDS often results because of disillusionment with standard medical treatment, which to date has provided no cure. Combined with traditional therapies, CAM may improve the patient's overall well-being. However, there can be adverse drug–drug interactions between certain CAM therapies (eg, St. John's wort) and some antiretroviral medications.

CAM can be divided into four categories:

- Spiritual or psychological therapies may include humor, hypnosis, faith healing, guided imagery, and positive affirmations.
- Nutritional therapies may include vegetarian or macrobiotic diets, vitamin C or beta-carotene supplements, and turmeric, which contains curcumin, a food spice supplement. Chinese herbs, such as traditional herbal mixtures, are also used, in addition to compound Q (a Chinese cucumber extract) and *Momordica charantia* (bitter melon), which is given as an enema.
- Drug and biologic therapies include medications and other substances not approved by the FDA. Examples are *N*-acetylcysteine, pentoxifylline (Trental), and 1-chloro-2, 4-dinitrobenzene. Also included in this category are oxygen therapy, ozone therapy, and urine therapy.
- Treatment with physical forces and devices may include acupuncture, acupressure, massage therapy, reflexology, therapeutic touch, yoga, and crystals.

Although there is insufficient research on the effects of CAM, there is a growing body of literature reporting benefits for modalities involving nutrition, exercise, psychosocial treatment, and Chinese medicine.

Many patients who use these therapies do not report use of CAM to their health care providers. To obtain a complete health history, the nurse should ask about the patient's use of alternative therapies. Patients may need to be encouraged to report their use of CAM to their primary health care provider. Problems may arise, for example, when patients are using CAM while participating in clinical drug trials; alternative therapies can have significant adverse side effects, making it difficult to assess the effects of the medications in the clinical trial. The nurse needs to become familiar with the potential adverse side effects of these therapies. The nurse who suspects that CAM is causing a side effect needs to discuss this with the patient, the alternative therapy provider, and the primary health care provider. It is important for the nurse to view CAM with an open mind and to try to understand the importance of this treatment to the patient. This approach will improve communication with the patient and reduce conflict.

Supportive Care

Patients who are weak and debilitated as a result of chronic illness associated with HIV infection typically require many kinds of supportive care. Nutritional support may be as simple as providing assistance in obtaining or preparing meals. For patients with more advanced nutritional impairment resulting from decreased intake, wasting syndrome, or gastrointestinal malabsorption associated with diarrhea, parenteral feedings may be required. Imbalances that result from nausea, vomiting, and profuse diarrhea often necessitate IV fluid and electrolyte replacement.

Management of skin breakdown associated with KS, perianal skin excoriation, or immobility entails thorough and meticulous skin care that involves regular turning, cleansing, and applications of medicated ointments and dressings. To combat pain associated with skin breakdown, abdominal cramping, peripheral neuropathy, or KS, it is necessary to administer analgesic agents at regular intervals around the clock. Relaxation and guided imagery may be helpful in reducing pain and anxiety.

Pulmonary symptoms, such as dyspnea and shortness of breath, may be related to infection, KS, or fatigue. For patients with these symptoms, oxygen therapy, relaxation training, and energy conservation techniques may be effective. Patients with severe respiratory dysfunction may require mechanical ventilation. Before mechanical ventilation is instituted, the procedure is explained to the patient and the caregiver. If the patient decides to forgo mechanical ventilation, his or her wishes should be followed. Ideally, the patient has prepared an advance directive identifying preferences for treatments and end-of-life care, including hospice care. If the patient has not identified preferences in advance, treatment options are described so that the patient can make informed decisions and have those wishes respected.

NURSING PROCESS

The Patient With HIV/AIDS

The nursing care of patients with AIDS is challenging because of the potential for any organ system to be the target of infections or cancer. In addition, this disease is complicated by many emotional, social, and ethical issues. The plan of care for the patient with AIDS (Chart 52-10) is individualized to meet the needs of the patient.

The scope and standards of HIV/AIDS nursing developed by the Association of Nurses in AIDS Care (2007) guide nursing practice. Care includes many of the interventions and concerns cited in the section on supportive care.

Assessment

Nursing assessment includes identification of potential risk factors, including a history of risky sexual practices or IV/injection drug use. The patient's physical status and psychological status are assessed. All factors affecting immune system functioning are thoroughly explored.

Nutritional Status

Nutritional status is assessed by obtaining a dietary history and identifying factors that may interfere with oral intake, such as anorexia, nausea, vomiting, oral pain, or difficulty swallowing. In addition, the patient's ability to purchase and prepare food is assessed. Weight history (ie, changes over time); anthropometric measurements; and blood urea nitrogen (BUN), serum protein, albumin, and transferrin levels provide objective measurements of nutritional status.

Skin Integrity

The skin and mucous membranes are inspected daily for evidence of breakdown, ulceration, or infection. The oral cavity is monitored for redness, ulcerations, and the presence of creamy-white patches indicative of candidiasis. Assessment of the perianal area for excoriation and infection in patients with profuse diarrhea is important. Wounds are cultured to identify infectious organisms.

Respiratory Status

Respiratory status is assessed by monitoring the patient for cough, sputum production (ie, amount and color), shortness of breath, orthopnea, tachypnea, and chest pain. The presence and quality of breath sounds are investigated. Other measures of pulmonary function include chest x-ray results, arterial blood gas values, pulse oximetry, and pulmonary function test results.

Neurologic Status

Neurologic status is determined by assessing level of consciousness; orientation to person, place, and time; and memory lapses. Mental status is assessed as early as possible to provide a baseline. The patient is also assessed for sensory deficits (visual changes, headache, or numbness and tingling in the extremities), motor involvement (altered gait, paresis, or paralysis), and seizure activity.

Fluid and Electrolyte Balance

Fluid and electrolyte status is assessed by examining the skin and mucous membranes for turgor and dryness. Dehydration may be indicated by increased thirst, decreased urine output, postural hypotension, weak and rapid pulse, and urine specific gravity of 1.025 or more. Electrolyte imbalances, such as decreased serum sodium, potassium, calcium, magnesium, and chloride, typically result from profuse diarrhea. The patient is assessed for signs and symptoms of electrolyte deficits, including decreased mental status, muscle twitching, muscle cramps, irregular pulse, nausea and vomiting, and shallow respirations.

Knowledge Level

The patient's level of knowledge about the disease and the modes of disease transmission is evaluated. In addition, the level of knowledge of family and friends is assessed. The patient's psychological reaction to the diagnosis of HIV infection or AIDS is important to explore. Reactions vary among patients and may include denial, anger, fear, shame, withdrawal from social interactions, and depression. It is often helpful to gain an understanding of how the patient has dealt with illness and major life stresses in the past. The patient's resources for support are also identified.

CHART 52-10 PLAN OF NURSING CARE

Care of the Patient With AIDS

NURSING DIAGNOSIS: Diarrhea related to enteric pathogens or HIV infection
GOAL: Resumption of usual bowel habits

Nursing Interventions	Rationale	Expected Outcomes
1. Assess patient's normal bowel habits.	1. Provides baseline for evaluation	• Exhibits return to normal bowel patterns
2. Assess for diarrhea: frequent, loose stools; abdominal pain or cramping, volume of liquid stools, and exacerbating and alleviating factors.	2. Detects changes in status, quantifies loss of fluid, and provides basis for nursing measures	• Reports decreasing episodes of diarrhea and abdominal cramping • Identifies and avoids foods that irritate the gastrointestinal tract
3. Obtain stool cultures and administer antimicrobial therapy as prescribed.	3. Identifies pathogenic organism; therapy targets specific organism	• Appropriate therapy is initiated as prescribed • Exhibits normal stool cultures
4. Initiate measures to reduce hyperactivity of bowel: a. Maintain food and fluid restrictions as prescribed. Suggest BRAT diet (*b*ananas, *r*ice, *a*pplesauce, *t*ea and *t*oast). b. Discourage smoking. c. Avoid bowel irritants such as fatty or fried foods, raw vegetables, and nuts. Offer small, frequent meals.	4. Promotes bowel rest, which may decrease acute episodes a. Reduces stimulation of bowel b. Eliminates nicotine, which acts as bowel stimulant c. Prevents stimulation of bowel and abdominal distention and promotes adequate nutrition	• Maintains adequate fluid intake • Maintains body weight and reports no additional weight loss • States rationale for avoiding smoking • Enrolls in program to stop smoking • Uses medication as prescribed
5. Administer anticholinergic antispasmodics and opioids or other medications as prescribed.	5. Decreases intestinal spasms and motility	• Maintains adequate fluid status • Exhibits normal skin turgor, moist mucous membranes, adequate urine output, and absence of excessive thirst
6. Maintain fluid intake of at least 3 L/day unless contraindicated.	6. Prevents hypovolemia	

NURSING DIAGNOSIS: Risk for infection related to immunodeficiency
GOAL: Absence of infection

Nursing Interventions	Rationale	Expected Outcomes
1. Monitor for infection: fever, chills, and diaphoresis; cough; shortness of breath; oral pain or painful swallowing; creamy-white patches in oral cavity; urinary frequency, urgency, or dysuria; redness, swelling, or drainage from wounds; vesicular lesions on face, lips, or perianal area.	1. Allows for early detection of infection, essential for prompt initiation of treatment. Repeated and prolonged infections contribute to patient's debilitation.	• Identifies reportable signs and symptoms of infection • Reports signs and symptoms of infection if present • Exhibits and reports absence of fever, chills, and diaphoresis
2. Teach patient or caregiver about need to report possible infection.	2. Allows early detection of infection	• Exhibits normal (clear) breath sounds without adventitious breath sounds • Maintains weight
3. Monitor white blood cell count and differential.	3. Identifies elevated WBC possibly associated with infection	• Reports adequate energy level without excessive fatigue • Reports absence of shortness of breath and cough
4. Obtain cultures of wound drainage, skin lesions, urine, stool, sputum, mouth, and blood as prescribed. Administer antimicrobial therapy as prescribed.	4. Assists in determining offending organism to initiate appropriate treatment	• Exhibits pink, moist oral mucous membranes without fissures or lesions • Takes appropriate therapy as prescribed
5. Instruct patient in ways to prevent infection: a. Clean kitchen and bathroom surfaces with disinfectants. b. Clean hands thoroughly after exposure to body fluids. c. Avoid exposure to others' body fluids or sharing eating utensils. d. Turn, cough, and deep breathe, especially when activity is decreased.	5. Minimizes exposure to infection and transmission of HIV infection to others	• Experiences no infection • States rationale for strategies to avoid infection • Modifies activities to reduce exposure to infection or infectious persons • Practices "safer sex" • Avoids sharing eating utensils and toothbrush • Exhibits normal body temperature

Continued

CHART 52-10 PLAN OF NURSING CARE

Care of the Patient With AIDS (Continued)

Nursing Interventions	Rationale	Expected Outcomes
e. Maintain cleanliness of perianal area. f. Avoid handling pet excreta or cleaning litter boxes, bird cages, or aquariums. g. Cook meat and eggs thoroughly. 6. Maintain aseptic technique when performing invasive procedures such as venipunctures, bladder catheterizations, and injections.	6. Prevents hospital-acquired infections	• Uses recommended techniques to maintain cleanliness of skin, skin lesions, and perianal area • Has others handle pet excreta and cleanup • Uses recommended cooking techniques
NURSING DIAGNOSIS: Ineffective airway clearance related to *Pneumocystis* pneumonia, increased bronchial secretions, and decreased ability to cough related to weakness and fatigue **GOAL:** Improved airway clearance		
1. Assess and report signs and symptoms of altered respiratory status, tachypnea, use of accessory muscles, cough, color and amount of sputum, abnormal breath sounds, dusky or cyanotic skin color, restlessness, confusion, or somnolence.	1. Indicates abnormal respiratory function	• Maintains normal airway clearance: Respiratory rate <20 breaths/min Unlabored breathing without use of accessory muscles and flaring nares (nostrils) Skin color pink (without cyanosis) Alert and aware of surroundings Arterial blood gas values normal Normal breath sounds without adventitious breath sounds
2. Obtain sputum sample for culture prescribed. Administer antimicrobial therapy as prescribed.	2. Aids in identification of pathogenic organisms	• Begins appropriate therapy
3. Provide pulmonary care (cough, deep breathing, postural drainage, and vibration) every 2 to 4 hours.	3. Prevents stasis of secretions and promotes airway clearance	• Takes medication as prescribed • Reports improved breathing
4. Assist patient in attaining semi- or high Fowler's position.	4. Facilities breathing and airway clearance	• Maintains clear airway
5. Encourage adequate rest periods.	5. Maximizes energy expenditure and prevents excessive fatigue	• Coughs and takes deep breaths every 2–4 hours as recommended
6. Initiate measures to decrease viscosity of secretions: a. Maintain fluid intake of at least 3 L/day unless contraindicated. b. Humidify inspired air as prescribed. c. Consult with physician concerning use of mucolytic agents delivered through nebulizer or IPPB treatment.	6. Facilitates expectoration of secretions; prevents stasis of secretions	• Demonstrates appropriate positions and practices postural drainage every 2–4 hours • Reports reduced breathing difficulty when in semi- or high Fowler's position • Practices energy-conserving strategies and alternates rest with activity • Demonstrates reduction in thickness (viscosity) of pulmonary secretions • Reports increased ease in coughing up sputum
7. Perform tracheal suctioning as needed.	7. Removes secretions if patient is unable to do so	• Uses humidified air or oxygen as prescribed and indicated
8. Administer oxygen therapy as prescribed.	8. Increases availability of oxygen	• Indicates need for assistance with removal of pulmonary secretions
9. Assist with endotracheal intubation; maintain ventilator settings as prescribed.	9. Maintains ventilation	• Understands need for and cooperates with endotracheal intubation and use of a mechanical ventilator • Verbalizes concerns about respiratory difficulty, intubation, and mechanical ventilation
NURSING DIAGNOSIS: Imbalanced nutrition, less than body requirements, related to decreased oral intake **GOAL:** Improvement of nutritional status		
1. Assess for malnutrition with height, weight, age, BUN, serum protein, albumin, and transferrin levels, hemoglobin, hematocrit, and cutaneous anergy.	1. Provides objective measurement of nutritional status	• Identifies factors limiting oral intake and uses resources to promote adequate dietary intake

Continued on following page

CHART 52-10 PLAN OF NURSING CARE
Care of the Patient With AIDS (Continued)

Nursing Interventions	Rationale	Expected Outcomes
2. Obtain dietary history, including likes and dislikes and food intolerances.	2. Defines need for nutritional education; helps individualize interventions	• Reports increased appetite • States understanding of nutritional needs
3. Assess factors that interfere with oral intake.	3. Provides basis and directions for interventions	• Identifies ways to reduce factors that limit oral intake
4. Consult with dietitian to determine patient's nutritional needs.	4. Facilitates meal planning	• Rests before meals • Eats in pleasant, odor-free environment
5. Reduce factors limiting oral intake: a. Encourage patient to rest before meals. b. Plan meals so that they do not occur immediately after painful or unpleasant procedures. c. Encourage patient to eat meals with visitors or others when possible. d. Encourage patient to prepare simple meals or to obtain assistance with meal preparation if possible. e. Serve small, frequent meals: 6 per day. f. Limit fluids 1 hour before meals and with meals.	5. Addresses factors limiting intake: a. Minimizes fatigue, which can decrease appetite b. Decreases noxious stimuli c. Limits social isolation d. Limits energy expenditure e. Prevents overwhelming patient f. Reduces satiety	• Arranges meals to coincide with visitors' visits • Reports increased dietary intake • Uses oral hygiene before meals • Takes analgesic agents before meals as prescribed • Identifies ways to increase protein and caloric intake • Identifies foods high in protein and calories • Consumes foods high in protein and calories • Reports decreased rate of weight loss
6. Instruct patient in ways to supplement nutrition: consume protein-rich foods (meat, poultry, fish) and carbohydrates (pasta, fruit, breads).	6. Provides additional proteins and calories	• Maintains adequate intake • States rationale for enteral or parenteral nutrition if needed
7. Consult with physician and dietitian about alternative feeding (enteral or parenteral nutrition).	7. Provides nutritional support if patient is unable to take sufficient amounts by mouth	• Demonstrates skill in preparing alternate sources of nutrition
8. Consult with social worker or community liaison about financial assistance if patient cannot afford food.	8. Increases availability of resources and nutrition	

NURSING DIAGNOSIS: Deficient knowledge related to means of preventing HIV transmission
GOAL: Increased knowledge concerning means of preventing disease transmission

Nursing Interventions	Rationale	Expected Outcomes
1. Instruct patient, family, and friends about routes of transmission of HIV.	1. Knowledge about disease transmission can help prevent spread of disease; may also alleviate fears.	• Patient, family, and friends state means of transmission.
2. Instruct patient, family, and friends about means of preventing transmission of HIV: a. Avoid sexual contact with multiple partners, and use precautions if sexual partner's HIV status is not certain. b. Use condoms during sexual intercourse (vaginal, anal, oral–genital); avoid mouth contact with the penis, vagina, or rectum; avoid sexual practices that can cause cuts or tears in the lining of the rectum, vagina, or penis. c. Avoid sex with sex workers and others at high risk.	2. Reduces transmission risk a. The risk of infection increases with the number of sexual partners, male or female, and sexual contact with those who engage in high-risk behaviors. b. Risk of HIV transmission is reduced. c. Many sex workers are infected with HIV through sexual contact with multiple partners or IV/injection drug use.	• Reports and demonstrates practices to reduce exposure of others to HIV • Demonstrates knowledge of safer sexual practices • Identifies means of preventing disease transmission • States that sexual partners are informed about patient's positive HIV status in blood • Avoids IV/injection drug use and sharing of drug equipment with others

Continued

CHART 52-10 PLAN OF NURSING CARE

Care of the Patient With AIDS (Continued)

Nursing Interventions	Rationale	Expected Outcomes
d. Do not use IV/injection drugs; if addicted and unable or unwilling to change behavior, use clean needles and syringes.	d. Clean needles and syringes are the only way to prevent HIV transmission for those who continue to use drugs. Taking precautions is important for those who are antibody positive to prevent transmitting HIV.	
e. Women who may have been exposed to HIV through sexual or drug practices should consult with a physician before becoming pregnant; consider use of antiretroviral agents if pregnant.	e. HIV can be transmitted from mother to child in utero; use of antiretroviral agents during pregnancy significantly reduces perinatal transmission of HIV.	
NURSING DIAGNOSIS: Social isolation related to stigma of the disease, withdrawal of support systems, isolation procedures, and fear of infecting others **GOAL:** Decreased sense of social isolation		
Nursing Interventions	**Rationale**	**Expected Outcomes**
1. Assess patient's usual patterns of social interaction.	1. Establishes basis for individualized interventions	• Shares with others the need for valued social interaction • Demonstrates interest in events, activities, and communication • Verbalizes feelings and reactions to diagnosis, prognosis, and life changes • Identifies modes of transmission of HIV • States ways of preventing transmission of AIDS virus to others while maintaining contact with valued friends and relatives • Reveals AIDS diagnosis to others when appropriate • Identifies resources (ie, family, friends, and support groups) • Uses resources when appropriate • Accepts offers of assistance and support • Reports decreased sense of isolation • Maintains contacts with those of importance to him or her • Develops or continues hobbies that effectively serve as diversion or distraction
2. Observe for behaviors indicative of social isolation, such as decreased interaction with others, hostility, noncompliance, sad affect, and stated feelings of rejection or loneliness.	2. Promotes early detection of social isolation, which may be manifested in several ways	
3. Provide instruction concerning modes of transmission of HIV.	3. Provides accurate information, corrects misconceptions, and alleviates anxiety	
4. Assist patient to identify and explore resources for support and positive mechanisms for coping (eg, contact with family, friends, AIDS task force).	4. Enables mobilization of resources and supports	
5. Allow time to be with patient other than for medications and procedures.	5. Promotes feelings of self-worth and provides social interaction	
6. Encourage participation in diversional activities such as reading, television, or hand crafts.	6. Provides distraction	
COLLABORATIVE PROBLEMS: Opportunistic infections; impaired breathing; wasting syndrome and fluid and electrolyte imbalances; adverse reaction to medications **GOAL:** Absence of complications		
Nursing Interventions	**Rationale**	**Expected Outcomes**
Opportunistic Infections		
1. Monitor vital signs.	1. Changes in vital signs such as increases in pulse rate, respirations, blood pressure, and temperature may indicate infection.	• Exhibits stable vital signs • Experiences control of infection • Identifies signs and symptoms correctly and experiences no complications • Identifies signs and symptoms that are reportable to the physician • Takes medications as prescribed
2. Obtain laboratory specimens and monitor test results.	2. Smears and cultures can identify causative agents such as bacteria, fungi, and protozoa, and sensitivity studies can identify antibiotics or other medications effective against the causative agent.	

Continued on following page

CHART 52-10 PLAN OF NURSING CARE
Care of the Patient With AIDS (Continued)

Nursing Interventions	Rationale	Expected Outcomes
3. Instruct the patient and caregiver about signs and symptoms of infection and the need to report them early.	3. Early recognition of symptoms facilitates prompt treatment and avoids extra complications.	
Impaired Breathing		
1. Monitor respiratory rate and pattern.	1. Rapid shallow breathing, diminished breath sounds, and shortness of breath may indicate respiratory failure resulting in hypoxia.	• Maintains stable respiratory rate and pattern within the normal limits • Exhibits no adventitious lung sounds; normal breath sounds
2. Auscultate the chest for breath sounds and abnormal lung sounds.	2. Crackles and wheezes may indicate fluid in the lungs, which disrupts respiratory function and alters the blood's oxygen-carrying capacity.	• Has stable pulse rate and blood pressure within normal limits, and exhibits no evidence of hypoxia
3. Monitor pulse rate, blood pressure, and oxygen saturation levels.	3. Changes in pulse rate, blood pressure, and oxygen levels may indicate the development of respiratory or cardiac failure.	• Oxygen saturation levels within acceptable range
Wasting Syndrome and Fluid and Electrolyte Disturbances		
1. Monitor weight and laboratory values for nutritional status.	1. Weight loss, malnutrition, and anemia are common in HIV infection and increase risk for superinfection.	• Maintains stable weight • Eats a nutritious diet
2. Monitor intake and output and laboratory values for fluid and electrolyte imbalance (potassium, sodium, calcium, phosphorus, magnesium, and zinc).	2. Chronic diarrhea, inadequate oral intake, vomiting, and profuse sweating deplete electrolytes. Small intestine inflammation may impair the absorption of fluids and electrolytes.	• Attains and maintains hemoglobin, hematocrit, and ferritin levels within normal limits • Sustains fluid and electrolyte balance within normal limits
3. Monitor for and report signs and symptoms of dehydration.	3. Fluid loss results in decreased circulating volume leading to tachycardia, dry skin and mucous membranes, poor skin turgor, elevated urine specific gravity, and thirst. Early detection allows early treatment.	• Exhibits no signs and symptoms of dehydration
Reactions to Medications		
1. Monitor for medication interactions.	1. People with HIV infection receive many medications for HIV and for disease complications. Early detection of medication interactions is necessary to prevent complications.	• Experiences no serious side effects or complications from medications • Correctly describes medication regimen and complies with therapy, including adaptations in eating routines and type of food used with prescribed medications
2. Monitor for and promptly report side effects from antiretroviral agents.	2. Side effects from antiretroviral agents can be life-threatening. Serious side effects include anemia, pancreatitis, peripheral neuropathy, mental confusion, and persistent nausea and vomiting. Corrective measures need to be instituted.	
3. Instruct the patient and caregiver in the medication regimen.	3. Knowledge of the medication purpose, correct administration, side effects, and strategies to manage or prevent side effects promote safety and greater compliance with treatment.	

Diagnosis

Nursing Diagnoses

The list of potential nursing diagnoses is extensive because of the complex nature of this disease. However, based on assessment data, major nursing diagnoses for the patient may include the following:

- Impaired skin integrity related to cutaneous manifestations of HIV infection, excoriation, and diarrhea
- Diarrhea related to enteric pathogens or HIV infection
- Risk for infection related to immunodeficiency
- Activity intolerance related to weakness, fatigue, malnutrition, impaired fluid and electrolyte balance, and hypoxia associated with pulmonary infections

- Disturbed thought processes related to shortened attention span, impaired memory, confusion, and disorientation associated with HIV encephalopathy
- Ineffective airway clearance related to PCP, increased bronchial secretions, and decreased ability to cough related to weakness and fatigue
- Pain related to impaired perianal skin integrity secondary to diarrhea, KS, and peripheral neuropathy
- Imbalanced nutrition, less than body requirements, related to decreased oral intake
- Social isolation related to stigma of the disease, withdrawal of support systems, isolation procedures, and fear of infecting others
- Anticipatory grieving related to changes in lifestyle and roles and unfavorable prognosis
- Deficient knowledge related to HIV infection, means of preventing HIV transmission, and self-care

Collaborative Problems/Potential Complications

Based on the assessment data, possible complications may include the following:

- Opportunistic infections
- Impaired breathing or respiratory failure
- Wasting syndrome and fluid and electrolyte imbalance
- Adverse reaction to medications

Planning and Goals

Goals for the patient may include achievement and maintenance of skin integrity, resumption of usual bowel patterns, absence of infection, improved activity tolerance, improved thought processes, improved airway clearance, increased comfort, improved nutritional status, increased socialization, expression of grief, increased knowledge regarding disease prevention and self-care, and absence of complications.

Nursing Interventions

Promoting Skin Integrity

The skin and oral mucosa are assessed routinely for changes in appearance, location and size of lesions, and evidence of infection and breakdown. The patient is encouraged to maintain a balance between rest and mobility whenever possible. Patients who are immobile are assisted to change position every 2 hours. Devices such as alternating-pressure mattresses and low-air-loss beds are used to prevent skin breakdown. Patients are encouraged to avoid scratching; to use nonabrasive, nondrying soaps; and to apply nonperfumed skin moisturizers to dry skin. Regular oral care is also encouraged.

Medicated lotions, ointments, and dressings are applied to affected skin surfaces as prescribed. Adhesive tape is avoided. Skin surfaces are protected from friction and rubbing by keeping bed linens free of wrinkles and avoiding tight or restrictive clothing. Patients with foot lesions are advised to wear cotton socks and shoes that do not cause the feet to perspire. Antipruritic, antibiotic, and analgesic agents are administered as prescribed.

The perianal region is assessed frequently for impairment of skin integrity and infection. The patient is instructed to keep the area as clean as possible. The perianal area is cleaned after each bowel movement with nonabrasive soap and water to prevent further excoriation and breakdown of the skin and infection. If the area is very painful, soft cloths or cotton sponges may be less irritating than washcloths. In addition, sitz baths or gentle irrigation may facilitate cleaning and promote comfort. The area is dried thoroughly after cleaning. Topical lotions or ointments may be prescribed to promote healing. Wounds are cultured if infection is suspected, so that the appropriate antimicrobial treatment can be initiated. Debilitated patients may require assistance in maintaining hygienic practices.

Promoting Usual Bowel Patterns

Bowel patterns are assessed for diarrhea. The nurse monitors the frequency and consistency of stools and the patient's reports of abdominal pain or cramping associated with bowel movements. Factors that exacerbate frequent diarrhea are also assessed. The quantity and volume of liquid stools are measured to document fluid volume losses. Stool cultures are obtained to identify pathogenic organisms.

The patient is counseled about ways to decrease diarrhea. The physician may recommend restriction of oral intake to rest the bowel during periods of acute inflammation associated with severe enteric infections. As the patient's dietary intake is increased, foods that act as bowel irritants, such as raw fruits and vegetables, popcorn, carbonated beverages, spicy foods, and foods of extreme temperatures, should be avoided. Small, frequent meals help to prevent abdominal distention. Medications, such as anticholinergic agents, antispasmodic, agents, or opioids, can be prescribed to decrease diarrhea by decreasing intestinal spasms and motility. Administering antidiarrheal agents on a regular schedule may be more beneficial than administering them on an as-needed basis. Antibiotics and antifungal agents may also be prescribed to combat pathogens identified by stool cultures. Assessment of self-care strategies being used is essential.

Preventing Infection

The patient and caregivers are instructed to monitor for signs and symptoms of infection: fever; chills; night sweats; cough with or without sputum production; shortness of breath; difficulty breathing; oral pain or difficulty swallowing; creamy-white patches in the oral cavity; unexplained weight loss; swollen lymph nodes; nausea; vomiting; persistent diarrhea; frequency, urgency, or pain on urination; headache; visual changes or memory lapses; redness, swelling, or drainage from skin wounds; and vesicular lesions on the face, lips, or perianal area. The nurse also monitors laboratory test results that indicate infection, such as the white blood cell count and differential. Cultures of specimens from wound drainage, skin lesions, urine, stool, sputum, mouth, and blood are obtained to identify pathogenic organisms and the most appropriate antimicrobial therapy. The patient is instructed to avoid others with active infections such as upper respiratory infections.

Improving Activity Tolerance

Activity tolerance is assessed by monitoring the patient's ability to ambulate and perform activities of daily living. Patients may be unable to maintain their usual levels of

activity because of weakness, fatigue, shortness of breath, dizziness, and neurologic involvement. Assistance in planning daily routines that maintain a balance between activity and rest may be necessary. In addition, patients benefit from instructions about energy conservation techniques, such as sitting while washing or while preparing meals. Personal items that are frequently used should be kept within the patient's reach. Measures such as relaxation and guided imagery may be beneficial because they decrease anxiety, which contributes to weakness and fatigue.

Collaboration with other members of the health care team may uncover other factors associated with increasing fatigue and strategies to address them. For example, if fatigue is related to anemia, administering epoetin alfa (Epogen) as prescribed may relieve fatigue and increase activity tolerance.

Maintaining Thought Processes

The patient is assessed for alterations in mental status that may be related to neurologic involvement, metabolic abnormalities, infection, side effects of treatment, and coping mechanisms. Manifestations of neurologic impairment may be difficult to distinguish from psychological reactions to HIV infection, such as anger and depression.

If the patient experiences altered mental or cognitive status, family and support network members are instructed to speak to the patient in simple, clear language and give the patient sufficient time to respond to questions. The patient's support network is encouraged to orient the patient to the daily routine by talking about what is taking place during daily activities and encouraged to provide the patient with a regular daily schedule for medication administration, grooming, meal times, bedtimes, and awakening times. Posting the schedule in a prominent area (eg, on the refrigerator), providing night lights for the bedroom and bathroom, and planning safe leisure activities allow the patient to maintain a regular routine in a safe manner. Activities that the patient previously enjoyed are encouraged. These should be easy to accomplish and fairly short in duration. The nurse encourages the social support network to remain calm and not to argue with the patient while protecting the patient from injury. Around-the-clock supervision may be necessary, and strategies can be implemented to prevent the patient from engaging in potentially dangerous activities, such as driving, using the stove, or mowing the lawn. Strategies for improving or maintaining functional abilities and for providing a safe environment are used for patients with HIV encephalopathy (see Chart 52-9).

Improving Airway Clearance

Respiratory status, including rate, rhythm, use of accessory muscles, and breath sounds; mental status; and skin color must be assessed at least daily. Any cough and the quantity and characteristics of sputum are documented. Sputum specimens are analyzed for infectious organisms. Pulmonary therapy (coughing, deep breathing, postural drainage, percussion, and vibration) is provided as often as every 2 hours to prevent stasis of secretions and to promote airway clearance. Because of weakness and fatigue, many patients require assistance in attaining a position (such as a high Fowler's or semi-Fowler's position) that facilitates breathing and airway clearance. Adequate rest is essential to minimize energy expenditure and prevent excessive fatigue. The fluid volume status is evaluated so that adequate hydration can be maintained. Unless contraindicated because of renal or cardiac disease, daily intake of 3 L of fluid is encouraged. Humidified oxygen may be prescribed, and nasopharyngeal or tracheal suctioning, intubation, and mechanical ventilation may be necessary to maintain adequate ventilation.

Relieving Pain and Discomfort

The patient is assessed for the quality and severity of pain associated with impaired perianal skin integrity, the lesions of KS, and peripheral neuropathy. In addition, the effects of pain on elimination, nutrition, sleep, affect, and communication are explored, along with exacerbating and relieving factors. Cleaning the perianal area, as previously described, can promote comfort. Topical anesthetic medications or ointments may be prescribed. Use of soft cushions or foam pads may increase comfort while sitting. The patient is instructed to avoid foods that act as bowel irritants. Antispasmodic and antidiarrheal medications may be prescribed to reduce the discomfort and frequency of bowel movements. If necessary, systemic analgesic agents may also be prescribed. Pain from KS is frequently described as a sharp, throbbing pressure, and heaviness, if lymphedema is present. Pain management may include use of nonsteroidal anti-inflammatory drugs (NSAIDs) and opioids plus nonpharmacologic approaches such as relaxation techniques. When NSAIDs are administered to patients who are receiving zidovudine, hepatic and hematologic status must be monitored.

The patient with pain related to peripheral neuropathy frequently describes it as burning, numbness, and "pins and needles." Pain management approaches may include opioids, tricyclic antidepressants, and anti-embolism stockings to equalize pressure. Tricyclic antidepressants have been found to be helpful in controlling the symptoms of neuropathic pain. They also potentiate the actions of opioids and can be used to relieve pain without increasing the dose of the opioid.

Improving Nutritional Status

Nutritional status is assessed by monitoring weight, dietary intake, and serum albumin, BUN, protein, and transferrin levels. The patient is also assessed for factors that interfere with oral intake, such as anorexia, oral and esophageal candidal infection, nausea, pain, weakness, fatigue, and lactose intolerance (Dudek, 2006). Based on the results of assessment, the nurse can implement specific measures to facilitate oral intake. The dietitian is consulted to determine the patient's nutritional requirements.

Control of nausea and vomiting with antiemetic medications administered on a regular basis may increase the patient's dietary intake. Inadequate food intake resulting from pain caused by oral lesions or a sore throat may be managed by administering prescribed opioids and viscous lidocaine (the patient is instructed to rinse the mouth and swallow). Additionally, the patient is encouraged to eat foods that are easy to swallow and to avoid rough, spicy, or sticky food items and foods that are excessively hot or cold. Oral hygiene before and after meals is encouraged. If fatigue

and weakness interfere with intake, the nurse encourages the patient to rest before meals. If the patient is hospitalized, meals should be scheduled so that they do not occur immediately after painful or unpleasant procedures. The patient with diarrhea and abdominal cramping is encouraged to avoid foods that stimulate intestinal motility and abdominal distention, such as fiber-rich foods or lactose, if the patient is intolerant to lactose. The patient is instructed about ways to enhance the nutritional value of meals. Adding eggs, butter, or fortified milk (milk to which powdered skim milk has been added to increase the caloric content) to gravies, soups, or milkshakes can provide additional calories and protein. Supplements such as puddings, powders, milkshakes, and Advera (previously described) may also be useful (Dudek, 2006). Patients who cannot maintain their nutritional status through oral intake may require enteral feedings or parenteral nutrition.

Decreasing the Sense of Isolation

People with AIDS are at risk for double stigmatization. They have what society refers to as a "dreaded disease," and they may have a lifestyle that differs from what is considered acceptable by many people. Many people with AIDS are young adults at a developmental stage that is usually associated with establishing intimate relationships, personal goals, and career goals, as well as having and raising children. Their focus changes as they are faced with a disease that threatens their life expectancy with no cure. In addition, they may be forced to reveal hidden lifestyles or behaviors to family, friends, coworkers, and health care providers. As a result, people with HIV infection may be overwhelmed with emotions such as anxiety, guilt, shame, and fear. They also may be faced with multiple losses, such as loss of financial security; normal roles and functions; self-esteem; privacy; ability to control bodily functions; ability to interact meaningfully with the environment; and sexual functioning, as well as rejection by sexual partners, family, and friends. Some patients may harbor feelings of guilt because of their lifestyle or because they may have infected others in current or previous relationships. Other patients may feel anger toward sexual partners who transmitted the virus to them. Infection control measures used in the hospital or at home may further contribute to the patient's emotional isolation. Any or all of these stressors may cause the patient with AIDS to withdraw both physically and emotionally from social contact.

Nurses are in a key position to provide an atmosphere of acceptance and understanding for people with AIDS and their families and partners. The patient's usual level of social interaction is assessed as early as possible to provide a baseline for monitoring changes in behaviors that suggest social isolation (eg, decreased interaction with staff or family, hostility, nonadherence). Patients are encouraged to express feelings of isolation and loneliness, with the assurance that these feelings are not unique or abnormal.

Providing information about how to protect themselves and others may help patients avoid social isolation. Patients, family, and friends must be reassured that AIDS is not spread through casual contact. Education of ancillary personnel, nurses, and physicians helps reduce factors that might contribute to patients' feelings of isolation. Patient care conferences that address the psychosocial issues associated with AIDS may help sensitize the health care team to patients' needs.

Coping With Grief

The nurse can help the patient verbalize feelings and explore and identify resources for support and mechanisms for coping, especially when the patient is grieving anticipated losses. The patient is encouraged to maintain contact with family, friends, and coworkers and to use local or national AIDS support groups and hotlines. If possible, losses are identified and addressed. The patient is encouraged to continue usual activities whenever possible. Consultations with mental health counselors are useful for many patients.

Monitoring and Managing Potential Complications

OPPORTUNISTIC INFECTIONS. Patients who are immunosuppressed are at risk for opportunistic infections. Therefore, anti-infective agents may be prescribed and laboratory tests obtained to monitor their effect. Signs and symptoms of opportunistic infections, including fever, malaise, difficulty breathing, nausea or vomiting, diarrhea, difficulty swallowing, and any occurrences of swelling or discharge, should be reported as treated as indicated.

RESPIRATORY FAILURE. Impaired breathing is a major complication that increases the patient's discomfort and anxiety and may lead to respiratory and cardiac failure. The respiratory rate and pattern are monitored, and the lungs are auscultated for abnormal breath sounds. The patient is instructed to report shortness of breath and increasing difficulty in carrying out usual activities. Pulse rate and rhythm, blood pressure, and oxygen saturation are monitored. Suctioning and oxygen therapy may be prescribed to ensure an adequate airway and to prevent hypoxia. Mechanical ventilation may be necessary for the patient who cannot maintain adequate ventilation as a result of pulmonary infection, fluid and electrolyte imbalance, or respiratory muscle weakness. Arterial blood gas values are used to guide ventilator settings. If the patient is intubated, methods must be established to allow communication with the nurse and others. Attention must be given to assisting the patient receiving mechanical ventilation to cope with the stress associated with intubation and ventilator assistance. The possible need for mechanical ventilation in the future should be discussed early in the course of the disease, when the patient is able to make known his or her preferences about treatment. The use of mechanical ventilation should be consistent with the patient's decisions about end-of-life treatment. (Further discussion of end-of-life care can be found in Chapter 17.)

CACHEXIA AND WASTING. Wasting syndrome and fluid and electrolyte disturbances, including dehydration, are common complications of HIV infection and AIDS. The patient's nutritional and electrolyte status is evaluated by monitoring weight gains or losses, skin turgor, ferritin levels, hemoglobin and hematocrit values, and electrolyte levels. Fluid and electrolyte status is monitored on an ongoing basis; fluid intake and output and urine specific gravity may be monitored daily if the patient is hospitalized with complications. The skin is assessed for dryness and adequate

turgor. Vital signs are monitored for decreased systolic blood pressure or increased pulse rate on sitting or standing. Signs and symptoms of electrolyte disturbances, such as muscle cramping, weakness, irregular pulse, decreased mental status, nausea, and vomiting, are documented and reported to the physician. Serum electrolyte values are monitored, and abnormalities are reported.

The nurse helps the patient select foods that will replenish electrolytes, such as oranges and bananas (potassium) and cheese and soups (sodium) (Dudek, 2006). A fluid intake of 3 L or more per day, unless contraindicated, is encouraged to replace fluid lost with diarrhea, and measures to control diarrhea are initiated. If fluid and electrolyte imbalances persist, the nurse administers IV fluids and electrolytes as prescribed. Effects of parenteral therapy are monitored.

SIDE EFFECTS OF MEDICATIONS. Adverse reactions are of concern in patients who receive many medications to treat HIV infection or its complications. Many medications can cause severe toxic effects. Information about the purpose of the medications, their correct administration, side effects, and strategies to manage or prevent side effects is provided. Patients and their caregivers need to know which signs and symptoms of side effects should be reported immediately to their primary health care provider (see Table 52-3).

In addition to medications used to treat HIV infection, other medications that may be required include opioids, tricyclic antidepressants, and NSAIDs for pain relief; medications for treatment of opportunistic infections; antihistamines (diphenhydramine [Benadryl]) for relief of pruritus; acetaminophen (Tylenol) or aspirin for management of fever; and antiemetic agents for control of nausea and vomiting. Concurrent use of these medications can cause many drug interactions, resulting in hepatic and hematologic abnormalities. Therefore, careful monitoring of laboratory test results is essential.

During each contact with the patient, it is important for the nurse to ask not only about side effects but also about how well the patient is managing the medication regimen. The nurse may be able to assist the patient in organizing and planning the medication schedule to promote adherence to the treatment regimen.

Promoting Home and Community-Based Care

TEACHING PATIENTS SELF-CARE. Patients, families, and friends are instructed about the routes of transmission of HIV. As discussed earlier, the nurse discusses precautions the patient can use to avoid transmitting HIV sexually (see Charts 52-2 and 52-3) or through sharing of body fluids. Patients and their families or caregivers must receive instructions about how to prevent disease transmission, including handwashing techniques and methods for safely handling and disposing of items soiled with body fluids. Clear guidelines about avoiding and controlling infection, regular health care appointments, symptom management, nutrition, rest, and exercise are necessary. The importance of personal and environmental hygiene is emphasized. Caregivers are taught many of the guidelines (standard precautions) described in Chart 52-4. Kitchen and bathroom surfaces should be cleaned regularly with disinfectants to prevent growth of fungi and bacteria. Patients with pets are encouraged to have another person clean areas soiled by animals, such as bird cages and litter boxes. If this is not possible, patients should use gloves and should wash their hands after they clean the area. Patients are advised to avoid exposure to others who are sick or who have been recently vaccinated. The importance of avoiding smoking, excessive alcohol, and over-the-counter and street drugs is emphasized. Patients who are HIV positive or who inject drugs are instructed not to donate blood. IV/injection drug users who are unwilling to stop using drugs are advised to avoid sharing drug equipment with others.

Caregivers in the home are taught how to administer medications, including IV preparations. The medication regimens used for patients with HIV infection and AIDS are often complex and expensive. Patients receiving combination therapies for treatment of HIV infection and its complications require careful teaching about the importance of taking medications as prescribed and explanations and assistance in fitting the medication regimen into their lives (see Chart 52-7). If the patient requires enteral or parenteral nutrition, instruction is provided to the patient and family about how to administer nutritional therapies at home. Home care nurses provide ongoing teaching and support for the patient and family.

CONTINUING CARE. Many people with AIDS remain in their community and continue their usual daily activities, whereas others can no longer work or maintain their independence. Families or caregivers may need assistance in providing supportive care. There are many community-based organizations that provide a variety of services for people living with HIV infection and AIDS; nurses can help identify these services.

Community health nurses, home care nurses, and hospice nurses are in an excellent position to provide the support and guidance so often needed in the home setting. As hospital costs continue to rise and insurance coverage continues to decline, the complexity of home care increases. Home care nurses are key to the safe and effective administration of parenteral antibiotics, chemotherapy, and nutrition in the home.

During home visits, the nurse assesses the patient's physical and emotional status and home environment. The patient's adherence to the therapeutic regimen is assessed, and strategies are suggested to assist with adherence. The patient is assessed for progression of disease and for adverse side effects of medications. Previous teaching is reinforced, and the importance of keeping follow-up appointments is stressed.

Complex wound care or respiratory care may be required in the home. Patients and families are often unable to meet these skilled care needs without assistance. Nurses may refer patients to community programs that offer a range of services for patients, friends, and families, including help with housekeeping, hygiene, and meals; transportation and shopping; individual and group therapy; support for caregivers; telephone networks for the homebound; and legal and financial assistance. These services are typically provided by both professionals and nonprofessional volunteers. A social worker may be consulted to identify sources of financial support, if needed.

Home care and hospice nurses are increasingly called on to provide physical and emotional support to patients and families as patients with AIDS enter the terminal stages of disease. This support takes on special meaning when people with AIDS lose friends and when family members fear the disease or feel anger concerning the patient's lifestyle. The nurse encourages the patient and family to discuss end-of-life decisions and to ensure that care is consistent with those decisions, all comfort measures are employed, and the patient is treated with dignity at all times.

Evaluation

Expected Patient Outcomes

Expected patient outcomes may include:

1. Maintains skin integrity
2. Resumes usual bowel habits
3. Experiences no infections
4. Maintains adequate level of activity tolerance
5. Maintains usual level of thought processes
6. Maintains effective airway clearance
7. Experiences increased sense of comfort and less pain
8. Maintains adequate nutritional status
9. Experiences decreased sense of social isolation
10. Progresses through grieving process
11. Reports increased understanding of AIDS and participates in self-care activities as possible
12. Remains free of complications

Detailed outcomes are included in the plan of nursing care for a patient with AIDS (see Chart 52-10).

EMOTIONAL AND ETHICAL CONCERNS

Nurses in all settings are called on to provide care for patients with HIV infection. In doing so, they encounter not only the physical challenges of this epidemic but also emotional and ethical concerns. The concerns raised by health care professionals involve issues such as fear of infection, responsibility for giving care, values clarification, confidentiality, developmental stages of patients and caregivers, and poor prognostic outcomes.

Many patients with HIV infection have engaged in "stigmatized" behaviors. Because these behaviors challenge some traditional religious and moral values, nurses may feel reluctant to care for these patients. In addition, health care providers may still have fear and anxiety about disease transmission despite education concerning infection control and the low incidence of transmission to health care providers (see Chart 52-5). Nurses are encouraged to examine their personal beliefs and to use the process of values clarification to approach controversial issues. The American Nurses Association's Code of Ethics for Nurses (2008) can also be used to help resolve ethical dilemmas that might affect the quality of care given to patients with HIV infection and AIDS.

Nurses are responsible for protecting the patient's right to privacy by safeguarding confidential information. Inadvertent disclosure of confidential patient information may result in personal, financial, and emotional hardships for the patient. The controversy surrounding confidentiality concerns the circumstances in which information may be disclosed to others. Health care team members need accurate patient information to conduct assessment, planning, implementation, and evaluation of patient care. Failure to disclose HIV status could compromise the quality of patient care. Sexual partners of HIV-infected patients should know about the potential for infection and the need to engage in safer sex practices, as well as the possible need for testing and health care. Nurses are advised to discuss concerns about confidentiality with nurse administrators and to consult professional nursing organizations such as the Association of Nurses in AIDS Care and legal experts in their state to identify the most appropriate course of action. Chart 52-11 explores issues related to revealing one's HIV status.

AIDS has had a high mortality rate, but advances in antiretroviral and multidrug therapy have demonstrated promise in slowing or controlling disease progression. It is not known whether current treatment regimens will remain effective, because viral drug resistance has developed with

CHART 52-11 *Ethics and Related Issues: Revealing One's HIV Status*

Should All People Who Are Infected With HIV Be Required to Reveal This Status to All Their Sexual and Drug-Sharing Contacts?

Situation

The human immunodeficiency virus (HIV) causes HIV infection, which progresses to AIDS. Because sexual contacts and needle-sharing partners are at risk for developing the disease, would a policy that requires notification of contacts infringe on the liberty and privacy of the known HIV-infected person?

Dilemma

The person's right to privacy conflicts with notifying all people who are contacts either through sexual or needle-sharing behavior (autonomy versus justice). The person's right to privacy conflicts with society's need to contain the deadly virus and stem a deadly epidemic (autonomy versus justice).

Discussion

1. What arguments would you offer in favor of notifying all the person's contacts?
2. What arguments would you offer against notifying all or some of the person's contacts?
3. Each state has various laws that pertain to whether contacts can be notified and who is responsible for notifying contacts. Is there a law for contact notification in the state in which you live? If there is such a law in your state, who is responsible for contact notification?
4. What would you do if the person responsible for contact notification refuses to do so based on his or her own beliefs for confidentiality of HIV infection status?
5. How would you respond if the HIV-positive person said that he or she is afraid to notify his or her contact because of fear of a violent response?

most previous medications. Most nurses in the United States have never faced an epidemic in which so many young and middle-aged adults experience serious illness and may die during the usual course of the disease process. Nurses may struggle with the value and meaning of their professional roles as they witness repeated instances of deterioration. Exposure to so many deaths among patients at the same developmental stage as many nurses can create stress. Contributing to this stress are personal fears of contagion or disapproval of the patient's lifestyle and behaviors. Unlike cancer or other diseases, AIDS is associated with controversies challenging our legal and political systems as well as religious and personal beliefs. Nurses who feel stressed and overburdened may experience physical and mental distress in the form of fatigue, headache, changes in appetite and sleep patterns, helplessness, irritability, apathy, negativity, and anger.

Many strategies have been used by nurses to cope with the stress associated with caring for AIDS patients. Education and provision of up-to-date information help to alleviate apprehension and prepare nurses to deliver safe, high-quality patient care. Interdisciplinary meetings allow participants to support one another and provide comprehensive patient care. Staff support groups give nurses an opportunity to solve problems and explore values and feelings about caring for AIDS patients and their families; they also provide a forum for grieving. Other sources of support include nursing administrators, peers, and spiritual advisors.

CRITICAL THINKING EXERCISES

EBP **1** A 43-year-old woman who has been using IV/injection drugs regularly for 20 years says that she is not going to stop using drugs but wants to reduce her risk of HIV infection. How would you counsel her? What is the evidence base on safer sex and strategies to reduce risk from use of IV/injection drugs? Is there evidence about the effectiveness of needle exchange programs? How would you determine the strength of the evidence and how would you present information to her?

2 During a code response in the intensive care unit (ICU), a nursing student is inadvertently stuck with a needle used on a patient with AIDS who has a high viral load. The student is apprehensive. What should the clinical instructor, in consultation with the nurse manager of the ICU, do? What reporting and documentation are needed (be sure to consider the student's health record)? What testing, treatment, and counseling are indicated for the student? Who should pay for the treatment?

EBP **3** During a home visit to a family in which two adolescents are HIV positive through vertical transmission, you are instructing the adolescents, their siblings, and their adult caregivers about strategies to protect the teens from other infections and to protect other family members from HIV transmission. What is the evidence for strategies that you plan to discuss with the adolescents and their family members? What is the strength of that evidence, and what criteria would you use to evaluate the strength of the evidence?

4 A 48-year-old man who is bleeding from several stab wounds presents to the emergency department. He is intoxicated and combative. An OraQuick Rapid HIV-1 Antibody Test is performed as part of routine health care. What are the implications of this test? How would care in the emergency department be modified because of a positive test result? What are the ethical and legal ramifications of obtaining this test if the patient's ability to consent to testing is questioned?

5 You are making a home visit to a patient with AIDS who is exhibiting early signs of dementia and encephalopathy. Describe the aspects of the home environment that you would assess to ensure safety and adequate care. How would you modify your assessment if the patient lived alone in a third-floor apartment without an elevator? If the patient lived in a rural setting? If the patient had a physical disability that limited his ability to leave his apartment?

The Smeltzer suite offers these additional resources to enhance learning and facilitate understanding of this chapter:

- thePoint online resource, thepoint.lww.com/Smeltzer12E
- Student CD-ROM included with the book
- *Study Guide to Accompany Brunner & Suddarth's Textbook of Medical-Surgical Nursing*
- *Handbook for Brunner & Suddarth's Textbook of Medical-Surgical Nursing*

REFERENCES AND SELECTED READINGS

*Asterisk indicates nursing research.
**Double asterisk indicates classic reference.

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Diffuse Connective Tissue Diseases

Diffuse connective tissue disease refers to a group of systematic disorders that are chronic in nature and are characterized by diffuse inflammation and degeneration in the connective tissues. These disorders share similar clinical features and may affect some of the same organs. The characteristic clinical course is one of exacerbations and remissions. Although the diffuse connective tissue diseases have unknown causes, they are thought to be the result of immunologic abnormalities. They include **rheumatoid arthritis** (RA), SLE, scleroderma, polymyositis, and polymyalgia rheumatica.

RHEUMATOID ARTHRITIS

RA is an autoimmune disease of unknown origin that affects 1% of the population worldwide, with a female-to-male ratio between 2:1 and 4:1 (Khanna, Arnold, Pencharz, et al., 2006).

Pathophysiology

In RA, the autoimmune reaction (Fig. 54-1) primarily occurs in the synovial tissue. Phagocytosis produces enzymes within the joint. The enzymes break down collagen, causing edema, proliferation of the synovial membrane, and ultimately pannus formation. Pannus destroys cartilage and erodes the bone. The consequence is loss of articular surfaces and joint motion. Muscle fibers undergo degenerative changes. Tendon and ligament elasticity and contractile power are lost.

Clinical Manifestations

Clinical manifestations of RA vary, usually reflecting the stage and severity of the disease. Joint pain, swelling, warmth, erythema, and lack of function are classic symptoms. Palpation of the joints reveals spongy or boggy tissue. Often fluid can be aspirated from the inflamed joint. Characteristically, the pattern of joint involvement begins in the small joints of the hands, wrists, and feet (Bickley, 2007). As the disease progresses, the knees, shoulders, hips, elbows, ankles, cervical spine, and temporomandibular joints are affected. The onset of symptoms is usually acute. Symptoms are usually bilateral and symmetric. In addition to joint pain and swelling, another classic sign of RA is joint stiffness in the morning.

In the early stages of disease, even before bony changes occur, limitation in function can occur when there is active inflammation in the joints. Joints that are hot, swollen, and painful are not easily moved. The patient tends to guard or protect these joints by immobilizing them. Immobilization for extended periods can lead to contractures, creating soft tissue deformity.

Deformities of the hands and feet are common in RA (see Chapter 66, Fig. 66-6). The deformity may be caused by misalignment resulting from swelling, progressive joint destruction, or the subluxation (partial dislocation) that occurs when one bone slips over another and eliminates the joint space.

RA is a systemic disease with multiple extra-articular features. Most common are fever, weight loss, fatigue, anemia, lymph node enlargement, and Raynaud's phenomenon (cold- and stress-induced vasospasm causing episodes of digital blanching or cyanosis). Rheumatoid nodules may be noted in patients with more advanced RA, and they develop at some time in the course of the disease in about 25% of patients. These nodules are usually nontender and movable in the subcutaneous tissue. They usually appear over bony prominences such as the elbow, are varied in size, and can disappear spontaneously. Nodules occur only in people who have rheumatoid factor. The nodules often are associated with rapidly progressive and destructive disease. Other extra-articular features include arteritis, neuropathy, scleritis, pericarditis, splenomegaly, and Sjögren's syndrome (dry eyes and dry mucous membranes).

Assessment and Diagnostic Findings

Several factors can contribute to a diagnosis of RA: rheumatoid nodules, joint inflammation detected on palpation, and laboratory findings. The history and physical examination address manifestations such as bilateral and symmetric stiffness, tenderness, swelling, and temperature changes in the joints. The patient is also assessed for extra-articular changes; these often include weight loss, sensory changes, lymph node enlargement, and fatigue. Rheumatoid factor is present in about three fourths of patients with RA, but its presence alone is not diagnostic of RA, and its absence does not rule out the diagnosis. The ESR is significantly elevated in RA. The red blood cell count and C4 complement component are decreased. C-reactive protein and antinuclear antibody (ANA) test results may also be positive (Karpoff & Labus, 2008) (Chart 54-3). Arthrocentesis shows synovial fluid that is cloudy, milky, or dark yellow and contains numerous inflammatory components, such as leukocytes and complement.

X-rays show bony erosions and narrowed joint spaces. X-rays of the hands and feet should be performed at baseline to help establish the diagnosis of RA and then every 3 years to monitor the progression of the disease (Khanna, et al., 2006).

Medical Management

Early Rheumatoid Arthritis

Patients with RA should receive aggressive and early treatment (Khanna, et al., 2006). Treatment includes education, a balance of rest and exercise, and referral to appropriate community agencies (such as the Arthritis Foundation) for support. Medical management begins with therapeutic doses of salicylates or NSAIDs. When used in full therapeutic dosages, these medications provide both anti-inflammatory and analgesic effects.

Several cyclo-oxygenase 2 (COX-2) enzyme blockers, another class of NSAIDs, have been approved for treatment of RA. Cyclo-oxygenase is an enzyme that is involved in the inflammatory process. COX-2 medications block the enzyme involved in inflammation (COX-2) while leaving intact the enzyme involved in protecting the stomach lining (COX-1). As a result, COX-2 enzyme blockers are less likely to cause gastric irritation and ulceration than other

CHART 54-3 NURSING RESEARCH PROFILE

Managing Symptoms in Rheumatoid Arthritis

Sousa, K. H., Ryu, E., Kwok, O., et al. (2007). Development of a model to measure symptom status in persons living with rheumatoid arthritis. *Nursing Research, 56*(6), 434–440.

Purpose

Rheumatoid arthritis (RA) is a chronic, progressive, inflammatory disease of unknown etiology that causes disability as well as morbidity and mortality. RA has a constellation of symptoms (eg, blurred vision, pain, dizziness) that affect quality of life. The purpose of this study was to develop and validate a structured model to measure symptom status in patients with RA.

Design

This study was a secondary analysis of symptom checklists available from 901 women enrolled in the Arthritis, Rheumatism, and Aging Medical Information System. The symptom checklists contained a list of 31 symptoms, and participants were asked to check off all symptoms they had experienced in the previous 6 months. Factor analysis was used to develop the model to measure symptoms.

Findings

Results of the factor analysis supported a two-factor model for the measurement of symptom status. The factors were (1) RA pain symptoms and (2) general symptoms. The two factors were found to be significantly different from each other, and the RA pain symptoms factor had a stronger impact on functional health than general symptoms.

Nursing Implications

The first aspect of effective symptom management for patients with RA is nursing assessment. This study provides a model and validated structure for such an assessment. For example, the symptoms that form the RA pain cluster could serve as a baseline assessment for nurses to identify goals and interventions to help improve the functional health and quality of life of patients with RA.

NSAIDs; however, they are associated with increased risk of cardiovascular disease and must be used with caution (Karch, 2008).

A window of opportunity for symptom control and improved disease management occurs within the first 2 years after disease onset. Therefore, it is recommended that treatment with the DMARDs (antimalarials, gold, penicillamine, or sulfasalazine) begin within 3 months of disease onset. If symptoms are aggressive (ie, early bony erosions as seen on x-rays), methotrexate may be considered. Methotrexate (Rheumatrex) is currently the standard treatment of RA because of its success in preventing both joint destruction and long-term disability (Schmajuk, et al., 2007).

An alternative treatment approach for RA has emerged in the area of biologic therapies. Biologic response modifiers are a group of agents that consist of molecules produced by cells of the immune system or by cells that participate in the inflammatory reactions. Research (Voulgari, Alamanos, Nikas, et al., 2005) using tumor necrosis factor-alpha (TNF-α) inhibitors in combination with other medications has shown that patients demonstrate significant improvement. Examples of biologic response modifiers that are currently available are etanercept (Enbrel), infliximab (Remicade), adalimumab (Humira), golimumab (Simponi), and anakinra (Kineret). Etanercept, infliximab, adalimumab, and golimumab inhibit the function of TNF-α, a key cytokine known to play a role in the disease process in RA (Porth & Matfin, 2009), whereas anakinra inhibits the function of interleukin-1, another cytokine that contributes to the destruction of the joint. Research in this area is ongoing.

Additional analgesia may be prescribed for periods of extreme pain. Opioid analgesic agents are avoided because of the potential for continuing need for pain relief. Nonpharmacologic pain management techniques (eg, relaxation techniques, heat and cold applications) are taught.

Moderate, Erosive Rheumatoid Arthritis

For moderate, erosive RA, a formal program with occupational and physical therapy is prescribed to educate the patient about principles of joint protection, pacing activities, work simplification, range of motion, and muscle-strengthening exercises. The patient is encouraged to participate actively in the management program. The medication program is reevaluated periodically, and appropriate changes are made if indicated. Cyclosporine (Neoral), an immunosuppressant, may be added to enhance the disease-modifying effect of methotrexate.

Persistent, Erosive Rheumatoid Arthritis

For persistent, erosive RA, reconstructive surgery and corticosteroids are often used. Reconstructive surgery is indicated when pain cannot be relieved by conservative measures and the threat of loss of independence is eminent. Surgical procedures include synovectomy (excision of the synovial membrane), tenorrhaphy (suturing of a tendon), arthrodesis (surgical fusion of the joint), and **arthroplasty** (surgical repair and replacement of the joint). Surgery is not performed during disease flares.

Systemic corticosteroids are used when the patient has unremitting inflammation and pain or needs a "bridging" medication while waiting for the slower DMARDs (eg, methotrexate) to begin taking effect. Low-dose corticosteroid therapy is prescribed for the shortest time necessary to minimize side effects (Khanna, et al., 2006). Single large joints that are severely inflamed and fail to respond promptly to the measures outlined previously may be treated by local injection of a corticosteroid.

Advanced, Unremitting Rheumatoid Arthritis

For advanced, unremitting RA, immunosuppressive agents are prescribed because of their ability to affect the production

of antibodies at the cellular level. These include high-dose methotrexate (Rheumatrex), cyclophosphamide (Cytoxan), azathioprine (Imuran), and leflunomide (Arava). However, these medications are highly toxic and can produce bone marrow suppression, anemia, gastrointestinal disturbances, and rashes.

For most patients with RA, depression and sleep deprivation may require the short-term use of low-dose antidepressant medications, such as amitriptyline (Elavil), paroxetine (Paxil), or sertraline (Zoloft), to reestablish an adequate sleep pattern and to manage chronic pain.

The U.S. Food and Drug Administration (FDA) has approved a medical device for use in treating patients with more severe and long-standing RA who have had no response to or are intolerant of DMARDs. The device, a protein A immunoadsorption column (Prosorba), is used in 12 weekly 2-hour apheresis treatments to bind immunoglobulin G (IgG) (ie, circulating immune complex). In this unique population of patients, a significant improvement using the American College of Rheumatology criteria for improvement has been demonstrated (Eustice & Eustice, 2008).

Nutrition Therapy

Patients with RA frequently experience anorexia, weight loss, and anemia. A dietary history identifies usual eating habits and food preferences. Food selection should include the daily requirements from the basic food groups, with emphasis on foods high in vitamins, protein, and iron for tissue building and repair. For the patient who is extremely anorexic, small, frequent feedings with increased protein supplements may be prescribed. Supplemental vitamins and minerals may also be prescribed as needed (Klippel, Stone, Crofford, et al., 2008). Certain medications (ie, oral corticosteroids) used in RA treatment stimulate the appetite and, when combined with decreased activity, may lead to weight gain. Therefore, patients may need to be counseled about eating a healthy, calorie-restricted diet.

Nursing Management

Nursing care of the patient with RA follows the basic plan of care presented earlier (see Chart 54-2). The most common issues for the patient with RA include pain, sleep disturbance, fatigue, altered mood, and limited mobility (Sousa, Ryu, Kwok, et al., 2007). The patient with newly diagnosed RA needs information about the disease to make daily self-management decisions and to cope with having a chronic disease.

Monitoring and Managing Potential Complications

Medications used for treating RA may cause serious and adverse effects. These medication-induced complications may include bone marrow suppression, anemia, gastrointestinal disturbances, and rashes. The primary health care provider bases the prescribed medication regimen on clinical findings and past medical history, and then with the help of the nurse, monitors for side effects using periodic clinical assessments and laboratory testing. The nurse, who can be available for consultation between physician visits, works to help the patient recognize and deal with these side effects (see Table 54-3). The medication may need to be stopped or the dose reduced. If the patient experiences an increase in symptoms while the complication is being resolved or a new medication is being initiated, the nurse's counseling regarding symptom management may relieve potential anxiety and distress.

Promoting Home and Community-Based Care

Teaching Patients Self-Care

Patient teaching is an essential aspect of nursing care of the patient with RA to enable the patient to maintain as much independence as possible, to take medications accurately and safely, and to use adaptive devices correctly. Patient teaching focuses on the disorder itself, the possible changes related to the disorder, the therapeutic regimen prescribed to treat it, the potential side effects of medications, strategies to maintain independence and function, and patient safety in the home (Chart 54-4).

CHART 54-4

HOME CARE CHECKLIST

The Patient With Rheumatoid Arthritis

At the completion of the home care instruction, the patient or caregiver will be able to:	PATIENT	CAREGIVER
• Explain the nature of the disease and principles of disease management.	✔	✔
• Describe the medication regimen (name of medications, dosage, schedule of administration, precautions, potential side effects, and desired effects).	✔	✔
• Identify monitoring procedures and strategies that should be implemented.	✔	✔
• Identify sources of additional information, if necessary.	✔	✔
• Demonstrate accurate and safe self-administration of medications.	✔	✔
• Describe and demonstrate use of pain management techniques.	✔	✔
• Demonstrate use of joint protection techniques in activities of daily living (ADLs).	✔	✔
• Demonstrate ability to perform self-care activities independently or with assistive devices.	✔	
• Demonstrate a safe exercise program.	✔	
• Demonstrate a relaxation technique.	✔	

The patient and family are encouraged to verbalize their concerns and ask questions. Because RA commonly affects young women, major concerns may be related to the effects of the disease on childbearing potential, caring for family, or work responsibilities. The patient with a chronic illness may seek a "cure" or have questions about alternative therapies. One alternative therapy, an expensive one, used by many patients with RA is elk antler velvet. The velvet from elk antlers is harvested, ground, and put into capsules. In one randomized clinical trial of 168 patients with early stages of RA, researchers found that elk velvet has no clinical efficacy in the symptom management of the disease (Allen, Oberle, Grace, et al., 2008).

Pain, fatigue, and depression can interfere with the patient's ability to learn and should be addressed before teaching is initiated. Various educational strategies may then be used, depending on the patient's previous knowledge base, interest level, degree of comfort, social or cultural influences, and readiness to learn. The nurse instructs the patient about basic disease management and necessary adaptations in lifestyle. Because suppression of inflammation and autoimmune responses requires the use of anti-inflammatory, disease-modifying antirheumatic, and immunosuppressive agents, the patient is taught about prescribed medications, including type, dosage, rationale, potential side effects, self-administration, and required monitoring procedures. If hospitalized, the patient is encouraged to practice new self-management skills with support from caregivers and significant others. The nurse then reinforces disease management skills during each patient contact. Barriers to compliance are assessed, and measures are taken to promote adherence to medications and the treatment program.

Continuing Care

Depending on the severity of the disorder and the patient's resources and supports, referral for home care may or may not be warranted. However, the patient who is elderly or frail, has RA that limits function significantly, and lives alone may need referral for home care.

The impact of RA on everyday life is not always evident when the patient is seen in the hospital or in an ambulatory care setting. The increased frequency with which nurses see patients in the home provides opportunities for recognizing problems and implementing interventions aimed at improving the quality of life of patients with RA.

During home visits, the nurse has the opportunity to assess the home environment and its adequacy for patient safety and management of the disorder. Adherence to the treatment program can be more easily monitored in the home setting, where physical and social barriers to adherence are more readily identified. For example, a patient who also has diabetes and requires insulin may be unable to fill the syringe accurately or unable to administer the insulin because of impaired joint mobility. Appropriate adaptive equipment needed for increased independence is often identified more readily when the nurse sees how the patient functions in the home. Any barriers to adherence are identified, and appropriate referrals are made.

For patients at risk for impaired skin integrity, the home care nurse can closely monitor skin status and also instruct, provide, or supervise the patient and family in preventive skin care measures. The nurse also assesses the patient's need for assistance in the home and supervises home health aides who may meet many of the needs of the patient with RA. Referrals to physical and occupational therapists may be made as problems are identified and limitations increase. A home care nurse may visit the home to make sure the patient can function as independently as possible despite mobility problems and can safely manage treatments, including pharmacotherapy. The patient and family should be informed about support services such as Meals on Wheels and local Arthritis Foundation chapters.

Because many of the medications used to suppress inflammation are injectable, the nurse may administer the medication to the patient or teach self-injection. These frequent contacts allow the nurse to reinforce other disease management techniques.

The nurse also assesses the patient's physical and psychological status, adequacy of symptom management, and adherence to the management plan. Patients should know which type of rheumatic disease they have, not just that they have "arthritis" or "arthritis of the knee." The importance of attending follow-up appointments is emphasized to the patient and family, and they should be reminded about the importance of participating in other health promotion activities and health screening. Patients with chronic disorders such as RA often focus on the chronic disease and neglect general health issues.

SYSTEMIC LUPUS ERYTHEMATOSUS

The overall prevalence of SLE is estimated to be 1 per 2500 persons. It occurs 10 times more frequently in women than in men and approximately three times more frequently in African Americans than in Caucasians (Wandstrat, Carr-Johnson, Branch, et al., 2006).

Pathophysiology

SLE is a result of disturbed immune regulation that causes an exaggerated production of autoantibodies. This immunoregulatory disturbance is brought about by some combination of genetic factors, hormonal factors (as evidenced by the usual onset during the childbearing years), and environmental factors (eg, sunlight, thermal burns). Certain medications, such as hydralazine (Apresoline), procainamide (Pronestyl), isoniazid (INH), chlorpromazine (Thorazine), and some antiseizure medications, have been implicated in chemical or drug-induced SLE.

Specifically, B cells and T cells both contribute to the immune response in SLE (Croker & Kimberly, 2005). B cells are instrumental in promoting the onset and flares of the disease (Tieng & Peeva, 2008).

Clinical Manifestations

SLE is an autoimmune systemic disease that can affect any body system. Involvement of the musculoskeletal system, with arthralgias and arthritis (synovitis), is a common

chapter 57

Management of Patients With Burn Injury

LEARNING OBJECTIVES

On completion of this chapter, the learner will be able to:

1. Discuss the incidence of burn injury in the United States today.
2. Describe the factors that affect the severity of burn injury.
3. Describe the local and systemic effects of a major burn injury.
4. Compare and contrast the potential fluid and electrolyte alterations of the emergent/resuscitative and acute phases of burn management.
5. Describe the goals of burn care and the nurse's role in wound assessment, wound cleansing, topical antibacterial therapy, wound dressing, débridement, excision, and skin grafting.
6. Describe the nurse's role in pain management, restoration of function, psychological support of the patient and family, nutritional support, pulmonary care, and patient and family education.
7. Describe patient care and the recovery process for burn patients

GLOSSARY

AlloDerm: processed dermis from human cadaver skin; can be used as dermal layer for skin grafts
autograft: a graft derived from one part of a patient's body and used on another part of that same patient's body
Biobrane: synthetic dressing composed of a nylon, Silastic membrane combined with a collagen derivative
carboxyhemoglobin: a compound of carbon monoxide and hemoglobin, formed in the blood with exposure to carbon monoxide
collagen: a protein present in skin, tendon, bone, cartilage, and connective tissue
contracture: shrinkage of burn scar through collagen maturation
cultured epithelial autograft (CEA): autologous epidermal cells that proliferate in culture and then are regrafted onto the patient
débridement: removal of foreign material and devitalized tissue until surrounding healthy tissue is exposed
donor site: the area from which skin is taken to provide a skin graft for another part of the body
eschar: devitalized tissue resulting from a burn
escharotomy: a linear excision made through eschar to release constriction of underlying tissue
excision: surgical removal of tissue
fasciotomy: an incision made through the fascia to release constriction of underlying muscle
heterograft: graft (ie, pigskin) obtained from an animal of a species other than that of the recipient; also called a xenograft
homograft: a graft transferred from one human (living or cadaveric) to another human; also called allograft
hydrotherapy: cleansing of wounds through use of bath, shower, shower cart table, or immersion
hypertrophic scar: excessive scar formation that rises above the level of the skin
Integra: synthetic dermal substitute
rule of nines: method for calculating body surface area burned by dividing the body into multiples of nine

The nurse who cares for a patient with a burn injury requires a high level of knowledge about the physiologic changes that occur after a burn, as well as astute assessment skills to detect subtle changes in the patient's condition. The patient's health history affects burn care. This makes each burn patient very unique and provides a variety of challenges to the patient's plan of care. In addition, the nurse provides sensitive, compassionate care to patients who are critically ill and initiates rehabilitation early in the course of care. The nurse must also be able to communicate effectively with patients who have burn injuries, family members in crisis, and members of the entire interdisciplinary burn management team. Care of the patient with a burn requires knowledge and skill throughout the care continuum from injury to recovery. This ensures quality care, improved patient outcomes, and optimal quality of life.

Overview of Burn Injury

Incidence

A burn injury can affect people of all age groups, in all socioeconomic groups. An estimated 500,000 people are treated for minor burn injury annually (Pitts, Niska, Xu, et al., 2008). The number of patients who are hospitalized each year with burn injuries is more than 40,000. This includes approximately 25,000 people who require hospitalization in specialized burn centers across the country. As emergency transportation and awareness of burn specialized hospitals has increased, the number of patients referred to these centers has risen. The remaining 5,000 hospitals see an average of three burns per year. Of those people admitted to burn centers, 47% of their injuries occurred at home, 27% on the road, 8% are occupational, 5% are recreational, and the remaining 13% from other sources. Forty percent of these injuries were flame related, 30% scald injuries, 4% electrical, 3% chemical, with the remaining unspecified (Miller, Bessey, Lentz, et al., 2008).

Males have greater then twice the chance of burn injury than women, and the most frequent age group for contact burns is between 20 to 40 years of age (Miller, et al., 2008). The National Fire Protection Association reports 4,000 fire and burn deaths each year. Of these, 3,500 deaths occur from residential fires and the remaining 500 from other sources such as motor vehicle crashes, scalds, or electrical and chemical sources. The overall mortality rate, for all ages and for total body surface area (TBSA) burned is 4.9% (Miller, et al., 2008).

Gerontologic Considerations

Reduced mobility, coordination, strength, and sensation and changes in vision place elderly people at higher risk for burn injury. Difficulties cooking and bathing and other activities of daily living are associated with flame and scald injury in this age group. These changes also place older people at risk for severe burn because they have difficulty in extinguishing the fire and removing themselves from the burn source (Sheridan, 2007a).

Morbidity and mortality rates associated with burns are greater in elderly patients than in younger patients when comparing injuries with similar severity. In 2007 patients over the age of 60 who had a 60% TBSA or greater had an overall mortality rate of 96% (Sheridan, 2007a). Predisposing factors and the health history in the older adult influence the complexity of care for the patient. Pulmonary function is limited in the older adult, therefore, airway exchange, lung elasticity, and ventilation can be affected. This can be further affected by a history of smoking. Decreased cardiac function and coronary artery disease increase the risk of complications in elderly patients with burn injuries. Malnutrition and presence of diabetes mellitus or other endocrine disorders present nutritional challenges and require close monitoring. Varying degrees of orientation may present themselves on admission or through the course of care, making assessment of pain and anxiety a challenge for the burn team. The skin of the elderly is thinner and less elastic, which affects the depth of injury and its ability to heal (Sheridan, 2007a).

An important goal of nurses in community and home settings is to provide education on the prevention of burn injury, especially among the elderly (Chart 57-1). Nurses

CHART 57-1 HEALTH PROMOTION
Burn Prevention

- Advise that matches and lighters be kept out of the reach of children.
- Emphasize the importance of never leaving children unattended around fire or in bathroom/bathtub.
- Advise the installation and maintenance of smoke detectors on every level of the home, changing batteries annually on birthday.
- Recommend the development and practice of a home exit fire drill with all members of the household.
- Advise setting the water heater temperature no higher than 120°F.
- Caution against smoking in bed, while using home oxygen, or against falling asleep while smoking.
- Caution against throwing flammable liquids onto an already burning fire.
- Caution against using flammable liquids to start fires.
- Caution against removing the radiator cap from a hot car engine.
- Recommend avoidance of overhead electrical wires and underground wires when working outside.
- Advise that hot irons and curling irons be kept out of the reach of children.
- Caution against running electric cords under carpets or rugs.
- Recommend storage of flammable liquids well away from a fire source, such as a pilot light.
- Advocate caution when cooking, being aware of loose clothing hanging over the stove top.
- Recommend having a working fire extinguisher in the home and knowing how to use it.

need to assess an elderly patient's ability to safely perform activities of daily living, assist elderly patients and families to modify their environment to ensure safety, and make referrals as needed.

Outlook for Survival and Recovery

The National Center for Injury Prevention and Control of the Centers for Disease Control and Prevention (CDC) identifies fire or burn injury as the fifth most common cause of death from unintentional injury in the United States and the third leading cause of death in the home from injury (CDC, 2008).

Great strides in research have helped to increase the survival rate of patients with burn injuries. Mortality has fallen to levels never thought possible. Long-term outcomes can now be explored because patients with very large burns are surviving their injuries. Research in areas such as fluid resuscitation, emergency burn treatment, inhalation injury and management, nutritional needs and changes in wound care practice with early excision, skin grafting, and use of skin substitutes have contributed greatly to the decrease in burn deaths. Continued research and advances in the areas of critical care, rehabilitation, psychosocial, and scar management are essential for continued progress in burn care.

Severity

The severity of each burn injury is determined by multiple factors that when assessed help the burn team estimate the likelihood that a patient will survive and plan for the care for each patient. These factors include age of the patient; depth of the burn; amount of surface area of the body that is burned; the presence of inhalation injury; presence of other injuries; location of the injury in special care areas such as the face, the perineum, hands, or feet; and the presence of a past medical history.

Age

Young children and the elderly continue to have increased morbidity and mortality when compared to other age groups with similar injuries and present a challenge for burn care. This is an important factor when determining the severity of injury and possible outcome for the patient.

Burn Depth

Burns are classified according to the depth of tissue destruction as superficial partial-thickness injuries, deep partial-thickness injuries, or full-thickness injuries (Table 57-1). These three categories are similar to, but not the same as, first-, second-, and third-degree burn classifications. Although the term fourth-degree burn is not used universally, it occurs with prolonged flame contact or high-voltage injury that destroys all layers of the skin and damages tendons and muscles.

In a superficial partial-thickness burn, the epidermis is destroyed or injured and a portion of the dermis may be injured. A deep partial-thickness burn involves destruction of the epidermis and upper layers of the dermis and injury to deeper portions of the dermis. Capillary refill follows tissue blanching. Hair follicles remain intact. A full-thickness burn involves total destruction of epidermis and dermis and, in some cases, destruction of underlying tissue, muscle, and bone. Wound color ranges widely from pale white to red, brown, or charred black. The burned area is painless and lacks sensation because nerve fibers are destroyed. The wound appears leathery; hair follicles and sweat glands are destroyed (Fig. 57-1). The severity of this burn is often deceiving to patients because they have no pain in the injury area. These wounds require skin grafting for healing.

Burn depth determines whether epithelialization will occur. Determining burn depth can be difficult even for the experienced burn care provider. The following factors are

Table 57-1 CHARACTERISTICS OF BURNS ACCORDING TO DEPTH

Depth of Burn and Causes	Skin Involvement	Symptoms	Wound Appearance	Recuperative Course
Superficial Partial-Thickness (Similar to First Degree)				
Sunburn Low-intensity flash	Epidermis; possibly a portion of dermis	Tingling Hyperesthesia (supersensitivity) Pain that is soothed by cooling	Reddened; blanches with pressure; dry Minimal or no edema Possible blisters	Complete recovery within a week; no scarring Peeling
Deep Partial-Thickness (Similar to Second Degree)				
Scalds Flash flame Contact	Epidermis, upper dermis, portion of deeper dermis	Pain Hyperesthesia Sensitive to cold air	Blistered, mottled red base; broken epidermis; weeping surface Edema	Recovery in 2 to 4 weeks Some scarring and depigmentation contractures Infection may convert it to full thickness
Full-Thickness (Similar to Third Degree)				
Flame Prolonged exposure to hot liquids Electric current Chemical Contact	Epidermis, entire dermis, and sometimes subcutaneous tissue; may involve connective tissue, muscle, and bone	Pain free Shock Hematuria (blood in the urine) and possibly hemolysis (blood cell destruction) Possible entrance and exit wounds (electrical burn)	Dry; pale white, leathery, or charred Broken skin with fat exposed Edema	Eschar sloughs Grafting necessary Scarring and loss of contour and function; contractures Loss of digits or extremity possible

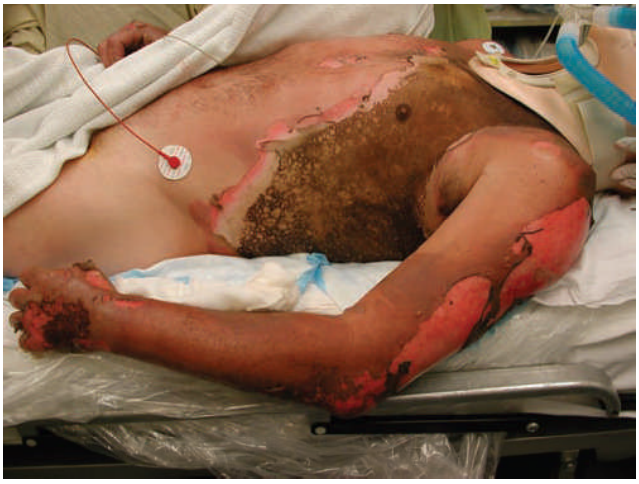

Figure 57-1 Full-thickness injury to chest and upper extremity. Epidermis, varying levels of the dermis and subcutaneous tissue is injured. Used with permission. Lehigh Valley Health Network, Allentown, PA.

considered in determining the depth of a burn: how the injury occurred, causative agent (such as flame or scalding liquid), temperature of the burning agent, duration of contact with the agent, and thickness of the skin.

Extent of Body Surface Area Injured

Various methods are used to estimate the TBSA affected by burns; among them are the rule of nines, the Lund and Browder method, and the palmer method. These methods assist the burn team in making decisions about treatment and transfer of the patient to a burn center.

Rule of Nines

A common method, the **rule of nines** (Fig. 57-2), is a quick way to estimate the extent of burns in adults. The system divides the body into multiples of nine. The sum total of these parts equals the total body surface area and is an important measurement in the severity of injury (Shukla & Sheridan, 2008).

Lund and Browder Method

A more precise method of estimating the extent of a burn is the Lund and Browder method, which recognizes the percentage of surface area of various anatomic parts, especially the head and legs, as it relates to the age of the patient. By dividing the body into very small areas and providing an estimate of the proportion of TBSA accounted for by each body part, one can obtain a reliable estimate of TBSA burned. The initial evaluation is made on arrival of the patient at the hospital and is revised within the first 72 hours because demarcation of the wound and its depth presents itself more clearly by this time.

Palmer Method

In patients with scattered burns, or for a quick prehospital assessment, the palmer method may be used to estimate the extent of the burns. The size of the patient's palm, not including the surface area of the digits, is approximately 1% of the TBSA. The patient's palm without the fingers is equivalent to 0.5% TBSA and serves as a general measurement for all age groups (Shukla & Sheridan, 2008).

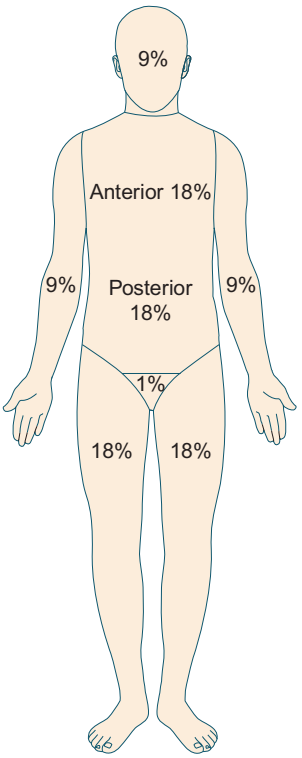

Figure 57-2 The rule of nines: Estimated percentage of total body surface area (TBSA) in the adult is arrived at by sectioning the body surface into areas with a numerical value related to nine. (Note: The anterior and posterior head total 9% of TBSA.)

Pathophysiology

Burn injury is a result of heat transfer from one site to another. Tissue destruction results from coagulation, protein denaturation, or ionization of cellular contents (Fig. 57-3). The skin and the mucosa of the upper airways are sites of tissue destruction. Deep tissues, including the viscera, can be damaged by electrical burns (Chart 57-2) or by prolonged contact with a heat source. Disruption of the skin can lead to increased fluid loss, infection, hypothermia, scarring, compromised immunity, and changes in function, appearance, and body image.

The depth of the injury depends on the temperature of the burning agent and the duration of contact with the agent. For example, in the case of scald burns in adults, 1 second of contact with hot tap water at 68.9°C (156°F) may result in a burn that destroys both the epidermis and the dermis, causing a full-thickness (third-degree) injury. Fifteen seconds of exposure to hot water at 56.1°C (133°F) results in a similar full-thickness injury. Temperatures less than 44°C (111°F) can be tolerated for long periods without injury.

Burns that do not exceed 20% TBSA produce a primarily local response. Burns that exceed 20% TBSA may produce both a local and a systemic response and are considered major burn injuries. The systemic response is caused by

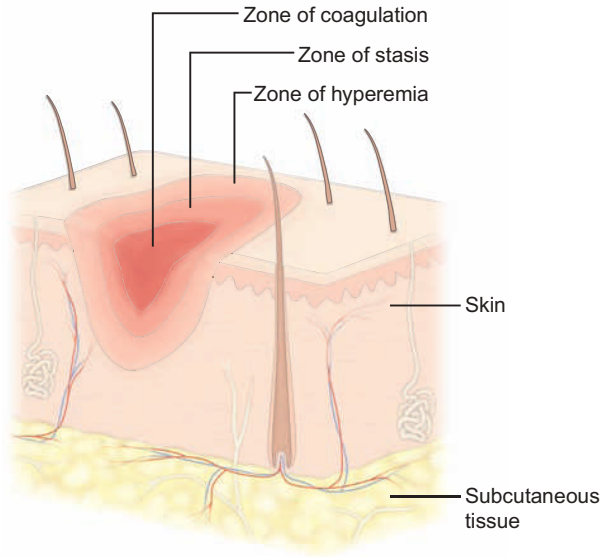

Figure 57-3 Zones of burn injury. Each burned area has three zones of injury. The inner zone (known as the area of coagulation, where cellular death occurs) sustains the most damage. The middle area, or zone of stasis, has a compromised blood supply, inflammation, and tissue injury. The outer zone—the zone of hyperemia—sustains the least damage.

the release of cytokines and other mediators into the systemic circulation. The release of local mediators and changes in blood flow, tissue edema, and infection can cause progression of the burn injury.

Pathophysiologic changes resulting from major burns during the initial burn-shock period include tissue hypoperfusion and organ hypofunction secondary to decreased cardiac output, followed by hyperdynamic and hypermetabolic phases. The incidence, magnitude, and duration of pathophysiologic changes in burns are proportional to the extent of burn injury, with a maximal response seen in burns covering 60% or more TBSA.

The initial systemic event after a major burn injury is hemodynamic instability, which results from loss of capillary integrity and a subsequent shift of fluid, sodium, and protein from the intravascular space into the interstitial spaces. Hemodynamic instability involves cardiovascular, fluid and electrolyte, blood volume, pulmonary, and other mechanisms.

Cardiovascular Alterations

Hypovolemia is the immediate consequence of fluid loss and results in decreased perfusion and oxygen delivery. Cardiac output decreases before any significant change in blood volume is evident. As fluid loss continues and vascular

Chart 57-2 • *Electrical Burns*

Electrical injury accounts for a small percentage of burn unit admissions each year, yet it is one of the most destructive types of burn injuries that can be sustained. The devastating effects of an electrical injury can cause lifelong neurovascular problems. Low-voltage injury (ie, less than 500 V exposure) generally does not cause significant damage or medical problems. Midrange exposure (ie, 200–1000 V) can cause local destruction to the tissue. High-voltage exposure (ie, greater than 1000 V) can cause loss of consciousness, fractures, compartment syndrome, arrhythmias, and is often associated with falls (Shukla & Sheridan, 2008).

Tissue and bone destruction often results in amputations and possible loss of life as the result of cardiac and respiratory abnormalities. A true electrical injury results when a current of electricity travels through the body and exits to the ground, causing internal damage to tissue and organs. Such an injury results in an entrance wound, which is the patient's point of contact with the source. The exit wound has a blow-out appearance, causing extensive damage to the surrounding tissue and structures. The amount of damage depends on the strength of the current and the length of the duration of contact with the source. An arc injury is the result of the electricity's traveling on the outside of the body or arcing around it. There is usually also a thermal injury due to clothing catching on fire. The surface injury from an electrical source is usually small compared with the damage under the surface of the skin. Electricity travels through areas of least resistance, nerves and blood vessels, to the most resistant, bone. The most severe damage occurs beneath the skin surface and is difficult to determine without surgical intervention (Shukla & Sheridan, 2008).

Once the patient is out of the path of the electricity, emergency care can safely be provided. The ABCs of emergency care are always followed. An electrical current immediately contracts muscles as it travels through the body, and cardiac dysrhythmias and spinal injuries often result from the muscular contraction. Cardiac dysrhythmias can occur in both low-voltage and high-voltage injury and therefore require electrocardiogram evaluation and monitoring. Those patients with loss of consciousness, dysrhythmias, or ST changes on ECG must be admitted and cardiac monitoring should occur (Arnoldo, Klein & Gibran, 2006). Until it is known that the patient has no fractures, it is imperative that a neck collar remain in place and that the patient is log-rolled to eliminate the chance of further spinal cord injury. With high-voltage electrical injuries, cervical spine immobilization is a priority until cervical spine injury is ruled out.

Prompt administration of intravenous (IV) fluids and monitoring of urine output are critical components of care. Patients with electrical burns are prone to acute renal failure because of the release of myoglobin resulting from the destruction of muscle and tissue. Myoglobin can constrict renal arteries and block urine flow through the kidneys. Patients can have gross hematuria on admission to the hospital. Administration of large amounts of IV fluids helps maintain the flow of urine. It is difficult to assess the amount of fluid a patient will require because the electrical injury creates such extensive internal damage. The nurse should expect 75 to 100 mL/h of urine output for a patient who is receiving fluid resuscitation. Creatine kinase (CK) is released by damaged muscle cells and it is measured during the early phases of care to assist in determining the degree of muscle injury.

In patients with electrical injuries, neurovascular checks of affected extremities are very important. Assessment of color, temperature, and sensation in the extremity as well as the monitoring of palpable or Doppler pulses should be done to assess adequate blood flow to the extremity. If indicated by clinical assessment, the measurement of compartment syndrome to determine deep tissue injury can be performed. Compartment pressures greater than 30 mm Hg can indicate poor tissue perfusion and the need for surgical decompression (Arnoldo, et al., 2006).

volume decreases, cardiac output continues to decrease and the blood pressure drops. This is the onset of burn shock. In response, the sympathetic nervous system releases catecholamines, resulting in an increase in peripheral resistance (vasoconstriction) and an increase in pulse rate. Peripheral vasoconstriction further decreases cardiac output.

Prompt fluid resuscitation maintains the blood pressure in the low to normal range and improves cardiac output. Despite adequate fluid resuscitation, cardiac filling pressures (central venous pressure, pulmonary artery pressure, and pulmonary artery wedge pressure) remain low during the burn-shock period. If inadequate fluid resuscitation occurs, distributive shock occurs (see Chapter 15).

Generally the greatest volume of fluid leak occurs in the first 24 to 36 hours after the burn, peaking by 6 to 8 hours. As the capillaries begin to regain their integrity, burn shock resolves and fluid returns to the vascular compartment. As fluid is reabsorbed from the interstitial tissue into the vascular compartment, blood volume increases. If renal and cardiac function is adequate, urinary output increases. Diuresis continues for several days to 2 weeks.

At the time of burn injury, some red blood cells may be destroyed and others damaged, resulting in anemia. Despite this, the hematocrit may be elevated due to plasma loss. Blood losses sustained during surgical procedures, wound care, and diagnostic studies and ongoing hemolysis further contribute to anemia. Blood transfusions are required periodically to maintain adequate hemoglobin levels for oxygen delivery. Abnormalities in coagulation, including a decrease in platelets (thrombocytopenia) and prolonged clotting and prothrombin times, also occur with burn injury.

Fluid and Electrolyte Alterations

Edema forms rapidly after a burn injury. A superficial burn will cause edema to form within 4 hours after injury, while a deeper burn will continue to form over a longer period of time up to 18 hours postinjury. This is caused by increased perfusion to the injured area and is reflective of the amount of vascular and lymphatic damage to the tissue. There is loss of capillary integrity, and fluid is localized to the burn itself, resulting in blister formation and edema only in the area of injury. Patients with more severe burns develop massive systemic edema (Greenhalgh, 2007). Reabsorption begins at about 4 hours and is complete by 4 days postburn injury. However, the reabsorption is dependent on the depth of injury to the tissue. Partial-thickness injury resolves more quickly due to a more functioning lymphatic system and increased perfusion when compared to the full-thickness injury (Greenhalgh, 2007). Edema in burn wounds can be reduced by avoiding excessive fluid administration during the early postburn period. Excessive fluid administration increases edema formation in both burned and nonburned tissue.

As the taut, burned tissue becomes unyielding to the edema underneath its surface, it begins to act like a tourniquet, especially if the burn is circumferential. As edema increases, pressure on small blood vessels and nerves in the distal extremities causes an obstruction of blood flow and consequent ischemia. This complication is similar to a compartment syndrome. The physician may need to perform an **escharotomy,** a surgical incision into the **eschar** (devitalized tissue resulting from a burn) to relieve the constricting effect of the burned tissue (Demling, 2005a).

Circulating blood volume decreases dramatically during burn shock. In addition, evaporative fluid loss through the burn wound may reach 3 to 5 L or more over a 24-hour period until the burn surfaces are covered.

During burn shock, serum sodium levels vary in response to fluid resuscitation. Usually, hyponatremia (sodium depletion) is present. Hyponatremia is also common during the first week of the acute phase, as water shifts from the interstitial space to the vascular space.

Immediately after burn injury, hyperkalemia (excessive potassium) results from massive cell destruction. Hypokalemia (potassium depletion) may occur later with fluid shifts and inadequate potassium replacement.

Pulmonary Alterations

Approximately 10% to 20% of patients admitted to burn centers have an inhalation injury. The presence of this injury increases the hospital length of stay, is a determinant of the severity of injury, and increases mortality and morbidity (Palmieri, 2007). An inhalation injury occurs when a person is trapped inside a burning structure or involved in an explosion that leads to the inhalation of superheated air and noxious gas (McCall & Cahill, 2005).

Deterioration in severely burned patients can occur without obvious evidence of a smoke inhalation injury. Bronchoconstriction (caused by release of histamine, serotonin, and thromboxane, a powerful vasoconstrictor) and chest constriction secondary to circumferential full-thickness chest burns cause this deterioration. Even without pulmonary injury, hypoxia (oxygen starvation) may be present. Early in the postburn period, catecholamine release in response to the stress of the burn injury alters peripheral blood flow, thereby reducing oxygen delivery to the periphery. Later, hypermetabolism and continued catecholamine release lead to increased tissue oxygen consumption, which can lead to hypoxia. To ensure that adequate oxygen is available to the tissues, supplemental oxygen may be needed.

Pulmonary injuries are categorized as upper airway injury or inhalation injury below the glottis. Upper airway injury results from inhalation of direct heat greater then 150°C (302°F) to the epithelium. This damage results in severe upper airway edema, which can cause obstruction of the upper airway, including the pharynx and larynx, in the early hours postburn (Palmieri, 2007). Because of the cooling effect of rapid vaporization in the pulmonary tract, direct heat injury does not normally occur below the level of the bronchus. Upper airway injury is treated by early nasotracheal or endotracheal intubation.

Inhalation injury below the glottis results from inhaling the products of incomplete combustion or noxious gases. Inhalation of noxious gases is often the source of death at the scene of a fire. These products include carbon monoxide, cyanide, ammonia, aldehydes, acrolein, sulfur dioxide, and isocyanates (Barillo, 2009; Kealey, 2009; Palmieri, 2007). Tissue hypoxia is the result of carbon monoxide inhalation. It combines with hemoglobin to form **carboxyhemoglobin.** The affinity of hemoglobin for carbon monoxide is 250 times greater than that for oxygen. This injury results

directly from chemical irritation of the pulmonary tissues at the alveolar level. Inhalation injuries below the glottis cause loss of ciliary action, hypersecretion, severe mucosal edema, and possibly bronchospasm. The pulmonary surfactant is reduced, resulting in atelectasis (collapse of alveoli). Expectoration of carbon particles in the sputum is the cardinal sign of this injury.

Treatment usually consists of early intubation and mechanical ventilation with 100% oxygen, which reduces the half-life of carboxyhemoglobin from 4 hours to 45 minutes (Kealey, 2009; Pham & Gibran, 2007). However, some patients require only oxygen therapy, depending on the extent of pulmonary injury and edema.

Restrictive pulmonary excursion may occur with full-thickness burns encircling the neck and thorax. Chest excursion may be greatly restricted, resulting in decreased tidal volume. In such situations, escharotomy is necessary.

Pulmonary abnormalities are not always immediately apparent. More than half of all patients with burn injuries with pulmonary involvement do not initially demonstrate pulmonary signs and symptoms. Any patient with possible inhalation injury must be observed for at least 24 hours for respiratory complications. Airway obstruction may occur very rapidly or develop in hours, even while fluid resuscitation is under way. Decreased lung compliance, decreased arterial oxygen levels, and respiratory acidosis may occur gradually over the first 5 days after a burn.

Indicators of possible upper airway injury include (1) injury occurring in an enclosed space; (2) burns of the face or neck; (3) singed nasal hair; (4) hoarseness, high-pitched voice change, dry cough, stridor; (5) sooty or bloody sputum; (6) labored breathing or tachypnea (rapid breathing) and other signs of reduced oxygen levels (hypoxemia); and (7) erythema and blistering of the oral or pharyngeal mucosa.

Diagnosis of lower airway inhalation injury includes monitoring of arterial blood gases and carboxyhemoglobin levels and direct observation of the airway by fiberoptic bronchoscopy to confirm the clinical diagnosis. Findings on bronchoscopy include airway edema, inflammation, necrosis, and soot in the airway (Edelman, White, Tyburski, et al., 2006). Less frequently, xenon scan and computed tomography scans can be used to aid diagnosis but are of questionable value (Pham & Gibran, 2007).

Pulmonary complications secondary to inhalation injuries include sloughing of the airway, increased secretions and inflammation, atelectasis, airway obstruction and ulceration, pulmonary edema, and tissue hypoxia. As a result, respiratory failure and acute respiratory distress syndrome (ARDS) and pneumonia can develop (Edelman, et al., 2006). Respiratory failure and ARDS are discussed in Chapter 23.

Renal Alterations

Renal function may be altered as a result of decreased blood volume. Destruction of red blood cells at the injury site results in free hemoglobin in the urine. If muscle damage occurs (eg, from electrical burns), myoglobin is released from the muscle cells and excreted by the kidneys. Adequate fluid volume replacement restores renal blood flow, increasing the glomerular filtration rate and urine volume. If there is inadequate blood flow through the kidneys, the hemoglobin and myoglobin occlude the renal tubules, resulting in acute tubular necrosis and renal failure (see Chapter 44).

Immunologic Alterations

The immunologic defenses of the body are greatly altered by a burn injury. Patients with burn injury are at high risk for infection and sepsis. The skin is the largest barrier to infection, and when it is compromised, the patient is continually exposed to the environment. The loss of skin integrity is compounded by the release of abnormal inflammatory factors, altered levels of immunoglobulins and serum complement, impaired neutrophil function, and a reduction in lymphocytes (lymphocytopenia). These alterations result in immunosuppression and increase the risk for sepsis. As a result, the major cause of death in the burn patient who survives after 24 hours is multiple organ dysfunction syndrome (MODS) (Greenhalgh, 2007). Most burn centers are specifically designed to provide an infection-controlled environment to protect the patient and minimize exposure to potentially harmful organisms.

Thermoregulatory Alterations

Loss of skin also results in an inability to regulate body temperature. Patients with burn injuries may therefore exhibit low body temperatures in the early hours after injury. Then, as hypermetabolism resets core temperature, the patient becomes hypothermic for much of the postburn period, even in the absence of infection. Most burn centers have heat panels at the bedside and additional heating sources to help maintain the patient's body temperature.

Gastrointestinal Alterations

Two potential gastrointestinal (GI) complications may occur: paralytic ileus (absence of intestinal peristalsis) and Curling's ulcer. Decreased peristalsis and bowel sounds are manifestations of paralytic ileus resulting from burn trauma. Gastric distention and nausea may lead to vomiting unless gastric decompression is initiated. Gastric bleeding secondary to massive physiologic stress may be signaled by occult blood in the stool, regurgitation of "coffee ground" material from the stomach, or bloody vomitus. These signs suggest gastric or duodenal erosion (Curling's ulcer).

Other alterations affect the GI tract after burn injury: the mucosal barrier becomes permeable, the permeability allows for overgrowth of GI bacteria, and the bacteria translocate to other organs, causing infection. Patients are unable to defend against their own bacteria due to immunosuppression. In addition, alcohol ingestion, which is common in the burn population, affects GI integrity and immune response, further increasing the risk of infection and possible bleeding complications (Gosain & Gamelli, 2005a).

Patients with large TBSA burns are also at risk for abdominal compartment syndrome (ACS). During resuscitation, fluid shifts into the abdominal cavity, causing increased abdominal distention, decreased urine output, hypotension, and respiratory insufficiency. The development of ACS is related to the volume of fluids administered. Factors such as the presence of inhalation injury, deep thermal injury, glucosuria, delayed or inadequate

resuscitation, and hemoglobinuria may necessitate additional fluids that may not be calculated by formulas. Bladder pressure is measured to determine the need for invasive intervention to treat increasing abdominal pressure. Bladder pressures greater than 25 mm Hg over time indicate increasing abdominal pressure. Although the prevention of this complication is not always possible, cautious and continuous measurement of fluids administered and urine output is essential (Hershberger, Hunt, Arnoldo, et al., 2007).

Management of Burn Injury

Burn care is typically categorized into three phases of care: emergent/resuscitative phase, acute/intermediate phase, and rehabilitation phase. Although priorities exist for each of the phases, the phases overlap, assessment, and management of specific problems and complications are not limited to these phases but take place throughout burn care. The three phases and the priorities for care are summarized in Table 57-2.

EMERGENT/RESUSCITATIVE PHASE

On-the-Scene Care

Preventing injury to the rescuer is the first priority of on-the-scene care. If needed, fire and emergency medical services should be requested at the first opportunity. Usually, rescue workers cover the wound, establish an airway, supply oxygen, and insert at least one large-bore intravenous (IV) line. Chart 57-3 describes the procedures and care required at the burn scene.

The burned person's appearance can be frightening at first. Although the local effects of a burn are the most evident, the systemic effects pose greater threats to life. A primary survey of the patient is carried out to assess the *a*irway (A), gas exchange or *b*reathing (B), and *c*irculatory status (C) as well as the need for cervical spine immobilization and cardiac monitoring for patients with high-voltage electrical injuries. The circulatory system must be assessed quickly. Apical pulse and blood pressure are monitored frequently. Tachycardia (abnormally rapid heart rate) and slight hypotension are expected soon after the burn. In the patient with extensive burns, neurologic status is assessed quickly. Often the patient is awake and alert initially, and vital information can be obtained at that time.

NURSING ALERT

Breathing must be assessed and a patent airway established immediately during the initial minutes of emergency care. Immediate therapy is directed toward establishing an airway and administering humidified 100% oxygen. If such a high concentration of oxygen is not available under emergency conditions, oxygen by mask or nasal cannula is given initially. If qualified personnel and equipment are available and the victim has severe respiratory distress or airway edema, the rescuers can insert an endotracheal tube and initiate manual ventilation.

NURSING ALERT

No food or fluid is given by mouth, and the patient is placed in a position that will prevent aspiration of vomitus, because nausea and vomiting typically occur due to paralytic ileus resulting from the stress of injury.

The secondary survey focuses on the completion of the total body system assessment, including the mechanism of injury, inhalation injury, and presence of corneal injury. A secondary head-to-toe survey of the patient is carried out to identify other potentially life-threatening injuries (Shukla & Sheridan, 2008).

Medical Management

The patient is transported to the nearest emergency department (ED). The hospital and physician are alerted that the patient is en route so that life-saving measures can be initiated immediately by a trained team and plans for referral to a burn center can be made.

Initial priorities in the ED remain airway, breathing, and circulation. For mild pulmonary injury, 100% humidified

Table 57-2 PHASES OF BURN CARE

Phase	Duration	Priorities
Emergent/resuscitative	From onset of injury to completion of fluid resuscitation	• First aid • Prevention of shock • Prevention of respiratory distress • Detection and treatment of concomitant injuries • Wound assessment and initial care
Acute/intermediate	From beginning of diuresis to near completion of wound closure	• Wound care and closure • Prevention or treatment of complications, including infection • Nutritional support
Rehabilitation	From major wound closure to return to individual's optimal level of physical and psychosocial adjustment	• Prevention of scars and contractures • Physical, occupational, and vocational rehabilitation • Functional and cosmetic reconstruction • Psychosocial counseling

Chart 57-3 • *Emergency Procedures at the Burn Scene*

- **Extinguish the flames.** When clothes catch fire, the flames can be extinguished if the person falls to the floor or ground and rolls ("stop, drop, and roll"); anything available to smother the flames, such as a blanket, rug, or coat, may be used. Standing still forces the person to breathe flames and smoke, and running fans the flames. If the burn source is electrical, the electrical source must be disconnected safely.
- **Cool the burn.** After the flames are extinguished, the burned area and adherent clothing are soaked with *cool* water, briefly, to cool the wound and halt the burning process. Once a burn has been sustained, the application of cool water is the best first-aid measure. However, *never* apply ice directly to the burn, *never* wrap the person in ice, and *never* use cold soaks or dressings for longer than several minutes; such procedures may worsen the tissue damage and lead to hypothermia in people with large burns.
- **Remove restrictive objects.** If possible, remove clothing immediately. Adherent clothing may be left in place once cooled. Other clothing and all jewelry, including all piercings, should be removed to allow for assessment and to prevent constriction secondary to rapidly developing edema.
- **Cover the wound.** The burn should be covered as quickly as possible to minimize bacterial contamination, to maintain body temperature, and decrease pain by preventing air from coming in contact with the injured surface. Sterile dressings are best, but any clean, dry cloth can be used as an emergency dressing. Ointments and salves should *not* be used. Other than the dressing, no medication or material should be applied to the burn wound.
- **Irrigate chemical burns.** Chemical burns resulting from contact with a corrosive material are irrigated immediately. Most chemical laboratories have a high-pressure shower for such emergencies. If such an injury occurs at home, brush off the chemical agent, remove clothes immediately, and rinse all areas of the body that have come in contact with the chemical. Rinsing can occur in the shower or any other source of continuous running water. If a chemical gets in or near the eyes, the eyes should be flushed with cool, clean water immediately. Outcomes for the patient with chemical burns are significantly improved by rapid, sustained flushing of the injury at the scene.

oxygen is administered and the patient is encouraged to cough so that secretions can be removed by suctioning. For more severe situations, it is necessary to remove secretions by bronchial suctioning and to administer bronchodilators and mucolytic agents. If edema of the airway develops, endotracheal intubation may be necessary. Continuous positive airway pressure and mechanical ventilation may also be required to achieve adequate oxygenation.

After adequate respiratory function and circulatory status have been established, the patient is assessed for cervical spinal injuries or head injury if he or she was involved in an explosion, a fall, a jump, or an electrical injury. Once the patient's condition is stable, attention is directed to the burn wound itself. All clothing and jewelry are removed. For chemical burns, flushing of the exposed areas is continued. The patient is checked for contact lenses. These are removed immediately if chemicals have contacted the eyes or if facial burns have occurred.

It is important to validate an account of the burn scenario provided by the patient, witnesses at the scene, and paramedics. Information needs to include the time of the burn injury, the source of the burn, the place where the burn occurred, how long the patient was in the burning structure, how the burn was treated at the scene, and any history of falling or jumping at the scene. A history of preexisting diseases, allergies, medications, and the use of drugs, alcohol, and tobacco is obtained at this point to aid in planning the patient's care. A large-bore (16- or 18-gauge) IV catheter should be inserted in a nonburned area (if not inserted earlier). Most patients will have a central venous catheter inserted so that large amounts of IV fluids can be administered quickly and central venous pressures can be monitored.

If the burn exceeds 20% to 25% TBSA, a nasogastric tube is inserted and connected to low intermittent suction. Often, patients with large burns become nauseated as a result of the GI effects of the burn injury, such as paralytic ileus, and the effects of medication, such as opioids. All patients who are intubated should have a nasogastric tube inserted to decompress the stomach and prevent vomiting.

The physician evaluates the patient's general condition, assesses the burn, determines the priorities of care, and directs the individualized plan of treatment, which is divided into systemic management and local care of the burned area. Nonsterile gloves, caps, masks and cover gowns are worn by personnel while assessing the exposed burned areas. Clean technique is maintained while assessing and treating the burn wounds.

Assessment of both the TBSA burned and the depth of the burn are completed after soot and debris have been gently cleansed from the burn wound. Careful attention is paid to keeping the patient warm during wound assessment and cleansing. Assessment is repeated frequently throughout burn wound care. Photographs may be taken of the burn areas initially and periodically throughout treatment; in this way, the initial injury and burn wound can be documented. Such documentation is invaluable for insurance and legal claims.

Clean sheets are placed under and over the patient to protect the burn wound from contamination, maintain body temperature, and reduce pain caused by air currents passing over exposed nerve endings. An indwelling urinary catheter is inserted to permit more accurate monitoring of urine output and renal function for patients with moderate to severe burns. Baseline height, weight, arterial blood gases, hematocrit, electrolyte values, blood alcohol level, drug panel, urinalysis, and chest x-rays are obtained. If the patient is elderly or has an electrical burn, a baseline electrocardiogram (ECG) is obtained. Because burns are contaminated wounds, tetanus prophylaxis is administered if

the patient's immunization status is not current or is unknown.

NURSING ALERT

If necessary, a blood pressure cuff can be placed around a patient's burned extremity. The cuff must be of the correct size with accommodations made for bulky dressings.

Although the major focus of care during the emergent phase is physical stabilization, the nurse must also attend to the patient's and family's psychological needs. Burn injury is a crisis, one that causes varying emotional responses. The patient's and family's coping abilities and available supports are assessed. Circumstances surrounding the burn injury should be considered when providing care. Individualized psychosocial support must be given to the patient and family. Because the patient is usually anxious and in pain, nurses should provide reassurance and support, explanations of procedures, and adequate pain relief. Because poor tissue perfusion accompanies burn injuries, only IV analgesia (usually morphine) is administered, titrated for the individual patient. If the patient wishes to see a spiritual advisor or counselor, one is notified.

Transfer to a Burn Center

Patients with the following types of injuries are referred to a burn center for evaluation and care: burns with partial-thickness injury greater than 10% or full-thickness burns in any age group; a burn in an area of the body that requires special attention, such as the face, hands, feet, genitalia, perineum, and over joints; and chemical, electrical, or inhalation burns. Patients with preexisting medical problems or who may have additional trauma that could complicate care should be referred. Children with burns are transferred if they cannot be managed by the available pediatric team. Lastly, any burn injury that carries special social, emotional, or rehabilitative need should be referred to a burn center where these needs are addressed more readily (Guidelines for the Operation of Burn Centers, 2007).

If the patient is to be transported to a burn center, the following measures, listed in order of importance, are instituted before transfer:

- A patent airway is ensured.
- Adequate peripheral circulation is established in any burned extremity.
- A secure IV catheter is inserted with lactated Ringer's solution infusing at the rate required to maintain a urine output of at least 30 mL per hour.
- An indwelling urinary catheter is inserted.
- Adequate pain relief is attained.
- Wounds are covered with a clean, dry sheet, and the patient is kept comfortably warm.

All assessments and treatments are documented, and this information is provided to the burn center personnel. The transferring facility must relay accurate vital signs, temperature, and intake and output totals to burn center personnel so that adequate fluid resuscitation measures will continue.

Management of Fluid Loss and Shock

Next to managing respiratory difficulties, the most urgent need is preventing irreversible shock by replacing lost fluids and electrolytes. As stated previously, survival of the patient with burn injury depends on adequate fluid resuscitation. Table 57-3 describes the fluid changes that occur in the emergent/resuscitative phase of care. Baseline weight and laboratory test results are obtained, and these parameters must be monitored closely in the immediate postburn (resuscitation) period. Both underresuscitation and overresuscitation are associated with poor outcome, and the optimal formula has not been identified; however, regardless of rate and composition of the fluids and colloids administered, diligent monitoring through the first 72 hours is critical to ensure optimal management (Pham, Cancio & Gilbran, 2008).

Fluid Replacement Therapy

The total volume and rate of IV fluid replacement are gauged by the patient's response and guided by the resuscitation formula. The adequacy of fluid resuscitation is determined by monitoring urine output totals, an index of renal

Table 57-3 FLUID AND ELECTROLYTE CHANGES IN THE EMERGENT/RESUSCITATIVE PHASE

Fluid accumulation phase (shock phase)
Plasma → interstitial fluid (edema at burn site)

Observation	Explanation
Generalized dehydration	Plasma leaks through damaged capillaries
Reduction of blood volume	Secondary to plasma loss, fall of blood pressure, and diminished cardiac output
Decreased urinary output	Secondary to: Fluid loss Decreased renal blood flow Sodium and water retention caused by increased adrenocortical activity Hemolysis of red blood cells, causing hemoglobinuria and myonecrosis or myoglobinuria
Potassium (K^+) excess	Massive cellular trauma causes release of K^+ into extracellular fluid (ordinarily, most K^+ is intracellular)
Sodium (Na^+) deficit	Large amount of Na^+ is lost in trapped edema fluid and exudate and by shift into cells as K^+ is released from cells (ordinarily most Na^+ is extracellular)
Metabolic acidosis (base-bicarbonate deficit)	Loss of bicarbonate ions accompanies sodium loss
Hemoconcentration (elevated hematocrit)	Liquid blood component is lost into extravascular space

perfusion. Urine output totals of 0.5 to 1.0 mL/kg/h for adults have been used as resuscitation goals (Pham, et al., 2008).

NURSING ALERT

Clinical parameters are far more important in resuscitation than any formula. Indeed, the patient's individual response is the key to assessing the adequacy of fluid resuscitation.

Additional gauges of fluid requirements and response to fluid resuscitation include hematocrit and hemoglobin and serum sodium levels. Within the first 24 hours after injury, if the hematocrit and the hemoglobin levels decrease or if the urinary output exceeds 50 mL/h, the rate of IV fluid administration may be decreased. One goal is to maintain serum sodium levels in the normal range during fluid replacement.

Appropriate resuscitation endpoints for patients with burn injuries remain unresolved, although some studies have examined hemodynamic and oxygen transport as resuscitation endpoints. Successful resuscitation is associated with increased delivery of oxygen and consumption of oxygen with declining serum lactate levels (Demling, 2005a). Factors associated with increased fluid requirements include delayed resuscitation, full-thickness injury, and presence of inhalation injuries. A State-of-the-Science in Burn Care conference was held in 2006 to identify the focus of research in the next decade. Overresuscitation was identified as a high-priority topic along with a need to identify endpoints for resuscitation (Pham, et al., 2008).

Fluid Requirements

The projected fluid requirements for the first 24 hours are calculated by the clinician based on the extent of the burn injury. Some combination of fluid categories may be used, including colloids (whole blood, plasma, and plasma expanders) and crystalloids/electrolytes (physiologic sodium chloride or lactated Ringer's solution). Adequate fluid resuscitation results in slightly decreased blood volume levels during the first 24 postburn hours and restoration of plasma levels to normal by the end of 48 hours. Formulas have been developed for estimating fluid loss based on the estimated percentage of burned TBSA and the weight of the patient. TBSA greater than 20% to 25% is associated with increased capillary permeability and intravascular fluid shifts that are most profound in the first 24 hours postburn (Pham, et al., 2008).

Oral and enteral resuscitation can be successful in adults with less than 20% TBSA burned (Atiyeh, Gunn & Hayek, 2005). Intravenous resuscitation is recommended when burn TBSA is greater then 20% (Pham, et al., 2008).

Although there is no consensus on the formulas for resuscitation, currently the most popular formula provides for the volume of an isotonic solution to be administered during the first 24 hours in a range of 2 to 4 mL/kg per percentage of TBSA burned. As with the other formulas, half of the calculated total should be given over the first 8 postburn hours, and the other half should be given over the next 16 hours. The hourly rate and volume of the infusion are modified based on the patient's response (Pham, et al., 2008). Clinicians should take note that the resuscitation formulas serve only as guidelines, and the patient's response to fluid therapy is the best parameter to use (Atiyeh, et al., 2005).

With large burns, there is a failure of the sodium–potassium pump (a physiologic mechanism involved in fluid–electrolyte balance) at the cellular level. Therefore, patients with very large burns may need proportionately more milliliters of fluid per percentage of burn than those with smaller burns. Also, patients with electrical injury, inhalation injury, or delayed fluid resuscitation and those who were burned while intoxicated may need additional fluids.

The following example illustrates the use of the consensus formula in a 70-kg (154-lb) patient with a 50% TBSA burn:

1. Formula: 2 to 4 mL/kg/% TBSA
2. 2 mL × 70 kg × 50 TBSA = 7000 mL/24 h
3. Plan to administer: first 8 hours = 3500 mL, or 437 mL/h; next 16 hours = 3500 mL, or 219 mL/h

Most fluid replacement formulas use isotonic electrolyte solutions. Regardless of which standard replacement formula is used, the patient receives approximately the same fluid volume and sodium replacement during the first 48 hours.

Another fluid replacement method requires hypertonic electrolyte solutions. The goal is to deliver smaller amounts of fluid and maintain the same urine output. Hypertonic resuscitation increases the osmolarity of the blood and encourages a shift of fluid into the intravascular space from the interstitial space. Careful monitoring of serum sodium level is required to prevent hypernatremia and acute renal failure (Pham, et al., 2008).

The use of colloids during resuscitation has been the subject of much controversy. Administration of large volumes of crystalloid during resuscitation decreases the protein content in the blood. Proteins help to prevent the movement of fluid and decrease edema. The purpose of adding colloid to the formula is to decrease the amount of fluid needed and also prevent massive edema formation. In contrast, some clinicians believe that after 24 hours the integrity of the capillaries begins to be restored and the use of colloids during that time would not be advantageous. These theories require further study (Pham, et al., 2008).

NURSING ALERT

Formulas are only a guide. The patient's response, evidenced by heart rate, blood pressure, and urine output, is the primary determinant of actual fluid therapy and must be assessed at least hourly. Patient outcomes are improved by optimal fluid resuscitation.

Nursing Management

Nursing assessment in the emergent phase of burn injury focuses on the major priorities for any trauma patient; the burn wound is a secondary consideration. Aseptic management of the burn wounds and invasive lines continues.

The nurse monitors vital signs frequently. Respiratory status is monitored closely, and apical, carotid, and femoral pulses are evaluated particularly in areas of circumferential burn injury to an extremity. Cardiac monitoring is indicated initially or if the patient has a history of cardiac disease, electrical injury, or respiratory conditions.

If all extremities are burned, determining blood pressure may be difficult. A sterile dressing applied under the blood pressure cuff protects the wound from contamination. Because increasing edema makes blood pressure difficult to auscultate, a Doppler (ultrasound) device or a noninvasive electronic blood pressure device may be helpful. In patients with severe burns, an arterial catheter is used for blood pressure measurement and for collecting blood specimens. Peripheral pulses of burned extremities are checked hourly; the Doppler device is useful for this. Elevation of burned upper extremities above the level of the heart is crucial to decrease edema. Elevation of the lower extremities on pillows and of the upper extremities on pillows or by suspension using IV poles may be helpful.

Large-bore IV catheters and an indwelling urinary catheter are inserted, if not already in place, and the nurse's assessment includes monitoring of fluid intake and output. Urine output, an indicator of renal perfusion, is monitored carefully and measured hourly. The amount of urine first obtained when the urinary catheter was inserted is recorded. This may assist in determining the extent of preburn renal function and fluid status.

Burgundy-colored urine suggests the presence of hemochromogen and myoglobin resulting from muscle damage. This is associated with deep burns caused by electrical injury or prolonged contact with flames. Glycosuria, a common finding in the early postburn hours, results from the release of stored glucose from the liver in response to stress.

Although not responsible for prescribing the fluids the nurse should be able to calculate the patient's expected fluid requirements. Infusion pumps are used to deliver a complex regimen of IV fluids prescribed. Administering and monitoring IV therapy are major nursing responsibilities. Strict monitoring of fluid intake and output is essential during the resuscitative phase along with reporting laboratory values and reporting patient responses to the physician.

Body temperature, body weight, preburn weight, and history of allergies, tetanus immunization, past medical and surgical disorders, current illnesses, and a list of current medications are essential to help guide medication needs for the patient. A head-to-toe assessment is performed, focusing on signs and symptoms of concomitant illness, associated injury, or developing complications. If the patient has facial burns, his or her eyes should be examined for injury to the corneas. An ophthalmologist is consulted for complete assessment via fluorescent staining.

Assessing the extent of the burn wound continues and is facilitated with anatomic diagrams (described previously). In addition, the nurse works with the physician to assess the depth of the wound and areas of full-thickness and partial-thickness injury. Assessment of the circumstances surrounding the injury is important. Obtaining a history of the burn injury can help in planning the care for the patient. Assessment should include the time of injury, mechanism of burn, whether the burn occurred in a closed space, the possibility of inhalation of noxious chemicals, and any related trauma. The neurologic assessment focuses on the patient's level of consciousness, psychological status, pain and anxiety levels, and behavior.

The patient's and family's understanding of the injury and treatment is assessed as well. A family meeting upon admission is helpful to explain the detail of the patient's injuries and the course of treatment. Ethical dilemmas, such as those discussed in Chart 57-4, may also occur during hospitalization.

Nursing care of the patient during the emergent/resuscitative phase of burn injury is detailed in the plan of nursing care in Chart 57-5.

Gerontologic Considerations

Comorbid conditions coupled with the burn injury contribute to the high mortality rates of patients 65 years and older. Demling (2005b) reported that more than 60% of elderly patients with burn injuries admitted to the hospital had moderate to severe protein–energy malnutrition, which contributed to an increase in infection compared with well-nourished elderly burn patients. Decreased function of the cardiovascular, renal, and pulmonary systems increases the need for close observation of elderly patients with even relatively minor burns during the emergent and acute phases. Acute renal failure is much more common in elderly patients than in those younger than 40 years of age. The margin of difference between hypovolemia and fluid overload is very small. Suppressed immunologic response, a high incidence of malnutrition, and an inability to withstand metabolic stressors (eg, a cold environment) further compromise the elderly person's ability to heal. As a result of these issues in elderly patients who sustain burn injury, close monitoring and prompt treatment of complications are mandatory.

ACUTE/INTERMEDIATE PHASE

The acute/intermediate phase of burn care follows the emergent/resuscitative phase and begins 48 to 72 hours after the burn injury. During this phase, attention is directed toward continued assessment and maintenance of respiratory and circulatory status, fluid and electrolyte balance, and GI function. Infection prevention, burn wound care (ie, wound cleaning, topical antibacterial therapy, wound dressing, dressing changes, wound débridement, and wound grafting), pain management, and nutritional support are priorities at this stage and are discussed in detail in the following sections.

Medical Management

Airway obstruction caused by upper airway edema can take as long as 48 hours to develop. Changes detected by x-ray and arterial blood gas analysis may occur as the effects of resuscitative fluid and the chemical reactions of smoke ingredients with lung tissues become apparent. Pulmonary complications are not unusual in burn injury. Those with ventilator-associated pneumonia (VAP) have a 40% mortality rate, increasing to 60% to 77% for VAP with an inhalation injury. Bronchial washing or bronchioalveolar lavage can assist in the diagnosis and treatment of pneumonia (Wahl, Ahrns, Brandt, et al., 2005). Ideally, the best practice

CHART 57-4 *Ethics and Related Issues*

How Much Is Enough and What Is Comfort Care?

Situation

A 71-year-old woman was flown to the nearest burn center. She lives alone and was cooking on top of a gas stove. She reached over the burner and caught her robe on fire. The flame consumed most of her upper body. She sustained a full-thickness burn to 42% of her body (face, neck, both arms, and chest). In addition, she turned to remove herself from the room quickly, slipped, and fell, fracturing two ribs and spraining her right ankle. Due to the nature of the fire she was enclosed in a burning area so she also sustained an upper airway inhalation injury. She was awake and alert at the scene and indicated a past medical history of diabetes, hypertension, and renal disease. She was in little pain at the time due to the depth of the burn injury; however, she was asking the team questions related to the survivability of her injury, "Am I going to die?"

Upon admission she was intubated to treat her upper airway edema and suspected inhalation injury. Family arrived and the burn surgeon explained the extent of injury. A decision was made to proceed with care and assess her progress postresuscitation. They were prepared for a future decision regarding withdrawal of care if survival becomes futile.

Dilemma

Initial resuscitation has occurred and approximately 4 days postburn the patient begins to show signs of renal failure. She has had one surgical procedure for débridement and grafting to her chest, and although grafts are intact, they do not look healthy. She is showing increased signs of discomfort and pain due to the operative procedure and the addition of another wound (the new donor site). The family decides in a team meeting that they do not want to initiate hemodialysis and therefore place the patient on comfort measures. Approximately 3 days later she remains on comfort care and has increased signs of restlessness and pain. The nurse caring for the patient requests an order from the physician for an increase in pain medication from 5 mg morphine to 10 mg IV. Within 2 hours after administration the patient expired without discomfort. Six months later the facility is contacted by an attorney and informed that the family is taking legal action against the nurse and physician for performing euthanasia on their mother.

Discussion

The burn team is faced with these types of ethical dilemmas on a regular basis. Whenever possible the team should attempt to elicit the patient's wishes as early as possible, including any advanced directives. This should be considered again prior to intubation unless it is an emergency situation. The problem in this case is that due to absence of pain it is often difficult for a patient to understand the severity of his or her burn injury. The patient begins to experience increased pain as care progresses and increased medication is expected. Once the patient is unable to make decisions for herself, family members assume this responsibility. Clear and documented discussions with the family are essential for communication. Decision makers should always make decisions based on what the patient would want them to do, not their individual concerns. They often have difficulty making comfort care decisions; not all family members always agree. If death does not occur rapidly after a decision, they have feelings of remorse and guilt.

Discussion Questions

1. As a member of the team when the patient arrived at your facility, what would you have done differently?
2. Do you believe this was a survivable injury?
3. Given the failing renal status of the patient, do you believe additional medication should have been administered?
4. Do you believe there is reason for the family to seek legal action for their mother's death?

is to remove the endotracheal tube as soon as possible so that a route for pathogens is not accessible to the lungs. The arterial blood gas values and other parameters determine the need for intubation and mechanical ventilation.

As capillaries regain integrity, 48 or more hours after the burn, fluid moves from the interstitial to the intravascular compartment and diuresis begins (Table 57-4). If cardiac or renal function is inadequate, for example in an elderly patient or in a patient with preexisting cardiac disease, fluid overload occurs and symptoms of congestive heart failure may result (see Chapter 30).

Cautious administration of fluids and electrolytes continues during this phase of burn care because of the shifts in fluid from the interstitial to the intravascular compartment, losses of fluid from large burn wounds, and the patient's physiologic responses to the burn injury. Blood components are administered as needed to treat blood loss and anemia.

Fever is common in patients after burn shock resolves. A resetting of the core body temperature in severely burned patients results in a body temperature a few degrees higher than normal for several weeks after the burn. Bacteremia and septicemia also cause fever in many patients. Acetaminophen (Tylenol) and hypothermia blankets and ancillary heating devices may be required to maintain body temperature in a range of 37.2°C to 38.3°C (99°F to 101°F) so as to reduce metabolic stress and tissue oxygen demand.

Table 57-4 FLUID AND ELECTROLYTE CHANGES IN THE ACUTE PHASE

Fluid remobilization phase (state of diuresis)
Interstitial fluid → plasma

Observation	Explanation
Hemodilution (decreased hematocrit)	Blood cell concentration is diluted as fluid enters the intravascular compartment; loss of red blood cells destroyed at burn site.
Increased urinary output	Fluid shift into intravascular compartment increases renal blood flow and causes increased urine formation.
Sodium (Na^+) deficit	With diuresis, sodium is lost with water; existing serum sodium is diluted by water influx.
Potassium (K^+) deficit (occurs occasionally in this phase)	Beginning on the fourth or fifth postburn day, K^+ shifts from extracellular fluid into cells.
Metabolic acidosis	Loss of sodium depletes fixed base; relative carbon dioxide content increases.

CHART 57-5

PLAN OF NURSING CARE
Care of the Patient During the Emergent/Resuscitative Phase of Burn Injury

NURSING DIAGNOSIS: Impaired gas exchange related to carbon monoxide poisoning, smoke inhalation, and upper airway obstruction
GOAL: Maintenance of adequate tissue oxygenation

Nursing Interventions	Rationale	Expected Outcomes
1. Provide humidified oxygen.	1. Humidified oxygen provides moisture to injured tissues; supplemental oxygen increases alveolar oxygenation.	• Absence of dyspnea • Respiratory rate between 12 and 20 breaths/min • Lungs clear on auscultation • Arterial oxygen saturation greater than 96% by pulse oximetry • Arterial blood gas levels within normal limits
2. Assess breath sounds, and respiratory rate, rhythm, depth, and symmetry. Monitor patient for signs of hypoxia.	2. These factors provide baseline data for further assessment and evidence of increasing respiratory compromise.	
3. Observe for the following: a. Erythema or blistering of lips or buccal mucosa b. Singed nostrils c. Burns of face, neck, or chest d. Increasing hoarseness e. Soot in sputum or tracheal tissue in respiratory secretions	3. These signs indicate possible inhalation injury and risk of respiratory dysfunction.	
4. Monitor arterial blood gas values, pulse oximetry readings, and carboxyhemoglobin levels.	4. Increasing $PaCO_2$ and decreasing PaO_2 and O_2 saturation may indicate need for mechanical ventilation.	
5. Report labored respirations, decreased depth of respirations, or signs of hypoxia to physician immediately.	5. Immediate intervention is indicated for respiratory difficulty.	
6. Prepare to assist with intubation and escharotomies.	6. Intubation allows mechanical ventilation. Escharotomy enables chest excursion in circumferential chest burns.	
7. Monitor mechanically ventilated patient closely.	7. Monitoring allows early detection of decreasing respiratory status or complications of mechanical ventilation.	

NURSING DIAGNOSIS: Ineffective airway clearance related to edema and effects of smoke inhalation
GOAL: Maintain patent airway and adequate airway clearance

Nursing Interventions	Rationale	Expected Outcomes
1. Maintain patent airway through proper patient positioning, removal of secretions, and artificial airway if needed.	1. A patent airway is crucial to respiration.	• Patent airway • Respiratory secretions are minimal, colorless, and thin • Respiratory rate, pattern, and breath sounds normal
2. Provide humidified oxygen.	2. Humidity liquefies secretions and facilitates expectoration.	
3. Encourage patient to turn, cough, and deep breathe. Encourage patient to use incentive spirometry. Suction as needed.	3. These activities promote mobilization and removal of secretions.	

NURSING DIAGNOSIS: Fluid volume deficit related to increased capillary permeability and evaporative losses from the burn wound
GOAL: Restoration of optimal fluid and electrolyte balance and perfusion of vital organs

Nursing Interventions	Rationale	Expected Outcomes
1. Observe vital signs (including central venous pressure or pulmonary artery pressure, if indicated) and urine output, and be alert for signs of hypovolemia or fluid overload.	1. Hypovolemia is a major risk immediately after the burn injury. Overresuscitation might cause fluid overload.	• Serum electrolytes within normal limits • Urine output between 0.5 and 1.0 mL/kg/h • Blood pressure higher than 90/60 mm Hg • Heart rate less than 120 beats/min • Exhibits clear sensorium • Voids clear yellow urine with specific gravity within normal limits
2. Monitor urine output at least hourly and weigh patient daily.	2. Output and weight provide information about renal perfusion, adequacy of fluid replacement, and fluid requirement and fluid status.	

Continued on following page

CHART 57-5 PLAN OF NURSING CARE

Care of the Patient During the Emergent/Resuscitative Phase of Burn Injury (Continued)

Nursing Interventions	Rationale	Expected Outcomes
3. Maintain IV lines and regulate fluids at appropriate rates, as prescribed.	3. Adequate fluids are necessary to maintain fluid and electrolyte balance and perfusion of vital organs.	
4. Observe for symptoms of deficiency or excess of serum sodium, potassium, calcium, phosphorus, and bicarbonate.	4. Rapid shifts in fluid and electrolyte status are possible in the postburn period.	
5. Elevate head of patient's bed and elevate burned extremities.	5. Elevation promotes venous return.	
6. Notify physician immediately of decreased urine output, blood pressure, central venous, pulmonary artery, or pulmonary artery wedge pressures, or increased pulse rate.	6. Because of the rapid fluid shifts in burn shock, fluid deficit must be detected early so that distributive shock does not occur.	
NURSING DIAGNOSIS: Hypothermia related to loss of skin microcirculation and open wounds **GOAL:** Maintenance of adequate body temperature		
1. Provide a warm environment through use of heat shield, space blanket, heat lights, or blankets.	1. A stable environment minimizes evaporative heat loss.	• Body temperature remains 36.1°C to 38.3°C (97°F to 101°F) • Absence of chills or shivering
2. Work quickly when wounds must be exposed.	2. Minimal exposure minimizes heat loss from wound.	
3. Assess core body temperature frequently.	3. Frequent temperature assessments help detect developing hypothermia.	
NURSING DIAGNOSIS: Pain related to tissue and nerve injury and emotional impact of injury **GOAL:** Control of pain		
1. Use pain intensity scale to assess pain level (ie, 1 to 10). Differentiate restlessness due to pain from restlessness due to hypoxia.	1. Pain level provides baseline for evaluating effectiveness of pain relief measures. Hypoxia can cause similar signs and must be ruled out before analgesic medication is administered.	• States pain level is decreased • Absence of nonverbal cues of pain
2. Administer intravenous opioid analgesics as prescribed. Observe for respiratory depression in the patient who is not mechanically ventilated. Assess response to analgesic.	2. Intravenous administration is necessary because of altered tissue perfusion from burn injury.	
3. Provide emotional support and reassurance.	3. Emotional support is essential to reduce fear and anxiety resulting from burn injury. Fear and anxiety increase the perception of pain.	
NURSING DIAGNOSIS: Anxiety related to fear and the emotional impact of burn injury **GOAL:** Minimization of patient's and family's anxiety		
1. Assess patient's and family's understanding of burn injury, coping skills, and family dynamics.	1. Previous successful coping strategies can be fostered for use in the present crisis. Assessment allows planning of individualized interventions.	• Patient and family verbalize understanding of emergent burn care • Able to answer simple questions
2. Individualize responses to the patient's and family's coping level.	2. Reactions to burn injury are extremely variable. Interventions must be appropriate to the patient's and family's present level of coping.	

Continued

CHART 57-5 PLAN OF NURSING CARE

Care of the Patient During the Emergent/Resuscitative Phase of Burn Injury (Continued)

Nursing Interventions	Rationale	Expected Outcomes
3. Explain all procedures to the patient and the family in clear, simple terms.	3. Increased understanding alleviates fear of the unknown. High levels of anxiety may interfere with understanding of complex explanations.	
4. Maintain adequate pain relief.	4. Pain increases anxiety.	
5. Consider administering prescribed antianxiety medications if the patient remains extremely anxious despite nonpharmacologic interventions.	5. Anxiety levels during the emergent phase may exceed the patient's coping abilities. Medication decreases physiologic and psychological anxiety responses.	

COLLABORATIVE PROBLEMS: Acute respiratory failure, distributive shock, acute renal failure, compartment syndrome, paralytic ileus, Curling's ulcer
GOAL: Absence of complications

Nursing Interventions	Rationale	Expected Outcomes
Acute Respiratory Failure		
1. Assess for increasing dyspnea, stridor, changes in respiratory patterns.	1. Such signs reflect deteriorating respiratory status.	• Arterial blood gas values within acceptable limits: PaO_2 greater than 80 mm Hg, $PaCO_2$ less than 50 mm Hg
2. Monitor pulse oximetry, arterial blood gas values for decreasing PaO_2 and oxygen saturation, and increasing $PaCO_2$.	2. Such signs reflect decreased oxygenation status.	• Breathes spontaneously with adequate tidal volume
3. Monitor chest x-ray results.	3. X-ray may disclose pulmonary injury.	• Chest x-ray findings normal
4. Assess for restlessness, confusion, difficulty attending to questions, or decreasing level of consciousness.	4. Such manifestations may indicate cerebral hypoxia.	• Absence of cerebral signs of hypoxia
5. Report deteriorating respiratory status immediately to physician.	5. Acute respiratory failure is life-threatening, and immediate intervention is required.	
6. Prepare to assist with intubation or escharotomies as indicated.	6. Intubation allows mechanical ventilation. Escharotomies allow improved chest excursion with respirations.	
Distributive Shock		
1. Assess for decreasing urine output and blood pressure as well as increasing pulse rate. (If hemodynamic monitoring is used, assess for decreasing pulmonary artery and pulmonary artery wedge pressures and cardiac output.)	1. Such signs and symptoms may indicate distributive shock and inadequate intravascular volume.	• Urine output between 0.5 and 1.0 mL/kg/h • Blood pressure within patient's normal range (usually greater than 90/60 mm Hg) • Heart rate within patient's normal range (usually less than110/min)
2. Assess for progressive edema as fluid shifts occur.	2. As fluid shifts into the interstitial spaces in burn shock, edema occurs and may compromise tissue perfusion.	• Pressures and cardiac output remain within normal limits
3. Adjust fluid resuscitation in collaboration with the physician in response to physiologic findings.	3. Optimal fluid resuscitation prevents distributive shock and improves patient outcomes.	
Acute Renal Failure		
1. Monitor urine output and blood urea nitrogen (BUN) and serum creatinine levels.	1. These values reflect renal function.	• Adequate urine output • BUN and serum creatinine values remain normal
2. Report decreased urine output or increased BUN and creatinine values to physician.	2. These laboratory values indicate possible renal failure.	
3. Assess urine for hemoglobin or myoglobin.	3. Hemoglobin or myoglobin in the urine points to an increased risk of renal failure.	

Continued on following page

CHART 57-5 PLAN OF NURSING CARE

Care of the Patient During the Emergent/Resuscitative Phase of Burn Injury (Continued)

Nursing Interventions	Rationale	Expected Outcomes
4. Administer increased fluids as prescribed.	4. Fluids help to flush hemoglobin and myoglobin from renal tubules, decreasing the potential for renal failure.	
Compartment Syndrome		
1. Assess peripheral pulses hourly with Doppler ultrasound device.	1. Assessment with Doppler device substitutes for auscultation and indicates characteristics of arterial blood flow.	• Absence of paresthesias or symptoms of ischemia of nerves and muscles • Peripheral pulses detectable by Doppler
2. Assess warmth, capillary refill, sensation, and movement of extremity hourly. Compare affected with unaffected extremity.	2. These assessments indicate characteristics of peripheral perfusion.	
3. Remove blood pressure cuff after each reading.	3. Cuff may act as a tourniquet as extremities swell.	
4. Elevate burned extremities.	4. Elevation reduces edema formation.	
5. Report loss of pulse or sensation or presence of pain to physician immediately.	5. These signs and symptoms may indicate inadequate tissue perfusion.	
6. Prepare to assist with escharotomies.	6. Escharotomies relieve the constriction caused by swelling under circumferential burns and improve tissue perfusion.	
Paralytic Ileus		
1. Maintain nasogastric tube on low intermittent suction until bowel sounds resume.	1. This measure relieves gastric and abdominal distention, also prevents vomiting.	• Absence of abdominal distention • Normal bowel sounds within 48 hours
2. Auscultate for bowel sounds, abdominal distention.	2. As bowel sounds resume, feeding may be slowly initiated. Abdominal distention reflects inadequate decompression.	
Curling's Ulcer		
1. Assess gastric aspirate for pH and blood.	1. Acidic pH indicates need for antacids or histamine blockers. Blood indicates possible gastric bleeding.	• Absence of abdominal distention • Normal bowel sounds within 48 hours • Gastric aspirate and stools do not contain blood
2. Assess stools for occult blood.	2. Blood in stools may indicate gastric or duodenal ulcer.	
3. Administer histamine blockers and antacids as prescribed.	3. Such medications reduce gastric acidity and risk of ulceration.	

Central venous, peripheral arterial, or pulmonary artery thermodilution catheters may be required for monitoring venous and arterial pressures, pulmonary artery pressures, pulmonary capillary wedge pressures, or cardiac output. It is not uncommon for patients with major burns to have multiple invasive line sites due to the amount and frequency of fluid and medication that need to be administered. Whenever possible, burned areas of the body are avoided as sites for insertion of invasive lines.

Infection Prevention

Infection progressing to sepsis is the major cause of death in patients who have survived the first few days after a major burn. Burn patients are at risk for infection for a few reasons. The loss of skin removes their ability to protect themselves from the environment. The longer length of stay in the hospital predisposes them to hospital-associated infections. The number and frequency of invasive procedures, both in the patient room and in the operating room, increase the risk of infection. Lastly, immunosuppression that accompanies extensive burn injury places patients at high risk. The nursing goal is to provide protection and safety in the patients' environment to ultimately prevent or control infection in the burn population (Hodle, Richter & Thompson, 2006).

The burn wound is an excellent medium for bacterial growth and proliferation. The burn eschar is nonviable tissue and has no blood supply; therefore, neither leukocytes or antibodies nor systemic antibiotics can reach the area. More than 1 billion bacteria per gram of tissue may

be present and subsequently spread to the bloodstream or release their toxins, which reach distant sites. *Pseudomonas* is the major challenge in 44% of burn centers. Other common organisms are methicillin-resistant *Staphylococcus aureus* (MRSA) and *Acinetobacter*. Important but somewhat less common are *Staphylococcus* and vancomycin-resistant enterococci (VRE) (Hodle, et al., 2006). *Staphylococcus* and *Enterococci* are responsible for more than 50% of nosocomial bloodstream infections in patients with burn injuries. Other bacteria that are important in burn care include *Proteus*, *Escherichia coli*, and *Klebsiella*. Bloodstream infections are confirmed if two positive blood cultures are obtained or if one positive culture is obtained in the presence of clinical signs of sepsis (Greenhalgh, 2007). Fungi such as *Candida albicans* also grow easily in burn wounds.

Infection impedes burn wound healing by promoting excessive inflammation and damaging tissue. When the burn wound is healing through spontaneous reepithelialization or is being prepared for skin grafting, it must be protected from sepsis. Characteristics of burn wound sepsis are 10^5 bacteria per gram of tissue, inflammation or destruction of unburned skin, and invasive infection with or without signs of sepsis (Greenhalgh, 2007).

A primary source of bacterial infection is the patient's intestinal tract, the source of most microbes. The intestinal mucosa normally serves as a barrier to keep the internal environment free from a variety of pathogens. After a severe burn injury, the intestinal mucosa becomes markedly permeable (Gosain & Gamelli, 2005b), allowing microbial flora and endotoxins to pass freely into the systemic circulation and causing infection. Early enteral feeding is one strategy to help avoid increased intestinal permeability and prevent early endotoxin translocation (De-Souza & Greene, 2005).

A major secondary source of pathogenic microbes is the environment. Compliance with infection-control policies has been identified as the single greatest challenge along with the abundance of resistant organisms (Hodle, et al., 2006). Burn centers are designed with specific measures to reduce the risk of infection: private rooms and bathrooms; increased airflow within patient rooms to create a more positive airflow; low humidification to prevent bacterial growth; limited use of cloth (eg, patient privacy curtains, window treatments); accessibility of proper protective equipment such as caps, masks, gowns and gloves; convenient and available hand washing/hand hygiene areas; and room design with antidust and dirt collection areas.

Use of cap, gown, mask, and gloves is essential while caring for the patient with open burn wounds. Aseptic technique is used when caring directly for burn wounds. Gowns and gloves are worn by all caregivers and visitors; hand hygiene is used before and after leaving the patient room. Special instruction is given to all visitors with the goal of preventing the spread of infection because of the immunosuppression experienced by patients with burns.

Bacteria are found on all skin surfaces; by itself, their presence does not determine burn infection. Constant monitoring and observation of the wound (eg, changes in the wound, presence of purulent drainage, pain, and increasing depth of burn wound) are needed to detect wound infection. Tissue specimens may be obtained for culture to monitor colonization (Greenhalgh, 2007). Antibiotics are seldom prescribed prophylactically because of the risk of promoting resistant strains of bacteria. Systemic antibiotics are administered when there is documented burn wound sepsis or other positive cultures such as urine, sputum, or blood. Sensitivity of the organisms to the prescribed antibiotics should be determined before administration. Careful attention is paid to antibiotic use in the burn unit because inappropriate use of antibiotics significantly affects the microbial flora present in the burn unit and increases the risk of drug resistance.

Wound Cleaning

Various measures are used to clean the burn wound, such as **hydrotherapy.** If the patient is ambulatory, the wounds can be cleansed in a shower. The wounds of nonambulatory patients can be cleansed using shower carts—mobile stretchers made with removable sides, drainage holes, and positioning capabilities. Retractable shower hoses suspended from walls and ceilings provide the nurse with easy access to a water source for washing the wounds. Unstable patients may have their wounds washed at the bedside. Total immersion hydrotherapy is rarely performed. Because of the high risk for infection and sepsis, the use of plastic liners, water filters, and thorough decontamination of hydrotherapy equipment and wound care areas is required to prevent cross-contamination. The temperature of the water is maintained at 37.8°C (100°F), and the temperature of the room should be maintained between 26.6°C and 29.4°C (80°F to 85°F). The duration of wound cleansing and dressing change is determined by the patient's ability to tolerate the treatment and to maintain a satisfactory body temperature.

During the bath, the patient is encouraged to be as active as possible. Hydrotherapy provides an excellent opportunity for exercising the extremities and cleaning the entire body. When the patient is removed, any residue adhering to the body is washed away with a clear water spray or shower. Unburned areas, including the hair, must be washed regularly as well. At the time of wound cleaning, all skin is inspected for any hints of redness, breakdown, or local infection. Hair in and around the burn area, except the eyebrows, should be clipped short or shaved. Intact blisters should be left alone and débrided only if they rupture or break.

Conscientious management of the burn wound is essential. When nonviable loose skin is removed, aseptic conditions must be established. Wound cleansing is usually performed daily in wound areas that are not undergoing surgical intervention. Mechanical débridement can be performed to remove loose nonviable tissue. However, surgical removal as soon as possible is preferred.

After the burn wounds are cleaned, they are gently patted dry, and the prescribed method of wound care is performed. Whatever the method is used, the goal is to protect the wound from overwhelming proliferation of pathogenic organisms and invasion of deeper tissues until either spontaneous healing or skin grafting can be achieved.

Patient comfort and ability to participate in the prescribed treatment are also important considerations. During the treatment, the patient is assessed for signs of chilling, fatigue, changes in hemodynamic status, and pain unrelieved by analgesic medications or relaxation techniques.

Topical Antibacterial Therapy

Variations in topical wound care for nonsurgical burn wounds exist among burn centers across the country and choices are made based on the individualized needs of each patient. There is general agreement that some form of antimicrobial therapy applied to the burn wound is an acceptable method of local care in extensive burn injury. Silver sulfadiazine is considered the gold standard for protecting wounds from infection (Caruso, Foster, Blome-Eberwein, et al., 2006). Topical antibacterial therapy becomes more important in the deep dermal and full-thickness injury because they are more prone to infection. Silver has been introduced into a variety of topical treatments because of its broad spectrum effectiveness against *Staphylococcus aureus* and *Pseudomonas aeruginosa* (Pham & Gibran, 2007). The goal of topical therapy is to provide a dressing that

- Is effective against gram-positive and gram-negative organisms and fungi,
- Penetrates the eschar but is not systemically toxic,
- Does not lose effectiveness or allow another infection to develop,
- Is cost-effective, available, and acceptable to the patient,
- Is easy to apply and remove, and decreases the frequency of dressing changes, decreases pain, and minimizes nursing time.

Table 57-5 describes three commonly used topical agents, silver sulfadiazine (Silvadene), silver nitrate, and mafenide acetate (Sulfamylon), two of which contain silver. Many other topical agents are available, including povidone–iodine ointment 10% (Betadine), gentamicin sulfate, nitrofurazone (Furacin), Dakin's solution, acetic acid, and antifungal agents (miconazole, clotrimazole, and mupirocin [Bactroban]). Bacitracin or a triple antibiotic agent may be used for facial burns or on skin grafts initially.

No single topical medication is universally effective, and use of different agents at different times in the postburn period may be necessary. Prudent use and alternation of antimicrobial agents result in less-resistant strains of bacteria, greater effectiveness of the agents, and a decreased risk of sepsis.

Table 57-5 OVERVIEW OF SELECTED TOPICAL ANTIBACTERIAL AGENTS USED FOR BURN WOUNDS

Agent	Indication/Comment	Application	Nursing Implications
Silver sulfadiazine 1% (Silvadene) water-soluble cream	• Most bactericidal agent • Minimal penetration of eschar	Apply 1/16-inch layer of cream with a sterile glove 1–3 times daily.	• Watch for leukopenia 2–3 days after initiation of therapy. (Leukopenia usually resolves within 2–3 days.) • Anticipate formation of pseudo-eschar (proteinaceous gel), which is removed easily after 72 hours.
Mafenide acetate 5% to 10% (Sulfamylon) hydrophilic-based cream	• Effective against gram-negative and gram-positive organisms • Diffuses rapidly through eschar • In 10% strength, it is the agent of choice for electrical burns because of its ability to penetrate thick eschar	Apply thin layer with sterile glove twice a day and leave open as prescribed; if the wound is dressed, change the dressing every 6 hours as prescribed.	• Monitor arterial blood gas levels and discontinue as prescribed, if acidosis occurs. Mafenide acetate is a strong carbonic anhydrase inhibitor that may reduce renal buffering and cause metabolic acidosis. • Premedicate the patient with an analgesic before applying mafenide acetate because this agent causes severe burning pain for up to 20 minutes after application.
Silver nitrate 0.5% aqueous solution	• Bacteriostatic and fungicidal • Does not penetrate eschar	Apply solution to gauze dressing and place over wound. Keep the dressing wet but covered with dry gauze and dry blankets to decrease vaporization. Remoisten every 2 hours, and redress wound twice a day.	• Monitor serum sodium (Na^+) and potassium (K^+) levels and replace as prescribed. Silver nitrate solution is hypotonic and acts as wick for sodium and potassium. • Protect bed linen and clothing from contact with silver nitrate, which stains everything it touches black.
Acticoat	• Effective against gram-negative and gram-positive organisms and some yeasts and molds • Delivers a uniform, antimicrobial concentration of silver to the burn wound	Moisten with sterile water only (never use normal saline). Apply directly to wound. Cover with absorbent secondary dressing. Remoisten every 3–4 hours with sterile water.	• Do not use oil-based products or topical antimicrobials with Acticoat burn dressing. Keep Acticoat moist, not saturated. May produce a "pseudo-eschar" from silver after application. • Can be left in place for 3–5 days. Also available in Acticoat 7, which can be left in place for up to 7 days without the need to change the dressing.

Wound Dressing

After wound cleaning, the burned areas are patted dry and the prescribed topical agent is applied; the wound is then covered with several layers of dressings. A light dressing is used over joint areas to allow for motion (unless the particular area has a graft and motion is contraindicated). A light dressing is also applied over areas for which a splint has been designed to conform to the body contour for proper positioning. Circumferential dressings should be applied distally to proximally. If the hand or foot is burned, the fingers and toes should be wrapped individually to promote adequate healing.

Burns to the face may be left open to air once they have been cleaned and the topical agent has been applied. Careful attention must be given to ensure that the topical agent does not interfere with the eyes or mouth. A light dressing can be applied to the face to absorb excess exudates that might run into the eyes, causing irritation.

Occlusive dressings may be used over areas with new skin grafts to protect the graft and promote an optimal condition for its adherence to the recipient site. An occlusive dressing is a thin gauze that is impregnated with a topical antimicrobial agent or is applied after application of a topical antimicrobial agent. Ideally, these dressings remain in place for 3 to 5 days, at which time they are removed for examination of the graft. When occlusive dressings are applied, precautions are taken to prevent two body surfaces from touching, such as fingers or toes, ear and scalp, the areas under the breasts, any point of flexion, or between the genital folds. Functional body alignment positions are maintained by using splints or by regular repositioning of the patient.

NURSING ALERT

Dressings impede circulation if they are too tightly wrapped. The peripheral pulses must be checked frequently and burned extremities elevated on two pillows. Extremities are always wrapped from distal to proximal to the heart. If the patient's pulse is diminished, this is a critical situation and must be addressed immediately.

Dressings that adhere to the wound can be removed more comfortably and without damaging healing tissue by moistening the wound with tap water. The remaining dressings are carefully and gently removed. The patient may participate in removing the dressings, providing some degree of control over this painful procedure. The wounds are then cleaned and débrided to remove any remaining topical agent, exudate, and dead skin. Sterile scissors and forceps may be used to trim loose eschar and encourage separation of devitalized skin. During this procedure, the wound and surrounding skin are carefully inspected. The color, odor, size, exudate, signs of reepithelialization, and other characteristics of the wound and the eschar and any changes from the previous dressing change are noted.

Wound Débridement

The goals of, **débridement,** the removal of devitalized tissue, are:

- Removal of tissue contaminated by bacteria and foreign bodies, thereby protecting the patient from invasion of bacteria
- Removal of devitalized tissue or burn eschar in preparation for grafting and wound healing

There are three types of débridement—natural, mechanical, and surgical.

Natural Débridement

With natural débridement, the dead tissue separates from the underlying viable tissue spontaneously. Bacteria that are present at the interface of the burned tissue and the viable tissue underneath gradually liquefy the fibrils of **collagen** that hold the eschar in place for the first or second postburn weeks. Proteolytic and other natural enzymes cause this phenomenon. However, use of antibacterial topical agents tends to slow this natural process of eschar separation and slows the healing process.

Mechanical Débridement

Mechanical débridement involves the use of surgical scissors, scalpels, and forceps to separate and remove the eschar. This technique can be performed by skilled physicians, nurses, or physical therapists and is usually done with daily dressing changes. If bleeding occurs, hemostatic agents or pressure can be used to stop the bleeding from small vessels. Wet-to-dry dressings are not advocated in burn care because of the chance of removing viable cells along with necrotic tissue. Dressing changes alone aid the removal of wound debris.

Chemical Débridement

Topical enzymatic débridement agents are available to promote débridement of the burn wounds. Because such agents usually do not have antimicrobial properties, they should be used together with topical antibacterial therapy to protect the patient from bacterial invasion. Heavy metals such as silver deactivate the débriding agent; therefore, caution is necessary to ensure that the débriding agent does not interfere with the topical antimicrobial agent. Separate dressings are used to prevent this from occurring.

Surgical Débridement

Early surgical excision to remove devitalized tissue along with early burn wound closure is now recognized as one of the most important factors contributing to survival in a patient with a major burn injury. Aggressive surgical wound closure has reduced the incidence of burn wound sepsis, thus improving survival rates (Burke, 2005). Early excision is carried out before the natural separation of eschar is allowed to occur.

Surgical débridement is an operative procedure involving either primary **excision** (surgical removal of tissue) of the full thickness of the skin down to the fascia (tangential excision) or shaving of the burned skin layers gradually down to freely bleeding, viable tissue. Surgical excision is initiated early in burn wound management. This may be performed within the first few days after the burn or as soon as the patient is hemodynamically stable and edema has decreased. Ideally, the wound is then covered immediately with a skin graft, if needed, and an occlusive dressing. If the wound bed is not ready for a skin graft at the time of excision, a temporary biologic dressing may be used until a skin graft can be applied during subsequent surgery.

The use of surgical excision carries with it risks and complications, especially with large burns. The procedure creates a high risk of extensive blood loss (as much as 100 to 125 mL of blood per percentage of body surface excised) and lengthy operating and anesthesia times. However, when conducted in a timely and efficient manner, surgical excision results in shorter hospital stays and possibly a decreased risk of complications from invasive burn wound sepsis.

Gerontologic Considerations

Eschar separation in full-thickness burns is typically delayed in elderly patients, and older patients are frequently poor risks for surgical excision. For these reasons, prolonged hospitalization, immobilization, and associated problems are common. If the elderly patient can tolerate surgery, early excision with skin grafting is the treatment of choice because it decreases the mortality rate in this population. If the patient is not a surgical candidate, chemical débridement is often chosen to enhance the removal of eschar over time. Prevention of complications of prolonged hospitalization, immobility, and surgery is essential in the care of the elderly burn patient.

Wound Grafting

The patient with deep partial-thickness or full-thickness burns may be a candidate for skin grafting. If so, temporary coverage of the burn wound is necessary until coverage with a graft of the patient's own skin (**autograft**) is possible. The purposes of wound coverage are to decrease the risk of infection; prevent further loss of protein, fluid, and electrolytes through the wound; and minimize heat loss through evaporation. Several methods of wound coverage are available; some are temporary until grafting with permanent coverage is possible. Wound coverage may consist of biologic, biosynthetic, synthetic, and autologous methods or a combination of these approaches.

The main areas for skin grafting include the face (for cosmetic and psychological reasons); functional areas, such as the hands and feet; and areas that involve joints. Grafting permits earlier functional ability and reduces wound **contractures.** When burns are very extensive, the order in which areas are grafted is chosen based on the ability to achieve wound closure as soon as possible, and, therefore, the chest and abdomen or back may be grafted first to reduce the burn surface.

Granulation tissue fills the space created by the wound, creates a barrier to bacteria, and serves as a bed for epithelial cell growth. Richly vascular granulation tissue is pink, firm, shiny, and free of exudate and debris. It should have a bacterial count of less than 100,000/g of tissue to optimize graft success. If the wound is not ready for skin grafting, the burn wound is excised and allowed to granulate. Once the wound is excised, a wound covering is applied to keep the wound bed moist and promote the granulation process.

Biologic Dressings (Homografts and Heterografts)

Biologic dressings have several uses. In extensive burns, they provide temporary wound coverage and protect the granulation tissue until autografting is possible. Biologic dressings are commonly used in patients with large areas of burn and little remaining normal skin for donor sites. They can be used as a test graft in preparation for the patient's own skin graft to determine if the bed will accept the graft. Once the biologic dressing appears to be "taking," or adhering to the granulating surface with minimal underlying exudation, the patient is ready for an autologous skin graft.

Biologic dressings also provide temporary immediate coverage for clean, superficial burns and decrease the wound's evaporative water and protein loss. They decrease pain by protecting nerve endings and are an effective barrier against water loss and entry of bacteria. When applied to superficial partial-thickness wounds, they seem to speed healing. Biologic materials can be left open or covered. They stay in place for varying lengths of time but are removed in instances of infection or rejection. Another advantage for the patient is that these dressings often require fewer dressing changes, therefore, decreasing pain. They can also be used in the outpatient environment.

Biologic dressings consist of **homografts** (or allografts) and **heterografts** (or xenografts). Homografts are skin obtained from living or recently deceased humans. Heterografts consist of skin taken from animals (usually pigs). Most biologic dressings are used as temporary coverings of burn wounds and are eventually rejected because of the body's immune reaction to them as foreign.

Homografts tend to be the most expensive biologic dressings. They are available from skin banks in fresh and cryopreserved (frozen) forms. Homografts are thought to provide the best infection control of all the biologic or biosynthetic dressings available. Revascularization occurs within 48 hours, and the graft may be left in place for several weeks.

Pigskin is available from commercial suppliers. It is available fresh, frozen, or lyophilized (freeze dried) for longer shelf life. Pigskin is used for temporary covering of clean wounds such as superficial partial-thickness wounds and donor sites. Although pigskin does not vascularize, it does adhere to clean superficial wounds and provides excellent pain control while the underlying wound epithelializes (Atiyeh, et al., 2005).

Biosynthetic and Synthetic Dressings

Problems with availability, sterility, and cost have prompted the search for biosynthetic and synthetic skin substitutes, which may eventually replace biologic dressings as temporary wound coverings. A widely used synthetic dressing is **Biobrane,** which is composed of a nylon, silastic membrane combined with a collagen derivative. The material is semitransparent and sterile. It has an indefinite shelf life and is less costly than homograft or pigskin. Like biologic dressings, Biobrane protects the wound from fluid loss and bacterial invasion (Fig. 57-4).

Biobrane adheres to the wound fibrin, which binds to the nylon–collagen material. Within 5 days, cells migrate into the nylon mesh. In general, adherence to the wound surface correlates directly with low bacterial counts. When the Biobrane dressing adheres to the wound, the wound remains stable. Biobrane can remain in place until spontaneous epithelialization and wound healing occur. It can be laid on top of a wide-meshed autograft to protect the wound until the autograft epithelium grows out to close the interstices. As the Biobrane gradually separates, it is trimmed, leaving a healed wound.

Another temporary wound covering is BCG Matrix. This dressing combines beta-glucan, a complex carbohydrate, with collagen in a meshed reinforced wound dressing. Beta-glucan is known to stimulate macrophages, which are vital in the inflammatory process of healing. BCG Matrix is

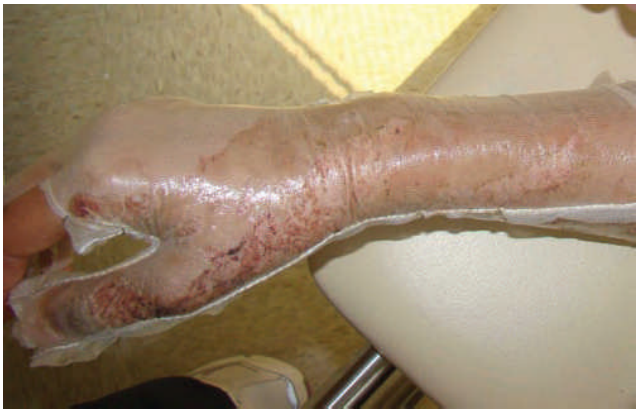

Figure 57-4 Biobrane dressing for partial-thickness burn wound. Biobrane dressing applied to a clean wound bed on the hand. Used with permission. Lehigh Valley Health Network, Allentown, PA.

a temporary wound covering intended for use with partial-thickness burns and donor sites. It is applied immediately after cleaning and débridement. If the burn wound surface remains free of infection, BCG Matrix can be left in place until healing is complete (Atiyeh, et al., 2005).

Several other synthetic dressings are available for burn wound care. Op-Site, a thin, transparent, polyurethane elastic film, can be used to cover clean partial-thickness wounds and donor sites. This dressing is occlusive and waterproof but permeable to water vapor and air; this permeability not only provides protection from microbial contamination but also allows for the exchange of gases, which occurs much more quickly in a moist environment. Other synthetic dressings used for burn wounds include Tegaderm, N-Terface, and DuoDerm.

Skin Substitutes

In an attempt to develop the ideal burn wound covering product, skin substitutes have been created that surgically replace the epidermis and the dermis. It is believed that skin substitutes enhance the healing process of an open wound when autologous skin is unavailable or limited for use. These products are often the choice when donor sites are inadequate or unavailable.

A **cultured epithelial autograft (CEA)** provides permanent coverage of large wounds when harvesting of skin for autografting is not an option. This involves a biopsy of the patient's skin in an unburned area. Keratinocytes are isolated, and epithelial cells are cultured in a laboratory. The original epithelial cell reproduces multiple plated sheets of CEA to cover an already surgically excised wound. These cells are then attached to the burn wound surface, and extreme care is taken until they have adhered to the wound surface. Varying degrees of success have been reported and results are encouraging. However, the disadvantages of the CEA are that the grafts are thin and fragile and can shear easily. Patients have longer hospital stays and higher hospital costs and require more surgical procedures than those treated by traditional methods. In addition, patients require more reconstructive procedures in the first 1 to 2 years after injury. Therefore, CEA use is very limited and is reserved for burn patients whose donor sites are limited (Pham & Gibran, 2007).

Two dermal substitutes are **Integra** Artificial Skin and **AlloDerm**. Artificial skin (Integra) is the newest type of dermal substitute (Fig. 57-5). A dermal analogue, Integra is

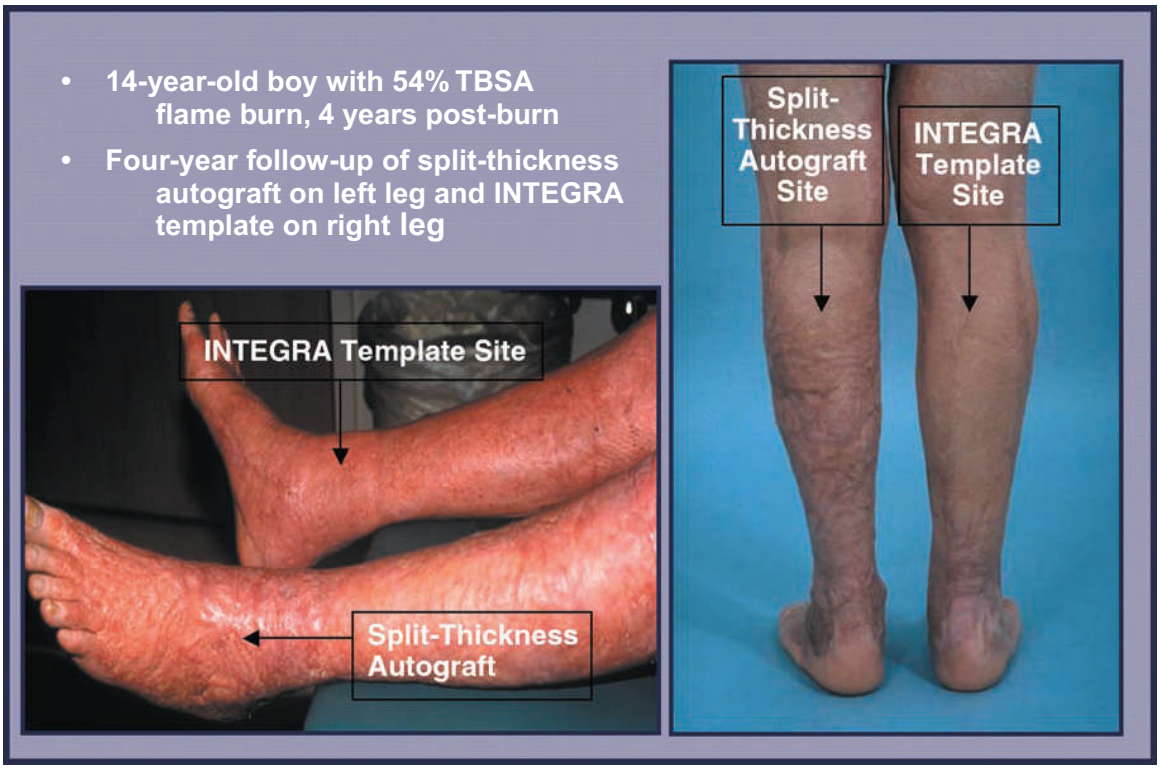

Figure 57-5 Comparison of Integra template site (*right leg*) to split-thickness autograft site (*left leg*). Used with permission from Glenn Warden, MD.

composed of two main layers. The epidermal layer, consisting of silicone, acts as a bacterial barrier and prevents water loss from the dermis. The dermal layer is composed of animal collagen. It interfaces with the open wound surface and allows migration of fibroblasts and capillaries into the material. This "neodermis" becomes a permanent structure. The artificial dermis is biodegraded and reabsorbed. The outer silicone membrane is removed 2 weeks after application and is replaced with the patient's own skin in the form of a thin epidermal skin graft. When a thinner autologous donor graft is used, donor site healing is quicker. Long-term effects of Integra include minimal contracture formation. The graft site is very pliable, almost eliminating the need for repeated cosmetic surgery. Most important, Integra has resulted in less hypertrophic scarring, thus reducing the need for compression devices once the burn wound has healed. Because Integra allows for earlier excision and coverage of the burn wound, metabolic demands of the patient are reduced. Integra allows for the increased survivability of patients with large burn injuries and improves the functional and cosmetic qualities of the healed burns. The combination of Integra with cultured skin substitutes has demonstrated promise in burn management (Pham & Gibran, 2007).

Another promising dermal substitute is AlloDerm. It is processed dermis from human cadaver skin, which can be used as the dermal layer for skin grafts. When a **donor site** (the area from which skin is taken to provide a skin graft for another part of the body) is harvested for an autologous skin graft, both the epidermal and the dermal layers of skin are removed from the donor site. AlloDerm provides a permanent dermal layer replacement. Its use allows the burn surgeon to harvest a thinner skin graft, consisting of the epidermal layer only. The patient's epidermal layer is placed directly over the dermal base (AlloDerm). The new graft is then treated according to the burn unit's protocol. Use of AlloDerm has also resulted in less scarring and contractures with healed grafts; donor sites heal more quickly than conventional donor sites because only the epidermal layer has been harvested. This is important when donor sites are limited because of extensive burns (Boyce, Greenhalgh, Palmieri, et al., 2006; Pham & Gibran, 2007).

Autografts

Autografts remain the preferred material for definitive burn wound closure after excision. Autografts are the ideal means of covering burn wounds because the grafts are the patient's own skin and therefore are not rejected by the patient's immune system. They can be split-thickness (Fig. 57-6), full-thickness, pedicle flaps, or epithelial grafts. Full-thickness autografts and pedicle flaps are commonly used for reconstructive surgery, which may take place months or years after the initial injury.

Split-thickness autografts can be applied in sheets or they can be expanded by meshing so that they cover 1.5 to 9 times more than a given donor site area. Skin meshers enable the surgeon to cut tiny slits into a sheet of donor skin, making it possible to cover large areas with smaller amounts of donor skin. These expanded grafts adhere to the recipient site more easily than sheet grafts and prevent the accumulation of blood, serum, air, or purulent material under the graft. However, any kind of graft other than a sheet graft contributes to scar formation as it heals. Use of expanded grafts may be necessary in large wounds but should be viewed as a compromise in terms of cosmesis.

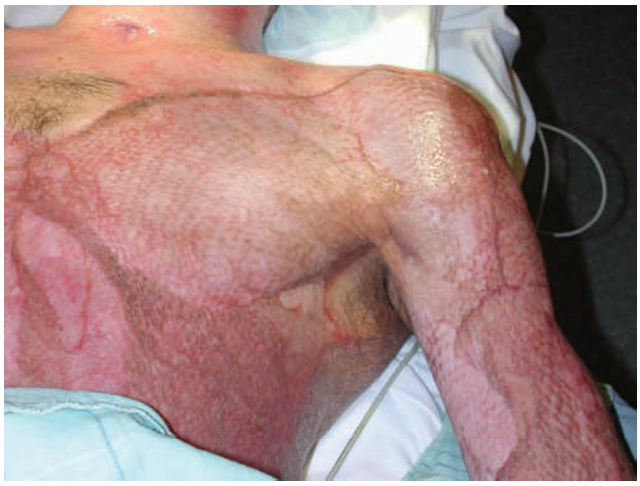

Figure 57-6 Healed split-thickness skin graft to the chest and upper extremity. Used with permission. Lehigh Valley Health Network, Allentown, PA.

If blood, serum, air, fat, or necrotic tissue lies between the recipient site and the graft, there may be partial or total loss of the graft. Infection or mishandling of the graft and trauma during dressing changes account for most other instances of graft loss. Use of split-thickness grafts allows the remaining donor site to retain sweat glands and hair follicles and minimizes donor site healing time.

Care of the Graft Site. Protection is the key goal of caring for skin grafts postoperatively. Occlusive dressings are commonly used initially after grafting to immobilize the graft. Occupational therapists may be helpful in constructing splints to immobilize newly grafted areas to prevent dislodging of the graft. Homografts, heterografts, or synthetic dressings may also be used to protect grafts.

The first dressing change is usually performed 2 to 5 days after surgery, or earlier in the case of clinical signs of infection, purulent drainage, or a foul odor. Infection, bleeding beneath the graft, and shearing force are the most common reasons for graft loss in the early postoperative period.

The patient is positioned and turned carefully to avoid disturbing the graft or putting pressure on the graft site. If an extremity has been grafted, it is elevated to minimize edema. The patient begins exercising the grafted area 5 to 7 days after grafting.

Care of the Donor Site. Donor sites are clean new wounds that are usually very painful. Caregivers need to recognize this additional source of pain and possible site of infection. Since the donor site is a clean wound created in a surgical environment, it should heal easily unless other complications exist. A moist gauze dressing is applied at the time of surgery to maintain pressure and to stop any oozing. After the donor skin is excised, a thrombostatic agent such as thrombin or epinephrine may be applied directly to the site. The donor site may be covered in several ways, from single-

layer gauze impregnated with petrolatum, scarlet red, or bismuth to new biosynthetic dressings such as Biobrane or BCG Matrix. Acticoat can also be used as a dressing on donor sites. With all types of covering, donor sites must remain clean, dry, and free from pressure. Because a donor site is a partial-thickness wound, it is very painful and will heal spontaneously within 7 to 14 days with proper care.

Pain Management

The ability to quantify pain may be difficult in any patient; however, the patient who has experienced a burn has many additional challenges. A burn injury is considered one of the most painful types of trauma that a patient can endure. The nature of the injury requires multiple procedures, débridement, surgery and treatments. All of these experiences vary in length and intensity, therefore creating variations in sensation. Additional pain occurs with each skin graft because a new painful donor site is created. In addition, moving, changing position, and receiving occupational and physical therapy cause addition discomfort. This is a constantly changing source of pain throughout the entire healing process, and to the patient it appears to be never ending. Therefore, the pain management plan for any patient needs to be flexible, evaluated regularly using standardized scales, and individualized to meet the patient's needs (Faucher & Furukawa, 2006, Connor-Ballard, 2009a, 2009b).

The American Burn Association's guidelines for pain management are: there must be an organized approach to pain management that addresses background, procedural, and breakthrough pain; the goal should be for patients to be comfortable and alert; and pain needs to be differentiated from anxiety (Faucher & Furukawa, 2006).

Background pain is a continuous level of discomfort even when the patient is inactive or not undergoing any procedures. The goal of treatment is to provide a long-acting analgesic that will provide even coverage for this long-term discomfort. It is helpful to use escalating doses when initiating the medication to reach the level of pain control that is acceptable to the patient. The use of patient-controlled analgesia (PCA) gives control to the patient and achieves this goal. Breakthrough pain is described as acute, intense, and episodic pain. It is generally related to an activity or movement of the affected area. Short-acting agents are used to achieve pain control in addition to the baseline treatment the patient receives for background pain. Procedural pain is discomfort that occurs with procedures such as daily wound treatments, invasive line insertions, physical and occupational therapy. The goal is to plan proper sedation that will place the patient in a state of comfort throughout the procedure. Depending on the agent, an anesthesia provider can be helpful in achievement of pain relief (Faucher & Furukawa, 2006).

Most severe burns are a combination of partial-thickness and full-thickness burns and the depth influences the amount of pain the patient experiences. Superficial and deep partial-thickness burns are very painful because the nerve endings are exposed, resulting in excruciating pain with exposure to temperature, pressure, and movement. In a full-thickness burn the nerve endings are destroyed, and upon admission there is numbness and decreased sensation to the area. Thus, severe injuries are often underestimated by the patient because the pain at that time is minimal. Educating patients and their families about burn pain and its relationship to the depth of injury as well as the pain management plan is an important priority for the nurse.

The pharmacologic treatment for the management of burn pain includes the use of opioids, nonsteroidal anti-inflammatory drugs (NASIDs), anxiolytics, and anesthetic agents. These and other pain management strategies are discussed in Chapter 13. Treatment of anxiety with benzodiazepines is used along with opioids to achieve both a pain-free and anxiety-free experience. The use of anesthetics in a nonoperative setting (ie, moderate sedation) requires administration by qualified personnel. Recent advances include the use of agents with rapid onset and short duration, which have been very effective in pain control during a planned procedure (Faucher & Furukawa, 2006).

Nonpharmacologic pain control can be achieved by using relaxation techniques, distraction, guided imagery, hypnosis, therapeutic touch, humor, music therapy, and more recently virtual reality techniques (see Chapter 13). These techniques can be used either alone or in conjunction with medications to achieve an acceptable level of comfort for the burn patient (Hansen, Gauld, Wathen, et al., 2008).

Nutritional Support

Burn injuries produce profound metabolic abnormalities fueled by the exaggerated stress response to the injury. The body's response has been classified as hyperdynamic, hypermetabolic, and hypercatabolic. Hypermetabolism can affect morbidity and mortality by increasing the risk for infection and slowing the healing rate. Patients' metabolic demands vary with the extent of the burn injury and age (Demling, 2005b). Hypermetabolism is evident immediately after a burn injury. The degree of the response depends on the size of the burn and the patient's age, body composition, size, and genetic response to insult (Jeschke, Chinkes, Finnerty, et al., 2008). Persistent hypermetabolism may last up to 1 year after burn injury.

Major metabolic abnormalities after a burn injury include increased catabolic hormones (cortisol and catechols); decreased anabolic hormones (human growth factor and testosterone); a marked increase in the metabolic rate; a sustained increase in body temperature; a marked increase in glucose demands; rapid skeletal muscle breakdown with amino acids serving as the energy source; lack of ketosis, indicating that fat is not a major source of calories; and catabolism that does not respond to nutrient intake (Pereira, Murphy & Herndon, 2005). Therefore, it is essential to control the stress response by increasing the anabolic process through adequate nutrition and increased muscle activity, decreasing heat loss from wounds, and maintaining a warm environment. Controlling secondary stressors, such as pain and anxiety, also helps control the stress response.

The most important nutritional intervention is to provide energy and nutrients for prevention of infection and promotion of wound healing (Wolfe, 2007). Healing of the burn wound consumes large quantities of energy. Patients with burns greater than 40% TBSA have resting metabolic rates twice that of normal (Pereira, et al., 2005). Effective nutrition management depends on how well the energy

expenditure due to the burn injury can be estimated and matched with appropriate amounts of micronutrients, carbohydrates, lipids, and protein. The nutritional support required is based on the patient's preburn status and the TBSA burned.

Several formulas exist for estimating the daily metabolic expenditure and caloric requirements of patients with burn injuries. The most commonly used formulas are the Harris-Benedict equation, which determines basal energy requirements based on activity and burn size, Ireton-Jones formula, and the Modified Schoefield (Masters & Wood, 2008). Protein requirements may range from 2.0 to 3.0 g of protein per kilogram of body weight every 24 hours, which is 15% to 25% of the caloric intake. Although variation exists, most burn centers use a low fat and high carbohydrate enteral feeding, approximately 55% to 85% carbohydrate and 3% to 20% fat. Carbohydrates are included to meet caloric requirements and to spare protein, which is essential for wound healing. The patient may also receive added vitamins and minerals in excess of the normal requirements (Masters & Wood, 2008).

Feeding usually begins immediately or at least within 24 to 72 hours postburn injury. Nutrition can be administered either by the enteral or parenteral route, or a combination of both. The goal is to reach the patient's nutritional needs, which are usually in proportion to the burn size. These feedings are continued until the patient can adequately consume the recommended daily requirements by mouth (Wolfe, 2007). When the oral route is used, high-protein, high-calorie meals and supplements are given. Dietary consultations are useful in helping patients meet their nutritional needs. Daily calorie counts aid in assessing the adequacy of nutritional intake.

Patients lose a great deal of weight during recovery from severe burns. Reserve fat deposits are catabolized, fluids are lost, and caloric intake may be limited. Because a burn injury decreases the patient's resistance to infection and disease, the nutritional status must be improved and maintained even though the patient has a poor appetite and is weak. One goal of nutrition management is to decrease or stop the catabolic process and promote protein anabolism.

In addition, research is focused on aggressive alteration of the hyperglycemic response and administration of insulin therapy to promote wound healing. Other treatment modalities include early excision and skin grafting of the burn wound, aggressive prevention or treatment of infections, and adequate exercise with physical therapy to lessen muscle wasting and increase strength. Additional pharmacologic modalities used to alter the hypermetabolic state of burn injury include the use of oxandrolone (Oxandrin), an anabolic steroid; an adrenergic antagonist (propranolol [Inderal]); and the anabolic protein, recombinant human growth hormone (Pereira, et al., 2005).

Indications for parenteral nutrition include weight loss greater than 10% of normal body weight, inadequate intake of enteral nutrition due to clinical status, prolonged wound exposure, and malnutrition or debilitated condition before injury. The risk of infection at the site of the central venous catheter required for parenteral nutrition must be considered.

Nursing Management

Continued assessment of the patient during the early weeks after the burn injury focuses on hemodynamic alterations, wound healing, pain and psychosocial responses, and early detection of complications. Assessment of respiratory and fluid status remains the highest priority for detection of potential complications.

The nurse assesses vital signs frequently. Continued assessment of peripheral pulses is essential for the first few postburn days while edema continues to increase, potentially damaging peripheral nerves and restricting blood flow. Close observation of the hourly fluid intake and urinary output as well as blood pressure and cardiac rhythm is essential during this phase and changes should be reported to the burn surgeon promptly.

The patient with an inhalation injury will require regular monitoring of level of consciousness, pulmonary function, and ability to ventilate. When inadequate ventilation and airway edema require the patient to be intubated and placed on a ventilator, frequent suctioning and assessment of the airway are priorities.

Restoring Normal Fluid Balance

To reduce the risk of fluid overload and consequent heart failure and pulmonary edema, the nurse closely monitors IV and oral fluid intake, using IV infusion pumps to minimize the risk of rapid fluid infusion. To monitor changes in fluid status, careful intake and output and daily weights are obtained. Changes, including those of blood pressure and pulse rate, are reported to the physician (invasive hemodynamic monitoring is avoided because of the high risk of infection).

Preventing Infection

A major part of the nurse's role during the acute phase of burn care is detection and prevention of infection. The nurse is responsible for providing a clean and safe environment and for closely scrutinizing the burn wound to detect early signs of infection. Culture results and white blood cell counts are monitored.

Aseptic technique is used for wound care procedures. Sterile technique is used for any invasive procedures, such as insertion of IV lines and urinary catheters or tracheal suctioning. Meticulous hand hygiene before and after each patient contact is also an essential component of preventing infection, even though gloves are worn to provide care.

The nurse protects the patient from sources of contamination, including other patients, staff members, visitors, and equipment. Invasive lines and tubing must be routinely changed according to recommendations of the CDC. Tube feeding reservoirs, ventilator circuits, and drainage containers are replaced regularly. Fresh flowers, plants, and fresh fruit baskets are not permitted in the patient's room because of the risk of microorganism growth. Visitors are screened to avoid exposure of the immunocompromised patient to pathogens.

Patients can inadvertently promote migration of microorganisms from one burned area to another by touching their wounds or dressings. Bed linens also can spread infection through either colonization with wound microorgan-

isms or fecal contamination. Regular bathing of unburned areas and changing of linens can help prevent infection.

Maintaining Adequate Nutrition

Oral fluids should be initiated slowly after bowel sounds resume. The patient's tolerance is recorded. If vomiting and distention do not occur, fluids may be increased gradually and the patient may be advanced to a normal diet or to tube feedings.

The nurse collaborates with the dietitian or nutrition support team to plan a protein- and calorie-rich diet that is acceptable to the patient. Family members may be encouraged to bring nutritious and favorite foods to the hospital. High-calorie nutritional supplements such as Ensure and Resource may be provided. Caloric intake must be documented. Vitamin and mineral supplements may be prescribed.

If caloric goals cannot be met by oral feeding, a feeding tube is inserted and used for continuous or bolus feedings of specific formulas. The volume of residual gastric secretions should be checked to ensure absorption.

The patient should be weighed each day and the results graphed. The patient can use this information to set goals for nutritional intake and to monitor weight loss and gain. Ideally, the patient will lose no more than 5% of preburn weight if aggressive nutritional management is implemented.

Promoting Skin Integrity

Wound care is usually the single most time-consuming element of burn care after the emergent phase. The physician prescribes the desired topical antibacterial agents and specific biologic, biosynthetic, or synthetic wound coverings and plans for surgical excision and grafting. The nurse needs to make astute assessments of wound status, use creative approaches to wound dressing, and support the patient during the emotionally distressing and very painful experience of wound care.

Assessment of the burn wound requires an experienced eye, hand, and sense of smell. Important wound assessment features include size, color, odor, eschar, exudate, epithelial buds (small pearl-like clusters of cells on the wound surface), bleeding, granulation tissue, the status of graft take, healing of the donor site, and the condition of the surrounding skin. Any significant changes in the wound are reported to the physician because they usually indicate burn infection and require immediate intervention.

A diagram, updated daily by the nurse responsible for the patient's care, helps inform all those concerned about the latest wound care procedures in use for the patient.

The nurse also assists the patient and family by providing instruction, support, and encouragement to take an active part in dressing changes and wound care when appropriate. Discharge planning needs for wound care are anticipated early in the course of burn management, and the strengths of the patient and family are assessed and used in preparing for the patient's eventual discharge and home care.

Relieving Pain and Discomfort

Pain measures are continued during the acute phase of burn recovery. Analgesic agents and anxiolytic medications are administered as prescribed. Frequent assessment of pain and discomfort is essential. To increase its effectiveness, analgesic medication is provided before the pain becomes severe. Nursing interventions such as teaching the patient relaxation techniques, giving the patient some control over wound care and analgesia, and providing frequent reassurance are helpful. Guided imagery and distraction (eg, video programs or video games) can be used to alter the patient's perceptions of and responses to pain. Other pain-relieving approaches include hypnosis, music therapy, and virtual reality.

The nurse assesses the patient's sleep patterns daily. Lack of sleep and rest interferes with healing, comfort, and restoration of energy. If necessary, sedatives are prescribed on a regular basis in addition to analgesics and anxiolytics.

The nurse works quickly to complete treatments and dressing changes to reduce pain and discomfort. The patient is encouraged to take analgesic medications before painful procedures. The patient's response to the medication and other interventions is assessed and documented.

Healing burn wounds are typically described by patients as itchy and tight. Oral antipruritic agents, a cool environment, frequent lubrication of the skin with water or a silica-based lotion, exercise and splinting to prevent skin contracture, and diversional activities all help promote comfort in this phase.

Promoting Physical Mobility

An early priority is to prevent complications of immobility. Deep breathing, turning, and proper positioning are essential nursing practices that prevent atelectasis and pneumonia, control edema, and prevent pressure ulcers and contractures. These interventions are modified to meet the patient's needs. Low-air-loss and rotation beds may be useful, and early sitting and ambulation are encouraged. If the lower extremities are burned, elastic pressure bandages should be applied before the patient is placed in an upright position. These bandages promote venous return and minimize edema formation. Prevention of deep vein thrombosis (DVT) is an important factor in care. Patients with burn injuries are at high risk because of their hypercoagulability, loss of vascular integrity, immobility, multiple invasive lines and need for other operative procedures. In fact, there is a 1% to 23% incidence of DVT in burn patients documented. There is cautious use of heparin due to the bleeding potential; however, most burn centers use prophylactic therapy including sequential graduated compression devices in the high-risk groups (Faucher & Conlon, 2007).

The burn wound is in a dynamic state for at least 1 year after wound closure. During this time, aggressive efforts must be made to prevent contracture and hypertrophic scarring. Both passive and active range-of-motion exercises are initiated from the day of admission and are continued after grafting, within prescribed limitations. Splints or functional devices may be applied to the extremities for contracture control. The nurse monitors the splinted areas for signs of vascular insufficiency, nerve compression, and skin breakdown. Occupational and physical therapists are consulted to develop a patient-specific plan of care throughout hospitalization and recovery.

Strengthening Coping Strategies

In the acute phase of burn care, the patient is facing the reality of the burn injury and is grieving over obvious losses. Depression, anger, regression, and manipulative behavior

are common responses of patients who have burn injuries. Withdrawal from participation in required treatments and regression must be viewed with an understanding that such behavior may help the patient cope with an enormously stressful event. Although most patients recover emotionally from a burn injury, some have more difficult psychological reactions to the injury and its outcomes (Kildal, Willebrand, Andersson, et al., 2004). There is evidence that psychological distress and depression are common in people who have experienced burns; however, more studies are needed in this area (Fauerbach, Pruzinsky & Saxe, 2007).

Difficulty coping along with other psychological stressors often limits the patient's physical and psychological recovery (Fauerbach, Lezotte, Hills, et al., 2005). Patients who experience a burn injury tend to have high rates of involvement in risky behaviors (eg, alcohol and substance abuse, depression) before the injury (Appleby, 2005). Intrusive thoughts of the burn event and reliving it over and over may also occur and can indicate posttraumatic stress disorder (PTSD).

Much of the patient's energy goes into maintaining vital physical functions and wound healing in the early postburn weeks, leaving little emotional energy for coping in a more effective manner. The nurse can assist the patient to develop effective coping strategies by setting specific expectations for behavior, promoting truthful communication to build trust, helping the patient practice appropriate strategies, and giving positive reinforcement when appropriate.

The patient frequently vents feelings of anger. At times the anger may be directed inward because of a sense of guilt, perhaps for causing the fire or even for surviving when loved ones perished. The anger may be directed outward toward those who escaped unharmed or toward those who are now providing care. One way to help the patient handle these emotions is to enlist someone to whom the patient can vent feelings without fear of retaliation. A nurse, social worker, psychiatric liaison nurse, or spiritual advisor or counselor who is not involved in direct care activities may fill this role successfully.

Patients with burn injuries are very dependent on health care team members during the long period of treatment and recovery. However, even when physically unable to contribute much to self-care, they should be included in decisions regarding care and encouraged to assert their individuality in terms of preferences and recognition of their unique identities. As the patient improves in mobility and strength, the nurse works with the patient to set realistic expectations for self-care, including self-feeding, assistance with wound care procedures, exercise, and planning for the future. Many patients respond positively to the use of contractual agreements and other strategies that recognize their independence and their specific role as part of the health care team moving toward the goal of self-care. Consultation with psychiatric/mental health care providers may be helpful to assist the patient in developing effective coping strategies.

Supporting Patient and Family Processes

Family functioning is disrupted with burn injury. One of the nurse's responsibilities is to support the patient and family and to address their spoken and unspoken concerns. Family members need to be instructed about ways that they can support the patient as adaptation to burn trauma occurs. The family also needs support from the health care team. The burn injury has tremendous psychological, economic, and practical impact on the patient and family. Referrals for social services or psychological counseling should be made as appropriate. This support continues into the rehabilitation phase. Some burn centers offer a peer support program that involves a burn survivor visiting the patient while hospitalized to provide support. Many survivors enjoy the opportunity to help others through this experience.

Patients who experience major burns are commonly sent to burn centers far from home. Because burn injuries are sudden and unexpected, family roles are disrupted. Therefore, both the patient and the family need thorough information about the patient's burn care and expected course of treatment. Patient and family education begins at the initiation of burn management. Barriers to learning are assessed and considered in teaching. The preferred learning styles of both the patient and family are assessed. This information is used to tailor teaching activities. The nurse assesses the ability of the patient and family to grasp and cope with the information. Verbal information is supplemented with videos, models, or printed materials if available. Patient and family education is a priority in the acute and rehabilitation phases.

Nurses must remain sensitive to the possibility of changing family dynamics. It is not unusual for the provider in the family to be the one who is injured. Roles begin to change, which adds more stress to the family. In addition, families are often relocated due to loss of property from the fire. Social services play an integral part in providing support at this time.

Monitoring and Managing Potential Complications

Heart Failure and Pulmonary Edema

The patient is assessed for fluid overload, which may occur as fluid is mobilized from the interstitial compartment back into the intravascular compartment. If the cardiac and renal systems cannot compensate for the excess vascular volume, heart failure and pulmonary edema may result. The patient is assessed for signs of heart failure, including decreased cardiac output, oliguria, jugular vein distention, edema, and the onset of an S_3 or S_4 heart sound. If invasive hemodynamic monitoring is used, increasing central venous, pulmonary artery, and wedge pressures indicate increased fluid volume.

Crackles in the lungs and increased difficulty with respiration may indicate a fluid buildup in the lungs, which is reported promptly to the physician. In the meantime, the patient is positioned comfortably, with the head of the bed raised (if not contraindicated because of other treatments or injuries) to promote lung expansion and gas exchange. Management of this complication includes providing supplemental oxygen, administering IV diuretic agents, carefully assessing the patient's response, and providing vasoactive medications, if indicated.

Sepsis

The signs of early systemic sepsis are subtle and require a high index of suspicion and very close monitoring of changes in the patient's status. Early signs of sepsis may include increased temperature, increased pulse rate,

widened pulse pressure, and flushed dry skin in unburned areas. As with many observations of the patient with a burn injury, one needs to look for patterns or trends in the data. (See Chapter 15 for a more detailed discussion of septic shock.)

Wound and blood cultures are performed as prescribed, and results are reported to the physician immediately. The nurse also observes for and reports early signs of sepsis and promptly intervenes, administering prescribed IV fluids and antibiotics to prevent septic shock, a complication with a high mortality rate. Antibiotics must be administered as scheduled to maintain proper blood concentrations. Serum antibiotic levels are monitored for evidence of maximal effectiveness, and the patient is monitored for toxic side effects.

Acute Respiratory Failure and Acute Respiratory Distress Syndrome

The patient's respiratory status is monitored closely for increased difficulty in breathing, change in respiratory pattern, or onset of adventitious (abnormal) sounds. Typically, at this stage, signs and symptoms of injury to the respiratory tract become apparent. Respiratory failure may follow. As described previously, signs of hypoxia (decreased oxygen to the tissues), decreased breath sounds, wheezing, tachypnea, stridor, and sputum tinged with soot (or in some cases containing sloughed tracheal tissue) are among the many possible findings. Patients receiving mechanical ventilation must be assessed for a decrease in tidal volume and lung compliance. The key sign of the onset of ARDS is hypoxemia while receiving 100% oxygen, with decreased lung compliance and significant shunting. The physician should be notified immediately of deteriorating respiratory status.

Medical management of the patient with acute respiratory failure requires intubation and mechanical ventilation (if not already in use). If ARDS has developed, higher oxygen levels, positive end-expiratory pressure, and pressure support are used with mechanical ventilation to promote gas exchange across the alveolar–capillary membrane (see Chapter 25).

Visceral Damage. The nurse must be alert to signs of necrosis of visceral organs due to electrical injury. Tissues affected are usually located between the entrance and exit wounds of the electrical burn. All patients with electrical burns should undergo cardiac monitoring, with dysrhythmias being reported to the physician. Careful attention must also be paid to signs or reports of pain related to deep muscle ischemia. To minimize the severity of complications, visceral ischemia must be detected as early as possible. In the operating room, the burn surgeon may perform **fasciotomies** to relieve the swelling and ischemia in the muscles and fascia and to promote oxygenation of the injured tissues. Because of the deep incisions involved with fasciotomies, the patient must be monitored carefully for signs of excessive blood loss and hypovolemia.

Gerontologic Considerations

In elderly patients, a careful history of preburn medications and preexisting illnesses is essential. Nursing assessment of the elderly patient with burns should include particular attention to pulmonary function, response to fluid resuscitation, and signs of mental confusion or disorientation. Fever may be absent in the presence of complications such as sepsis. Therefore, surveillance for other signs of infection becomes even more important. Nursing care of the elderly patient with burn injuries promotes early mobilization, aggressive pulmonary care, and attention to preventing complications.

REHABILITATION PHASE

Rehabilitation begins immediately after the burn has occurred and often extends for years after injury. The emphasis on early rehabilitation cannot be overestimated. In this final phase of care, the focus becomes rehabilitation, reconstruction, and reintegration of the burn survivor (Sheridan, 2007b). In addition, the burn team focuses on late complications (Table 57-6).

Burn rehabilitation is time-consuming and challenging and is very specific to the severity and location of injury as well as the patient's needs and goals. These goals vary based on phase of care and need to be addressed frequently to ensure constant progress. During hospitalization the goals include maintaining range of motion (ROM), preventing contractures through splinting techniques, decreasing edema, and the preventing skin breakdown through proper positioning. As the acute phase comes to a close, patients become more aware of their injuries and the challenges they face. The goals are functional and aimed at activities of daily living such as ambulation and participation in self-care as well as scar management and returning to work or school (Chart 57-6). Occupational and physical therapists are essential to optimizing patient goals and outcomes (Sheridan, 2007b).

Psychological Support

A patient's outlook, motivation, and support system are important to his or her overall well-being and ability to progress through the rehabilitation phase. There are three basic phases of psychological recovery from a burn injury. During the critical phase, patients often are confused from medication they are taking, but they have an underlying sense of fear, anxiety, and pain. In the acute phase, patients recognize that survival is expected. They have periods of depression due to their awareness of the functional and body image challenges ahead and can recognize all they have lost. Thirty percent of burn patients develop some form of PTSD. The symptoms and psychological responses to traumatic events including PTSD are discussed further in Chapter 7.

The final stage of psychological recovery occurs within 1 to 2 years following discharge. This is an emotional time as the patient and family begin to live with new physical limitations and challenges in relationships. The role of various team members and the support from peer burn survivors during this time cannot be understated (Sheridan, 2007b).

Psychological treatment plans should include a full assessment of these issues and a targeted plan with the appropriate resources to promote the patient's social and vocational reintegration and improved quality of life (Wallis, Renneberg, Ripper, et al., 2006). Burn injuries can have a major impact

Table 57-6 COMPLICATIONS IN REHABILITATION PHASE OF BURN CARE

Complications	Contributing Factors	Interventions
Neuropathies, peripheral neuropathies, mononeuropathies, multimononeuropathies, nerve entrapment	Electrical injury, large deep burns, improper positioning, edema, scar tissue	Assess peripheral pulses and sensation (neurovascular checks). Prevent edema and pressure by elevation, positioning, and prevention of constricting dressings. Assess splints for proper fit and application. Consult occupational therapy (OT) and physical therapy (PT) for positioning.
Heterotopic ossification (abnormal formation of bone in response to soft tissue trauma)	Prolonged immobility	Perform gentle range-of-motion exercises.
Hypertrophic scarring	Partial-thickness and full-thickness burns	Keep skin pliable and soft. Apply pressure garments as prescribed. Massage.
Contractures	Partial-thickness and full-thickness burns	Maintain position of joints in alignment. Perform gentle range of motion exercises. Consult OT and PT for exercises and positioning recommendations.
Wound breakdown	Shearing, pressure, inadequate nutrition	Teach patient about importance of good nutrition. Protect wound from pressure and shearing forces.
Gait deviations	Pain, burn wound, donor site, scarring of joints, electrical injury of the brain	Provide adequate pain management. Consult OT and PT. Promote ambulation and mobility training.
Complex regional pain syndrome (previous reflex sympathetic dystrophy [RSD])	Trauma and burns	Provide adequate pain management. Consult OT and PT for exercises. Promote gentle motion of affected extremities.
Joint instability	Burn wound, burn scar and contractures	Maintain joint through appropriate application of splints. Monitor joint pinning if indicated. Consult OT and PT.

on quality of life. Changes in physical activity as well as social and psychological adjustments, such as returning to school and employment status, may be challenging. It is important throughout this process to assess and address the family needs. When one member of a family sustains a major burn injury, the entire family is affected. Separation, feelings of helplessness, loss, and psychological dysfunction may be experienced in varying degrees. Family and friends need support, education, and guidance in assisting the patient to return to their optimal health (Ceranoglu & Stern, 2006).

CHART 57-6 NURSING RESEARCH PROFILE

Life After Burn Injury

Moi, A. L. & Gjengedal, E. (2008). Life after burn injury: Striving for regained freedom. *Qualitative Health Research, 18*(12), 1621–1630.

Purpose

Second only to the patient's survival, the priority of burn care today is optimal quality of life of survivors. The purpose of this study was to identify and describe the meaning of the experience of life after major burn injury.

Design

This qualitative study used a phenomenological perspective to describe and explore the meaning that 14 people who survived severe burn injury attributed to the experience. They were recruited from an outpatient clinic at a burn center in Norway. The researchers intentionally recruited participants of both genders (men = 11, women = 4), across a wide age range (19 to 74, with mean age of 46 years), who had experienced different types of burns (flames = 9, electrical injury = 3, scalding = 2), and received different treatments of their burns. In-depth unstructured interviews were conducted 10 to 35 months after the burn injury; the interviews were audiotaped, transcribed, and analyzed using Giorgi's phenomenological method.

Findings

The major finding was the effort on the part of participants to regain freedom that included reduced or absent bodily or social restrictions and a meaningful life that was the same as or better than before the injury occurred. The experiences described as supporting this goal included (1) facing the extreme and trying to restore order and minimize damage, (2) having a disrupted life history with the loss of memory that occurred during the immediate postburn period, (3) accepting the unchangeable, and (4) changing what could be changed. Some participants indicated that experiencing and surviving their injury gave them a new view of life and made their lives richer.

Nursing Implications

The experience of surviving a severe burn injury is life-altering. The researchers suggest that patients should be given the opportunity to tell their stories and to express their views. Positive feelings and growth on the part of patients should be recognized by burn-care staff, particularly during the later phases of burn care. Patients should be encouraged to share their experiences and views. The researchers also suggest that patients and families be provided with information about what to expect as they move through the phases of burn care.

Abnormal Wound Healing

Partial-thickness wounds involving the epidermis and superficial dermis tend to heal without scarring. However, deep partial-thickness and full-thickness wounds involving the dermis and subcutaneous tissue heal with varying degrees of scarring due to abnormal healing (Arnt, Dover & Alam, 2006).

Normal scarring occurs in a superficial tissue injury and begins forming within 7 to 10 days postinjury and progresses over the next 6 to 12 months. Abnormal scarring occurs after a longer period of wound healing and forms either hypertrophic or keloid scars.

Hypertrophic and Keloid Scars

Hypertrophic scars form within the boundaries of the initial wound and push outward on the perimeter of the wound. They are common in areas over joints and in the younger population. These scars may be hypopigmented or hyperpigmented (Arnt, et al., 2006).

The scar becomes red (because of its hypervascular nature), raised, and hard. A keloid is an irregularly formed scar that extends beyond the margins of the original wound. They are large, nodular, and ropelike, often causing itching and tenderness. They are more common in dark pigmented skin, uncommon in children and the elderly, and have familial tendencies. Scars occur in all forms and arise in different areas, making some more undesirable than others. Therefore, prevention and treatment is individualized to the patient's needs (Arnt, et al., 2006).

Prevention and Treatment of Scars

Treatment modalities that are theoretically based on wound healing and scar formation are used to prevent scar contractures and excess hypertrophic tissue. Compression is introduced early in burn wound treatment. Elastic bandage wraps are used initially to help promote adequate circulation, but they can also be used as the first form of compression followed by elasticized tubular bandage until the patient can be measured for a customized garment (Fig. 57-7). Tools of therapy include pressure, use of topical silicone, scar massage, and steroid injections (Sheridan, 2007b). Application of elastic pressure garments loosens collagen bundles and encourages parallel orientation of the collagen to the skin surface. As pressure continues over time, there is a restructuring of the collagen and a decrease in vascularity and cellularity. Although this therapy is somewhat controversial, pressure has shown to be beneficial in controlling scar formation over time. Garments are worn continuously (ie, 23 hours a day). Many areas of the body are difficult to compress due to the contours or location of the injury. Silicone sheets are helpful for these small troublesome areas and are placed beneath the garment to enhance scar compression. Gentle superficial scar massage can be performed with a moisturizer several times a day. This is helpful in smaller areas and is convenient for the patient. The use of steroid injections into the scar may be helpful in areas of scar development, but they are difficult and painful. Pruritus is a common discomfort in the healed burn wound and can last up to 6 months after healing has occurred. It is treated with moisturizers, massage, oral and topical antihistamines, and topical compresses or baths. This is a troublesome part of recovery and requires further research (Sheridan, 2007b).

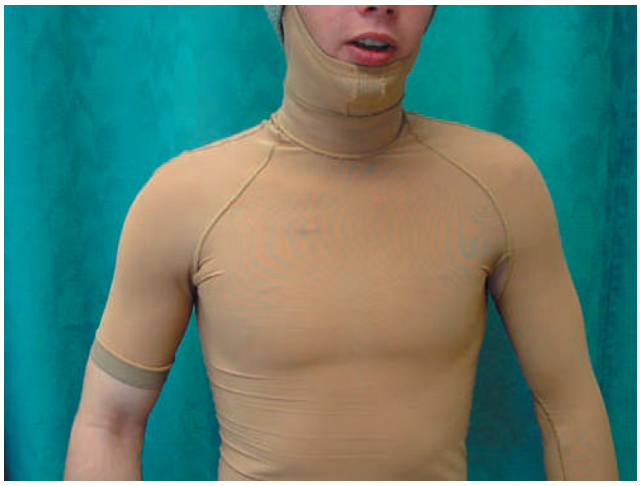

Figure 57-7 Pressure garments. Application of pressure garments helps prevent hypertrophic burn scarring. Used with permission of Jobst Institute, Inc., Toledo, OH.

Burn reconstruction is a treatment option after all scars have matured and is discussed within the first few years after injury. This decision requires individualized planning, realistic expectations, and patience. The procedures utilized by the surgeon include contracture release and skin grafting, use of tissue expansion, and skin flaps to cover or reconstruct the defect area (Sheridan, 2007b).

NURSING PROCESS

Care of the Patient During the Rehabilitation Phase

Assessment

The nurse obtains information about the patient's education level, occupation, leisure activities, cultural background, religion, and family interactions early. The patient's self-concept, mental status, emotional response to the injury and hospitalization, level of intellectual functioning, previous hospitalizations, response to pain and pain relief measures, and sleep pattern are also essential components of a comprehensive assessment. Information about the patient's general self-concept, self-esteem, and coping strategies in the past are valuable in addressing emotional needs.

Ongoing physical assessments related to rehabilitation goals include range of motion of affected joints, functional abilities in activities of daily living, early signs of skin breakdown from splints or positioning devices, evidence of neuropathies (neurologic damage), activity tolerance, and quality or condition of healing skin. The patient's participation in care and ability to demonstrate self-care in such areas as ambulation, eating, wound cleaning, and applying pressure wraps are documented on a regular basis. In addition to these assessment parameters, specific complications and treatments require additional specific assessments; for

example, the patient undergoing primary excision requires postoperative assessment.

Diagnosis

Nursing Diagnoses

Based on the assessment data, priority nursing diagnoses in the long-term rehabilitation phase of burn care may include the following:

- Activity intolerance related to pain on exercise, limited joint mobility, muscle wasting, and limited endurance
- Disturbed body image related to altered physical appearance and self-concept
- Deficient knowledge about postdischarge home care and recovery needs

Collaborative Problems/Potential Complications

Based on the assessment data, potential complications that may develop in the rehabilitation phase include:

- Contractures
- Inadequate psychological adaptation to burn injury

Planning and Goals

The major goals for the patient include increased participation in activities of daily living; increased understanding of the injury, treatment, and planned follow-up care; adaptation and adjustment to alterations in body image, self-concept, and lifestyle; and absence of complications.

Nursing Interventions

Promoting Activity Tolerance

Nursing interventions that must be carried out according to a strict regimen and the pain that accompanies movement take their toll on the patient. The patient may become confused and disoriented and lack the energy or motivation to participate optimally in care. The nurse must schedule care in such a way that the patient has periods of uninterrupted sleep. A good time for planned patient rest is after the stress of dressing changes and exercise, while pain interventions and sedatives are still effective. This plan must be communicated to family members and other care providers.

The patient may have insomnia related to frequent nightmares about the burn injury or to other fears and anxieties about the outcome of the injury. The nurse listens to and reassures the patient and administers hypnotic agents, as prescribed, to promote sleep.

Reducing metabolic stress by relieving pain, preventing chilling or fever, and promoting the physical integrity of all body systems help the patient conserve energy for therapeutic activities and wound healing.

The nurse incorporates physical therapy exercises in the patient's care to prevent muscle atrophy and to maintain the mobility required for daily activities. The patient's activity tolerance, strength, and endurance gradually increase if activity occurs over increasingly longer periods. Fatigue, fever, and pain tolerance are monitored and used to determine the amount of activity to be encouraged on a daily basis. Activities such as family visits and recreational or play therapy (eg, video games, radio, television) can provide diversion, improve the patient's outlook, and increase tolerance for physical activity. In elderly patients and those with chronic illnesses and disabilities, rehabilitation must take into account preexisting functional abilities and limitations.

Improving Body Image and Self-Concept

Patients who have survived burn injuries frequently suffer profound losses. These include not only a loss of body image due to disfigurement but also losses of personal property, homes, loved ones, and ability to work. They lack the benefit of anticipatory grief often seen in a patient who is approaching surgery or dealing with the terminal illness of a loved one.

As care progresses, the patient who is recovering from burns becomes aware of daily improvement and begins to exhibit basic concerns: Will I be disfigured or be disabled? How long will I be in the hospital? What about my job and family? Will I ever be independent again? How can I pay for my care? Was my burn the result of my carelessness?

As the patient expresses such concerns, the nurse must take time to listen and to provide realistic support. The nurse can refer the patient to a support group, such as those usually available at regional burn centers or through organizations such as the Phoenix Society (see Resources at the end of the chapter). Through participation in such groups, the patient will meet others with similar experiences and learn coping strategies to help him or her deal with losses. Interaction with other burn survivors allows the patient to see that adaptation to the burn injury is possible. If a support group is not available, visits from other survivors of burn injuries can be helpful to the patient coping with such a traumatic injury.

A major responsibility of the nurse is to constantly assess the patient's psychosocial reactions. Questions to consider include the following: What are the patient's fears and concerns? Does the patient fear loss of control of care, independence, or sanity itself? Is the patient afraid of rejection by family and loved ones? Does he or she fear being unable to cope with pain or physical appearance? Does the patient have concerns about sexuality, including sexual function? Being aware of these anxieties and understanding the basis of the patient's fears enable the nurse to provide support and to cooperate with other members of the health care team in developing a plan to help the patient deal with these feelings. Journaling can be helpful for patients to express themselves and track their progress with psychological healing.

When caring for a patient with a burn injury, the nurse needs to be aware that there are prejudices and misunderstandings in society about those who are viewed as different. Opportunities and accommodations available to others are often denied those who are disfigured. These include social participation, employment, prestige, various roles, and status. The health care team must actively promote a healthy body image and self-concept in patients with burn injuries so that they can accept or challenge others' perceptions of those who are disfigured or disabled. Survivors themselves must show others who they are, how they function, and how they want to be treated.

The nurse can help patients practice their responses to people who may stare or inquire about their injury once

they are discharged from the hospital. The nurse can help patients build self-esteem by recognizing their uniqueness—for example, with small gestures such as providing a birthday cake, combing the patient's hair before visiting hours, giving information about the availability of a cosmetician to enhance appearance, and teaching the patient ways to direct attention away from a disfigured body to the self within. Consultants such as psychologists, social workers, vocational counselors, and teachers are valuable participants in assisting burn patients to regain their self-esteem.

Monitoring and Managing Potential Complications

CONTRACTURES. With early and aggressive physical and occupational therapy, contractures are rarely a long-term complication. However, surgical intervention is indicated if a full range of motion in the burn patient is not achieved. (See Chapter 11 for a discussion of prevention of contractures.)

IMPAIRED PSYCHOLOGICAL ADAPTATION TO THE BURN INJURY. Some patients, particularly those with limited coping skills or psychological function or a history of psychiatric problems before the burn injury, may not achieve adequate psychological adaptation to the burn injury. Psychological counseling or psychiatric referral may be made to assess the patient's emotional status, to help the patient develop coping skills, and to intervene if major psychological issues or ineffective coping is identified.

Promoting Home and Community-Based Care

TEACHING PATIENTS SELF-CARE. As the inpatient phase of recovery becomes shorter, the focus of rehabilitative interventions is directed toward outpatient care, home care, or care in a rehabilitation center. Throughout the phases of burn care, efforts are made to prepare the patient and family for the care that will continue at home. They are instructed about the measures and procedures that they will need to perform. For example, patients commonly have small areas of clean, open wounds that are healing slowly. They are instructed to wash these areas daily with mild soap and water and to apply the prescribed topical agent or dressing.

In addition to instructions about wound care, patients and families require careful written and verbal instructions about pain management, nutrition, and prevention of complications. Information about specific exercises and use of pressure garments and splints is reviewed with both the patient and the family, and written instructions are provided for their use at home. The patient and family are taught to recognize abnormal signs and report them to the physician. The patient and family are assisted in planning for the patient's continued care by identifying and acquiring supplies and equipment that are needed at home (Chart 57-7).

CONTINUING CARE. Follow-up care after discharge by the multidisciplinary team is necessary. Patients who receive care in a burn center usually return to the burn clinic or center periodically for evaluation by the burn team, modification of home care instructions, and planning for reconstructive surgery. Other patients receive ongoing care from the burn surgeon who cared for them during the acute phase of their management. Still other patients require the services of a rehabilitation center and may be transferred to such a center for aggressive rehabilitation before going home. Many patients require outpatient physical or occupational therapy, often several times weekly. It is often the nurse who is responsible for coordinating all aspects of care and ensuring that the patient's needs are met. Such coordination is an important aspect of assisting the patient to achieve independence.

Patients who return home after a severe burn injury, those who cannot manage their own burn care, and those with inadequate support systems need referral for home care. During visits to the patient at home, the home care nurse assesses the patient's physical and psychological status as well as the adequacy of the home setting for safe and adequate care. The nurse monitors the patient's progress and adherence to the plan of care and notes any problems that interfere with the patient's ability to carry out the care. During the visit, the nurse assists the patient and family with wound care and exercises. Patients with severe or persistent depression or difficulty adjusting to changes in their social or occupational roles are identified and referred to the burn team for possible referral to a psychologist, psychiatrist, or vocational counselor.

The burn team or home care nurse identifies community resources that may be helpful for the patient and family. Several burn patient support groups and other organizations throughout the United States offer services for burn survivors. They provide contact with caring people (often people who have themselves recovered from burn injuries) who can visit the patient in the hospital or home or telephone the patient and family periodically to provide support and counseling about skin care, cosmetics, and problems related to psychosocial adjustment. Such organizations, and many regional burn centers, sponsor group meetings and social functions at which outpatients are welcome. Some also provide reentry programs or burn retreats and are active in burn prevention activities.

Evaluation

Expected Patient Outcomes

Expected patient outcomes may include the following:

1. Demonstrates activity tolerance required for desired daily activities
   a. Obtains adequate sleep daily
   b. Reports absence of nightmares or sleep disturbances
   c. Shows gradually increasing tolerance and endurance in physical activities
   d. Can concentrate during conversations
   e. Has energy available to sustain desired daily activities
2. Adapts to altered body image
   a. Verbalizes accurate description of alterations in body image and accepts physical appearance
   b. Demonstrates interest in resources that may improve body appearance and function
   c. Uses cosmetics, wigs, and prostheses as desired to achieve acceptable appearance
   d. Socializes with significant others, peers, and usual social group

CHART 57-7

HOME CARE CHECKLIST
The Patient With a Burn Injury

At the completion of the home care instruction, the patient or caregiver will be able to:	PATIENT	CAREGIVER
Mental Health		
Identify strategies to promote own mental health; for example:		
• Remember that changes in lifestyle take time.	✔	✔
• Resume previous interests and activities gradually.	✔	
• Take one day at a time to regain physical and mental strength.	✔	
• Be aware of own feelings and fears and discuss them with selected others.	✔	✔
• Expect concerns, frustrations, and depression about changes in appearance.	✔	✔
• Be honest with self, family, and friends about needs, hopes, and fears.	✔	✔
• Realize that emotional adjustment to the burn injury will occur with time.	✔	✔
Burn Skin Precautions and Wound Care		
Identify the following skin precautions and wound care:		
• Wear sun block with the highest SPF possible to protect burned skin from the sun.	✔	
• Avoid further trauma to burned skin; leave unbroken blisters that may form.	✔	✔
• Lubricate healed burned skin with mild lotion (as prescribed); avoid scratching.	✔	
• Wear wide-brimmed hats if face has been burned to protect the area from the sun.	✔	
• Use only mild soap and lotion (ie, products without perfume) on burned areas.	✔	✔
Exercise		
Describe the following guidelines for exercise:		
• Do as much for self as possible.	✔	
• Adhere to the exercise regimen given by the therapist.	✔	
• Participate in exercise every day, several times a day, even when "not feeling like it."	✔	
Nutrition		
Identify the following guidelines for nutrition:		
• Eat a diet high in calories and protein.	✔	
• Drink adequate volume of fluids to prevent constipation associated with use of analgesic medications.	✔	
Pain Management		
Describe the following steps for managing pain:		
• Avoid situations that require alertness (analgesic agents may produce drowsiness).	✔	
• Take analgesic medication as prescribed (30 minutes before painful procedures such as dressing changes).	✔	
• Use relaxation and distraction to relieve pain and discomfort.	✔	
Thermoregulation		
Identify strategies to compensate for inability to regulate body temperature:		
• Dress to accommodate cold and hot weather or environment.	✔	
• Avoid extremes of temperature.	✔	

CHART 57-7 HOME CARE CHECKLIST

The Patient With a Burn Injury (Continued)

	PATIENT	CAREGIVER
Clothing Considerations		
State the following strategies in selection of clothing to wear:		
• Avoid tight clothing over burned areas.	✔	
• Select white cotton, loose-fitting clothing so that dyes in colored clothes do not irritate healing skin.	✔	
• Wear clothing and gloves to protect healing skin from unnecessary bruises, bumps, and scratches.	✔	
Management of Burn Scar		
Describe the following strategies to manage burn scar:		
• Massage and stretch skin to maintain/increase its elasticity.	✔	✔
• Use lotion for massage as recommended by therapist.	✔	✔
• Wear compression garments 23 hours a day.	✔	
Resumption of Sexual Relations		
Identify the following guidelines regarding resumption of sexual relationships:		
• Realize that resumption of sexual relationships is the rule rather than the exception.	✔	✔
• Expect sensitivity of and around the genital area for several months if these areas were burned.	✔	
• Resume sexual activity slowly; endurance will increase with time.	✔	

Adapted with permission from Orlando Regional Medical Center Burn Unit's Personal Guide to Burn Care.

e. Seeks and achieves return to role in family, school, and community as a contributing member

3. Demonstrates knowledge of required self-care and follow-up care
   a. Describes surgical procedures and treatments accurately
   b. Verbalizes detailed plan for follow-up care
   c. Demonstrates ability to perform wound care and prescribed exercises
   d. Returns for follow-up appointments as scheduled
   e. Identifies resource people and agencies to contact for specific problems
4. Exhibits no complications
   a. Demonstrates full range of motion
   b. Shows no signs of withdrawal or depression
   c. Displays no psychotic behaviors

Outpatient Burn Care

Increasing numbers of patients receive treatment of burns in outpatient settings in an effort to coordinate specific burn care needs and to decrease healthcare costs and length of hospitalization. The increased availability of outpatient surgery and access to expert burn care in outpatient settings make this option possible for the treatment of minor burn care as well as a destination for the discharged burn patient.

The goals for treatment in an outpatient setting may include burn wound management, pain management, scar and reconstructive care, and rehabilitation. However, a number of factors must be considered in determining if outpatient care is appropriate for the patient: age, past medical history, the extent and depth of the burn, location of the burn wounds, the availability of family support systems and community resources, the patient's compliance and the distance from home, and availability of transportation from home to the outpatient setting.

The frequency of follow-up visits is individualized and based on these factors. The initial outpatient visit for a discharged burn patient is usually scheduled within 2 or 3 days after hospital discharge and then biweekly until there is evidence of a successful outcome. After healing has occurred, appointments are monthly and eventually every 4 to 6 weeks for continued assessment of pain, physical limitations, and scar maturation. Patient and family education is very important and should include verbal and written instructions as well as return demonstration of the burn or scar care required. These include the wound treatment, pain management, treatment for itching, provision of adequate nutrition, and promotion of exercise and rest. Instruction is also provided about the signs and symptoms of infection that should be reported to the burn team. The importance of notifying the outpatient setting about early complications and of keeping follow-up appointments is emphasized to the patient and family. Physical therapy and occupational therapy are often provided in the outpatient

burn setting. The rehabilitation goals are to increase range of motion and to strengthen and build the patient's endurance. This is accomplished with a specific plan of care and includes routine visits for up to 2 years following the injury. Adaptation to lifestyle changes and emotional status should be assessed during the outpatient visits and proper referrals made for counseling services. These assessments are difficult to recognize due to the infrequent nature of the visits, and, therefore, it is helpful to incorporate family response and interactions into the assessment. The health care team must also be alert to issues of substance abuse, safety concerns, suicidal thoughts, depression, and PTSD.

CRITICAL THINKING EXERCISES

1 A 35-year-old woman was scalded in the bathtub, where she sustained 40% full-thickness burns to her lower legs, right arm, and back. It is not known how long the woman was in the tub. She apparently had a seizure while showering and fell onto the hot water faucet. On admission to the emergency department, the woman's temperature is 35.5°C (94°F) and her weight is 111 lb (50 kg). She has diabetes as well as a history of uncontrolled seizure activity. What are the priorities in her medical and nursing care during the emergent phase of burn care? What assessment parameters would you monitor closely?

2 An 82-year-old man who is wheelchair dependent and has a history of chronic obstructive pulmonary disease was smoking while using oxygen at home. He sustained superficial partial-thickness burns to his face, including his nose, lips, and chin. This is his second admission for the same type of injury in less than 1 year. His pulse oximetry is 91% and his vital signs are stable. Before he is intubated in the emergency room, he asks for a cigarette and states he wants to go home. What are this patient's immediate care needs? What referrals for inpatient services should be arranged before his discharge? What important factors need to be addressed as part of his discharge plan?

3 A 19-year-old, 233 lb (105 kg) man sustained partial-thickness and full-thickness burn injuries to his face, neck, and both hands and forearms circumferentially that occurred while he was working on his car while smoking a cigarette. Using the rule of nines, estimate the percentage of TBSA burned and estimate his fluid resuscitation needs. What immediate concerns would you have for his airway? How would you handle the care of the circumferential injury of his forearms?

EBP **4** A 52-year-old man suffered an electrical burn when he touched a high-voltage wire inside a closet while on the job. There was an explosion and he was thrown backward. The current entered his right hand and exited his left knee, leaving a large deficit in his knee. He also sustained a 45% TBSA flame burn when his clothing ignited. When asked to rate the intensity of his pain, he reports a "10" on a 10-point pain scale. What immediate concerns would you have related to his cardiopulmonary and neurological status? What strategies would you use to relieve his pain? What is the evidence that supports the pain relief strategies that you identified and the strength of that evidence?

5 A 28-year-old woman involved in a house fire is brought to the emergency department by her boyfriend. There is no information from the scene. She is complaining of severe pain in her neck and back. She sustained a full-thickness burn of her lower extremities and her lower back. She is asking about the status of her two children who perished in the fire. What would you be concerned about related to her complaints that requires action from the burn team? What are the psychological and emotional issues that need to be addressed? Who might you consult to assist in the psychological management of this patient?

The Smeltzer suite offers these additional resources to enhance learning and facilitate understanding of this chapter:

- thePoint online resource, thepoint.lww.com/Smeltzer12E
- Student CD-ROM included with the book
- *Study Guide to Accompany Brunner & Suddarth's Textbook of Medical-Surgical Nursing*
- *Handbook for Brunner & Suddarth's Textbook of Medical-Surgical Nursing*

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RESOURCES

Alisa Ann Ruch Burn Foundation, www.aarbf.org
American Burn Association, www.ameriburn.org
American Red Cross, www.redcross.org
Burn Children Recovery Foundation, www.burnchildrenrecovery.org
Burn Foundation, www.burnfoundation.org/
Burn Institute, www.burninstitute.org
Burn Prevention, www.burnprevention.org
Chemical Educational Foundation, www.chemed.org
Firefighters Pacific Burn Institute, www.ffburn.org
Integra Life Sciences Corporation, www.integra-ls.com
International Association of Fire Fighters Burn Foundation, www.iaff.org
International Medical Education Foundation. www.burnsurgery.org
International Society for Burn Injuries, www.worldburn.org
National Burn Center Reporting System Report Form, U.S. Consumer Product Safety Commission, www.cpsc.gov/burnctr.html
National Fire Protection Association Fire, www.nfpa.org
Phoenix Society for Burn Survivors, Inc., www.phoenix-society.org
United States Fire Administration, www.usfa.dh.gov

unit 13

Sensorineural Function

Case Study • Applying Concepts From NANDA, NIC, and NOC

A Patient With Impaired Vision and Decreased Attention to One Side of the Body

Mr. Martin is a 60-year-old man who has had several strokes. Ophthalmologic testing reveals that he has homonymous hemianopsia of the left visual field and visual spatial neglect; as a result, he has limited vision in the left visual fields of both eyes. He has difficulty in many areas, such as bumping into objects and ignoring the left side of his body.

Visit thePoint to view a concept map that illustrates the relationships that exist between the nursing diagnoses, interventions, and outcomes for the patient's clinical problems.

chapter 62

Management of Patients With Cerebrovascular Disorders

LEARNING OBJECTIVES

On completion of this chapter, the learner will be able to:

1. Describe the incidence and social impact of cerebrovascular disorders.
2. Identify the risk factors for cerebrovascular disorders and related measures for prevention.
3. Compare the various types of cerebrovascular disorders: their causes, clinical manifestations, and medical management.
4. Apply the principles of nursing management to the care of a patient in the acute stage of an ischemic stroke.
5. Use the nursing process as a framework for care of a patient recovering from an ischemic stroke.
6. Use the nursing process as a framework for care of a patient with a hemorrhagic stroke.
7. Identify essential elements for family teaching and preparation for home care of the patient who has had a stroke.

GLOSSARY

agnosia: failure to recognize familiar objects perceived by the senses
aneurysm: a weakening or bulge in an arterial wall
aphasia: inability to express oneself or to understand language
apraxia: inability to perform previously learned purposeful motor acts on a voluntary basis
dysarthria: defects of articulation due to neurologic causes
expressive aphasia: inability to express oneself; often associated with damage to the left frontal lobe area
hemianopsia: blindness of half of the field of vision in one or both eyes
hemiplegia/hemiparesis: weakness/paralysis of one side of the body, or part of it, due to an injury in the motor area of the brain
infarction: a zone of tissue deprived of blood supply
Korsakoff's syndrome: disorder characterized by psychosis, disorientation, delirium, insomnia, and hallucinations
penumbra region: area of low cerebral blood flow
receptive aphasia: inability to understand what someone else is saying; often associated with damage to the temporal lobe area

Cerebrovascular disorders is an umbrella term that refers to a functional abnormality of the central nervous system (CNS) that occurs when the normal blood supply to the brain is disrupted. Stroke is the primary cerebrovascular disorder in the United States, and it is the third leading cause of death after heart disease and cancer. Approximately 780,000 people experience a stroke each year in the United States. Approximately 600,000 of these are new strokes, and 180,000 are recurrent strokes (Rosamond, Flegal, Furie, et al., 2008). About 5.6 million noninstitutionalized stroke survivors are alive today; stroke is a leading cause of serious, long-term disability in the United States. The financial impact of stroke is profound, with estimated direct and indirect costs of $65.5 billion in 2008 (Rosamond, et al., 2008).

Strokes can be divided into two major categories: ischemic (85%), in which vascular occlusion and significant hypoperfusion occur, and hemorrhagic (15%), in which there is extravasation of blood into the brain or subarachnoid space (Hinkle & Guanci, 2007). Although there are some similarities between the two broad types of stroke, differences exist in etiology, pathophysiology, medical management, surgical management, and nursing care. Table 62-1 compares ischemic and hemorrhagic strokes.

Ischemic Stroke

An ischemic stroke, cerebrovascular accident (CVA), or "brain attack" is a sudden loss of function resulting from disruption of the blood supply to a part of the brain. The term *brain attack* is being used to suggest to health care practitioners and the public that a stroke is an urgent health care issue similar to a heart attack. With the approval of thrombolytic therapy for the treatment of acute ischemic stroke in 1996 came a revolution in the care of patients after a stroke. Early treatment with thrombolytic therapy for ischemic stroke results in fewer stroke symptoms and less loss of function (National Institute of Neurologic Disorders and Stroke [NINDS], 1995). Currently approved thrombolytic therapy has a treatment window of only 3 hours after the onset of a stroke. Urgency is needed on the part of the public and health care practitioners for rapid transport of the patient to a hospital for assessment and administration of the medication.

Ischemic strokes are subdivided into five different types based on the cause: large artery thrombotic strokes (20%), small penetrating artery thrombotic strokes (25%), cardiogenic embolic strokes (20%), cryptogenic strokes (30%), and other (5%) (see Table 62-1). Large artery thrombotic strokes are caused by atherosclerotic plaques in the large blood vessels of the brain. Thrombus formation and occlusion at the site of the atherosclerosis result in ischemia and **infarction** (deprivation of blood supply) (Hinkle & Guanci, 2007).

Small penetrating artery thrombotic strokes affect one or more vessels and are the most common type of ischemic stroke. Small artery thrombotic strokes are also called lacunar strokes because of the cavity that is created after the death of infarcted brain tissue (American Association of Neuroscience Nurses [AANN], 2008).

Cardiogenic embolic strokes are associated with cardiac dysrhythmias, usually atrial fibrillation. Embolic strokes can also be associated with valvular heart disease and thrombi in the left ventricle. Emboli originate from the heart and circulate to the cerebral vasculature, most commonly the left middle cerebral artery, resulting in a stroke. Embolic strokes may be prevented by the use of anticoagulation therapy in patients with atrial fibrillation.

The last two classifications of ischemic strokes are cryptogenic strokes, which have no known cause, and strokes from other causes, such as illicit drug use, coagulopathies, migraine, and spontaneous dissection of the carotid or vertebral arteries.

Pathophysiology

In an ischemic brain attack, there is disruption of the cerebral blood flow due to obstruction of a blood vessel. This disruption in blood flow initiates a complex series of cellular metabolic events referred to as the ischemic cascade (Fig. 62-1).

The ischemic cascade begins when cerebral blood flow decreases to less than 25 mL per 100 g of blood per minute. At this point, neurons are no longer able to maintain aerobic respiration. The mitochondria must then switch to anaerobic respiration, which generates large amounts of lactic acid, causing a change in the pH. This switch to the less efficient anaerobic respiration also renders the neuron incapable of producing sufficient quantities of adenosine triphosphate (ATP) to fuel the depolarization processes. The membrane pumps that maintain electrolyte balances begin to fail, and the cells cease to function.

Early in the cascade, an area of low cerebral blood flow, referred to as the **penumbra region,** exists around the area of infarction. The penumbra region is ischemic brain tissue that may be salvaged with timely intervention. The ischemic cascade threatens cells in the penumbra because

Table 62-1 COMPARISON OF MAJOR TYPES OF STROKE

Item	Ischemic	Hemorrhagic
Causes	Large artery thrombosis Small penetrating artery thrombosis Cardiogenic embolic Cryptogenic (no known cause) Other	Intracerebral hemorrhage Subarachnoid hemorrhage Cerebral aneurysm Arteriovenous malformation
Main presenting symptoms	Numbness or weakness of the face, arm, or leg, especially on one side of the body	"Exploding headache" Decreased level of consciousness
Functional recovery	Usually plateaus at 6 months	Slower, usually plateaus at about 18 months

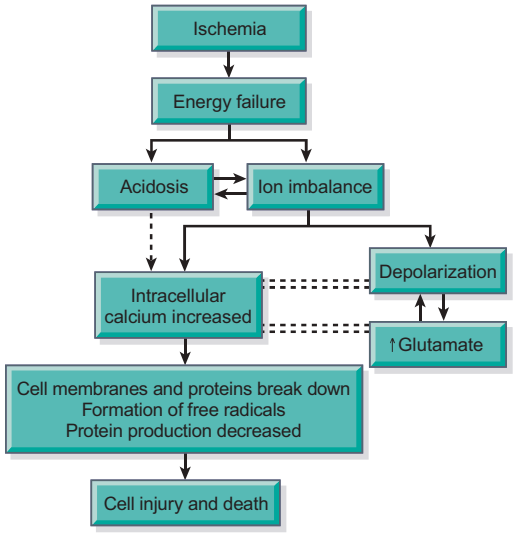

Figure 62-1 Processes contributing to ischemic brain cell injury. Courtesy of National Stroke Association, Englewood, Colorado.

membrane depolarization of the cell wall leads to an increase in intracellular calcium and the release of glutamate. The influx of calcium and the release of glutamate, if continued, activate a number of damaging pathways that result in the destruction of the cell membrane, the release of more calcium and glutamate, vasoconstriction, and the generation of free radicals. These processes enlarge the area of infarction into the penumbra, extending the stroke. A person experiencing a stroke typically loses 1.9 million neurons each minute that a stroke is not treated, and the ischemic brain ages 3.6 years each hour without treatment (Saver, 2006).

Each step in the ischemic cascade represents an opportunity for intervention to limit the extent of secondary brain damage caused by a stroke. The penumbra area may be revitalized by administration of tissue plasminogen activator (t-PA). Medications that protect the brain from secondary injury are called neuroprotectants. A number of ongoing clinical trials focus on neuroprotective medications and strategies to improve stroke recovery and survival (Lapchak & Araujo, 2007).

Clinical Manifestations

An ischemic stroke can cause a wide variety of neurologic deficits, depending on the location of the lesion (which vessels are obstructed), the size of the area of inadequate perfusion, and the amount of collateral (secondary or accessory) blood flow (see Chapter 60 for discussion of anatomy and brain blood supply). The patient may present with any of the following signs or symptoms:

- Numbness or weakness of the face, arm, or leg, especially on one side of the body
- Confusion or change in mental status
- Trouble speaking or understanding speech
- Visual disturbances
- Difficulty walking, dizziness, or loss of balance or coordination
- Sudden severe headache

Motor, sensory, cranial nerve, cognitive, and other functions may be disrupted. Table 62-2 reviews the neurologic deficits frequently seen in patients with strokes. Table 62-3 compares the symptoms and behaviors seen in right hemispheric stroke with those seen in left hemispheric stroke.

Motor Loss

A stroke is an upper motor neuron lesion and results in loss of voluntary control over motor movements. Because the upper motor neurons decussate (cross), a disturbance of voluntary motor control on one side of the body may reflect damage to the upper motor neurons on the opposite side of the brain. The most common motor dysfunction is **hemiplegia** (paralysis of one side of the body) caused by a lesion of the opposite side of the brain. **Hemiparesis,** or weakness of one side of the body, is another sign. The concept of upper and lower motor neuron lesions is described in more detail in Table 60-4 in Chapter 60.

In the early stage of stroke, the initial clinical features may be flaccid paralysis and loss of or decrease in the deep tendon reflexes. When these deep reflexes reappear (usually by 48 hours), increased tone is observed along with spasticity (abnormal increase in muscle tone) of the extremities on the affected side.

Communication Loss

Other brain functions affected by stroke are language and communication. In fact, stroke is the most common cause of aphasia. The following are dysfunctions of language and communication:

- **Dysarthria** (difficulty in speaking), caused by paralysis of the muscles responsible for producing speech
- Dysphasia (impaired speech) or **aphasia** (loss of speech), which can be **expressive aphasia, receptive aphasia,** or global (mixed) aphasia
- **Apraxia** (inability to perform a previously learned action), as may be seen when a patient makes verbal substitutions for desired syllables or words

Perceptual Disturbances

Perception is the ability to interpret sensation. Stroke can result in visual-perceptual dysfunctions, disturbances in visual-spatial relations, and sensory loss.

Visual-perceptual dysfunctions are caused by disturbances of the primary sensory pathways between the eye and visual cortex. Homonymous **hemianopsia** (loss of half of the visual field) may occur from stroke and may be temporary or permanent. The affected side of vision corresponds to the paralyzed side of the body.

Disturbances in visual-spatial relations (perceiving the relationship of two or more objects in spatial areas) are frequently seen in patients with right hemispheric damage.

Sensory Loss

The sensory losses from stroke may take the form of slight impairment of touch, or it may be more severe, with loss of proprioception (ability to perceive the position and motion of body parts) as well as difficulty in interpreting visual, tactile, and auditory stimuli. **Agnosias** are deficits in the ability to recognize previously familiar objects perceived by one or more of the senses.

Table 62-2 NEUROLOGIC DEFICITS OF STROKE: MANIFESTATIONS AND NURSING IMPLICATIONS

Neurologic Deficit	Manifestation	Nursing Implications/Patient Teaching Applications
Visual Field Deficits		
Homonymous hemianopsia (loss of half of the visual field)	• Unaware of persons or objects on side of visual loss • Neglect of one side of the body • Difficulty judging distances	Place objects within intact field of vision. Approach the patient from side of intact field of vision. Instruct/remind the patient to turn head in the direction of visual loss to compensate for loss of visual field. Encourage the use of eyeglasses if available. When teaching the patient, do so within patient's intact visual field.
Loss of peripheral vision	• Difficulty seeing at night • Unaware of objects or the borders of objects	Place objects in center of patient's intact visual field. Encourage the use of a cane or other object to identify objects in the periphery of the visual field. Driving ability will need to be evaluated.
Diplopia	• Double vision	Explain to the patient the location of an object when placing it near the patient. Consistently place patient care items in the same location.
Motor Deficits		
Hemiparesis	• Weakness of the face, arm, and leg on the same side (due to a lesion in the opposite hemisphere)	Place objects within the patient's reach on the nonaffected side. Instruct the patient to exercise and increase the strength on the unaffected side.
Hemiplegia	• Paralysis of the face, arm, and leg on the same side (due to a lesion in the opposite hemisphere)	Encourage the patient to provide range-of-motion exercises to the affected side. Provide immobilization as needed to the affected side. Maintain body alignment in functional position. Exercise unaffected limb to increase mobility, strength, and use.
Ataxia	• Staggering, unsteady gait • Unable to keep feet together; needs a broad base to stand	Support patient during the initial ambulation phase. Provide supportive device for ambulation (walker, cane). Instruct the patient not to walk without assistance or supportive device.
Dysarthria	• Difficulty in forming words	Provide the patient with alternative methods of communicating. Allow the patient sufficient time to respond to verbal communication. Support patient and family to alleviate frustration related to difficulty in communicating.
Dysphagia	• Difficulty in swallowing	Test the patient's pharyngeal reflexes before offering food or fluids. Assist the patient with meals. Place food on the unaffected side of the mouth. Allow ample time to eat.
Sensory Deficits		
Paresthesia (occurs on the side opposite the lesion)	• Numbness and tingling of extremity • Difficulty with proprioception	Instruct patient that sensation may be altered. Provide range of motion to affected areas and apply corrective devices as needed.
Verbal Deficits		
Expressive aphasia	• Unable to form words that are understandable; may be able to speak in single-word responses	Encourage patient to repeat sounds of the alphabet. Explore the patient's ability to write as an alternative means of communication.
Receptive aphasia	• Unable to comprehend the spoken word; can speak but may not make sense	Speak slowly and clearly to assist the patient in forming the sounds. Explore the patient's ability to read as an alternative means of communication.
Global (mixed) aphasia	• Combination of both receptive and expressive aphasia	Speak clearly and in simple sentences; use gestures or pictures when able. Establish alternative means of communication.
Cognitive Deficits		
	• Short- and long-term memory loss • Decreased attention span • Impaired ability to concentrate • Poor abstract reasoning • Altered judgment	Reorient patient to time, place, and situation frequently. Use verbal and auditory cues to orient patient. Provide familiar objects (family photographs, favorite objects). Use noncomplicated language. Match visual tasks with a verbal cue; holding a toothbrush, simulate brushing of teeth while saying, "I would like you to brush your teeth now." Minimize distracting noises and views when teaching the patient. Repeat and reinforce instructions frequently.
Emotional Deficits		
	• Loss of self-control • Emotional lability	Support patient during uncontrollable outbursts. Discuss with the patient and family that the outbursts are due to the disease process.
	• Decreased tolerance to stressful situations • Depression • Withdrawal • Fear, hostility, and anger • Feelings of isolation	Encourage patient to participate in group activity. Provide stimulation for the patient. Control stressful situations, if possible. Provide a safe environment. Encourage patient to express feelings and frustrations related to disease process.

Table 62-3 COMPARISON OF LEFT AND RIGHT HEMISPHERIC STROKES

Left Hemispheric Stroke	Right Hemispheric Stroke
Paralysis or weakness on right side of body	Paralysis or weakness on left side of body
Right visual field deficit	Left visual field deficit
Aphasia (expressive, receptive, or global)	Spatial-perceptual deficits
Altered intellectual ability	Increased distractibility
Slow, cautious behavior	Impulsive behavior and poor judgment
	Lack of awareness of deficits

Adapted from Hickey, J. V. (2009). *The clinical practice of neurological and neurosurgical nursing* (6th ed., p. 600). Philadelphia: Lippincott Williams & Wilkins.

Cognitive Impairment and Psychological Effects

If damage has occurred to the frontal lobe, learning capacity, memory, or other higher cortical intellectual functions may be impaired. Such dysfunction may be reflected in a limited attention span, difficulties in comprehension, forgetfulness, and a lack of motivation. These changes can cause the patient to become easily frustrated during rehabilitation. Depression is common and may be exaggerated by the patient's natural response to this catastrophic event. Emotional lability, hostility, frustration, resentment, lack of cooperation, and other psychological problems may occur.

Assessment and Diagnostic Findings

Any patient with neurologic deficits needs a careful history and a complete physical and neurologic examination. Initial assessment focuses on airway patency, which may be compromised by loss of gag or cough reflexes and altered respiratory pattern; cardiovascular status (including blood pressure, cardiac rhythm and rate, carotid bruit); and gross neurologic deficits.

Patients may present to the acute care facility with temporary neurologic symptoms. A transient ischemic attack (TIA) is a neurologic deficit typically lasting less than 1 hour. A TIA is manifested by a sudden loss of motor, sensory, or visual function. The symptoms result from temporary ischemia (impairment of blood flow) to a specific region of the brain but when brain imaging is performed there is no evidence of ischemia. A TIA may serve as a warning of impending stroke. Lack of evaluation and treatment of a patient who has experienced previous TIAs may result in a stroke and irreversible deficits (Lewandowski, Rao & Silver, 2008).

The initial diagnostic test for a stroke is usually a noncontrast computed tomography (CT) scan performed emergently to determine if the event is ischemic or hemorrhagic (the category of stroke determines treatment). Further diagnostic workup for ischemic stroke involves attempting to identify the source of the thrombi or emboli. A 12-lead electrocardiogram (ECG) and a carotid ultrasound are standard tests. Other studies may include CT angiography or magnetic resonance imaging and angiography (MRI and MRA) of the brain and neck vessels; transcranial Doppler flow studies; transthoracic or transesophageal echocardiography; xenon-enhanced CT scan; and single photon emission CT (SPECT) scan (Adams, Zoppo, Alberts, et al., 2007).

Prevention

Primary prevention of ischemic stroke remains the best approach. Leading a healthy lifestyle, which includes not smoking, maintaining a healthy weight, following a healthy diet (including modest alcohol consumption), and daily exercise, can reduce the risk of having a stroke by about one half (Chiuve, Rexrode, Spiegelman, et al., 2008). The risk of coronary heart disease and stroke has decreased in women on the Dietary Approaches to Stop Hypertension (DASH) diet. The DASH diet is high in fruits and vegetables, moderate in low-fat dairy products, and low in animal protein (has a substantial amount of plant protein from legumes and nuts) (Fung, Chiuve, McCullough, et al., 2008). Stroke risk screenings are an ideal opportunity to lower stroke risk by identifying people or groups of people who are at high risk for stroke and by educating patients and the community about recognition and prevention of stroke. Research findings suggest that low-dose aspirin may lower the risk of stroke in women who are at risk (Ridker, Cook, Lee, et al., 2005).

Advanced age, gender, and race are well-known nonmodifiable risk factors for stroke. High-risk groups include people older than 55 years of age; the incidence of stroke more than doubles in each successive decade. Men have a higher rate of stroke than that of women. Another high-risk group is African Americans; the incidence of first stroke in African Americans is almost twice that in Caucasian Americans (Rosamond, et al., 2008).

Modifiable risk factors for ischemic stroke include hypertension, atrial fibrillation, hyperlipidemia, obesity, smoking, and diabetes (Chart 62-1). For people who are at high risk, interventions that alter modifiable factors, such as treating hypertension and hyperglycemia and stopping smoking, reduce stroke risk. Other treatable conditions that increase risk of stroke are asymptomatic carotid stenosis and valvular heart disease (eg, endocarditis, prosthetic heart valves). Periodontal disease has also been linked to stroke risk. The association between periodontal disease and stroke may result from the host inflammatory response and the chronic bacterial infection, but the exact mechanism is not fully understood. Periodontal disease is a treatable and preventable condition.

Several methods of preventing recurrent stroke have been identified for patients with TIAs or ischemic stroke. Patients with moderate to severe carotid stenosis are treated with carotid endarterectomy (Sacco, Adams, Albers, et al., 2006). In patients with atrial fibrillation, which increases the risk of emboli, administration of warfarin (Coumadin), an anticoagulant that inhibits clot formation, may prevent both thrombotic and embolic strokes.

CHART 62-1 *Modifiable Risk Factors for Ischemic Stroke*

- Hypertension (controlling hypertension, the major risk factor, is the key to preventing stroke)
- Atrial fibrillation
- Hyperlipidemia
- Diabetes mellitus (associated with accelerated atherogenesis)
- Smoking
- Asymptomatic carotid stenosis
- Obesity
- Excessive alcohol consumption

Medical Management

Patients who have experienced a TIA or stroke should have medical management for secondary prevention. Those with atrial fibrillation (or cardioembolic strokes) are treated with dose-adjusted warfarin (Coumadin) unless contraindicated. The international normalized ratio (INR) target is 2 to 3. If warfarin is contraindicated, aspirin is the best option, although other medications may be used if both are contraindicated (Karch, 2008).

Platelet-inhibiting medications, including aspirin, extended-release dipyridamole (Persantine) plus aspirin, clopidogrel (Plavix), and ticlopidine (Ticlid), decrease the incidence of cerebral infarction in patients who have experienced TIAs and stroke from suspected embolic or thrombotic causes. The specific medication that is used is based on the patient's health history.

Research has found that medications classified as 3-hydroxy-2-methyl-glutaryl-coenzyme A reductase inhibitors (also known as statins) reduce coronary events and strokes. Benefits were independent of cholesterol levels, and these medications are now widely used for stroke prevention. The U.S. Food and Drug Administration (FDA) has recently updated indications for a statin medication, such as simvastatin (Zocor), to include secondary stroke prevention (Nassief & Marsh, 2008). After the acute stroke period, antihypertensive medications are also used, if indicated, for secondary stroke prevention. Angiotensin-converting enzyme (ACE) inhibitors and thiazide diuretics may also have benefits in stroke prevention (Luders, 2007).

Ongoing research is focusing on several aspects of the medical management of acute ischemic stroke. The FDA has approved a clot retrieval device (shaped like a tiny corkscrew) that opens the blocked artery and restores blood flow to the brain (Felton, Ogden, Pena, et al., 2005). Other clot retrieval devices are under investigation, including catheters using vacuum and ultrasound techniques to assist in the removal of clots in the brain.

Thrombolytic Therapy

Thrombolytic agents are used to treat ischemic stroke by dissolving the blood clot that is blocking blood flow to the brain. Recombinant t-PA is a genetically engineered form of t-PA, a thrombolytic substance made naturally by the body. It works by binding to fibrin and converting plasminogen to plasmin, which stimulates fibrinolysis of the atherosclerotic lesion. Rapid diagnosis of stroke and initiation of thrombolytic therapy (within 3 hours) in patients with ischemic stroke leads to a decrease in the size of the stroke and an overall improvement in functional outcome after 3 months (Adams, et al., 2007; NINDS, 1995). Ongoing clinical trials continue to investigate other thrombolytic agents (Lapchak & Araujo, 2007).

To realize the full potential of thrombolytic therapy, community education directed at recognizing the symptoms of stroke and obtaining appropriate emergency care is necessary to ensure rapid transport to a hospital and initiation of therapy within the 3-hour period. Delays make the patient ineligible for thrombolytic therapy, because revascularization of necrotic tissue (which develops after 3 hours) increases the risk of cerebral edema and hemorrhage.

Enhancing Prompt Diagnosis

After being notified by emergency medical service personnel, the emergency department contacts the appropriate staff (neurologist, neuroradiologist, radiology department, nursing staff, ECG, and laboratory technicians) and informs them of the patient's imminent arrival at the hospital. Many institutions have acute stroke teams that respond rapidly, ensuring that treatment occurs within the allotted period (AANN, 2008).

Initial management requires the definitive diagnosis of an ischemic stroke by brain imaging and a careful history to determine whether the patient meets the criteria for t-PA therapy (Chart 62-2). Some of the absolute contraindications for thrombolytic therapy include symptom onset greater than 3 hours before admission, a patient who is anticoagulated (with an INR above 1.7), or a patient who has recently had any type of intracranial pathology (eg, previous stroke, head injury, trauma). Once it is determined that the patient is a candidate for t-PA therapy, no anticoagulants are administered for the next 24 hours.

Before receiving t-PA, the patient is assessed using the National Institutes of Health Stroke Scale (NIHSS), a standardized assessment tool that helps evaluate stroke severity (Table 62-4). Total NIHSS scores range from 0 (normal) to 42 (severe stroke) (Kasner, 2006). Certification in the administration of the scale is recommended and is available for nurses and other health care professionals.

Dosage and Administration

The patient is weighed to determine the dose of t-PA. The dosage for t-PA is 0.9 mg/kg, with a maximum dose of 90 mg. Ten percent of the calculated dose is administered as an intravenous (IV) bolus over 1 minute. The remaining dose (90%) is administered IV over 1 hour via an infusion pump.

The patient is admitted to the intensive care unit or an acute stroke unit, where continuous cardiac monitoring and frequent neurologic assessments are conducted. Vital signs are obtained frequently, with particular attention to blood

Chart 62-2 • *Eligibility Criteria for t-PA Administration*

- Age 18 years or older
- Clinical diagnosis of ischemic stroke
- Time of onset of stroke known and is 3 hours or less
- Systolic blood pressure ≤185 mm Hg; diastolic ≤110 mm Hg
- Not a minor stroke or rapidly resolving stroke
- No seizure at onset of stroke
- Not taking warfarin (Coumadin)
- Prothrombin time ≤15 seconds or INR ≤1.7
- Not receiving heparin during the past 48 hours with elevated partial thromboplastin time
- Platelet count ≥100,000/mm^3
- No prior intracranial hemorrhage, neoplasm, arteriovenous malformation, or aneurysm
- No major surgical procedures within 14 days
- No stroke, serious head injury, or intracranial surgery within 3 months
- No gastrointestinal or urinary bleeding within 21 days

Table 62-4 SUMMARY OF NATIONAL INSTITUTES OF HEALTH STROKE SCALE (NIHSS)

Category	Description	Score
1a. Level of consciousness (LOC)	Alert	0
	Arousable by minor stimulation	1
	Obtunded, strong stimulation to attend	2
	Unresponsive, or reflexic responses only	3
1b. LOC questions (month, age)	Answers both correctly	0
	Answers one correctly	1
	Both incorrect	2
1c. LOC commands (open, close eyes; make fist, let go)	Obeys both correctly	0
	Obeys one correctly	1
	Both incorrect	2
2. Best gaze (eyes open—patient follows examiner's finger or face)	Normal	0
	Partial gaze palsy	1
	Forced deviation	2
3. Visual (introduce visual stimulus/threat to patient's visual field quadrants)	No visual loss	0
	Partial hemianopsia	1
	Complete hemianopsia	2
	Bilateral hemianopsia	3
4. Facial palsy (show teeth, raise eyebrows and squeeze eyes shut)	Normal	0
	Minor	1
	Partial	2
	Complete	3
5a. Motor; arm—left (elevate extremity to 90° and score drift/movement)	No drift	0
	Drift but maintains in air	1
	Unable to maintain in air	2
	No effort against gravity	3
	No movement	4
	Amputation, joint fusion (explain)	N/A
5b. Motor; arm—right (elevate extremity to 90° and score drift/movement)	No drift	0
	Drift but maintains in air	1
	Unable to maintain in air	2
	No effort against gravity	3
	No movement	4
	Amputation, joint fusion (explain)	N/A
6a. Motor; leg—left (elevate extremity to 30° and score drift/movement)	No drift	0
	Drift but maintains in air	1
	Unable to maintain in air	2
	No effort against gravity	3
	No movement	4
	Amputation, joint fusion (explain)	N/A
6b. Motor; leg—right (elevate extremity to 30° and score drift/movement)	No drift	0
	Drift but maintains in air	1
	Unable to maintain in air	2
	No effort against gravity	3
	No movement	4
	Amputation, joint fusion (explain)	N/A
7. Limb ataxia (finger-to-nose and heel-to-shin testing)	Absent	0
	Present in one limb	1
	Present in two limbs	2
8. Sensory (pinprick to face, arm, trunk, and leg—compare side to side)	Normal	0
	Mild to moderate loss	1
	Severe to total loss	2
9. Best language (name items, describe a picture and read sentences)	No aphasia	0
	Mild to moderate aphasia	1
	Severe aphasia	2
	Mute	3
10. Dysarthria (evaluate speech clarity by having patient repeat words)	Normal	0
	Mild to moderate dysarthria	1
	Severe dysarthria, mostly unintelligible or worse	2
	Intubated or other physical barrier	N/A
11. Extinction and inattention (use information from prior testing to score)	No abnormality	0
	Visual, tactile, auditory, or other extinction to bilateral simultaneous stimulation	1
	Profound hemiattention or extinction to more than one modality.	2
Total score		

Adapted from the version available at the National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, MD 20892, www.ninds.nih.gov/doctors/NIH_Stroke_Scale.pdf. It is recommended that the full scale with all instructions be used.

pressure (with the goal of lowering the risk of intracranial hemorrhage). An example of a standard protocol would be to obtain vital signs every 15 minutes for the first 2 hours, every 30 minutes for the next 6 hours, then every hour until 24 hours after treatment. Blood pressure should be maintained with the systolic pressure less than 180 mm Hg and the diastolic pressure less than 105 mm Hg (Adams, et al., 2007). Airway management is instituted based on the patient's clinical condition and arterial blood gas values.

Side Effects

Bleeding is the most common side effect of t-PA administration, and the patient is closely monitored for any bleeding (IV insertion sites, urinary catheter site, endotracheal tube, nasogastric tube, urine, stool, emesis, other secretions). A 24-hour delay in placement of nasogastric tubes, urinary catheters, and intra-arterial pressure catheters is recommended. Intracranial bleeding is a major complication that occurred in approximately 6.4% of patients in the initial t-PA study (NINDS, 1995). A number of factors are associated with the occurrence of symptomatic intracranial bleeding: age greater than 70 years, baseline NIHSS score greater than 20, serum glucose concentration 300 mg/dL or higher, and edema or mass effect observed on the patient's initial CT scan.

Therapy for Patients With Ischemic Stroke Not Receiving t-PA

Not all patients are candidates for t-PA therapy. Other treatments may include anticoagulant administration (IV heparin or low-molecular-weight heparin). Because of the risks associated with anticoagulation, their general use is no longer recommended for patients with acute ischemic stroke, whether treated with t-PA or not (Adams, et al., 2007).

Careful maintenance of cerebral hemodynamics to maintain cerebral perfusion is extremely important after a stroke. Increased intracranial pressure (ICP) from brain edema, and associated complications, may occur after a large ischemic stroke. Interventions during this period include measures to reduce ICP, such as administering an osmotic diuretic (eg, mannitol), maintaining the partial pressure of carbon dioxide ($PaCO_2$) within the range of 30 to 35 mm Hg, and positioning to avoid hypoxia. Other treatment measures include the following:

- Elevation of the head of the bed to promote venous drainage and to lower increased ICP
- Possible hemicraniectomy for increased ICP from brain edema in a very large stroke
- Intubation with an endotracheal tube to establish a patent airway, if necessary
- Continuous hemodynamic monitoring (the goals for blood pressure remain controversial for a patient who has not received thrombolytic therapy; antihypertensive treatment may be withheld unless the systolic blood pressure exceeds 220 mm Hg or the diastolic blood pressure exceeds 120 mm Hg)
- Neurologic assessment to determine if the stroke is evolving and if other acute complications are developing; such complications may include seizures, bleeding from anticoagulation, or medication-induced bradycardia, which can result in hypotension and subsequent decreases in cardiac output and cerebral perfusion pressure

An acute ischemic stroke clinical pathway is shown in Appendix B.

Managing Potential Complications

Adequate cerebral blood flow is essential for cerebral oxygenation. If cerebral blood flow is inadequate, the amount of oxygen supplied to the brain will decrease, and tissue ischemia will result. Adequate oxygenation begins with pulmonary care, maintenance of a patent airway, and administration of supplemental oxygen as needed. The importance of adequate gas exchange in these patients cannot be overemphasized as many are at risk for aspiration pneumonia.

Other potential complications after a stroke include urinary tract infections, cardiac dysrhythmias, and complications of immobility.

Surgical Prevention of Ischemic Stroke

The main surgical procedure for selected patients with TIAs and mild stroke is carotid endarterectomy, which is currently the most frequently performed noncardiac vascular procedure. A carotid endarterectomy is the removal of an atherosclerotic plaque or thrombus from the carotid artery to prevent stroke in patients with occlusive disease of the extracranial cerebral arteries (Fig. 62-2). This surgery is indicated for patients with symptoms of TIA or mild stroke found to be caused by severe (70% to 99%) carotid artery stenosis or moderate (50% to 69%) stenosis with other significant risk factors (Chaturvedi, Bruno, Feasby, et al., 2005).

Carotid stenting, with or without angioplasty, is a less invasive procedure that is used, at times, for severe stenosis. It is used for selected patients who are at high risk for surgery, and its efficacy continues to be investigated. In a recent

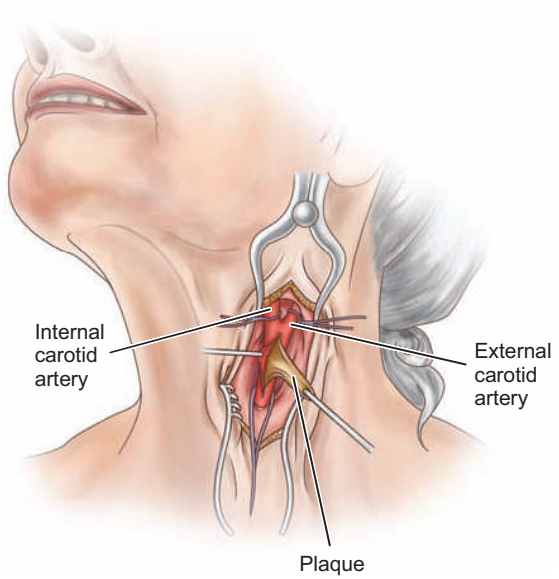

Figure 62-2 Plaque, a potential source of emboli in transient ischemic attack and stroke, is surgically removed from the carotid artery.

Table 62-5 SELECTED COMPLICATIONS OF CAROTID ENDARTERECTOMY AND NURSING INTERVENTIONS

Complication	Characteristics	Nursing Interventions
Incision hematoma	Occurs in 5.5% of patients. Large or rapidly expanding hematomas require emergency treatment. If the airway is obstructed by the hematoma, the incision may be opened at the bedside.	Monitor neck discomfort and wound expansion. Report swelling, subjective feelings of pressure in the neck, difficulty breathing.
Hypertension	Poorly controlled hypertension increases the risk of postoperative complications, including hematoma and hyperperfusion syndrome. There is an increased incidence of neurologic impairment and death due to intracerebral hemorrhage. May be related to surgically induced abnormalities of carotid baroreceptor sensitivity.	Risk is highest in the first 48 h after surgery. Check blood pressure frequently and report deviations from baseline. Observe for and report new onset of neurologic deficits.
Postoperative hypotension	Occurs in approximately 5% of patients. Treated with fluids and low-dose phenylephrine infusion. Usually resolves in 24–48 h. Patients with hypotension should have serial ECGs to rule out myocardial infarction.	Monitor blood pressure and observe for signs and symptoms of hypotension.
Hyperperfusion syndrome	Occurs when cerebral vessel autoregulation fails. Arteries accustomed to diminished blood flow may be permanently dilated; increased blood flow after endarterectomy coupled with insufficient vasoconstriction leads to capillary bed damage, edema, and hemorrhage.	Observe for severe unilateral headache improved by sitting upright or standing.
Intracerebral hemorrhage	Occurs infrequently, but is often fatal (60%) or results in serious neurologic impairment. Can occur secondary to hyperperfusion syndrome. Increased risk with advanced age, hypertension, presence of high-grade stenosis, poor collateral flow, and slow flow in the region of the middle cerebral artery.	Monitor neurologic status and report any changes in mental status or neurologic functioning immediately.

study, 334 patients with severe carotid artery stenosis and at high risk for surgery underwent stenting with the use of an emboli protection device or carotid endarterectomy. This study demonstrated that this procedure is not inferior to carotid endarterectomy as it resulted in similar long-term outcomes (Gurm, Yadav, Fayad, et al., 2008).

Nursing Management

The primary complications of carotid endarterectomy are stroke, cranial nerve injuries, infection or hematoma at the incision, and carotid artery disruption. It is important to maintain adequate blood pressure levels in the immediate postoperative period. Hypotension is avoided to prevent cerebral ischemia and thrombosis. Uncontrolled hypertension may precipitate cerebral hemorrhage, edema, hemorrhage at the surgical incision, or disruption of the arterial reconstruction. Medications are used to reduce the blood pressure to previous levels. Close cardiac monitoring is necessary, because these patients have a high incidence of coronary artery disease.

After carotid endarterectomy, a neurologic flow sheet is used to monitor and document assessment parameters for all body systems, with particular attention to neurologic status. The surgeon is notified immediately if a neurologic deficit develops. Formation of a thrombus at the site of the endarterectomy is suspected if there is a sudden increase in neurologic deficits, such as weakness on one side of the body. The patient should be prepared for repeat endarterectomy.

Difficulty in swallowing, hoarseness, or other signs of cranial nerve dysfunction must be assessed. The nurse focuses on assessment of the following cranial nerves: facial (VII), vagus (X), spinal accessory (XI), and hypoglossal (XII). Some edema in the neck after surgery is expected; however, extensive edema and hematoma formation can obstruct the airway. Emergency airway supplies, including those needed for a tracheostomy, must be available. Table 62-5 provides more information about potential complications of carotid surgery.

NURSING PROCESS

The Patient Recovering From an Ischemic Stroke

The acute phase of an ischemic stroke may last 1 to 3 days, but ongoing monitoring of all body systems is essential as long as the patient requires care. The patient who has had a stroke is at risk for multiple complications, including deconditioning and other musculoskeletal problems, swallowing difficulties, bowel and bladder dysfunction, inability to perform self-care, and skin breakdown. After the stroke is complete, management focuses on the prompt initiation of rehabilitation for any deficits.

Assessment

During the acute phase, a neurologic flow sheet is maintained to provide data about the following important measures of the patient's clinical status:

- Change in level of consciousness or responsiveness as evidenced by movement, resistance to changes of position, and response to stimulation; orientation to time, place, and person
- Presence or absence of voluntary or involuntary movements of the extremities; muscle tone; body posture; and position of the head

- Stiffness or flaccidity of the neck
- Eye opening, comparative size of pupils and pupillary reactions to light, and ocular position
- Color of the face and extremities; temperature and moisture of the skin
- Quality and rates of pulse and respiration; arterial blood gas values as indicated, body temperature, and arterial pressure
- Ability to speak
- Volume of fluids ingested or administered; volume of urine excreted each 24 hours
- Presence of bleeding
- Maintenance of blood pressure within the desired parameters

After the acute phase, the nurse assesses mental status (memory, attention span, perception, orientation, affect, speech/language), sensation/perception (usually the patient has decreased awareness of pain and temperature), motor control (upper and lower extremity movement), swallowing ability, nutritional and hydration status, skin integrity, activity tolerance, and bowel and bladder function. Ongoing nursing assessment continues to focus on any impairment of function in the patient's daily activities, because the quality of life after stroke is closely related to the patient's functional status.

Diagnosis

Nursing Diagnoses

Based on the assessment data, the major nursing diagnoses for a patient with a stroke may include the following:

- Impaired physical mobility related to hemiparesis, loss of balance and coordination, spasticity, and brain injury
- Acute pain (painful shoulder) related to hemiplegia and disuse
- Self-care deficits (bathing, hygiene, toileting, dressing, grooming, and feeding) related to stroke sequelae
- Disturbed sensory perception (kinesthetic, tactile or visual) related to altered sensory reception, transmission, and/or integration
- Impaired swallowing
- Impaired urinary elimination related to flaccid bladder, detrusor instability, confusion, or difficulty in communicating
- Disturbed thought processes related to brain damage
- Impaired verbal communication related to brain damage
- Risk for impaired skin integrity related to hemiparesis, hemiplegia, or decreased mobility
- Interrupted family processes related to catastrophic illness and caregiving burdens
- Sexual dysfunction related to neurologic deficits or fear of failure

Collaborative Problems/Potential Complications

Potential complications include:

- Decreased cerebral blood flow due to increased ICP
- Inadequate oxygen delivery to the brain
- Pneumonia

Planning and Goals

Although rehabilitation begins on the day the patient has the stroke, the process is intensified during convalescence and requires a coordinated team effort. It is helpful for the team to know what the patient was like before the stroke: his or her illnesses, abilities, mental and emotional state, behavioral characteristics, and activities of daily living (ADLs). It is also helpful for clinicians to be knowledgeable about the relative importance of predictors of stroke outcome (age, NIHSS score, and level of consciousness at time of admission) in order to provide stroke survivors and their families with realistic goals (Adams, et al., 2007).

The major goals for the patient (and family) may include improved mobility, avoidance of shoulder pain, achievement of self-care, relief of sensory and perceptual deprivation, prevention of aspiration, continence of bowel and bladder, improved thought processes, achieving a form of communication, maintaining skin integrity, restored family functioning, improved sexual function, and absence of complications.

Nursing Interventions

Nursing care has a significant impact on the patient's recovery. Often, many body systems are impaired as a result of the stroke, and conscientious care and timely interventions can prevent debilitating complications. During and after the acute phase, nursing interventions focus on the whole person. In addition to providing physical care, the nurse encourages and fosters recovery by listening to the patient and asking questions to elicit the meaning of the stroke experience.

Improving Mobility and Preventing Joint Deformities

A patient with hemiplegia has unilateral paralysis (paralysis on one side). When control of the voluntary muscles is lost, the strong flexor muscles exert control over the extensors. The arm tends to adduct (adductor muscles are stronger than abductors) and to rotate internally. The elbow and the wrist tend to flex, the affected leg tends to rotate externally at the hip joint and flex at the knee, and the foot at the ankle joint supinates and tends toward plantar flexion.

Correct positioning is important to prevent contractures; measures are used to relieve pressure, assist in maintaining good body alignment, and prevent compressive neuropathies, especially of the ulnar and peroneal nerves. Because flexor muscles are stronger than extensor muscles, a splint applied at night to the affected extremity may prevent flexion and maintain correct positioning during sleep. (See Chapter 11 for additional information.)

PREVENTING SHOULDER ADDUCTION. To prevent adduction of the affected shoulder while the patient is in bed, a pillow is placed in the axilla when there is limited external rotation; this keeps the arm away from the chest. A pillow is placed under the arm, and the arm is placed in a neutral (slightly flexed) position, with distal joints positioned higher than the more proximal joints (ie, the elbow is positioned higher than the shoulder and the wrist higher than the elbow). This helps to prevent edema and the resultant joint fibrosis that will limit range of motion if the patient regains control of the arm (Fig. 62-3).

POSITIONING THE HAND AND FINGERS. The fingers are positioned so that they are barely flexed. The hand is placed in slight supination (palm faces upward), which is its most functional position. If the upper extremity is flaccid, a splint can be used to support the wrist and hand in a functional

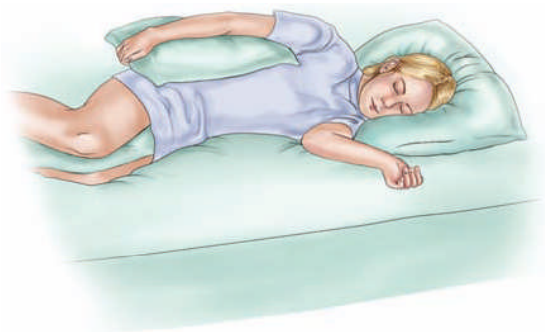

Figure 62-3 Correct positioning to prevent shoulder adduction.

position. If the upper extremity is spastic, a hand roll is not used, because it stimulates the grasp reflex. In this instance a dorsal wrist splint is useful in allowing the palm to be free of pressure. Every effort is made to prevent hand edema.

Spasticity, particularly in the hand, can be a disabling complication after stroke. Researchers have reported that repeated intramuscular injections of botulinum toxin type A into wrist and finger muscles reduced upper limb spasticity after stroke, resulting in significant and sustained improvements in muscle tone, lessened disability, and improved quality of life (Elovic, Brashear, Kaelin, et al., 2008). Other treatments for spasticity may include stretching and splinting.

CHANGING POSITIONS. The patient's position should be changed every 2 hours. To place a patient in a lateral (side-lying) position, a pillow is placed between the legs before the patient is turned. To promote venous return and prevent edema, the upper thigh should not be acutely flexed. The patient may be turned from side to side, but if sensation is impaired, the amount of time spent on the affected side should be limited.

If possible, the patient is placed in a prone position for 15 to 30 minutes several times a day. A small pillow or a support is placed under the pelvis, extending from the level of the umbilicus to the upper third of the thigh (Fig. 62-4). This position helps promote hyperextension of the hip joints, which is essential for normal gait and helps prevent knee and hip flexion contractures. The prone position also helps drain bronchial secretions and prevents contractural deformities of the shoulders and knees. During positioning, it is important to reduce pressure and change position frequently to prevent pressure ulcers.

ESTABLISHING AN EXERCISE PROGRAM. The affected extremities are exercised passively and put through a full range of motion four or five times a day to maintain joint mobility, regain motor control, prevent contractures in the paralyzed extremity, prevent further deterioration of the neuromuscular system, and enhance circulation. Exercise is helpful in preventing venous stasis, which may predispose the patient to thrombosis and pulmonary embolus.

Figure 62-4 Prone position with pillow support helps prevent hip flexion.

Repetition of an activity forms new pathways in the CNS and therefore encourages new patterns of motion. At first, the extremities are usually flaccid. If tightness occurs in any area, the range-of-motion exercises should be performed more frequently (see Chapter 11).

The patient is observed for signs and symptoms that may indicate pulmonary embolus or excessive cardiac workload during exercise; these include shortness of breath, chest pain, cyanosis, and increasing pulse rate with exercise. Frequent short periods of exercise always are preferable to longer periods at infrequent intervals. Regularity in exercise is most important. Improvement in muscle strength and maintenance of range of motion can be achieved only through daily exercise.

The patient is encouraged and reminded to exercise the unaffected side at intervals throughout the day. It is helpful to develop a written schedule to remind the patient of the exercise activities. The nurse supervises and supports the patient during these activities. The patient can be taught to put the unaffected leg under the affected one to assist in moving it when turning and exercising. Flexibility, strengthening, coordination, endurance, and balancing exercises prepare the patient for ambulation. Quadriceps muscle setting and gluteal setting exercises are started early to improve the muscle strength needed for walking; these are performed at least five times daily for 10 minutes at a time.

PREPARING FOR AMBULATION. As soon as possible, the patient is assisted out of bed and an active rehabilitation program is started. The patient is first taught to maintain balance while sitting and then to learn to balance while standing. If the patient has difficulty in achieving standing balance, a tilt table, which slowly brings the patient to an upright position, can be used. Tilt tables are especially helpful for patients who have been on bed rest for prolonged periods and have orthostatic blood pressure changes.

If the patient needs a wheelchair, the folding type with hand brakes is the most practical because it allows the patient to manipulate the chair. The chair should be low enough to allow the patient to propel it with the uninvolved foot and narrow enough to permit it to be used in the home. When the patient is transferred from the wheelchair, the brakes must be applied and locked on both sides of the chair.

The patient is usually ready to walk as soon as standing balance is achieved. Parallel bars are useful in these first efforts. A chair or wheelchair should be readily available in case the patient suddenly becomes fatigued or feels dizzy.

The training periods for ambulation should be short and frequent. As the patient gains strength and confidence, an adjustable cane can be used for support. Generally, a three- or four-pronged cane provides a stable support in the early phases of rehabilitation.

Preventing Shoulder Pain

As many as 72% of patients who have had a stroke have pain in the shoulder (Duncan, Zorowitz, Bates, et al., 2005). That pain may prevent them from learning new skills and

affect their quality of life (Chae, Mascarenhas, Yu, et al., 2007). Shoulder function is essential in achieving balance and performing transfers and self-care activities. Three problems can occur: painful shoulder, subluxation of the shoulder, and shoulder–hand syndrome.

A flaccid shoulder joint may be overstretched by the use of excessive force in turning the patient or from overstrenuous arm and shoulder movement. To prevent shoulder pain, the nurse should never lift the patient by the flaccid shoulder or pull on the affected arm or shoulder. Overhead pulleys should also be avoided. If the arm is paralyzed, subluxation (incomplete dislocation) at the shoulder can occur as a result of overstretching of the joint capsule and musculature by the force of gravity when the patient sits or stands in the early stages after a stroke. This results in severe pain. Shoulder–hand syndrome (painful shoulder and generalized swelling of the hand) can cause a frozen shoulder and ultimately atrophy of subcutaneous tissues. When a shoulder becomes stiff, it is usually painful.

Many shoulder problems can be prevented by proper patient movement and positioning. The flaccid arm is positioned on a table or with pillows while the patient is seated. Some clinicians advocate the use of a properly worn sling when the patient first becomes ambulatory, to prevent the paralyzed upper extremity from dangling without support. Range-of-motion exercises are important in preventing painful shoulder. Overstrenuous arm movements are avoided. The patient is instructed to interlace the fingers, place the palms together, and push the clasped hands slowly forward to bring the scapulae forward; he or she then raises both hands above the head. This is repeated throughout the day. The patient is instructed to flex the affected wrist at intervals and move all the joints of the affected fingers. The patient is encouraged to touch, stroke, rub, and look at both hands. Pushing the heel of the hand firmly down on a surface is useful. Elevation of the arm and hand is also important in preventing dependent edema of the hand. Patients with continuing pain after attempted movement and positioning may require the addition of analgesia to their treatment program. Other treatments may include injections to the shoulder joint with corticosteroid medications, electrical stimulation, heat or ice, and soft tissue massage (Duncan, et al., 2005).

Medications are helpful in the management of poststroke pain. Amitriptyline hydrochloride (Elavil) has been used, but it can cause cognitive problems, has a sedating effect, and is not effective in all patients. The antiseizure medications lamotrigine (Lamictal) and pregabalin (Lyrica) have been found to be effective for poststroke pain, and they may serve as alternatives for patients who cannot tolerate amitriptyline (Vranken, Dijkgraaf, Kruis, et al., 2008).

Enhancing Self-Care

As soon as the patient can sit up, personal hygiene activities are encouraged. The patient is helped to set realistic goals; if feasible, a new task is added daily. The first step is to carry out all self-care activities on the unaffected side. Such activities as combing the hair, brushing the teeth, shaving with an electric razor, bathing, and eating can be carried out with one hand and should be encouraged. Although the patient may feel awkward at first, these motor skills can be learned by repetition, and the unaffected side will become stronger with use. The nurse must be sure that the patient does not neglect the affected side. Assistive devices will help make up for some of the patient's deficits (Chart 62-3). A small towel is easier to control while drying after bathing, and boxed paper tissues are easier to use than a roll of toilet tissue.

Chart 62-3 • *Assistive Devices to Enhance Self-Care After Stroke*

Eating Devices

- Nonskid mats to stabilize plates
- Plate guards to prevent food from being pushed off plate
- Wide-grip utensils to accommodate a weak grasp

Bathing and Grooming Devices

- Long-handled bath sponge
- Grab bars, nonskid mats, handheld shower heads
- Electric razors with head at 90 degrees to handle
- Shower and tub seats, stationary or on wheels

Toileting Aids

- Raised toilet seat
- Grab bars next to toilet

Dressing Aids

- Velcro closures
- Elastic shoelaces
- Long-handled shoe horn

Mobility Aids

- Canes, walkers, wheelchairs
- Transfer devices such as transfer boards and belts

Return of functional ability is important to the patient recovering after a stroke. An early baseline assessment of functional ability with an instrument such as the Functional Independence Measure (FIM) is important in team planning and goal setting for the patient. The FIM is a widely used instrument in stroke rehabilitation and provides valuable information about motor, social, and cognitive function (Kasner, 2006). The patient's morale may improve if ambulatory activities are carried out in street clothes. The family is instructed to bring in clothing that is preferably a size larger than that normally worn. Clothing fitted with front or side fasteners or Velcro closures is the most suitable. The patient has better balance if most of the dressing activities are carried out while seated.

Perceptual problems may make it difficult for the patient to dress without assistance because of an inability to match the clothing to the body parts. To assist the patient, the nurse can take steps to keep the environment organized and uncluttered, because the patient with a perceptual problem is easily distracted. The clothing is placed on the affected side in the order in which the garments are to be put on. Using a large mirror while dressing promotes the patient's awareness of what he or she is putting on the affected side. The patient has to make many compensatory movements when dressing; these can produce fatigue and painful twisting of the intercostal muscles. Support and encouragement

are provided to prevent the patient from becoming overly fatigued and discouraged. Even with intensive training, not all patients can achieve independence in dressing.

Managing Sensory-Perceptual Difficulties

Patients with a decreased field of vision should be approached on the side where visual perception is intact. All visual stimuli (eg, clock, calendar, television) should be placed on this side. The patient can be taught to turn the head in the direction of the defective visual field to compensate for this loss. The nurse should make eye contact with the patient and draw his or her attention to the affected side by encouraging the patient to move the head. The nurse may also want to stand at a position that encourages the patient to move or turn to visualize who is in the room. Increasing the natural or artificial lighting in the room and providing eyeglasses are important aids to increasing vision.

The patient with homonymous hemianopsia (loss of half of the visual field) turns away from the affected side of the body and tends to neglect that side and the space on that side; this is called amorphosynthesis. In such instances, the patient cannot see food on half of the tray, and only half of the room is visible. It is important for the nurse to constantly remind the patient of the other side of the body, to maintain alignment of the extremities, and, if possible, to place the extremities where the patient can see them.

Assisting With Nutrition

Stroke can result in swallowing problems (dysphagia) due to impaired function of the mouth, tongue, palate, larynx, pharynx, or upper esophagus. Patients must be observed for paroxysms of coughing, food dribbling out of or pooling in one side of the mouth, food retained for long periods in the mouth, or nasal regurgitation when swallowing liquids. Swallowing difficulties place the patient at risk for aspiration, pneumonia, dehydration, and malnutrition.

A speech therapist will evaluate the patient's swallowing ability. If swallowing function is partially impaired, it may return over time, or the patient may be taught alternative swallowing techniques, advised to take smaller boluses of food, and taught about types of foods that are easier to swallow. The patient may be started on a thick liquid or puréed diet, because these foods are easier to swallow than thin liquids. Having the patient sit upright, preferably out of bed in a chair, and instructing him or her to tuck the chin toward the chest as he or she swallows will help prevent aspiration. The diet may be advanced as the patient becomes more proficient at swallowing. If the patient cannot resume oral intake, a gastrointestinal feeding tube is placed for ongoing tube feedings and medication administration.

Enteral tubes can be either nasogastric (placed in the stomach) or nasoenteral (placed in the duodenum) to reduce the risk of aspiration. Nursing responsibilities in feeding include elevating the head of the bed at least 30 degrees to prevent aspiration, checking the position of the tube before feeding, ensuring that the cuff of the tracheostomy tube (if in place) is inflated, and giving the tube feeding slowly. The feeding tube is aspirated periodically to ensure that the feedings are passing through the gastrointestinal tract. Retained or residual feedings increase the risk of aspiration. Patients with retained feedings may benefit from the placement of a gastrostomy tube or a percutaneous endoscopic gastrostomy tube. In a patient with a nasogastric tube, the feeding tube should be placed in the duodenum to reduce the risk of aspiration. For long-term feedings, a gastrostomy tube is preferred. Management of patients with tube feedings is discussed in Chapter 36.

Attaining Bowel and Bladder Control

After a stroke, the patient may have transient urinary incontinence due to confusion, inability to communicate needs, and inability to use the urinal or bedpan because of impaired motor and postural control. Occasionally after a stroke, the bladder becomes atonic, with impaired sensation in response to bladder filling. Sometimes control of the external urinary sphincter is lost or diminished. During this period, intermittent catheterization with sterile technique is carried out. After muscle tone increases and deep tendon reflexes return, bladder tone increases and spasticity of the bladder may develop. Because the patient's sense of awareness is clouded, persistent urinary incontinence or urinary retention may be symptomatic of bilateral brain damage. The voiding pattern is analyzed, and the urinal or bedpan is offered on this pattern or schedule. The upright posture and standing position are helpful for male patients during this aspect of rehabilitation.

Patients may have problems with bowel control, particularly constipation. Unless contraindicated, a high-fiber diet and adequate fluid intake (2 to 3 L/day) should be provided and a regular time (usually after breakfast) should be established for toileting. See Chapter 11 for additional information about bowel and bladder control.

Improving Thought Processes

After a stroke, the patient may have problems with cognitive, behavioral, and emotional deficits related to brain damage. However, in many instances, a considerable degree of function can be recovered, because not all areas of the brain are equally damaged; some remain more intact and functional than others.

After assessment that delineates the patient's deficits, the neuropsychologist, in collaboration with the primary care physician, psychiatrist, nurse, and other professionals, structures a training program using cognitive-perceptual retraining, visual imagery, reality orientation, and cueing procedures to compensate for losses.

The role of the nurse is supportive. The nurse reviews the results of neuropsychological testing, observes the patient's performance and progress, gives positive feedback, and, most importantly, conveys an attitude of confidence and hope. Interventions capitalize on the patient's strengths and remaining abilities while attempting to improve performance of affected functions. Other interventions are similar to those for improving cognitive functioning after a head injury (see Chapter 63).

Improving Communication

Aphasia, which impairs the patient's ability to express himself or herself and to understand what is being said, may become apparent in various ways. The cortical area that is responsible for integrating the myriad pathways required for

the comprehension and formulation of language is called Broca's area. It is located in a convolution adjoining the middle cerebral artery. This area is responsible for control of the combinations of muscular movements needed to speak each word. Broca's area is so close to the left motor area that a disturbance in the motor area often affects the speech area. This is why so many patients who are paralyzed on the right side (due to damage or injury to the left side of the brain) cannot speak, whereas those paralyzed on the left side are less likely to have speech disturbances.

The speech therapist assesses the communication needs of the stroke patient, describes the precise deficit, and suggests the best overall method of communication. Most language intervention strategies can be tailored for the individual patient. The patient is expected to take an active part in establishing goals.

A person with aphasia may become depressed. The inability to talk on the telephone, answer a question, or participate in conversation often causes anger, frustration, fear of the future, and hopelessness. Nursing interventions include strategies to make the atmosphere conducive to communication. This includes being sensitive to the patient's reactions and needs and responding to them in an appropriate manner, while always treating the patient as an adult. The nurse provides strong emotional support and understanding to allay anxiety and frustration.

A common pitfall is for the nurse or other health care team member to complete the thoughts or sentences of the patient. This should be avoided, because it causes the patient to become more frustrated at not being allowed to speak and may deter efforts to practice putting thoughts together and completing sentences. A consistent schedule, routines, and repetition help the patient to function despite significant deficits. A written copy of the daily schedule, a folder of personal information (birth date, address, names of relatives), checklists, and an audiotaped list help improve the patient's memory and concentration. The patient may also benefit from a communication board, which has pictures of common needs and phrases. The board may be translated into any language.

When talking with the patient, it is important for the nurse to gain the patient's attention, speak slowly, and keep the language of instruction consistent. One instruction is given at a time, and time is allowed for the patient to process what has been said. The use of gestures may enhance comprehension. Speaking is thinking out loud, and the emphasis is on thinking. Listening and sorting out incoming messages requires mental effort; the patient must struggle against mental inertia and needs time to organize a response.

In working with the patient with aphasia, the nurse must remember to talk to the patient during care activities. This provides social contact for the patient. Chart 62-4 describes points to keep in mind when communicating with the patient with aphasia.

Chart 62-4 • *Communicating With the Patient With Aphasia*

- Face the patient and establish eye contact.
- Speak in a normal manner and tone.
- Use short phrases, and pause between phrases to allow the patient time to understand what is being said.
- Limit conversation to practical and concrete matters.
- Use gestures, pictures, objects, and writing.
- As the patient uses and handles an object, say what the object is. It helps to match the words with the object or action.
- Be consistent in using the same words and gestures each time you give instructions or ask a question.
- Keep extraneous noises and sounds to a minimum. Too much background noise can distract the patient or make it difficult to sort out the message being spoken.

Maintaining Skin Integrity

The patient who has had a stroke may be at risk for skin and tissue breakdown because of altered sensation and inability to respond to pressure and discomfort by turning and moving. Preventing skin and tissue breakdown requires frequent assessment of the skin, with emphasis on bony areas and dependent parts of the body. During the acute phase, a specialty bed (eg, low-air-loss bed) may be used until the patient can move independently or assist in moving.

A regular turning schedule (eg, every 2 hours) is adhered to even if pressure-relieving devices are used to prevent tissue and skin breakdown. When the patient is positioned or turned, care must be used to minimize shear and friction forces, which cause damage to tissues and predispose the skin to breakdown.

The patient's skin must be kept clean and dry; gentle massage of healthy (nonreddened) skin and adequate nutrition are other factors that help to maintain normal skin and tissue integrity (see Chapter 11).

Improving Family Coping

Family members play an important role in the patient's recovery. Family members are encouraged to participate in counseling and to use support systems that will help with the emotional and physical stress of caring for the patient. Involving others in the patient's care and teaching stress management techniques and methods for maintaining personal health also facilitate family coping.

The family may have difficulty accepting the patient's disability and may be unrealistic in their expectations. They are given information about the expected outcomes and are counseled to avoid doing activities for the patient that he or she can do. They are assured that their love and interest are part of the patient's therapy.

The family needs to be informed that the rehabilitation of the hemiplegic patient requires many months and that progress may be slow. The gains made by the patient in the hospital or rehabilitation unit must be maintained. All caregivers should approach the patient with a supportive and optimistic attitude, focusing on the patient's remaining abilities. The rehabilitation team, the medical and nursing team, the patient, and the family must all be involved in developing attainable goals for the patient at home.

Most relatives of patients with stroke handle the physical changes better than the emotional aspects of care. The family should be prepared to expect occasional episodes of

emotional lability. The patient may laugh or cry easily and may be irritable and demanding or depressed and confused. The nurse can explain to the family that the patient's laughter does not necessarily connote happiness, nor does crying reflect sadness, and that emotional lability usually improves with time.

Helping the Patient Cope With Sexual Dysfunction

Sexual functioning can be profoundly altered by stroke. Although research in this area of stroke management is limited, it appears that patients who have had a stroke consider sexual function important, and many have sexual dysfunction. Sexual dysfunction after stroke is multifactorial. There may be medical reasons for the dysfunction (neurologic and cognitive deficits, previous diseases, medications), as well as various psychosocial factors, including depression. A stroke is such a catastrophic illness that the patient experiences loss of self-esteem and value as a sexual being. These psychosocial factors play an important role in determining sexual drive, activity, and satisfaction after a stroke.

Nurses in the rehabilitation setting play a crucial role in beginning a dialogue between the patient and his or her partner about sexuality after a stroke. In-depth assessments to determine sexual history before and after the stroke should be followed by appropriate interventions. Interventions for the patient and partner focus on providing relevant information, education, reassurance, adjustment of medications, counseling regarding coping skills, suggestions for alternative sexual positions, and a means of sexual expression and satisfaction (Kautz, 2007).

Promoting Home and Community-Based Care

TEACHING PATIENTS SELF-CARE. Patient and family education is a fundamental component of rehabilitation. The nurse provides teaching about stroke, its causes and prevention, and the rehabilitation process. In both acute care and rehabilitation facilities, the focus is on teaching the patient to resume as much self-care as possible. This may entail using assistive devices or modifying the home environment to help the patient live with a disability.

An occupational therapist may be helpful in assessing the home environment and recommending modifications to help the patient become more independent. For example, a shower is more convenient than a tub for the patient with hemiplegia because most patients do not gain sufficient strength to get up and down from a tub. Sitting on a stool of medium height with rubber suction tips allows the patient to wash with greater ease. A long-handled bath brush with a soap container is helpful to the patient who has only one functional hand. If a shower is not available, a stool may be placed in the tub and a portable shower hose attached to the faucet. Handrails may be attached alongside the bathtub and the toilet. Other assistive devices include special utensils for eating, grooming, dressing, and writing (see Chart 62-3).

A program of physical therapy can be beneficial, whether it takes places in the home or in an outpatient program. Recent research has focused on techniques using robotics and constraint-induced movement therapy. Constraint-induced movement therapy involves constraint of the less affected upper limb with a mitt and intensely training the more affected limb. This technique has shown improved arm function in patients who had a stroke 3 to 9 months prior to receiving the treatment (Wolf, Winstein, Miller, et al., 2006). Robot-assisted therapy uses sensorimotor training of the upper limb. This method allows patients to train without the presence of a therapist.

CONTINUING CARE. The recovery and rehabilitation process after stroke may be prolonged and requires patience and perseverance on the part of both the patient and the family. Depending on the specific neurologic deficits resulting from the stroke, the patient at home may require the services of a number of health care professionals. The nurse often coordinates the care of the patient at home and considers the many educational needs of caregivers and patients. The family (often the spouse) requires education as well as assistance in planning and providing care.

The family is advised that the patient may tire easily, may become irritable and upset by small events, and may be less interested in events than expected. Emotional problems associated with stroke are often related to speech dysfunction and the frustrations of being unable to communicate. A speech therapist allows the family to be involved and gives the family practical instructions to help the patient between therapy sessions.

Depression is a common and serious problem in the patient who has had a stroke. Incidence of depression in patients who have had a stroke ranges from less than 10% to more than 50%. Risk factors include increased severity of stroke, a history of depression, and cognitive or physical impairment (Johnson, Minarik, Nyström, et al., 2006). Nurses should identify patients who may be at risk while they are in the hospital. In the home or in the rehabilitation setting, nurses may be involved in coordinating care and referring patients and family to appropriate resources. The family can help by continuing to support the patient and by giving positive reinforcement for the progress that is being made. Antidepressant therapy may help if depression dominates the patient's life.

Community-based stroke support groups may allow the patient and family to learn from others with similar problems and to share their experiences. Support groups take the form of in-person meetings as well as Internet-based support programs. The patient is encouraged to continue hobbies and recreational and leisure interests and to maintain contact with friends to prevent social isolation. All nurses coming in contact with the patient should encourage the patient to keep active, adhere to the exercise program, and remain as self-sufficient as possible.

The nurse should recognize the potential effects of caregiving on the family. Not all families have the adaptive coping skills and adequate psychological functioning necessary for the long-term care of another person. The patient's spouse may be elderly, with his or her own health concerns; in some instances, the patient may have been the provider of care to the spouse. A spouse may have to take on new roles and responsibilities in the relationship and around the home. He or she may also feel a sense of loss (of freedom and leisure time as well as of the marital relationship) (Chart 62-5).

Depression is common in caregivers of stroke survivors, and it may last 18 months or more (Berg, Palomäki,

CHART 62-5 NURSING RESEARCH PROFILE
Spouses Caring for Stroke Survivors

Coombs, U. (2007). Spousal caregiving for stroke survivors. *Journal of Neuroscience Nursing, 39*(2), 112–119.

Purpose

Survivors of a stroke may be discharged to an acute rehabilitation facility or skilled nursing facility. The ultimate goal after a stroke is to have survivors return home. A spouse plays an important role in caring for stroke survivors and also provides much-needed emotional support. The purpose of this study was to explore the phenomenon of spousal caregiving from the perspective of the spousal caregivers (the lived experience of spousal caregivers).

Design

This phenomenologic study was guided by the research question: What is it like for older caregivers to care for a spouse who has survived a stroke? This study used eight interviews from adults who were at least 50 years of age and were caring for a stroke survivor who was at least 1 year poststroke. Data were collected through taped interviews. Each participant had two separate interviews lasting 60 to 120 minutes using an interview guide containing five to six questions. Participants were asked to describe their caregiving experience, and the interview guide questions were used as prompts if needed.

Findings

Five females and three males participated, the mean age was 65 years, and all were married. The number of years caring for their spouses ranged from 1.5 to 5 years. The mean age of the stroke survivors was 68 years. Six themes emerged for this study: experiencing a profound sense of loss, adjusting to a new relationship with the spouse, taking on a new responsibility, feeling the demands of caregiving, having to depend on others, and maintaining hope and optimism.

Nursing Implications

The findings of this study may help nurses working in hospital, rehabilitation, community, or home health care settings understand the impact of caregiving on spouses. More attention should be given to the spouse in the acute care setting, and support resources should be provided. By being aware of the particular concerns of caregivers, nurses may anticipate problems and stresses that caregivers may encounter.

Lönnqvist, et al., 2005). Nurses should assess caregivers for signs of depression (Lightbody, Auton, Baldwin, et al., 2007). Caregivers who are depressed may be more likely to resort to physical or emotional abuse of the patient and to place the patient in a nursing home.

Caregivers may require reminders to attend to their own health concerns and well-being. Even healthy caregivers may find it difficult to maintain a schedule that includes being available around the clock. The nurse encourages the family to arrange for respite care services (planned short-term care to relieve the family from having to provide continuous 24-hour care), which may be available from an adult day care center. Some hospitals also offer weekend respite care that can provide caregivers with needed time for themselves. The nurse involved in home and continuing care also needs to remind the patient and family of the need for respite care as well as continuing health promotion and screening practices.

Evaluation

Expected Patient Outcomes

Expected patient outcomes may include the following:

1. Achieves improved mobility
   a. Avoids deformities (contractures and footdrop)
   b. Participates in prescribed exercise program
   c. Achieves sitting balance
   d. Uses unaffected side to compensate for loss of function of hemiplegic side
2. Reports absence of shoulder pain
   a. Demonstrates shoulder mobility; exercises shoulder
   b. Elevates arm and hand at intervals
3. Achieves self-care; performs hygiene care; uses adaptive equipment
4. Demonstrates techniques to compensate for altered sensory reception, such as turning the head to see people or objects
5. Demonstrates safe swallowing
6. Achieves normal bowel and bladder elimination
7. Participates in cognitive improvement program
8. Demonstrates improved communication
9. Maintains intact skin without breakdown
   a. Demonstrates normal skin turgor
   b. Participates in turning and positioning activities
10. Family members demonstrate a positive attitude and coping mechanisms
    a. Encourage patient in exercise program
    b. Take an active part in rehabilitation process
    c. Contact respite care programs or arrange for other family members to assume some responsibilities for care
11. Develops alternative approaches to sexual expression

Hemorrhagic Stroke

Hemorrhagic strokes account for 15% to 20% of cerebrovascular disorders and are primarily caused by intracranial or subarachnoid hemorrhage. Hemorrhagic strokes are caused by bleeding into the brain tissue, the ventricles, or the subarachnoid space. Primary intracerebral hemorrhage from a spontaneous rupture of small vessels accounts for approximately 80% of hemorrhagic strokes and is caused chiefly by uncontrolled hypertension. Subarachnoid hemorrhage results from a ruptured intracranial **aneurysm** (a weakening in the arterial wall) in about half the cases (Hickey, 2009).

Another common cause of intracerebral hemorrhage in the elderly is cerebral amyloid angiopathy, which involves damage caused by the deposit of beta-amyloid protein in the small and medium-sized blood vessels of the brain. Secondary intracerebral hemorrhage is associated with arteriovenous malformations (AVMs), intracranial aneurysms, intracranial neoplasms, or certain medications (eg, anticoagulants, amphetamines). The mortality rate has been reported to be as high as 48% at 30 days after an intracranial hemorrhage (Flaherty, Haverbusch, Sekar, et al., 2006). Patients who survive the acute phase of care usually have more severe deficits and a longer recovery phase compared to those with ischemic stroke.

Pathophysiology

The pathophysiology of hemorrhagic stroke depends on the cause and type of cerebrovascular disorder. Symptoms are produced when a primary hemorrhage, aneurysm, or AVM presses on nearby cranial nerves or brain tissue or, more dramatically, when an aneurysm or AVM ruptures, causing subarachnoid hemorrhage (hemorrhage into the cranial subarachnoid space). Normal brain metabolism is disrupted by the brain's exposure to blood; by an increase in ICP resulting from the sudden entry of blood into the subarachnoid space, which compresses and injures brain tissue; or by secondary ischemia of the brain resulting from the reduced perfusion pressure and vasospasm that frequently accompany subarachnoid hemorrhage.

Intracerebral Hemorrhage

An intracerebral hemorrhage, or bleeding into the brain tissue, is most common in patients with hypertension and cerebral atherosclerosis, because degenerative changes from these diseases cause rupture of the blood vessel. An intracerebral hemorrhage may also result from certain types of arterial pathology, brain tumors, and the use of medications (eg, oral anticoagulants, amphetamines, and illicit drug use).

Bleeding occurs most commonly in the cerebral lobes, basal ganglia, thalamus, brain stem (mostly the pons), and cerebellum (Hickey, 2009). Occasionally, the bleeding ruptures the wall of the lateral ventricle and causes intraventricular hemorrhage, which is frequently fatal.

Intracranial (Cerebral) Aneurysm

An intracranial (cerebral) aneurysm is a dilation of the walls of a cerebral artery that develops as a result of weakness in the arterial wall. The cause of aneurysms is unknown, although research is ongoing. An aneurysm may be due to atherosclerosis, which results in a defect in the vessel wall with subsequent weakness of the wall; a congenital defect of the vessel wall; hypertensive vascular disease; head trauma; or advancing age.

Any artery within the brain can be the site of a cerebral aneurysm, but these lesions usually occur at the bifurcations of the large arteries at the circle of Willis (Fig. 62-5). The cerebral arteries most commonly affected by an aneurysm are the internal carotid artery (ICA), anterior cerebral artery (ACA), anterior communicating artery (ACoA), posterior communicating artery (PCoA), posterior cerebral artery (PCA), and middle cerebral artery (MCA). Multiple cerebral aneurysms are not uncommon.

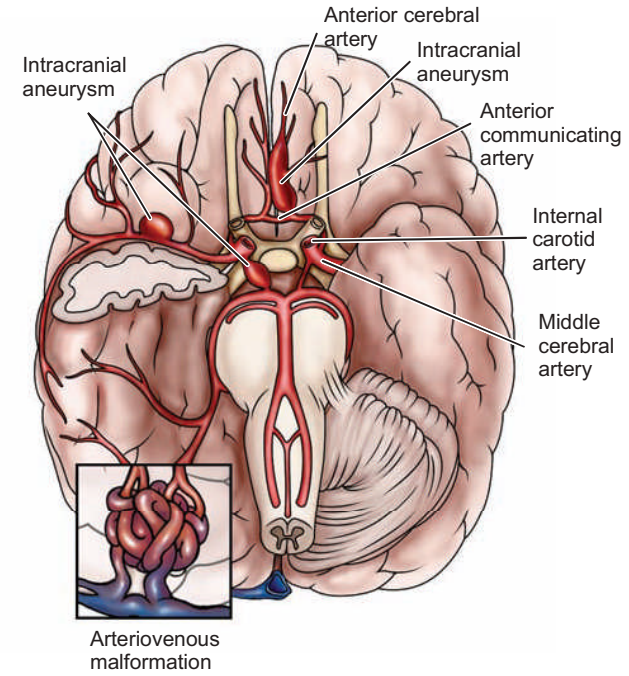

Figure 62-5 Common sites of intracranial aneurysms and an arteriovenous malformation.

Arteriovenous Malformations

Most AVMs are caused by an abnormality in embryonal development that leads to a tangle of arteries and veins in the brain that lacks a capillary bed (see Fig. 62-5). The absence of a capillary bed leads to dilation of the arteries and veins and eventual rupture. AVM is a common cause of hemorrhagic stroke in young people.

Subarachnoid Hemorrhage

A subarachnoid hemorrhage (hemorrhage into the subarachnoid space) may occur as a result of an AVM, intracranial aneurysm, trauma, or hypertension. The most common causes are a leaking aneurysm in the area of the circle of Willis and a congenital AVM of the brain.

Clinical Manifestations

The patient with a hemorrhagic stroke can present with a wide variety of neurologic deficits, similar to the patient with ischemic stroke. The conscious patient most commonly reports a severe headache. A comprehensive assessment reveals the extent of the neurologic deficits. Many of the same motor, sensory, cranial nerve, cognitive, and other functions that are disrupted after ischemic stroke are also altered after a hemorrhagic stroke. Table 62-2 reviews the neurologic deficits frequently seen in stroke patients. Table 62-3 compares the symptoms seen in right hemispheric stroke with those seen in left hemispheric stroke. Other symptoms that may be observed more frequently in patients with acute intracerebral hemorrhage (compared with ischemic stroke) are vomiting, an early sudden change in level of consciousness, and possibly focal seizures due to frequent brain stem involvement (Hickey, 2009).

In addition to the neurologic deficits (similar to those of ischemic stroke), the patient with an intracranial aneurysm or AVM may have some unique clinical manifestations. Rupture of an aneurysm or AVM usually produces a sudden, unusually severe headache and often loss of consciousness for a variable period of time. There may be pain and rigidity of the back of the neck (nuchal rigidity) and spine due to meningeal irritation. Visual disturbances (visual loss, diplopia, ptosis) occur if the aneurysm is adjacent to the oculomotor nerve. Tinnitus, dizziness, and hemiparesis may also occur.

At times, an aneurysm or AVM leaks blood, leading to the formation of a clot that seals the site of rupture. In this instance, the patient may show little neurologic deficit. In other cases, severe bleeding occurs, resulting in cerebral damage, followed rapidly by coma and death.

Prognosis depends on the neurologic condition of the patient, the patient's age, associated diseases, and the extent and location of the hemorrhage or intracranial aneurysm. Subarachnoid hemorrhage from an aneurysm is a catastrophic event with significant morbidity and mortality. Chart 62-6 presents ethical issues related to the patient with a severe hemorrhagic stroke.

Assessment and Diagnostic Findings

Any patient with suspected stroke should undergo a CT scan or MRI to determine the type of stroke, the size and location of the hematoma, and the presence or absence of ventricular blood and hydrocephalus. Cerebral angiography confirms the diagnosis of an intracranial aneurysm or AVM. These tests show the location and size of the lesion and provide information about the affected arteries, veins, adjoining vessels, and vascular branches. Lumbar puncture is performed if there is no evidence of increased ICP, the CT scan results are negative, and subarachnoid hemorrhage must be confirmed. Lumbar puncture in the presence of increased ICP could result in brain stem herniation or rebleeding. When diagnosing a hemorrhagic stroke in a patient younger than 40 years of age, some clinicians obtain a toxicology screen for illicit drug use.

CHART 62-6 *Ethics and Related Issues*

What Are the Ethical Issues Related to DNR Orders After Severe Stroke?

Situation

An 85-year-old patient is admitted with a large intracerebral hemorrhage, severe neurologic deficits, and a past medical history of coronary artery bypass graft surgery, hypertension, atrial fibrillation, and gout. The patient does not have an advanced directive. The attending physician suggests a do-not-resuscitate (DNR) order to the family.

Dilemma

The principle of autonomy for the patient (including death with dignity) conflicts with the principle of beneficence for the health care providers.

Discussion

1. What arguments would you pose in favor of the DNR order?
2. What arguments would you pose against the DNR order?
3. Does the family have the right to refuse?
4. Is a DNR order an example of "patient abandonment" by health care workers, or an attempt to limit treatment and avoid CPR in a patient with an anticipated poor outcome?

Prevention

Primary prevention of hemorrhagic stroke is the best approach and includes managing hypertension and ameliorating other significant risk factors. Control of hypertension, especially in people older than 55 years of age, reduces the risk of hemorrhagic stroke (Luders, 2007). Additional risk factors are increased age, male gender, and excessive alcohol intake. Stroke risk screenings provide an ideal opportunity to lower hemorrhagic stroke risk by identifying high-risk individuals or groups and educating patients and the community about recognition and prevention.

Complications

Potential complications of hemorrhagic stroke include rebleeding or hematoma expansion; cerebral vasospasm resulting in cerebral ischemia; acute hydrocephalus, which results when free blood obstructs the reabsorption of cerebrospinal fluid (CSF) by the arachnoid villi; and seizures.

Cerebral Hypoxia and Decreased Blood Flow

Immediate complications of a hemorrhagic stroke include cerebral hypoxia, decreased cerebral blood flow, and extension of the area of injury. Providing adequate oxygenation of blood to the brain minimizes cerebral hypoxia. Brain function depends on delivery of oxygen to the tissues. Administering supplemental oxygen and maintaining the hemoglobin and hematocrit at acceptable levels will assist in maintaining tissue oxygenation.

Cerebral blood flow is dependent on the blood pressure, cardiac output, and integrity of cerebral blood vessels. Adequate hydration (IV fluids) must be ensured to reduce blood viscosity and improve cerebral blood flow. Extremes of hypertension or hypotension need to be avoided to prevent changes in cerebral blood flow and the potential for extending the area of injury.

A seizure can also compromise cerebral blood flow, resulting in further injury to the brain. Observing for seizure activity and initiating appropriate treatment are important components of care after a hemorrhagic stroke.

Vasospasm

The development of cerebral vasospasm (narrowing of the lumen of the involved cranial blood vessel) is a serious complication of subarachnoid hemorrhage and is the leading cause of morbidity and mortality in those who survive the initial subarachnoid hemorrhage (Kosty, 2005). The mechanism responsible for vasospasm is not clear, but it is associated with increasing amounts of blood in the subarachnoid cisterns and cerebral fissures, as visualized by CT scan. Monitoring for vasospasm may be performed through the use of bedside transcranial Doppler ultrasonography (TCD) or follow-up cerebral angiography.

Vasospasm frequently occurs 3 to 14 days after initial hemorrhage, when the clot undergoes lysis (dissolution), and the chance of rebleeding is increased (Hickey, 2009). It leads to increased vascular resistance, which impedes cerebral blood flow and causes brain ischemia and infarction. The signs and symptoms reflect the areas of the brain involved. Vasospasm is often heralded by a worsening headache, a decrease in level of consciousness (confusion, lethargy, and disorientation), or a new focal neurologic deficit (aphasia, hemiparesis).

Management of vasospasm remains difficult and controversial. It is believed that early surgery to clip the aneurysm prevents rebleeding and that removal of blood from the basal cisterns around the major cerebral arteries may prevent vasospasm. Advances in technology have led to the introduction of interventional neuroradiology for the treatment of aneurysms. Endovascular techniques may be used in selected patients to occlude the artery supplying the aneurysm with a balloon, coils, or other techniques to occlude the aneurysm itself. As more studies on these techniques are completed, their use will increase.

Medication may be effective in the treatment of vasospasm. Based on one theory, that vasospasm is caused by an increased influx of calcium into the cell, medication therapy may be used to block or antagonize this action and prevent or reverse the action of vasospasm if already present. The most frequently used calcium channel blocker is nimodipine (Nimotop) (Devlin, 2008). Another therapy for vasospasm, referred to as "triple-H therapy," is aimed at minimizing the deleterious effects of the associated cerebral ischemia and includes (1) fluid volume expanders (hypervolemia), (2) induced arterial hypertension, and (3) hemodilution (Kosty, 2005).

Increased Intracranial Pressure

An increase in ICP can occur after either an ischemic or a hemorrhagic stroke but almost always follows a subarachnoid hemorrhage, usually because of disturbed circulation of CSF caused by blood in the basal cisterns. Neurologic assessments are performed frequently, and if there is evidence of deterioration from increased ICP (due to cerebral edema, herniation, hydrocephalus, or vasospasm), CSF drainage may be instituted by ventricular catheter drainage. Mannitol may be administered to reduce ICP. When mannitol is used as a long-term measure to control ICP, dehydration and disturbances in electrolyte balance (hyponatremia or hypernatremia; hypokalemia or hyperkalemia) may occur. Mannitol pulls water out of the brain tissue by osmosis and reduces total-body water through diuresis. The patient is monitored for signs of dehydration and for rebound elevation of ICP. Other interventions may include elevating the head of the bed, sedation, and hyperosmolar therapy (discussed in the vasospasm section) (Broderick, Connolly, Feldmann, et al., 2007; Presciutti, 2006).

Hypertension

Hypertension is the most common cause of intracerebral hemorrhage, and its treatment is critical. Specific goals for blood pressure management, which are individualized for each patient, remain controversial. Blood pressure goals may be dependent on the presence of increased ICP. Clinical trials are currently ongoing to further investigate control of blood pressure in intracerebral hemorrhage (Broderick, et al., 2007). Systolic blood pressure may be lowered to prevent hematoma enlargement. If blood pressure is elevated, antihypertensive therapy (labetalol [Trandate], nicardipine [Cardene], nitroprusside [Nitropress], hydralazine [Apresoline]) may be prescribed. During the administration of antihypertensives, arterial hemodynamic monitoring is important to detect and avoid a precipitous drop in blood pressure, which can produce brain ischemia. Stool softeners are used to prevent straining, which can elevate the blood pressure.

Medical Management

The goals of medical treatment for hemorrhagic stroke are to allow the brain to recover from the initial insult (bleeding), to prevent or minimize the risk of rebleeding, and to prevent or treat complications. Management may consist of bed rest with sedation to prevent agitation and stress, management of vasospasm, and surgical or medical treatment to prevent rebleeding. If the bleeding is caused by anticoagulation with warfarin (Coumadin), the INR may be corrected with fresh-frozen plasma and vitamin K. Because seizures can occur after intracerebral hemorrhage, antiseizure agents are often administered prophylactically for a brief period of time. Analgesic agents may be prescribed for head and neck pain. The patient is fitted with sequential compression devices or anti-embolism stockings to prevent deep vein thrombosis (DVT). Fever should be treated. Hyperglycemia should also be treated (an IV insulin drip may be required to achieve control) (Broderick, et al., 2007; Presciutti, 2006). After discharge most patients will require antihypertensive medications to decrease their risk of another intracerebral hemorrhage.

Surgical Management

In many cases, a primary intracerebral hemorrhage is not treated surgically. However, if the diameter of the hematoma exceeds 3 cm and the Glasgow Coma Scale score decreases, surgical evacuation is strongly recommended for the patient with a cerebellar hemorrhage (Broderick, et al., 2007). Surgical evacuation is most frequently accomplished via a craniotomy (see Chapter 61).

The patient with an intracranial aneurysm is prepared for surgical intervention as soon as his or her condition is considered stable. Surgical treatment of the patient with an unruptured aneurysm is an option. The goal of surgery is to prevent bleeding in an unruptured aneurysm or further bleeding in an already ruptured aneurysm. This objective is accomplished by isolating the aneurysm from its circulation or by strengthening the arterial wall. An aneurysm may be excluded from the cerebral circulation by means of a ligature or a clip across its neck. If this is not anatomically possible, the aneurysm can be reinforced by wrapping it with some substance to provide support and induce scarring.

Less invasive endovascular treatments are now being used for aneurysms. These procedures are performed by neurosurgeons in neurointerventional radiology facilities. Two procedures include endovascular treatment (occlusion of the parent artery) and aneurysm coiling (obstruction of the aneurysm site with a coil). Although these techniques are

associated with lower risks than intracranial surgery in general, secondary stroke and rupture of the aneurysm are still potential complications.

Postoperative complications include psychological symptoms (disorientation, amnesia, **Korsakoff's syndrome,** personality changes), intraoperative embolization, postoperative internal artery occlusion, fluid and electrolyte disturbances (from dysfunction of the neurohypophyseal system), and gastrointestinal bleeding.

NURSING PROCESS

The Patient With a Hemorrhagic Stroke

Assessment

A complete neurologic assessment is performed initially and includes evaluation for the following:

- Altered level of consciousness
- Sluggish pupillary reaction
- Motor and sensory dysfunction
- Cranial nerve deficits (extraocular eye movements, facial droop, presence of ptosis)
- Speech difficulties and visual disturbance
- Headache and nuchal rigidity or other neurologic deficits

All patients should be monitored in the intensive care unit after an intracerebral or subarachnoid hemorrhage. Neurologic assessment findings are documented and reported as indicated. The frequency of these assessments varies depending on the patient's condition. Any changes in the patient's condition require reassessment and thorough documentation; changes should be reported immediately.

Alteration in level of consciousness often is the earliest sign of deterioration in a patient with a hemorrhagic stroke. Because nurses have the most frequent contact with patients, they are in the best position to detect subtle changes. Mild drowsiness and slight slurring of speech may be early signs that the level of consciousness is deteriorating.

Diagnosis

Nursing Diagnoses

Based on the assessment data, the patient's major nursing diagnoses may include the following:

- Ineffective tissue perfusion (cerebral) related to bleeding or vasospasm
- Disturbed sensory perception related to medically imposed restrictions (aneurysm precautions)
- Anxiety related to illness and/or medically imposed restrictions (aneurysm precautions)

Collaborative Problems/Potential Complications

Based on the assessment data, potential complications that may develop include the following:

- Vasospasm
- Seizures
- Hydrocephalus
- Rebleeding
- Hyponatremia

Planning and Goals

The goals for the patient may include improved cerebral tissue perfusion, relief of sensory and perceptual deprivation, relief of anxiety, and the absence of complications.

Nursing Interventions

Optimizing Cerebral Tissue Perfusion

The patient is closely monitored for neurologic deterioration resulting from recurrent bleeding, increasing ICP, or vasospasm. A neurologic flow record is maintained. The blood pressure, pulse, level of consciousness (an indicator of cerebral perfusion), pupillary responses, and motor function are checked hourly. Respiratory status is monitored, because a reduction in oxygen in areas of the brain with impaired autoregulation increases the chances of a cerebral infarction. Any changes are reported immediately.

IMPLEMENTING ANEURYSM PRECAUTIONS. Cerebral aneurysm precautions are implemented for the patient with a diagnosis of aneurysm to provide a nonstimulating environment, prevent increases in ICP, and prevent further bleeding. The patient is placed on immediate and absolute bed rest in a quiet, nonstressful environment, because activity, pain, and anxiety elevate the blood pressure, which increases the risk for bleeding. Visitors, except for family, are restricted.

The head of the bed is elevated 15 to 30 degrees to promote venous drainage and decrease ICP. Some neurologists, however, prefer that the patient remain flat to increase cerebral perfusion.

Any activity that suddenly increases the blood pressure or obstructs venous return is avoided. This includes the Valsalva maneuver, straining, forceful sneezing, pushing oneself up in bed, acute flexion or rotation of the head and neck (which compromises the jugular veins), and cigarette smoking. Any activity requiring exertion is contraindicated. The patient is instructed to exhale through the mouth during voiding or defecation to decrease strain. No enemas are permitted, but stool softeners and mild laxatives are prescribed. Both prevent constipation, which would cause an increase in ICP, as would enemas. Dim lighting is helpful, because photophobia (visual intolerance of light) is common. Coffee and tea, unless decaffeinated, are usually eliminated.

Anti-embolism stockings or sequential compression devices may be prescribed to decrease the incidence of DVT resulting from immobility. The legs are observed for signs and symptoms of DVT (tenderness, redness, swelling, warmth, and edema), and abnormal findings are reported.

The nurse administers all personal care. The patient is fed and bathed to prevent any exertion that might increase the blood pressure. External stimuli are kept to a minimum, including no television, no radio, and no reading. Visitors are restricted in an effort to keep the patient as quiet as possible. This precaution must be individualized based on the patient's condition and response to visitors. A sign indicating this restriction should be placed on the door of the room, and the restrictions should be discussed with both patient and family. The purpose of aneurysm precautions should be thoroughly explained to both the patient (if possible) and family.

Relieving Sensory Deprivation and Anxiety

Sensory stimulation is kept to a minimum for patients on aneurysm precautions. For patients who are awake, alert, and oriented, an explanation of the restrictions helps reduce the patient's sense of isolation. Reality orientation is provided to help maintain orientation.

Keeping the patient well informed of the plan of care provides reassurance and helps minimize anxiety. Appropriate reassurance also helps relieve the patient's fears and anxiety. The family also requires information and support.

Monitoring and Managing Potential Complications

VASOSPASM. The patient is assessed for signs of possible vasospasm: intensified headaches, a decrease in level of responsiveness (confusion, disorientation, lethargy), or evidence of aphasia or partial paralysis. These signs may develop several days after surgery or on the initiation of treatment and must be reported immediately. If vasospasm is diagnosed, calcium channel blockers or fluid volume expanders may be prescribed.

SEIZURES. Seizure precautions are maintained for every patient who may be at risk for seizure activity. Should a seizure occur, maintaining the airway and preventing injury are the primary goals. Medication therapy is initiated at this time, if not already prescribed. The medication of choice for many years has been phenytoin (Dilantin). Its use is being questioned based on the results of research including 527 patients that suggested that phenytoin may increase functional and cognitive disability after subarachnoid hemorrhage (Naidech, Kreiter, Janjua, et al., 2005).

HYDROCEPHALUS. Blood in the subarachnoid space or ventricles impedes the circulation of CSF, resulting in hydrocephalus. A CT scan that indicates dilated ventricles confirms the diagnosis. Hydrocephalus can occur within the first 24 hours (acute) after subarachnoid hemorrhage or several days (subacute) to several weeks (delayed) later. Symptoms vary according to the time of onset and may be nonspecific. Acute hydrocephalus is characterized by sudden onset of stupor or coma and is managed with a ventriculostomy drain to decrease ICP. Symptoms of subacute and delayed hydrocephalus include gradual onset of drowsiness, behavioral changes, and ataxic gait. A ventriculoperitoneal shunt is surgically placed to treat chronic hydrocephalus. Changes in patient responsiveness are reported immediately.

REBLEEDING. The rate of recurrent hemorrhage is approximately 2% after a primary intracerebral hemorrhage. Hypertension is the most serious risk factor, suggesting the importance of appropriate antihypertensive treatment.

Aneurysm rebleeding occurs most frequently during the first 2 weeks after the initial hemorrhage and is considered a major complication. Symptoms of rebleeding include sudden severe headache, nausea, vomiting, decreased level of consciousness, and neurologic deficit. Rebleeding is confirmed by CT scan. Blood pressure is carefully maintained with medications. The most effective preventive treatment is to secure the aneurysm if the patient is a candidate for surgery or endovascular treatment.

HYPONATREMIA. After subarachnoid hemorrhage, hyponatremia is found in up to 30% of patients (Naval, Stevens, Mirski, et al., 2006). Laboratory data must be checked frequently, and hyponatremia (defined as a serum sodium concentration of less than 135 mEq/L) must be identified as early as possible. The patient's primary health care provider needs to be notified of a low serum sodium level that has persisted for 24 hours or longer. The patient is then evaluated for syndrome of inappropriate antidiuretic hormone (SIADH) or cerebral salt-wasting syndrome. (SIADH is described in Chapter 14.) Cerebral salt-wasting syndrome occurs when the kidneys are unable to conserve sodium and volume depletion results. The treatment most often is the use of hypertonic 3% saline.

Promoting Home and Community-Based Care

TEACHING PATIENTS SELF-CARE. The patient and family are provided with information that will enable them to cooperate with the care and restrictions required during the acute phase of hemorrhagic stroke and to prepare them to return home. Patient and family teaching includes information about the causes of hemorrhagic stroke and its possible consequences. In addition, the patient and family are informed about the medical treatments that are implemented, including surgical intervention if warranted, and the importance of interventions taken to prevent and detect complications (ie, aneurysm precautions, close monitoring of the patient). Depending on the presence and severity of neurologic impairment and other complications resulting from the stroke, the patient may be transferred to a rehabilitation unit or center for additional patient and family teaching about strategies to regain self-care ability. Teaching addresses the use of assistive devices or modification of the home environment to help the patient live with the disability. Modifications of the home may be required to provide a safe environment.

CONTINUING CARE. The acute and rehabilitation phase of care focuses on obvious needs, issues, and deficits for the patient with a hemorrhagic stroke. The patient and family are reminded of the importance of following recommendations to prevent further hemorrhagic stroke and keeping follow-up appointments with health care providers for monitoring of risk factors. Referral for home care may be warranted to assess the home environment and the ability of the patient and to ensure that the patient and family are able to manage at home. Home visits provide opportunities to monitor the physical and psychological status of the patient and the ability of the family to cope with any alterations in the patient's status. In addition, the home care nurse reminds the patient and family of the importance of continuing health promotion and screening practices. Chart 62-7 lists teaching points for the patient recovering from a stroke.

Evaluation

Expected Patient Outcomes

Expected patient outcomes may include the following:

1. Demonstrates intact neurologic status and normal vital signs and respiratory patterns
   a. Is alert and oriented to time, place, and person

CHART 62-7 HOME CARE CHECKLIST

The Patient Recovering From a Stroke

At the completion of the home care instruction, the patient or caregiver will be able to:	PATIENT	CAREGIVER
• Discuss measures to prevent subsequent strokes.	✔	✔
• Identify signs and symptoms of specific complications.	✔	✔
• Identify potential complications and discuss measures to prevent them (blood clots, aspiration, pneumonia, urinary tract infection, fecal impaction, skin breakdown, contracture).	✔	✔
• Identify psychosocial consequences of stroke and appropriate interventions.	✔	✔
• Identify safety measures to prevent falls.	✔	✔
• State names, doses, indications, and side effects of medications.	✔	✔
• Demonstrate adaptive techniques for accomplishing ADLs.	✔	✔
• Demonstrate swallowing techniques (for patients with dysphagia).	✔	✔
• Demonstrate care of enteric feeding tube, if applicable.	✔	✔
• Demonstrate home exercises, use of splints or orthotics, proper positioning, and frequent repositioning.	✔	✔
• Describe procedures for maintaining skin integrity.	✔	✔
• Demonstrate indwelling catheter care, if applicable. Describe a bowel and bladder elimination program as appropriate.	✔	✔
• Identify appropriate recreational or diversional activities, support groups, and community resources.	✔	✔

   b. Demonstrates normal speech patterns and intact cognitive processes
   c. Demonstrates normal and equal strength, movement, and sensation of all four extremities
   d. Exhibits normal deep tendon reflexes and pupillary responses
2. Demonstrates normal sensory perceptions
   a. States rationale for aneurysm precautions
   b. Exhibits clear thought processes
3. Exhibits reduced anxiety level
   a. Is less restless
   b. Exhibits absence of physiologic indicators of anxiety (eg, has normal vital signs; normal respiratory rate; absence of excessive, fast speech)
4. Is free of complications
   a. Exhibits absence of vasospasm
   b. Exhibits normal vital signs and neuromuscular activity without seizures
   c. Verbalizes understanding of seizure precautions
   d. Exhibits normal mental status and normal motor and sensory status
   e. Reports no visual changes

CRITICAL THINKING EXERCISES

1 A patient had symptoms of an ischemic stroke approximately 1 hour ago and is undergoing a CT scan. What are the time frames, criteria, and dosage for t-PA administration? What nursing assessments and actions would you take? What is your rationale for these assessments and actions?

2 A 58-year-old man is admitted with an ischemic stroke and has left-sided hemiplegia. He is concerned about how to resume sexual relations with his wife and the possibility of not being able to resume sexual intimacy. Identify possible causes of sexual dysfunction after a stroke. What interventions can the nurse implement to address his concerns?

3 A 50-year-old patient with a history of hypertension is expected to be discharged to home today after a 7-day stay for a stroke. She has residual right-sided weakness and a visual field deficit. What teaching would be indicated to prevent another stroke? What resources may be needed to enable her to go home as scheduled?

EBP **4** A patient is admitted to the hospital following a hemorrhagic stroke and is at high risk for vasospasm. What medical and nursing measures should be implemented to prevent vasospasm? Identify the evidence for and the criteria used to evaluate the strength of the evidence for the specific measures identified for prevention of vasospasm.

The Smeltzer suite offers these additional resources to enhance learning and facilitate understanding of this chapter:

- thePoint online resource, thepoint.lww.com/Smeltzer12E
- Student CD-ROM included with the book
- *Study Guide to Accompany Brunner & Suddarth's Textbook of Medical-Surgical Nursing*
- *Handbook for Brunner & Suddarth's Textbook of Medical-Surgical Nursing*

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**Asterisks indicate nursing research.*
***Double asterisks indicate classic references.*

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RESOURCES

American Association of Neuroscience Nurses, www.aann.org
American Stroke Association, a Division of the American Heart Association, www.strokeassociation.org
National Institute of Neurological Disorders and Stroke, www.ninds.nih.gov
National Stroke Association, www.stroke.org