# CHAPTER 10 Nursing Care of Clients with Altered Fluid, Electrolyte, and Acid–Base Balance

## LEARNING OUTCOMES

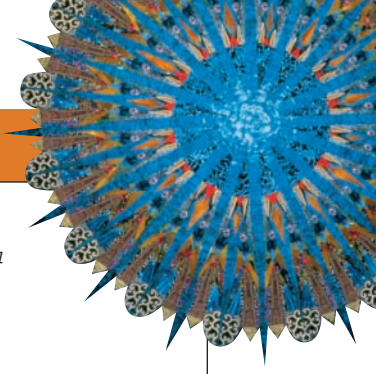
- Describe the functions and regulatory mechanisms that maintain water and electrolyte balance in the body.
- Compare and contrast the causes, effects, and care of the client with fluid volume or electrolyte imbalance.
- Explain the pathophysiology and manifestations of imbalances of sodium, potassium, calcium, magnesium, and phosphorus.
- Describe the causes and effects of acid–base imbalances.

## CLINICAL COMPETENCIES

- Assess and monitor fluid, electrolyte, and acid–base balance for assigned clients.
- Administer fluids and medications knowledgeably and safely.
- Determine priority nursing diagnoses, based on assessment data, to select and implement individualized nursing interventions.
- Provide client and family teaching about diet and medications used to restore, promote, and maintain fluid, electrolyte, and acid–base balance.
- Integrate interdisciplinary care into care of clients with altered fluid, electrolyte, and acid–base balance.

**MEDIALINK**

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



KEY TERMS

**acidosis,** *238*
**acids,** *238*
**active transport,** *200*
**alkalis,** *238*
**alkalosis,** *238*
**anasarca,** *209*
**anion gap,** *243*
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Changes in the normal distribution and composition of body fluids often occur in response to illness and trauma. These changes affect fluid balance of the intracellular and extracellular compartments of the body, the concentration of electrolytes within fluid compartments, and the body's hydrogen ion concentration (pH). Normal physiologic processes depend on a relatively stable state in the internal environment of the body. The fluid volume, electrolyte composition, and pH of both intracellular and extracellular spaces must remain constant within a relatively narrow range to maintain health and life.

**Homeostasis** is the body's tendency to maintain a state of physiologic balance in the presence of constantly changing conditions. Homeostasis is necessary if the body is to function optimally at a cellular level and as a total organism. Homeostasis depends on multiple factors in both the external and internal environments, such as available oxygen in the air and nutrients in food, as well as normal body temperature, respiration, and digestive processes. The normal volume, composition, distribution, and pH of body fluids reflect a state of homeostasis.

Changes in the normal volume of fluids, their composition, distribution, and relative acidity or alkalinity have the potential to disrupt most functional health patterns. Imbalances of fluids, electrolytes, and pH affect the ability to maintain activities of daily living (the Activity-Exercise Pattern), think clearly (the Cognitive-Perceptual Pattern), and engage in self-care (the Health Perception-Health Management Pattern). Conversely, alterations in a number of health patterns affect the ability to maintain homeostasis. Alterations in the Nutritional-Metabolic Pattern affect the ability to consume adequate food and fluids. Disruptions of the Elimination Pattern may lead to retention or loss of excess amounts of fluids and electrolytes. Disrupted heart or respiratory function, which falls within the Activity-Exercise Pattern, has the potential to affect fluid, electrolyte, and acid–base balance.

The goal in managing fluid, electrolyte, and acid–base imbalances is to reestablish and maintain a normal balance. Nursing care includes identifying and assessing clients who are likely to develop imbalances, monitoring clients for early manifestations, and implementing collaborative and nursing interventions to prevent or correct imbalances. Effective nursing interventions require an understanding of the multiple processes that maintain fluid, electrolyte, and acid–base balance and an understanding of the causes and treatment of imbalances that occur.

Mechanisms that maintain normal fluid and electrolyte balance are discussed first, followed by sections on fluid imbalances and electrolyte imbalances. Discussion of normal acid–base balance precedes discussion of acid–base imbalances. Case studies related to selected fluid, electrolyte, and acid–base disorders are found throughout the chapter.

MEDIALINK Fluid Balance Animation

## OVERVIEW OF NORMAL FLUID AND ELECTROLYTE BALANCE

Fluid and electrolyte balance in the body involves regulatory mechanisms that maintain the composition, distribution, and movement of fluids and electrolytes. This section provides an overview of fluid and electrolyte balance in the body. It is followed by discussion of fluid volume and electrolyte balance disorders.

### Body Fluid Composition

Body fluid is composed of water and various dissolved substances (solutes).

#### Water

Water is the primary component of body fluids. It functions in several ways to maintain normal cellular function. Water:

- Provides a medium for the transport and exchange of nutrients and other substances such as oxygen, carbon dioxide, and metabolic wastes to and from cells.
- Provides a medium for metabolic reactions within cells.
- Assists in regulating body temperature through the evaporation of perspiration.
- Provides form for body structure and acts as a shock absorber.
- Provides insulation.
- Acts as a lubricant.

Total body water constitutes about 60% of the total body weight, but this amount varies with age, gender, and the amount of body fat. Total body water decreases with aging; in people over age 65, body water may decrease to 45% to 50% of total body weight. Fat cells contain comparatively little water: In the

**TABLE 10–1 Balanced Fluid Gain and Loss for an Adult**

| | SOURCE | AMOUNT (mL) |
|---|---|---|
| Gain | Fluids taken orally | 1200 |
| | Water in food | 1000 |
| | Water as by-product of food metabolism | 300 |
| | | ↓ |
| | Total | 2500 |
| | | ↑ |
| Loss | Urine | 1500 |
| | Feces | 200 |
| | Perspiration | 300 |
| | Respiration | 500 |

person who is obese, the proportion of water to total body weight is less than in the person of average weight; in a person who is very thin, the proportion of water to total body weight is greater than in the person of average weight. Adult females have a greater ratio of fat to lean tissue mass than adult males; therefore, they have a lower percentage of body water content.

To maintain normal fluid balance, body water intake and output should be approximately equal. The average fluid intake and output usually is about 2500 mL over a 24-hour period. Food and fluids consumed provide the majority of water gain; carbohydrate metabolism and other metabolic processes produce an additional small amount.

Urine production and excretion account for most water loss. The average daily urine output is 1500 mL in adults. At least 400 mL of highly concentrated urine per day is required to excrete metabolic wastes produced by the body. *Insensible* water loss (which normally cannot be measured) occurs through the skin, lungs, and feces. These losses, while normally small, can increase significantly during exercise, when environmental temperatures are high, and during illness that increases the respiratory rate, perspiration, or gastrointestinal (GI) losses (particularly diarrhea). Table 10–1 shows the sources of fluid gain and loss.

### Electrolytes

Body fluids contain both water molecules and chemical compounds. These chemical compounds can either remain intact in solution or separate (dissociate) into discrete particles. **Electrolytes** are substances that dissociate in solution to form charged particles called ions. *Cations* are positively charged electrolytes; *anions* are negatively charged electrolytes. For example, sodium chloride (NaCl) in solution dissociates into a sodium ion, a cation carrying a positive charge ($Na^+$); and a chloride ion, an anion carrying a negative charge ($Cl^-$). Electrolytes may be *univalent,* with only one unit of electrical charge, such as sodium ($Na^+$) and chloride ($Cl^-$); or they may be *divalent,* carrying two units of electrical charge, such as magnesium ($Mg^{2+}$) and phosphate ($HPO_4^{2-}$).

Electrolytes have many functions. They:

- Assist in regulating water balance.
- Help regulate and maintain acid–base balance.
- Contribute to enzyme reactions.
- Are essential for neuromuscular activity.

The concentration of electrolytes in body fluids generally is measured in milliequivalents per liter of water (mEq/L). A *milliequivalent* is a measure of the chemical combining power of the ion. For example, 100 mEq of sodium ($Na^+$) can combine with 100 mEq of chloride ($Cl^-$) to form sodium chloride (NaCl). Sodium, potassium, and chloride usually are measured in milliequivalents. In some cases, the amount of an electrolyte in body fluid may be measured by weight in milligrams per 100 mL (1 deciliter [dL]) of water (mg/dL). Calcium, magnesium, and phosphorus often are measured by weight in milligrams per deciliter. Other laboratories use the International System of Measurements, or SI units.

## Body Fluid Distribution

Body fluid is classified by its location inside or outside of cells. *Intracellular fluid* (ICF) is found within cells. It accounts for approximately 40% of total body weight (Figure 10–1 ■). ICF is essential for normal cell function, providing a medium for metabolic processes. *Extracellular fluid* (ECF) is located outside of cells. It accounts for approximately 20% of the total body weight. ECF is classified by location:

- Interstitial fluid is located in the spaces between most of the cells of the body. It accounts for approximately 15% of total body weight.
- Intravascular fluid, called *plasma*, is contained within the arteries, veins, and capillaries. It accounts for approximately 5% of total body weight.

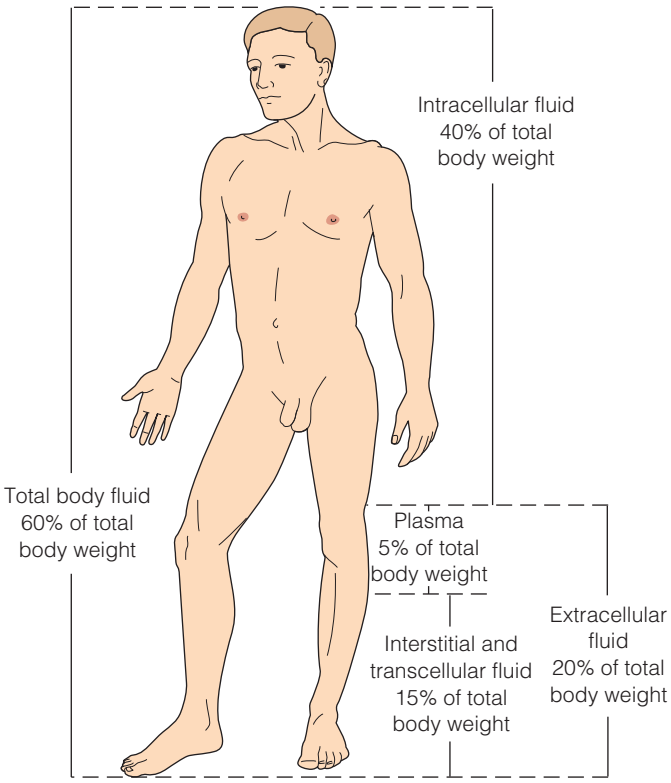


**Figure 10–1** ■ The major fluid compartments of the body.

- Transcellular fluid includes urine; digestive secretions; perspiration; and cerebrospinal, pleural, synovial, intraocular, gonadal, and pericardial fluids.

A trace amount of water is found in bone, cartilage, and other dense connective tissues; this water is not exchangeable with other body fluids.

ECF is the transport medium that carries oxygen and nutrients to and waste products from the cells. For example, plasma transports oxygen from the lungs and glucose from the digestive system to the tissues. These solutes diffuse through the capillary wall into the interstitial space, and from there across the cell membrane into the cells. Waste products of metabolism (e.g., carbon dioxide and hydrogen ion) diffuse from the intracellular space into the interstitial space, and from there into plasma via the capillary walls. Plasma then transports these waste products to the lungs and kidneys for elimination.

Although the overall concentration of solutes in ICF and ECF is nearly identical, the concentration of specific electrolytes differs significantly between these compartments, as shown in Figure 10–2 ■. ICF contains high concentrations of potassium ($K^+$), magnesium ($Mg^{2+}$), and phosphate ($PO_4^{2-}$), as well as other solutes such as glucose and oxygen. Sodium ($Na^+$), chloride ($Cl^-$), and bicarbonate ($HCO_3^-$) are the principal extracellular electrolytes. The high sodium concentration in ECF is essential to regulating body fluid volume. The concentration of potassium in ECF is low. There is a minimal difference in electrolyte concentration between plasma and interstitial fluid. Normal values for electrolytes in plasma are shown in Table 10–2.

The body fluid compartments are separated by several types of membranes:

- Cell membranes separate interstitial fluid from intracellular fluid.
- Capillary membranes separate plasma from interstitial fluid.
- Epithelial membranes separate transcellular fluid from interstitial fluid and plasma. These membranes include the mucosa of the stomach, intestines, and gallbladder; the pleural, peritoneal, and synovial membranes; and the tubules of the kidney.

A cell membrane consists of layers of lipid and protein molecules. The layering of these molecules controls the passage of fluid and solutes between the cell and interstitial fluid. The cell membrane is selectively permeable; that is, it allows the passage of water, oxygen, carbon dioxide, and small water-soluble molecules, but bars proteins and other intracellular colloids.

The capillary membrane separating the plasma from the interstitial space is made of squamous epithelial cells. Pores in the membrane allow solute molecules (such as glucose and sodium), dissolved gases, and water to cross the membrane. Minute amounts of albumin and other proteins can also pass through the pores of a capillary membrane, but normally plasma proteins stay in the intravascular compartment.

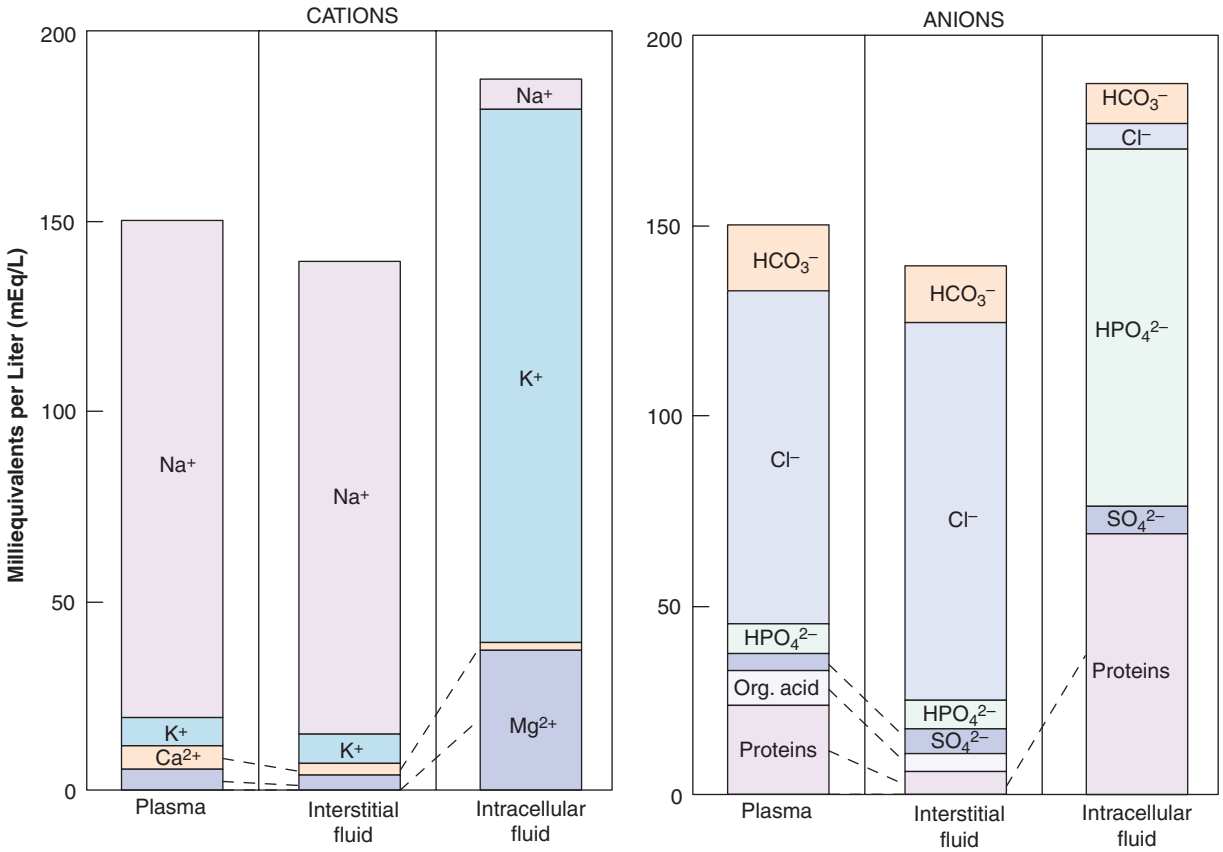


**Figure 10–2** ■ Electrolyte composition (cations and anions) of body fluid compartments.

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**TABLE 10–2 Normal Values for Electrolytes and Serum Osmolality**

| SERUM COMPONENT | VALUES | |
|---|---|---|
| | CONVENTIONAL | SI |
| Electrolytes | | |
| Sodium ($Na^+$) | 135–145 mEq/L | 135–145 mmol/L |
| Chloride ($Cl^-$) | 98–106 mEq/L | 98–106 mmol/L |
| Bicarbonate ($HCO_3^-$) | 22–26 mEq/L | 22–26 mmol/L |
| Calcium ($Ca^{2+}$) (total) | 8.5–10 mg/dL | 2.1–2.6 mmol/L |
| Potassium ($K^+$) | 3.5–5.0 mEq/L | 3.5–5.0 mmol/L |
| Phosphate/inorganic phosphorus ($PO_4^{-2}$) | 1.7–2.6 mEq/L (2.5–4.5 mg/dL) | 0.8–1.5 mmol/L |
| Magnesium ($Mg^{2+}$) | 1.6–2.6 mg/dL (1.3–2.1 mEq/L) | 0.8–1.3 mmol/L |
| Serum osmolality | 275–295 mOsm/kg | 275–295 mmol/kg |

## Body Fluid Movement

Four chemical and physiologic processes control the movement of fluid, electrolytes, and other molecules across membranes between the intracellular and interstitial space and the interstitial space and plasma. These processes are osmosis, diffusion, filtration, and active transport.

***OSMOSIS*** The process by which water moves across a selectively permeable membrane from an area of lower solute concentration to an area of higher solute concentration is **osmosis** (Figure 10–3 ■). A *selectively permeable membrane* allows water molecules to cross but is relatively impermeable to dissolved substances (*solutes*). Osmosis continues until the solute concentration on both sides of the membrane is equal. For example, if pure water and a sodium chloride solution are separated by a selectively permeable membrane, then water molecules will move across the membrane to the sodium chloride solution. Osmosis is the primary process that controls body fluid movement between the ICF and ECF compartments.

**Osmolarity and Osmolality** The concentration of a solution may be expressed as the osmolarity or osmolality of the solution. *Osmolarity* refers to the amount of solutes per liter of solution (by volume); it is reported in milliosmoles per liter (mOsm/L) in a solution. *Osmolality* refers to the number of solutes per kilogram of water (by weight); it is reported in milliosmoles per kilogram (mOsm/kg). Because osmotic activity in the body is regulated by the number of active particles (solutes) per kilogram of water, osmolality is used to describe the concentration of body fluids. The normal osmolality of both ICF and ECF ranges between 275 and 295 mOsm/kg. The osmolality of the ECF depends chiefly on sodium concentration. Serum osmolality may be estimated by doubling the serum sodium concentration (approximately 142 mEq/L). Glucose and urea contribute to the osmolality of ECF, although to a lesser extent than sodium.

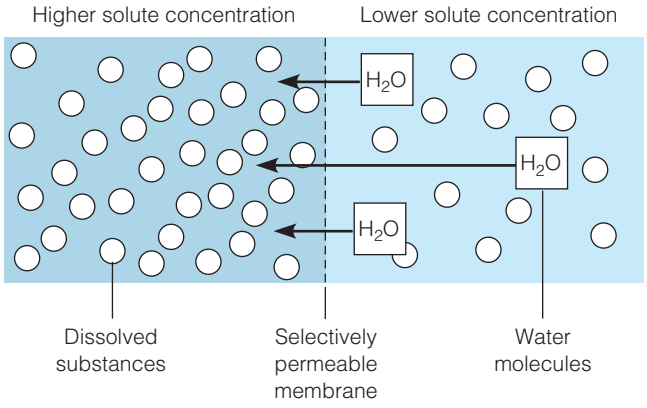


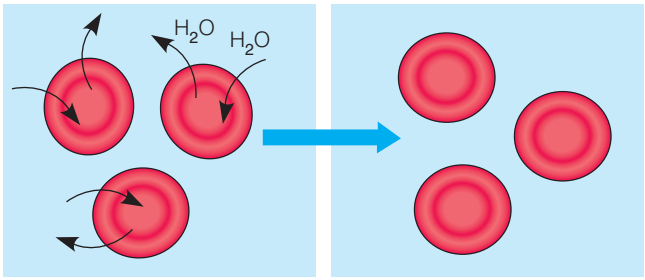

**Figure 10–3** ■ Osmosis. Water molecules move through a selectively permeable membrane from an area of low solute concentration to an area of high solute concentration.

MEDIALINK Osmosis Animation

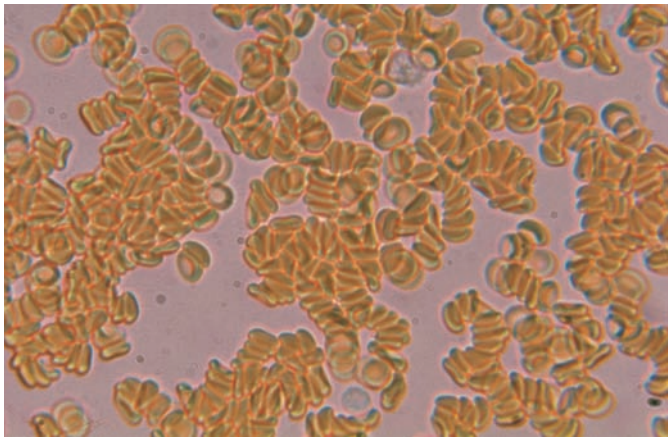
**Osmotic Pressure and Tonicity** The power of a solution to draw water across a membrane is known as the *osmotic pressure* of the solution. The composition of interstitial fluid and intravascular plasma is essentially the same except for a higher concentration of proteins in the plasma. These proteins (especially albumin) exert colloid osmotic pressure (also called oncotic pressure), pulling fluid from the interstitial space into the intravascular compartment. Because the osmolality of intravascular and interstitial fluid is essentially identical, the osmotic activity of plasma proteins is important in maintaining fluid balance between the interstitial and intravascular spaces, helping hold water within the vascular system.

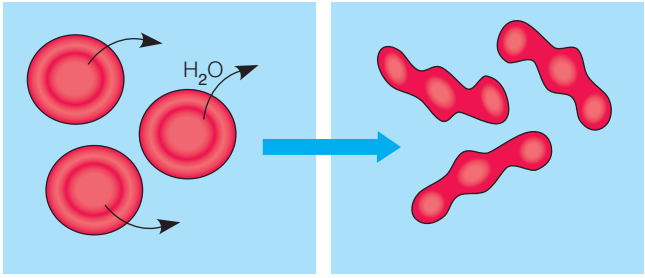
*Tonicity* refers to the effect a solution's osmotic pressure has on water movement across the cell membrane of cells within that solution. *Isotonic* solutions have the same concentration of solutes as plasma. Cells placed in an isotonic solution will neither shrink nor swell because there is no net gain or loss of water within the cell, and no change in cell volume (Figure 10–4A ■). Normal saline (0.9% sodium chloride solution) is an example of an isotonic solution.

*Hypertonic* solutions have a greater concentration of solutes than plasma. In their presence, water is drawn out of a cell, causing it to shrink (Figure 10–4B). A 3% sodium chloride solution is hypertonic. *Hypotonic* solutions (such as 0.45% sodium chloride) have a lower solute concentration than plasma (Figure 10–4C). When red blood cells are placed in a

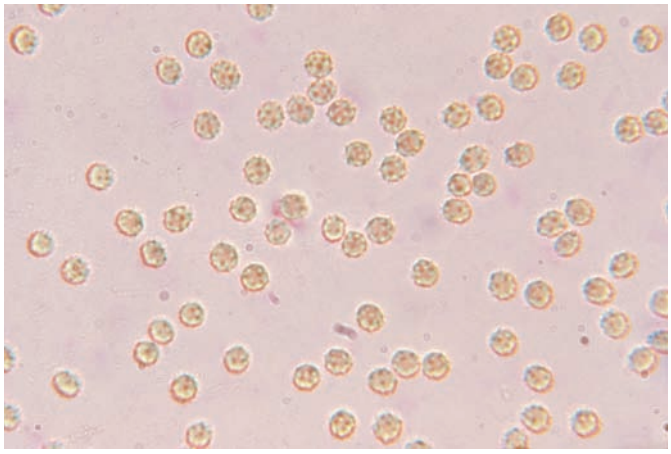


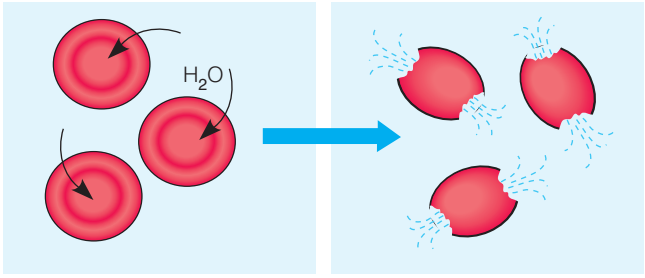

**A** Isotonic solution






**B** Hypertonic solution






**C** Hypotonic solution

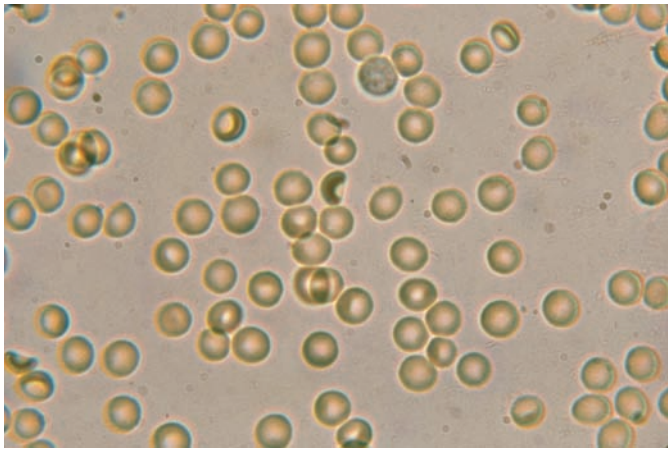


**Figure 10–4** ■ The effect of tonicity on red blood cells. *A,* In an isotonic solution, RBCs neither gain nor lose water, retaining their normal biconcave shape. *B,* In a hypertonic solution, cells lose water and shrink in size. *C,* In a hypotonic solution, cells absorb water and may burst (hemolysis).

hypotonic solution, water moves into the cells, causing them to swell and rupture (*hemolyze*).

The concepts of osmotic draw and tonicity are important in understanding the pathophysiologic changes that occur with fluid and electrolyte imbalances, as well as treatment measures. For example, an increased sodium concentration of extracellular fluid pulls water from the ICF compartment into the ECF compartment, causing cells to shrink. In this case, administering a hypotonic intravenous solution to reduce the sodium concentration and osmolality of ECF will facilitate water movement back into the cells.

***DIFFUSION*** The process by which solute molecules move from an area of high solute concentration to an area of low solute concentration to become evenly distributed is **diffusion** (Figure 10–5 ■). The two types of diffusion are simple and facilitated diffusion. *Simple diffusion* occurs by the random

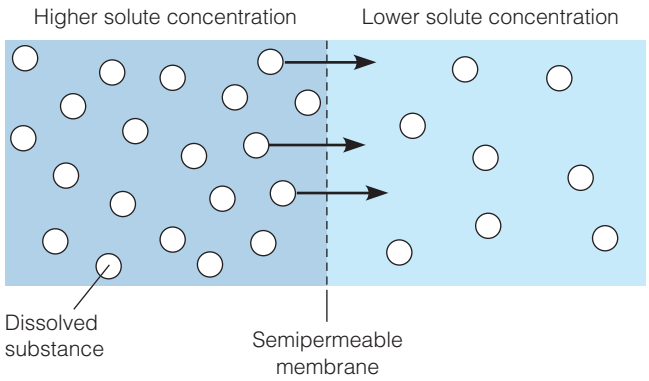



**Figure 10–5** ■ Diffusion. Solute molecules move through a semipermeable membrane from an area of high solute concentration to an area of low solute concentration.

movement of particles through a solution. Water, carbon dioxide, oxygen, and solutes move between plasma and the interstitial space by simple diffusion through the capillary membrane. Water and solutes move into the cell by passing through protein channels or by dissolving in the lipid cell membrane. *Facilitated diffusion*, also called carrier-mediated diffusion, allows large water-soluble molecules, such as glucose and amino acids, to diffuse across cell membranes. Proteins embedded in the cell membrane function as *carriers*, helping large molecules cross the membrane.

The rate of diffusion is influenced by a number of factors, such as the concentration of solute and the availability of carrier proteins in the cell membrane. The effect of both simple and facilitated diffusion is to establish equal concentrations of the molecules on both sides of a membrane.

***FILTRATION*** The process by which water and dissolved substances (solutes) move from an area of high hydrostatic pressure to an area of low hydrostatic pressure is **filtration**. This usually occurs across capillary membranes. *Hydrostatic pressure* is created by the pumping action of the heart and gravity against the capillary wall. Filtration occurs in the glomerulus of the kidneys, as well as at the arterial end of capillaries.

A balance of hydrostatic (filtration) pressure and osmotic pressure regulates the movement of water between the intravascular and interstitial spaces in the capillary beds of the body. Hydrostatic pressure within the arterial end of the capillary pushes water into the interstitial space. Hydrostatic pressure within the interstitial space opposes this movement to some degree. At the venous end of the capillary, the osmotic force of plasma proteins draws fluid back into the capillary (Figure 10–6 ■).

***ACTIVE TRANSPORT*** **Active transport** allows molecules to move across cell membranes and epithelial membranes against a concentration gradient. This movement requires energy (adenosine triphosphate, or ATP) and a carrier mechanism to maintain a higher concentration of a substance on one side of the membrane than on the other. The sodium-potassium pump is an important example of active transport (Figure 10–7 ■). High concentrations of potassium in intracellular fluids and of sodium in extracellular fluids are maintained because cells actively transport potassium from interstitial fluid (where the concentration of potassium is about 5 mEq/L) into intracellular fluid (where the potassium concentration is about 150 mEq/L).

## Body Fluid Regulation

Homeostasis requires several regulatory mechanisms and processes to maintain the balance between fluid intake and excretion. These include thirst, the kidneys, the renin–angiotensin–aldosterone mechanism, antidiuretic hormone, and atrial natriuretic peptide. These mechanisms affect the volume, distribution, and composition of body fluids.

### Thirst

Thirst is the primary regulator of water intake. Thirst plays an important role in maintaining fluid balance and preventing dehydration. The thirst center, located in the brain, is stimulated when the blood volume drops because of water losses or when serum osmolality (solute concentration) increases (Figure 10–8 ■).

The thirst mechanism is highly effective in regulating extracellular sodium levels. Increased sodium in ECF increases

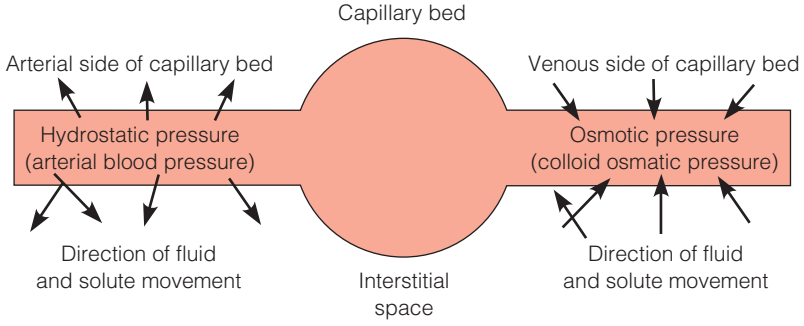



**Figure 10–6** ■ Fluid balance between the intravascular and interstitial spaces is maintained in the capillary beds by a balance of filtration at the arterial end and osmotic draw at the venous end.

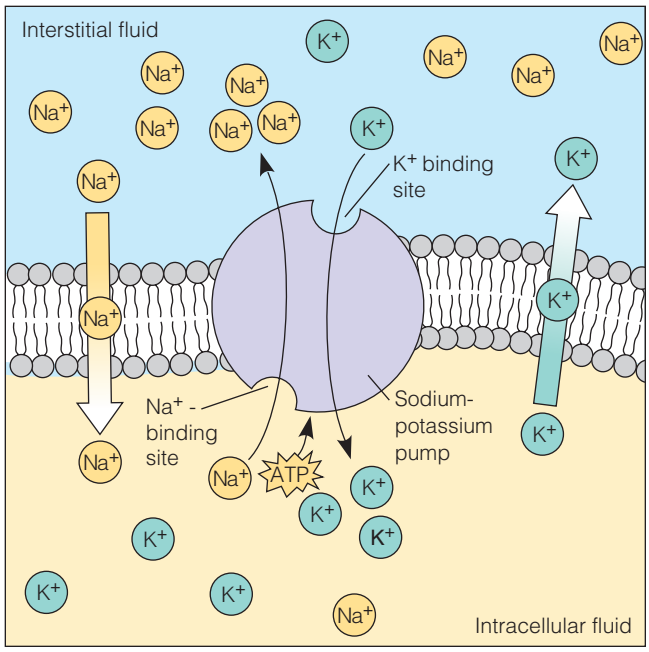



**Figure 10–7** ■ The sodium-potassium pump. Sodium and potassium ions are moved across the cell membranes against their concentration gradients. This active transport process is fueled by energy from ATP.

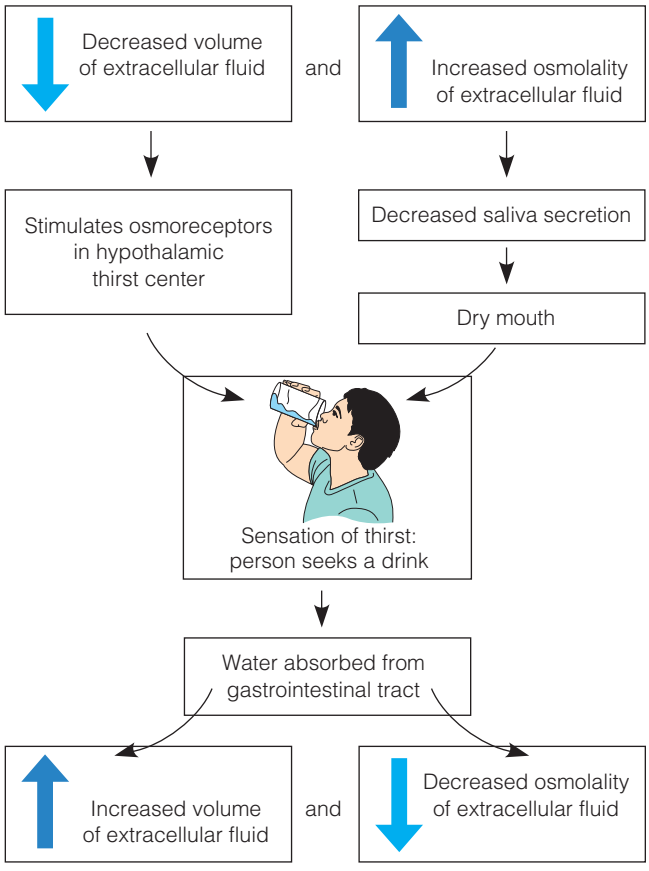



**Figure 10–8** ■ Factors stimulating water intake through the thirst mechanism.

serum osmolality, stimulating the thirst center. Fluid intake in turn reduces the sodium concentration of ECF and lowers serum osmolality. Conversely, a drop in serum sodium and low serum osmolality inhibit the thirst center.

**PRACTICE ALERT**

The thirst mechanism declines with aging, making older adults more vulnerable to dehydration and hyperosmolality (high serum osmolality). Clients with an altered level of consciousness or who are unable to respond to thirst also are at risk.

## Kidneys

The kidneys are primarily responsible for regulating fluid volume and electrolyte balance in the body. They regulate the volume and osmolality of body fluids by controlling the excretion of water and electrolytes. In adults, about 170 L of plasma are filtered through the glomeruli every day. By selectively reabsorbing water and electrolytes, the kidneys maintain the volume and osmolality of body fluids. About 99% of the glomerular filtrate is reabsorbed, and only about 1500 mL of urine is produced over a 24-hour period.

## Renin–Angiotensin–Aldosterone System

The renin–angiotensin–aldosterone system works to maintain intravascular fluid balance and blood pressure. A decrease in blood flow or blood pressure to the kidneys stimulates specialized receptors in the juxtaglomerular cells of the nephrons to produce *renin*, an enzyme. Renin converts angiotensinogen (a plasma protein) in the circulating blood into angiotensin I. Angiotensin I travels through the bloodstream to the lungs, where it is converted to angiotensin II by angiotensin-converting enzyme (ACE). Angiotensin II is a potent vasoconstrictor; it raises the blood pressure. It also stimulates the thirst mechanism to promote fluid intake and acts directly on the kidneys, causing them to retain sodium and water. Angiotensin II stimulates the adrenal cortex to release aldosterone. Aldosterone promotes sodium and water retention in the distal nephron of the kidney, restoring blood volume (Figure 10–9 ■).

## Antidiuretic Hormone

Antidiuretic hormone (ADH), released by the posterior pituitary gland, regulates water excretion from the kidneys. Osmoreceptors in the hypothalamus respond to increases in serum osmolality and decreases in blood volume, stimulating ADH production and release. ADH acts on the distal tubules of the kidney, making them more permeable to water and thus increasing water reabsorption. With increased water reabsorption, urine output falls, blood volume is restored, and serum osmolality drops as the water dilutes body fluids (Figure 10–10 ■).

In addition to decreased blood volume and increased serum osmolality, increased amounts of ADH are released in response to stress, pain, surgery and anesthesia, some medications such as morphine and barbiturates, and mechanical ventilation. Its release is inhibited by ethanol, medications such as phenytoin, as well as increased circulating blood volume and decreased serum osmolality (Suhayda & Walton, 2002).

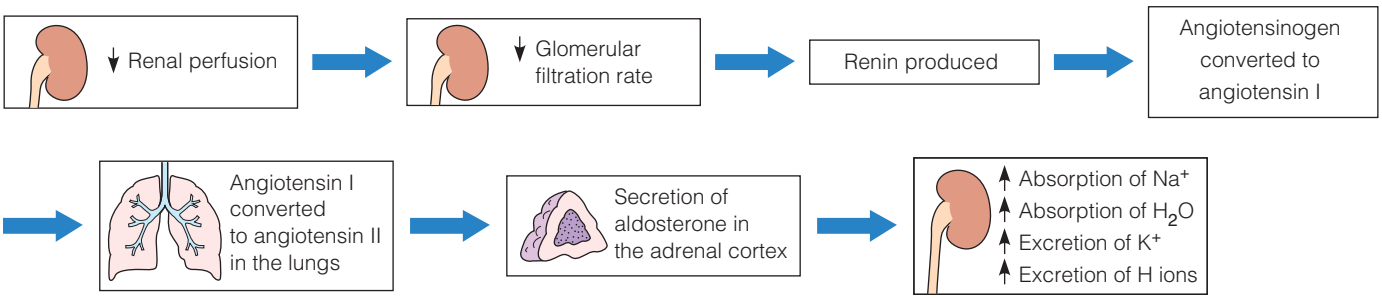



**Figure 10–9** ■ The renin–angiotensin–aldosterone system. Decreased blood volume and renal perfusion set off a chain of reactions, leading to release of aldosterone from the adrenal cortex. Increased levels of aldosterone regulate serum $K^+$ and $Na^+$, blood pressure, and water balance through effects on the kidney tubules.

Two disorders of ADH production illustrate the effect of ADH on water balance and urine output. First, diabetes insipidus is a condition characterized by deficient ADH production. The lack of ADH causes the distal tubules and collecting ducts of the kidney to be impermeable to water, so little water is reabsorbed into the bloodstream. As a result, copious, very dilute urine is excreted. Water loss leads to increased concentration of the plasma, or increased serum osmolality. ADH is not released in response to the serum hyperosmolality, but the thirst mechanism is stimulated and the client drinks additional fluids, maintaining high urine output. In the other condition, the syndrome of inappropriate ADH secretion (SIADH), excess ADH is released. Increased water reabsorption causes increased fluid volume and scant, concentrated urine output. These diseases of the pituitary gland are discussed in Chapter 19 ∞.

### Atrial Natriuretic Peptide

**Atrial natriuretic peptide (ANP)** is a hormone released by atrial muscle cells in response to distention from fluid overload. ANP affects several body systems, including the cardiovascular, renal, neural, gastrointestinal, and endocrine systems, but it primarily affects the renin–angiotensin–aldosterone system. ANP opposes this system by inhibiting renin secretion and blocking the secretion and sodium-retaining effects of aldosterone. As a result, ANP promotes sodium wasting and diuresis (increased urine output) and causes vasodilation.

## CHANGES IN THE OLDER ADULT

A number of changes commonly occurring with aging affect homeostasis. In older adults, the percentage of total body water is lower than in younger or middle age adults. Lean muscle mass is lower in older adults, and the percentage of body fat is higher; as a result water accounts for about 50% of the total body weight (TBW) of an older man and about 45% TBW of an older woman. Sodium and water regulation become less efficient with aging. Renal blood flow and glomerular filtration decline with aging; consequently, the kidneys are less able to effectively concentrate the urine and conserve sodium and water. The perception of thirst decreases, interfering with the thirst mechanism. Consequently, the older adult may become dehydrated without being aware of the need to increase fluid intake. Aging affects temperature regulation as well.

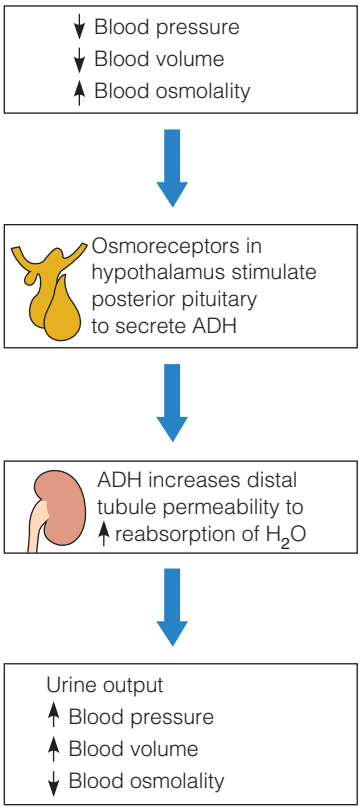



**Figure 10–10** ■ Antidiuretic hormone (ADH) release and effect. Increased serum osmolality or a fall in blood volume stimulates the release of ADH from the posterior pituitary. ADH increases the permeability of distal tubules, promoting water reabsorption.

Functional changes of aging also affect fluid balance:

- Fear of incontinence can lead to self-limiting of fluid intake.
- Physical disabilities associated with age-related illnesses, such as arthritis or stroke, may limit access to fluids.
- Cognitive impairments can interfere with recognition of thirst and the ability to respond to it.

Older adults who have self-care deficits, or who are confused, depressed, tube fed, on bed rest, or taking medications (such as sedatives, tranquilizers, diuretics, and laxatives) are at greatest risk for fluid volume imbalance. Older adults without air conditioning are at risk during extremely hot weather.

# FLUID AND ELECTROLYTE IMBALANCES

## FLUID IMBALANCE

## The Client with Fluid Volume Deficit

**Fluid volume deficit (FVD)** is a decrease in intravascular, interstitial, and/or intracellular fluid in the body. Fluid volume deficits may be due to excessive fluid losses, insufficient fluid intake, or failure of regulatory mechanisms and fluid shifts within the body. FVD is a relatively common problem that may exist alone or in combination with other electrolyte or acid–base imbalances. The term **dehydration** refers to loss of water alone, even though it often is used interchangeably with fluid volume deficit.

### FAST FACTS

- Water loss of as little as 1–2% impairs cognition and physical performance
- Loss of 7% of body water can lead to circulatory collapse
- Dehydration is one of the ten most common hospital admitting diagnoses for older adults (Suhayda & Walton, 2002)

### Pathophysiology

The most common cause of fluid volume deficit is excessive loss of gastrointestinal fluids from vomiting, diarrhea, gastrointestinal suctioning, intestinal fistulas, and intestinal drainage. Other causes of fluid losses include:

- Excessive renal losses of water and sodium from diuretic therapy, renal disorders, or endocrine disorders
- Water and sodium losses during sweating from excessive exercise or increased environmental temperature
- Hemorrhage
- Chronic abuse of laxatives and/or enemas.

Inadequate fluid intake may result from lack of access to fluids, inability to request or to swallow fluids, oral trauma, or altered thirst mechanisms. Older adults are at particular risk for fluid volume deficit (see Nursing Care of the Older Adult box on this page).

Fluid volume deficit can develop slowly or rapidly, depending on the type of fluid loss. Loss of extracellular fluid volume can lead to *hypovolemia,* decreased circulating blood volume. Electrolytes often are lost along with fluid, resulting in an *isotonic fluid volume deficit*. When both water and electrolytes are lost, the serum sodium level remains normal, although levels of other electrolytes such as potassium may fall. Fluid is drawn into the vascular compartment from the interstitial spaces as the body attempts to maintain tissue perfusion. This eventually depletes fluid in the intracellular compartment as well.

Hypovolemia stimulates regulatory mechanisms to maintain circulation. The sympathetic nervous system is stimulated, as is the thirst mechanism. ADH and aldosterone are released, prompting sodium and water retention by the kidneys. Severe fluid loss can lead to cardiovascular collapse.

Two other types of fluid volume deficit, hypovolemic fluid volume deficit and hypertonic fluid volume deficit, are discussed as effects of sodium imbalance in that section of this chapter.

### NURSING CARE OF THE OLDER ADULT
### Fluid Volume Deficit

Fluid volume deficit, or dehydration, is a common reason for hospitalization of people over age 65 who live either in the community or in a long-term care setting. Older adults have a significant number of risk factors for fluid volume deficit (see the preceding section of this chapter). In addition, the older adult has fewer intracellular reserves, contributing to rapid development of dehydration. Without intervention, mortality from dehydration can exceed 50% in the older adult population (Suhayda & Walton, 2002).

Manifestations of fluid volume deficit may be more difficult to recognize in the older adult. A change in mental status, memory, or attention may be an early sign. Skin turgor is less reliable as an indicator of dehydration, although assessing turgor over the sternum or on the inner aspect of the thigh may be more effective. Dry oral mucous membranes and tongue furrows also are indicative of dehydration. Orthostatic vital signs may not demonstrate typical changes in the dehydrated older adult.

***THIRD SPACING*** **Third spacing** is a shift of fluid from the vascular space into an area where it is not available to support normal physiologic processes. The trapped fluid represents a volume loss and is unavailable for normal physiologic processes. Fluid may be sequestered in the abdomen or bowel, or in such other actual or potential body spaces as the pleural or peritoneal space. Fluid may also become trapped within soft tissues following trauma or burns.

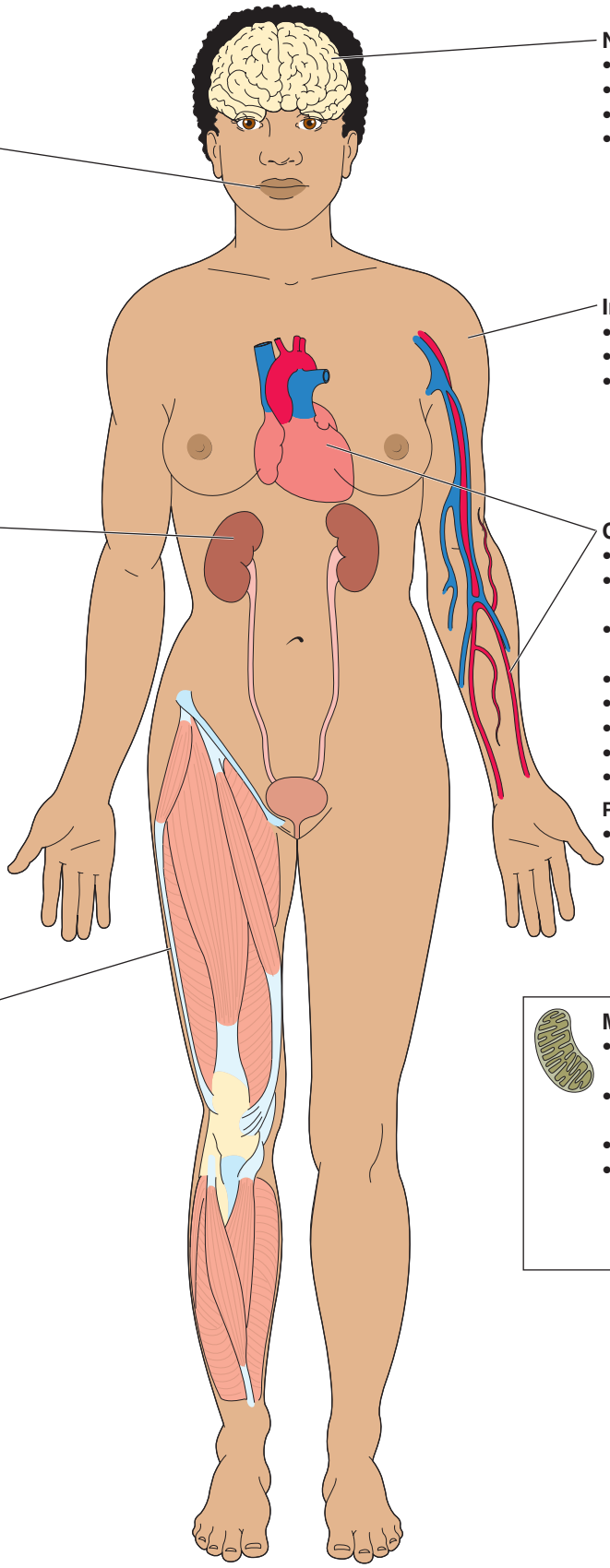
In many cases, fluid is sequestered in interstitial tissues and thus unavailable to support cardiovascular function. Surgery triggers adaptive stress responses and the release of stress hormones (ACTH, cortisol, and catecholamines). These hormones increase blood glucose levels to provide increased fuel for metabolic processes and lead to vasoconstriction that redistributes blood to vital organs (the heart and brain). Renal blood flow falls, stimulating the renin–angiotensin–aldosterone system. This promotes sodium and water retention to maintain intravascular volume. The blood vessel and tissue damage caused by surgery stimulate the release of inflammatory mediators such as histamine and prostaglandins. These substances lead to local vasodilation and increased capillary permeability, allowing fluid to accumulate in interstitial tissues.

Assessing the extent of FVD resulting from third spacing is difficult. It may not be reflected by changes in weight or intake-and-output records, and it may not become apparent until after organ malfunction occurs (Metheny, 2000).

### Manifestations

With a rapid fluid loss (such as hemorrhage or uncontrolled vomiting), manifestations of hypovolemia develop rapidly. When the loss of fluid occurs more gradually, the client's fluid volume may be very low before symptoms develop. The *Multisystem Effects of Fluid Volume Deficit* are illustrated on the following page.

# MULTISYSTEM EFFECTS **of Fluid Volume Deficit (FVD)**



**Neurologic**
- Altered mental status
- Anxiety, restlessness
- Diminished alertness/cognition
- Possible coma (severe FVD)

**Mucous Membranes**
- Dry; may be sticky
- ↓ tongue size, longitudinal furrows ↑

**Integumentary**
- Diminished skin turgor
- Dry skin
- Pale, cool extremities

**Urinary**
- ↓ urine output
- Oliguria (severe FVD)
- ↑ urine specific gravity

**Cardiovascular**
- Tachycardia
- Orthostatic hypotension (moderate FVD)
- Falling systolic/diastolic pressure (severe FVD)
- Flat neck veins
- ↓ venous filling
- ↓ pulse volume
- ↓ capillary refill
- ↑ hematocrit

**Potential Complication**
- Hypovolemic shock

**Musculoskeletal**
- Fatigue



**Metabolic Processes**
- ↓ body temperature (isotonic FVD)
- ↑ body temperature (dehydration)
- Thirst
- Weight loss
  2–5% mild FVD
  6–9% moderate FVD
  >10% severe FVD

Rapid weight loss is a good indicator of fluid volume deficit. Each liter of body fluid weighs about 1 kg (2.2 lb). The severity of the fluid volume deficit can be estimated by the percentage of rapid weight loss: a loss of 2% to 5% of total body weight represents a mild FVD; 6% to 9%, moderate FVD; and 10% or greater, severe FVD.

Loss of interstitial fluid causes skin turgor to diminish. When pinched, the skin of a client with FVD remains elevated. Loss of skin elasticity with aging makes this assessment finding less accurate in older adults. Tongue turgor is not generally affected by age; therefore, assessing the size, dryness, and longitudinal furrows of the tongue may be a more accurate indicator of FVD.

Postural or orthostatic hypotension is a sign of hypovolemia. A drop of more than 15 mmHg in systolic blood pressure when changing from a lying to standing position often indicates loss of intravascular volume. Venous pressure falls as well, causing flat neck veins, even when the client is recumbent. Loss of intravascular fluid causes the hematocrit to increase.

Compensatory mechanisms to conserve water and sodium and maintain circulation account for many of the manifestations of fluid volume deficit, such as tachycardia; pale, cool skin (vasoconstriction); and decreased urine output. The specific gravity of urine increases as water is reabsorbed in the tubules. Table 10–3 compares assessment findings for fluid deficit and fluid excess.

## INTERDISCIPLINARY CARE

The primary goals of care related to fluid volume deficit are to prevent deficits in clients at risk and to correct deficits and their underlying causes. Depending on the acuity of the imbalance, treatment may include replacement of fluids and electrolytes by the intravenous, oral, or enteral route. When possible, the oral or enteral route is preferred for administering fluids. In acute situations, however, intravenous fluid administration is necessary.

TABLE 10–3 **Comparison of Assessment Findings in Clients with Fluid Imbalance**

| ASSESSMENT | FLUID DEFICIT | FLUID EXCESS |
|---|---|---|
| Blood pressure | Decreased systolic<br>Postural hypotension | Increased |
| Heart rate | Increased | Increased |
| Pulse amplitude | Decreased | Increased |
| Respirations | Normal | Moist crackles<br>Wheezes |
| Jugular vein | Flat | Distended |
| Edema | Rare | Dependent |
| Skin turgor | Loose, poor turgor | Taut |
| Output | Low, concentrated | May be low or normal |
| Urine specific gravity | High | Low |
| Weight | Loss | Gain |

### Diagnosis

Laboratory and diagnostic tests may be ordered when fluid volume deficit is suspected. Such tests measure:

- *Serum electrolytes.* In an isotonic fluid deficit, sodium levels are within normal limits; when the loss is water only, sodium levels are high. Decreases in potassium are common.
- *Serum osmolality.* Measurement of serum osmolality helps to differentiate isotonic fluid loss from water loss. With water loss, osmolality is high; it may be within normal limits with an isotonic fluid loss.
- *Hemoglobin and hematocrit.* The hematocrit often is elevated due to loss of intravascular volume and hemoconcentration.
- *Urine specific gravity and osmolality.* As the kidneys conserve water, both the specific gravity and osmolality of urine increase.
- *Central venous pressure (CVP).* The CVP measures the mean pressure in the superior vena cava or right atrium, providing an accurate assessment of fluid volume status. The technique for measuring CVP is outlined in Box 10–1.

### Fluid Management

Oral rehydration is the safest and most effective treatment for fluid volume deficit in alert clients who are able to take oral fluids. Adults require a minimum of 1500 mL of fluid per day or approximately 30 mL per kg of body weight (ideal body weight is used to calculate fluid requirements for obese clients) for maintenance. Fluids are replaced gradually, particularly in older adults, to prevent rapid rehydration of the cells. In general, fluid deficits are replaced at a rate of approximately 30% to 50% of the deficit per 24 hours.

For mild fluid deficits in which the loss of electrolytes has been minimal (e.g., moderate exercise in warm weather), water alone may be used for fluid replacement. When the fluid deficit is more severe and when electrolytes have also been lost (e.g., FVD due to vomiting and/or diarrhea, strenuous exercise for longer than an hour or two), a carbohydrate/electrolyte solution such as a sports drink, ginger ale, or a rehydrating solution (e.g., Pedialyte or Rehydralyte) is more appropriate. These solutions provide sodium, potassium, chloride, and calories to help meet metabolic needs.

***INTRAVENOUS THERAPY*** When the fluid deficit is severe or the client is unable to ingest fluids, the intravenous route is used to administer replacement fluids. Table 10–4 describes the types, tonicity, and uses of commonly administered intravenous fluids. Isotonic electrolyte solutions (0.9% NaCl or Ringer's solution) are used to expand plasma volume in hypotensive clients or to replace abnormal losses, which are usually isotonic in nature. Normal saline (0.9% NaCl) tends to remain in the vascular compartment, increasing blood volume. When administered rapidly, however, this solution can precipitate acid–base imbalances, so balanced electrolyte solutions such as lactated Ringer's solution are preferred to expand plasma volume.

Five percent dextrose in water ($D_5W$) or 0.45% NaCl (one-half normal saline or 1/2 NS) are given to provide water to treat total body water deficits. $D_5W$ is isotonic (similar in tonicity to the plasma) when administered and thus does not provoke hemolysis

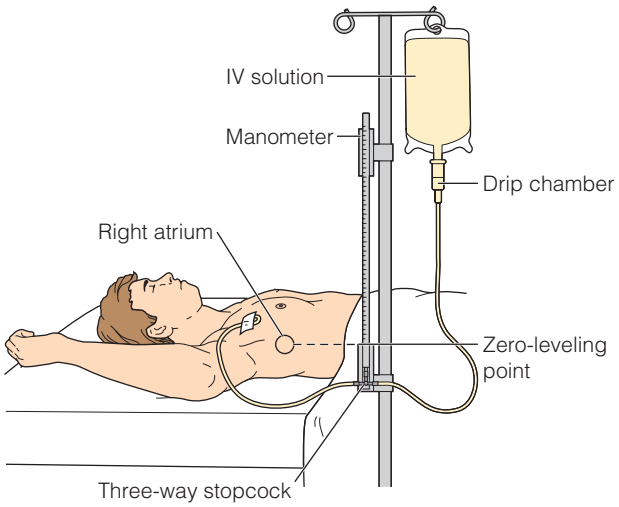
## BOX 10–1 Measuring Central Venous Pressure with a Manometer

CVP is a hemodynamic monitoring method for evaluating fluid volume status. It measures mean right atrial pressure by means of a catheter. The CVP catheter is inserted by a physician, most often at the client's bedside, into the antecubital, internal jugular, or subclavian vein. Either a hemodynamic monitoring system (see Chapter 32 ∞) or a manual system may be used to measure the CVP. Nursing responsibilities in measuring CVP are as follows:

1. Explain to the client and family what is being done.
2. Prior to the first measurement, take baseline vital signs, and measure the level of the right atrium on the client's thorax. This is usually at the fourth intercostal space on the lateral chest wall, midway between the anterior and posterior chest. This site, called the *phlebostatic axis,* is marked and used as the reference point for all measurements.
3. If possible, place the bed in the same position for each reading, usually with the client supine and the head of the bed flat. Elevating the head of the bed to as much as 60 degrees usually does not affect the accuracy of the CVP reading in clients who are hemodynamically stable (Urden et al., 2006).
4. Use a carpenter's level to check the level of the measuring device to make sure the transducer or the 0 on the manometer is level with the phlebostatic axis (see figure).
5. Remove any air bubbles in the line.
6. If using a manometer, turn the stopcock so that fluid flows into the manometer, filling it a few centimeters above the expected reading. Then turn the stopcock to open the line between the manometer and the client. The fluid level will fall and then reach a point at which it fluctuates with the client's respirations. This point is recorded as the CVP.
7. After the measurement is taken, turn the stopcock so that the fluid can again flow from the fluid source to the client.

### Normal Values

When CVP is measured by a manometer, normal values range from 2 to 8 cm water. With a hemodynamic monitoring system, the normal CVP range is 2 to 5 mmHg. A low CVP indicates inadequate venous return from fluid deficit and hypovolemia or due to peripheral vasodilation. A high CVP indicates fluid overload, cardiac problems that decrease cardiac contractility, or pulmonary disorders that increase pulmonary vascular resistance.




of red blood cells. The dextrose is metabolized to carbon dioxide and water, leaving free water available for tissue needs. Hypotonic saline solution (0.45% NaCl with or without added electrolytes) or 5% dextrose in 0.45% sodium chloride ($D_5$ 1/2 NS) are used as maintenance solutions. These solutions provide additional electrolytes such as potassium, a buffer (lactate or acetate) as needed, and water. When dextrose is added, they also provide a minimal number of calories.

***FLUID CHALLENGE*** A fluid challenge, the rapid administration of a designated amount of intravenous fluid, may be performed to evaluate fluid volume when urine output is low and cardiac or renal function is questionable. A fluid challenge helps prevent fluid volume overload resulting from intravenous fluid therapy when cardiac or renal function is compromised. Nursing responsibilities for a fluid challenge are as follows:

1. Obtain and document baseline vital signs, breath sounds, urine output, and mental status.
2. Administer (by IV infusion) an initial fluid volume of 200 to 300 mL over 5 to 10 minutes.
3. Reevaluate baseline data at the end of the 5- or 10-minute infusion period.
4. Administer additional fluid until a specified volume is infused or the desired hemodynamic parameters are achieved.

## NURSING CARE

Nurses are responsible for identifying clients at risk for fluid volume deficit, initiating and carrying out measures to prevent and treat fluid volume deficit, and monitoring the effects of therapy.

### Health Promotion

Health promotion activities focus on teaching clients to prevent fluid volume deficit. Discuss the importance of maintaining adequate fluid intake, particularly when exercising and during hot weather. Advise clients to use commercial sports drinks to replace both water and electrolytes when exercising during warm weather. Instruct clients to maintain fluid intake when ill, particularly during periods of fever or when diarrhea is a problem.

Discuss the increased risk for fluid volume deficit with older adults (see page 203) and provide information about prevention. Teach older adults (and their caretakers) that thirst decreases with aging and urge them to maintain a regular fluid intake of about 1500 mL per day, regardless of perception of thirst.

Carefully monitor clients at risk for abnormal fluid losses through routes such as vomiting, diarrhea, nasogastric suction, increased urine output, fever, or wounds. Monitor fluid intake in clients with decreased level of consciousness, disorientation, nausea and anorexia, and physical limitations.

TABLE 10–4 **Commonly Administered Intravenous Fluids**

| | FLUID AND TONICITY | USES |
|---|---|---|
| **Dextrose in Water Solutions** | 5% dextrose in water ($D_5W$)<br>Isotonic | Replaces water losses<br>Provides free water necessary for cellular rehydration<br>Lowers serum sodium in hypernatremia |
| | 10% dextrose in water ($D_{10}W$)<br>Hypertonic | Provides free water<br>Provides nutrition (supplies 340 kcal/L) |
| | 20% dextrose in water ($D_{20}W$)<br>Hypertonic | Supplies 680 kcal/L<br>May cause diuresis |
| | 50% dextrose in water ($D_{50}W$)<br>Hypertonic | Supplies 1700 kcal/L<br>Used to correct hypoglycemia |
| **Saline Solutions** | 0.45% sodium chloride<br>Hypotonic | Provides free water to replace hypotonic fluid losses<br>Maintains levels of plasma sodium and chloride |
| | 0.9% sodium chloride<br>Isotonic | Expands intravascular volume<br>Replaces water lost from extracellular fluid<br>Used with blood transfusions<br>Replaces large sodium losses (as from burns) |
| | 3% sodium chloride<br>Hypertonic | Corrects serious sodium depletion |
| **Combined Dextrose and Saline Solution** | 5% dextrose and 0.45% sodium chloride<br>Isotonic | Provides free water<br>Provides sodium chloride<br>Maintenance fluid of choice if there are no electrolyte imbalances |
| **Multiple Electrolyte Solutions** | Ringer's solution<br>Isotonic (electrolyte concentrations of sodium, potassium, chloride, and calcium are similar to plasma levels) | Expands the intracellular fluid<br>Replaces extracellular fluid losses |
| | Lactated Ringer's solution<br>Isotonic (similar in composition of electrolytes to plasma but does not contain magnesium) | Replaces fluid losses from burns and the lower gastrointestinal tract<br>Fluid of choice for acute blood loss |

## Assessment

Collect assessment data through the health history interview and physical examination.

- *Health history:* Risk factors such as medications, acute or chronic renal or endocrine disease; precipitating factors such as hot weather, extensive exercise, lack of access to fluids, recent illness (especially if accompanied by fever, vomiting, and/or diarrhea); onset and duration of symptoms.
- *Physical assessment:* Weight; vital signs including orthostatic blood pressure and pulse; peripheral pulses and capillary refill; jugular neck vein distention; skin color, temperature, turgor; level of consciousness and mentation; urine output. See Box 10–2 for physical assessment changes in the older adult.

### BOX 10–2 Assessing Older Adults: Fluid Volume Deficit

With aging, the elasticity of skin decreases. As a result, turgor diminishes, even in the well-hydrated older adult. This makes skin turgor less reliable when assessing for fluid volume deficit. In addition, some older adults experience postural hypotension, even when well hydrated. Allow the older adult to stand quietly for a full minute before rechecking blood pressure and pulse when measuring orthostatic vital signs.

- *Diagnostic tests:* Serum osmolality and electrolytes, hemoglobin and hematocrit (expect values to fall with rehydration), urine specific gravity and osmolality, central venous pressure readings.

## Nursing Diagnoses and Interventions

The focus for nursing diagnoses and interventions for the client with fluid volume deficit is on managing the effects of the deficit and preventing complications.

### Deficient Fluid Volume

Clients with a fluid volume deficit due to abnormal losses, inadequate intake, or impaired fluid regulation require close monitoring as well as immediate and ongoing fluid replacement.

- Assess intake and output accurately, monitoring fluid balance. In acute situations, hourly intake and output may be indicated. *Urine output should be 30 to 60 mL per hour (unless renal failure is present). Urine output of less than 30 mL per hour indicates inadequate renal perfusion and an increased risk for acute renal failure and inadequate tissue perfusion.*

**PRACTICE ALERT**

Report a urine output of less than 30 mL per hour to the primary healthcare provider.

## NURSING RESEARCH Evidence-Based Practice for Clients with Imbalanced Fluid Volume

Nurses caring for clients with a fluid volume imbalance frequently monitor both 24-hour intake and output records and daily weights. These measurements require caregiver time, and may be providing redundant data. Nurse managers on three nursing units compared the results of continuous 48-hour intake and output records with daily weights for a total of 73 selected clients on their units. Their findings suggest that even when compliance with recording accurate intake and output is optimal, it is an unreliable measure of actual fluid balance (Wise et al., 2000).

### IMPLICATIONS FOR NURSING

A significant shortage of licensed nurses is predicted for the early part of the 21st century. Tight nursing resources will require efficient nursing practice to maintain quality care. This study suggests that for the majority of clients (the exceptions being clients with kidney disease or who are on a fluid restriction), measuring accurate daily weights is a better indicator of fluid balance than intake and output records.

### CRITICAL THINKING IN CLIENT CARE

1. What factors can you identify that would affect the accuracy of intake and output records?
2. What measures can you and your institution take to ensure accurate daily weight measurements?
3. Compare intake and output records and daily weights for your assigned clients. Is the balance between intake and output accurately reflected by day-to-day weight changes? If not, what factors can you identify that might account for this discrepancy?

*Source:* Adapted from "Evaluating the Reliability and Utility of Cumulative Intake and Output" by L. C. Wise et al., 2000, *Journal of Nursing Care Quality, 14*(3), pp. 37–42.

- Assess vital signs, CVP, and peripheral pulse volume at least every 4 hours. *Hypotension, tachycardia, low CVP, and weak, easily obliterated peripheral pulses indicate hypovolemia.*
- Weigh daily under standard conditions (time of day, clothing, and scale). *In most instances (except third spacing), changes in weight accurately reflect fluid balance.* See the Nursing Research box on this page.
- Administer and monitor the intake of oral fluids as prescribed. Identify beverage preferences and provide these on a schedule. *Oral fluid replacement is preferred when the client is able to drink and retain fluids.*
- Administer intravenous fluids as prescribed using an electronic infusion pump. Monitor for indicators of fluid overload if rapid fluid replacement is ordered: dyspnea, tachypnea, tachycardia, increased CVP, jugular vein distention, and edema. *Rapid fluid replacement may lead to hypervolemia, resulting in pulmonary edema and cardiac failure, particularly in clients with compromised cardiac and renal function.*
- Monitor laboratory values: electrolytes, serum osmolality, blood urea nitrogen (BUN), and hematocrit. *Rehydration may lead to changes in serum electrolytes, osmolality, BUN, and hematocrit. In some cases, electrolyte replacement may be necessary during rehydration.*

### Ineffective Tissue Perfusion

A fluid volume deficit can lead to decreased perfusion of renal, cerebral, and peripheral tissues. Inadequate renal perfusion can lead to acute renal failure. Decreased cerebral perfusion leads to changes in mental status and cognitive function, causing restlessness, anxiety, agitation, excitability, confusion, vertigo, fainting, and weakness.

- Monitor for changes in level of consciousness and mental status. *Restlessness, anxiety, confusion, and agitation may indicate inadequate cerebral blood flow and circulatory collapse.*
- Monitor serum creatinine, BUN, and cardiac enzymes, reporting elevated levels to the physician. *Elevated levels may indicate impaired renal function or cardiac perfusion related to circulatory failure.*
- Turn at least every 2 hours. Provide good skin care and monitor for evidence of skin or tissue breakdown. *Impaired circulation to peripheral tissues increases the risk of skin breakdown. Turn frequently to relieve pressure over bony prominences. Keep skin clean, dry, and moisturized to help maintain integrity.*

### Risk for Injury

The client with fluid volume deficit is at risk for injury because of dizziness and loss of balance resulting from decreased cerebral perfusion secondary to hypovolemia.

- Institute safety precautions, including keeping the bed in a low position, using side rails as needed, and slowly raising the client from supine to sitting or sitting to standing position. *Using safety precautions and allowing time for the blood pressure to adjust to position changes reduce the risk of injury.*
- Teach client and family members how to reduce orthostatic hypotension:
  a. Move from one position to another in stages; for example, raise the head of the bed before sitting up, and sit for a few minutes before standing.
  b. Avoid prolonged standing.
  c. Rest in a recliner rather than in bed during the day.
  d. Use assistive devices to pick up objects from the floor rather than stooping.

  *Teaching measures to reduce orthostatic hypotension reduces the client's risk for injury. Prolonged bed rest increases skeletal muscle weakness and decreases venous tone, contributing to postural hypotension. Prolonged standing allows blood to pool in the legs, reducing venous return and cardiac output.*

## Using NANDA, NIC, and NOC

Chart 10–1 shows linkages between NANDA nursing diagnoses, nursing interventions classification (NIC), and nursing outcomes classification (NOC) for the client with fluid volume deficit.

## Community-Based Care

Depending on the severity of the fluid volume deficit, the client may be managed in the home or residential facility, or may be

NANDA, NIC, AND NOC LINKAGES

CHART 10–1 **The Client with Fluid Volume Deficit**

**NANDA**

Deficient Fluid Volume

↓

**NIC**

Fluid Management

Fluid Monitoring

Hypovolemia Management

Intravenous (IV) Therapy

Shock Management: Volume

↓

**NOC**

Electrolyte and Acid–Base Balance

Fluid Balance

Hydration

Data from *NANDA's Nursing Diagnoses: Definitions & classification 2005–2006* by NANDA International (2003), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

admitted to an acute care facility. Assess the client's understanding of the cause of the deficit and the fluids necessary for providing replacement. Address the following topics when preparing the client and family for home care:

- The importance of maintaining adequate fluid intake (at least 1500 mL per day; more if extra fluid is being lost through perspiration, fever, or diarrhea)
- Manifestations of fluid imbalance, and how to monitor fluid balance
- How to prevent fluid deficit:
  - Avoid exercising during extreme heat.
  - Increase fluid intake during hot weather.
  - If vomiting, take small frequent amounts of ice chips or clear liquids, such as weak tea, flat cola, or ginger ale.
  - Reduce intake of coffee, tea, and alcohol, which increase urine output and can cause fluid loss.
- Replacement of fluids lost through diarrhea with fruit juices or bouillon, rather than large amounts of tap water
- Alternate sources of fluid (such as gelatin, frozen juices, or ice cream) for effective replacement of lost fluids.

# THE CLIENT WITH FLUID VOLUME EXCESS

**Fluid volume excess** results when both water and sodium are retained in the body. Fluid volume excess may be caused by fluid overload (excess water and sodium intake) or by impairment of the mechanisms that maintain homeostasis. The excess fluid can lead to excess intravascular fluid (hypervolemia) and excess interstitial fluid (**edema**).

## Pathophysiology

Fluid volume excess usually results from conditions that cause retention of both sodium and water. These conditions include heart failure, cirrhosis of the liver, renal failure, adrenal gland disorders, corticosteroid administration, and stress conditions causing the release of ADH and aldosterone. Other causes include an excessive intake of sodium-containing foods, drugs that cause sodium retention, and the administration of excess amounts of sodium-containing intravenous fluids (such as 0.9% NaCl or Ringer's solution). This *iatrogenic* (induced by the effects of treatment) cause of fluid volume excess primarily affects clients with impaired regulatory mechanisms.

In fluid volume excess, both water and sodium are gained in about the same proportions as normally exists in extracellular fluid. The total body sodium content is increased, which in turn causes an increase in total body water. Because the increase in sodium and water is isotonic, the serum sodium and osmolality remain normal, and the excess fluid remains in the extracellular space.

Stress responses activated before, during, and immediately after surgery commonly lead to increased ADH and aldosterone levels, leading to sodium and water retention. In the immediate postoperative period, however, this additional fluid tends to be sequestered in interstitial tissues and unavailable to support cardiovascular and renal function (see earlier "Third Spacing" section in this chapter). This sequestered fluid is reabsorbed into the circulation within about 48 to 72 hours after surgery. Although it is then normally eliminated through a process of diuresis, clients with heart or kidney failure are at risk for developing fluid overload.

## Manifestations and Complications

Excess extracellular fluid leads to hypervolemia and circulatory overload. Excess fluid in the interstitial space causes peripheral or generalized edema. The following manifestations of fluid volume excess relate to both the excess fluid and its effects on circulation:

- The increase in total body water causes weight gain (more than 5% of body weight) over a short period.
- Circulatory overload causes manifestations such as:
  - A full, bounding pulse
  - Distended neck and peripheral veins
  - Increased central venous pressure (>11–12 cm of water)
  - Cough, **dyspnea** (labored or difficult breathing), **orthopnea** (difficulty breathing when supine)
  - Moist crackles (rales) in the lungs; pulmonary edema (excess fluid in pulmonary interstitial spaces and alveoli) if severe
  - Increased urine output (**polyuria**)
  - **Ascites** (excess fluid in the peritoneal cavity)
  - Peripheral edema, or if severe, **anasarca** (severe, generalized edema).
- Dilution of plasma by excess fluid causes a decreased hematocrit and BUN.
- Possible cerebral edema (excess water in brain tissues) can lead to altered mental status and anxiety.

Heart failure is not only a potential cause of fluid volume excess, but it is also a potential complication of the condition if the

heart is unable to increase its workload to handle the excess blood volume. Severe fluid overload and heart failure can lead to pulmonary edema, a medical emergency. See Chapter 32 for more information about heart failure and pulmonary edema.

## INTERDISCIPLINARY CARE

Managing fluid volume excess focuses on prevention in clients at risk, treating its manifestations, and correcting the underlying cause. Management includes limiting sodium and water intake and administering diuretics.

### Diagnosis

The following laboratory tests may be ordered.

- *Serum electrolytes* and *serum osmolality* are measured. Serum sodium and osmolality usually remain within normal limits.
- *Serum hematocrit* and *hemoglobin* often are decreased due to plasma dilution from excess extracellular fluid.

Additional tests of *renal* and *liver function* (such as serum creatinine, BUN, and liver enzymes) may be ordered to help determine the cause of fluid volume excess if it is unclear.

### Medications

Diuretics are commonly used to treat fluid volume excess. They inhibit sodium and water reabsorption, increasing urine output. The three major classes of diuretics, each of which acts on a different part of the kidney tubule, are as follows:

- Loop diuretics act in the ascending loop of Henle.
- Thiazide-type diuretics act on the distal convoluted tubule.
- Potassium-sparing diuretics affect the distal nephron.

The nursing implications for diuretics are outlined in the Medication Administration box below.

### Treatments

***FLUID MANAGEMENT*** Fluid intake may be restricted in clients who have fluid volume excess. The amount of fluid allowed per day is prescribed by the primary care provider. All fluid intake must be calculated, including meals and that used to administer medications orally or intravenously. Box 10–3 provides guidelines for clients with a fluid restriction.

***DIETARY MANAGEMENT*** Because sodium retention is a primary cause of fluid volume excess, a sodium-restricted diet of-

### BOX 10–3 Fluid Restriction Guidelines

- Subtract requisite fluids (e.g., ordered IV fluids, fluid used to dilute IV medications) from total daily allowance.
- Divide remaining fluid allowance:
  - Day shift: 50% of total
  - Evening shift: 25% to 33% of total
  - Night shift: Remainder
- Explain the fluid restriction to the client and family members.
- Identify preferred fluids and intake pattern of client.
- Place allowed amounts of fluid in small glasses (gives perception of a full glass).
- Offer ice chips (when melted, ice chips are approximately half the frozen volume).
- Provide frequent mouth care.
- Provide sugarless chewing gum (if allowed) to reduce thirst sensation.

### MEDICATION ADMINISTRATION Diuretics for Fluid Volume Excess

Diuretics increase urinary excretion of water and sodium. They are categorized into three major groups: loop diuretics, thiazide and thiazide-like diuretics, and potassium-sparing diuretics. Diuretics are used to enhance renal function and to treat vascular fluid overload and edema. Common side effects include orthostatic hypotension, dehydration, electrolyte imbalance, and possible hyperglycemia. Diuretics should be used with caution in the older adult. Examples of each major type follow.

#### LOOP DIURETICS

**Furosemide (Lasix)**
**Bumetanide (Bumex)**
**Ethacrynic Acid (Edecrin)**
**Torsemide (Demadex)**

Loop diuretics inhibit sodium and chloride reabsorption in the ascending loop of Henle (see Chapter 27 for the anatomy of the kidneys). As a result, loop diuretics promote the excretion of sodium, chloride, potassium, and water.

#### THIAZIDE AND THIAZIDE-LIKE DIURETICS

**Bendroflumethiazide (Naturetin)**
**Chlorothiazide (Diuril)**
**Hydrochlorothiazide (HydroDIURIL, Oretic)**
**Metolazone (Zaroxolyn)**
**Polythiazide (Renese)**
**Chlorthalidone (Hygroton)**
**Trichlormethiazide (Naqua)**
**Indapamide (Lozol)**

Thiazide and thiazide-like diuretics promote the excretion of sodium, chloride, potassium, and water by decreasing absorption in the distal tubule.

#### POTASSIUM-SPARING DIURETICS

**Spironolactone (Aldactone)**
**Amiloride HCl (Midamor)**
**Triamterene (Dyrenium)**

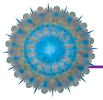
Potassium-sparing diuretics promote excretion of sodium and water by inhibiting sodium-potassium exchange in the distal tubule.

#### Health Education for the Client and Family

- The drug will increase the amount and frequency of urination.
- The drugs must be taken even when you feel well.
- Take the drugs in the morning and afternoon to avoid having to get up at night to urinate.
- Change position slowly to avoid dizziness.
- Report the following to your primary healthcare provider: dizziness; trouble breathing; or swelling of face, hands, or feet.
- Weigh yourself every day, and report sudden gains or losses.
- Avoid using the salt shaker when eating.
- If the drug increases potassium loss, eat foods high in potassium, such as orange juice and bananas.
- Do not use salt substitute if you are taking a potassium-sparing diuretic.

ten is prescribed. Americans typically consume more than 4 or 5 g of sodium every day; recommended sodium intake is 500 to 2400 mg per day. The primary dietary sources of sodium are the salt shaker, processed foods, and foods themselves (Box 10–4).

A mild sodium restriction can be achieved by instructing the client and primary food preparer in the household to reduce the amount of salt in recipes by half, avoid using the salt shaker during meals, and avoid foods that contain high levels of sodium (either naturally or because of processing). In moderate and severely sodium-restricted diets, salt is avoided altogether, as are all foods containing significant amounts of sodium.



## NURSING CARE

Nursing care focuses on preventing fluid volume excess in clients at risk and on managing problems resulting from its effects. See the Nursing Care Plan: A Client with Fluid Volume Excess on the following page.

### Health Promotion

Health promotion related to fluid volume excess focuses on teaching preventive measures to clients who are at risk (e.g., clients who have heart or kidney failure). Discuss the relationship between sodium intake and water retention. Provide guidelines for a low-sodium diet, and teach clients to carefully read food labels to identify "hidden" sodium, particularly in processed foods. Instruct clients at risk to weigh themselves on a regular basis, using the same scale, and to notify their primary care provider if they gain more than 5 lb in a week or less.

Carefully monitor clients receiving intravenous fluids for signs of hypervolemia. Reduce the flow rate and promptly report manifestations of fluid overload to the physician.

**BOX 10–4 Foods High in Sodium**

**High in Added Sodium**

**Processed Meat and Fish**
- Bacon
- Luncheon meat and other cold cuts
- Sausage
- Smoked fish

**Selected Dairy Products**
- Buttermilk
- Cheeses
- Cottage cheese
- Ice cream

**Processed Grains**
- Graham crackers
- Most dry cereals

**Most Canned Goods**
- Meats
- Soups
- Vegetables

**Snack Foods**
- Salted popcorn
- Potato chips/pretzels
- Nuts
- Gelatin desserts

**Condiments and Food Additives**
- Barbecue sauce
- Catsup
- Chili sauce
- Meat tenderizers
- Worcestershire sauce
- Saccharin
- Pickles
- Soy sauce
- Salted margarine
- Salad dressings

**Naturally High in Sodium**
- Brains
- Kidney
- Clams
- Crab
- Lobster
- Oysters
- Shrimp
- Dried fruit
- Spinach
- Carrots

### Assessment

Collect assessment data through the health history interview and physical examination.

- *Health history:* Risk factors such as medications, heart failure, acute or chronic renal or endocrine disease; precipitating factors such as a recent illness, change in diet, or change in medications. Recent weight gain; complaints of persistent cough, shortness of breath, swelling of feet and ankles, or difficulty sleeping when lying down.
- *Physical assessment:* Weight; vital signs; peripheral pulses and capillary refill; jugular neck vein distention; edema; lung sounds (crackles or wheezes), dyspnea, cough, and sputum; urine output; mental status.
- *Diagnostic tests:* Monitor serum electrolytes and osmolality, hemoglobin and hematocrit, urine specific gravity.

### Nursing Diagnoses and Interventions

Nursing diagnoses and interventions for the client with fluid volume excess focus on the multisystem effects of the fluid overload.

#### Excess Fluid Volume

Nursing care for the client with excess fluid volume includes collaborative interventions such as administering diuretics and maintaining a fluid restriction, as well as monitoring the status and effects of the excess fluid volume. This is particularly critical in older clients because of the age-related decline in cardiac and renal compensatory responses.

- Assess vital signs, heart sounds, CVP, and volume of peripheral arteries. *Hypervolemia can cause hypertension, bounding peripheral pulses, a third heart sound ($S_3$) due to the volume of blood flow through the heart, and high CVP readings.*
- Assess for the presence and extent of edema, particularly in the lower extremities, the back, sacral, and periorbital areas. *Initially, edema affects the dependent portions of the body—the lower extremities of ambulatory clients and the sacrum in bedridden clients. Periorbital edema indicates more generalized edema.*

**PRACTICE ALERT**

Assess urine output hourly. Maintain accurate intake and output records. Note urine output of less than 30 mL per hour or a positive fluid balance on 24-hour total intake and output calculations. Heart failure and inadequate renal perfusion may result in decreased urine output and fluid retention.

- Obtain daily weights at the same time of day, using approximately the same clothing and a balanced scale. *Daily weights are one of the most important gauges of fluid balance. Acute*

## NURSING CARE PLAN A Client with Fluid Volume Excess

Dorothy Rainwater is a 45-year-old Native American woman hospitalized with acute renal failure that developed as a result of acute glomerulonephritis. She is expected to recover, but she has very little urine output. Ms. Rainwater is a single mother of two teenage sons. Until her illness, she was active in caring for her family, her career as a high school principal, and community activities.

### ASSESSMENT

Mike Penning, Ms. Rainwater's nurse, notes that she is in the oliguric phase of acute renal failure, and that her urine output for the previous 24 hours is 250 mL; this low output has been constant for the past 8 days. She gained 1 lb (0.45 kg) in the past 24 hours. Laboratory test results from that morning are sodium, 155 mEq/L (normal 135 to 145 mEq/L); potassium, 5.3 mEq/L (normal 3.5 to 5.0 mEq/L); calcium, 7.6 mg/dL (normal 8.0 to 10.5 mg/dL), and urine specific gravity 1.008 (normal 1.010 to 1.030). Ms. Rainwater's serum creatinine and blood urea nitrogen (BUN) are high; however, her ABGs are within normal limits.

In his assessment of Ms. Rainwater, Mike notes the following:

- BP 160/92; P 102, with obvious neck vein distention; R 28, with crackles and wheezes; head of bed elevated 30 degrees; T 98.6°F
- Periorbital and sacral edema present; 3+ pitting bilateral pedal edema; skin cool, pale, and shiny
- Alert, oriented; responds appropriately to questions
- Client states she is thirsty, slightly nauseated, and extremely tired.

Ms. Rainwater is receiving intravenous furosemide and is on a 24-hour fluid restriction of 500 mL plus the previous day's urine output to manage her fluid volume excess.

### DIAGNOSES

- *Excess Fluid Volume* related to acute renal failure
- *Risk for Impaired Skin Integrity* related to fluid retention and edema
- *Risk for Impaired Gas Exchange* related to pulmonary congestion
- *Activity Intolerance* related to fluid volume excess, fatigue, and weakness

### EXPECTED OUTCOMES

- Regain fluid balance, as evidenced by weight loss, decreasing edema, and normal vital signs.
- Experience decreased dyspnea.
- Maintain intact skin and mucous membranes.
- Increase activity levels as prescribed.

### PLANNING AND IMPLEMENTATION

- Weigh at 0600 and 1800 daily.
- Assess vital signs and breath sounds every 4 hours.
- Measure intake and output every 4 hours.
- Obtain urine specific gravity every 8 hours.
- Restrict fluids as follows: 350 mL from 0700 to 1500; 300 mL from 1500 to 2300; 100 mL from 2300 to 0700. Prefers water or apple juice.
- Turn every 2 hours, following schedule posted at the head of bed. Inspect and provide skin care as needed; avoid vigorous massage of pressure areas.
- Provide oral care every 2 to 4 hours (can brush her own teeth, caution not to swallow water); use moistened applicators as desired.
- Elevate head of bed to 30 to 40 degrees; prefers to use own pillows.
- Assist to recliner chair at bedside for 20 minutes two or three times a day. Monitor ability to tolerate activity without increasing dyspnea or fatigue.

### EVALUATION

At the end of the shift, Mike evaluates the effectiveness of the plan of care and continues all diagnoses and interventions. Ms. Rainwater gained no weight, and her urinary output during his shift is 170 mL. Her urine specific gravity remains at 1.008. Her vital signs are unchanged, but her crackles and wheezes have decreased slightly. Her skin and mucous membranes are intact. Ms. Rainwater tolerated the bedside chair without dyspnea or fatigue.

### CRITICAL THINKING IN THE NURSING PROCESS

1. What is the pathophysiologic basis for Ms. Rainwater's increased respiratory rate, blood pressure, and pulse?
2. Explain how elevating the head of the bed 30 degrees facilitates respirations.
3. Suppose Ms. Rainwater says, "I would really like to have all my fluids at once instead of spreading them out." How would you reply, and why?
4. Outline a plan for teaching Ms. Rainwater about diuretics.

*See Evaluating Your Response in Appendix C.*

*weight gain or loss represents fluid gain or loss. Weight gain of 2 kg is equivalent to 2 L of fluid gain.*

- Administer oral fluids cautiously, adhering to any prescribed fluid restriction. Discuss the restriction with the client and significant others, including the total volume allowed, the rationale, and the importance of reporting all fluid taken. *All sources of fluid intake, including ice chips, are recorded to avoid excess fluid intake.*
- Provide oral hygiene at least every 2 hours. *Oral hygiene contributes to client comfort and keeps mucous membranes intact; it also helps relieve thirst if fluids are restricted.*
- Teach client and significant others about the sodium-restricted diet, and emphasize the importance of checking before bringing foods to the client. *Excess sodium promotes water retention; a sodium-restricted diet is ordered to reduce water gain.*
- Administer prescribed diuretics as ordered, monitoring the client's response to therapy. *Loop or high-ceiling diuretics such as furosemide can lead to rapid fluid loss and signs of hypovolemia and electrolyte imbalance.*
- Promptly report significant changes in serum electrolytes or osmolality or abnormal results of tests done to determine contributing factors to the fluid volume excess. *Gradual correction of serum electrolytes and osmolality is expected, however, aggressive diuretic therapy can lead to overcorrection.*

### Risk for Impaired Skin Integrity

Tissue edema decreases oxygen and nutrient delivery to the skin and subcutaneous tissues, increasing the risk of injury.

- Frequently assess skin, particularly in pressure areas and over bony prominences. *Skin breakdown can progress rapidly when circulation is impaired.*
- Reposition the client at least every 2 hours. Provide skin care with each position change. *Frequent position changes minimize tissue pressure and promote blood flow to tissues.*
- Provide an egg-crate mattress or alternating pressure mattress, foot cradle, heel protectors, and other devices to reduce pressure on tissues. *These devices, which distribute pressure away from bony prominences, reduce the risk of skin breakdown.*

### Risk for Impaired Gas Exchange

With fluid volume excess, gas exchange may be impaired by edema of pulmonary interstitial tissues. Acute pulmonary edema is a serious and potentially life-threatening complication of pulmonary congestion.

- Auscultate lungs for presence or worsening of crackles and wheezes; auscultate heart for extra heart sounds. *Crackles and wheezes indicate pulmonary congestion and edema. A gallop rhythm ($S_3$) may indicate diastolic overloading of the ventricles secondary to fluid volume excess.*
- Place in Fowler's position if dyspnea or orthopnea is present. *Fowler's position improves lung expansion by decreasing the pressure of abdominal contents on the diaphragm.*
- Monitor oxygen saturation levels and **arterial blood gases (ABGs)** for evidence of impaired gas exchange ($Sao_2 < 92\%$ to 95%; $Pao_2 < 80$ mmHg). Administer oxygen as indicated. *Edema of interstitial lung tissues can interfere with gas exchange and delivery to body tissues. Supplemental oxygen promotes gas exchange across the alveolar-capillary membrane, improving tissue oxygenation.*

## Community-Based Care

Teaching for home care focuses on managing the underlying cause of fluid volume excess and preventing future episodes of excess fluid volume. Address the following topics when preparing the client and family for home care:

- Signs and symptoms of excess fluid and when to contact the care provider
- Prescribed medications: when and how to take, intended and adverse effects, what to report to care provider
- Recommended or prescribed diet; ways to reduce sodium intake; how to read food labels for salt and sodium content; use of salt substitutes, if allowed (Box 10–5)
- If restricted, the amount and type of fluids to take each day; how to balance intake over 24 hours
- Monitoring weight; changes reported to care provider
- Ways to decrease dependent edema:
  a. Change position frequently.
  b. Avoid restrictive clothing.
  c. Avoid crossing the legs when sitting.
  d. Wear support stockings or hose.
  e. Elevate feet and legs when sitting.

**BOX 10–5 Client Teaching**

**Low-Sodium Diet**

- Reducing sodium intake will help the body excrete excess sodium and water.
- The body needs less than one-tenth of a teaspoon of salt per day.
- Approximately one-third of sodium intake comes from salt added to foods during cooking and at the table; one-fourth to one-third comes from processed foods; and the rest comes from food and water naturally high in sodium.
- Sodium compounds are used in foods as preservatives, leavening agents, and flavor enhancers.
- Many nonprescription drugs (such as analgesics, cough medicine, laxatives, and antacids) as well as toothpastes and mouthwashes contain high amounts of sodium.
- Low-sodium salt substitutes are not really sodium free and may contain half as much sodium as regular salt.
- Use salt substitutes sparingly; larger amounts often taste bitter instead of salty.
- The preference for salt will eventually diminish.
- Salt, monosodium glutamate, baking soda, and baking powder contain substantial amounts of sodium.
- Read labels.
- In place of salt or salt substitutes, use herbs, spices, lemon juice, vinegar, and wine as flavoring when cooking.

- How to protect edematous skin from injury:
  a. Do not walk barefoot.
  b. Buy good-fitting shoes; shop in the afternoon when feet are more likely to be swollen.
- Using additional pillows or a recliner to sleep, to relieve orthopnea.

# SODIUM IMBALANCE

Sodium is the most plentiful electrolyte in ECF, with normal serum sodium levels ranging from 135 to 145 mEq/L. Sodium is the primary regulator of the volume, osmolality, and distribution of ECF. It also is important to maintain neuromuscular activity. Because of the close interrelationship between sodium and water balance, disorders of fluid volume and sodium balance often occur together. Sodium imbalances affect the osmolality of ECF and water distribution between the fluid compartments. When sodium levels are low (hyponatremia), water is drawn into the cells of the body, causing them to swell. In contrast, high levels of sodium in ECF (hypernatremia) draw water out of body cells, causing them to shrink.

## Overview of Normal Sodium Balance

Most of the body's sodium comes from dietary intake. Although a sodium intake of 500 mg per day is usually sufficient to meet the body's needs, the average intake of sodium by adults in the United States is about 6 to 15 g per day (Porth, 2005). Other sources of sodium include prescription drugs and certain self-prescribed remedies. Sodium is primarily excreted

by the kidneys. A small amount is excreted through the skin and the gastrointestinal tract.

The kidney is the primary regulator of sodium balance in the body. The kidney excretes or conserves sodium in response to changes in vascular volume. A fall in blood volume prompts several mechanisms that lead to sodium and water retention:

- The renin–angiotensin–aldosterone system (see Figure 10–9) is stimulated. Angiotensin II prompts the renal tubules to reabsorb sodium. It also causes vasoconstriction, slowing blood flow through the kidney and reducing glomerular filtration. This further reduces the amount of sodium excreted. Angiotensin II promotes the release of aldosterone from the adrenal cortex. In the presence of aldosterone, more sodium is reabsorbed in the cortical collecting tubules of the kidney, and more potassium is eliminated in the urine.
- ADH is released from the posterior pituitary (see Figure 10–10). ADH promotes sodium and water reabsorption in the distal tubules of the kidney, reducing urine output and expanding blood volume.

In contrast, when blood volume expands, sodium and water elimination by the kidneys increases.

- The **glomerular filtration rate** (the rate at which plasma is filtered through the glomeruli of the kidney) increases, allowing more water and sodium to be filtered and excreted.
- ANP is released by cells in the atria of the heart. ANP increases sodium excretion by the kidneys.
- ADH release from the pituitary gland is inhibited. In the absence of ADH, the distal tubule is relatively impermeable to water and sodium, allowing more to be excreted in the urine. Table 10–5 summarizes the causes and effects of sodium imbalances.

## The Client with Hyponatremia

Hyponatremia is a serum sodium level of less than 135 mEq/L. Hyponatremia usually results from a loss of sodium from the body, but it may also be caused by water gains that dilute ECF.

### Pathophysiology

Excess sodium loss can occur through the kidneys, gastrointestinal tract, or skin. Diuretic medications, kidney diseases, or adrenal insufficiency with impaired aldosterone and cortisol production can lead to excessive sodium excretion in urine. Vomiting, diarrhea, and gastrointestinal suction are common causes of excess sodium loss through the GI tract. Sodium may also be lost when gastrointestinal tubes are irrigated with water instead of saline, or when repeated tap water enemas are administered (Porth, 2005). Excessive sweating or loss of skin surface (as with an extensive burn) can also cause excessive sodium loss.

Water gains that can lead to hyponatremia may occur with:

- Systemic diseases such as heart failure, renal failure, or cirrhosis of the liver
- Syndrome of inappropriate secretion of antidiuretic hormone (SIADH), in which water excretion is impaired
- Excessive administration of hypotonic intravenous fluids.

Hyponatremia causes a drop in serum osmolality. Water shifts from ECF into the intracellular space, causing cells to swell and reducing the osmolality of intracellular fluid. Many of the manifestations of hyponatremia can be attributed to cellular edema and hypo-osmolality.

### Manifestations

The manifestations of hyponatremia depend on the rapidity of onset, the severity, and the cause of the imbalance. If the condition develops slowly, manifestations are usually not experienced until the serum sodium levels reach 125 mEq/L. In addition, the manifestations of hyponatremia vary, depending on extracellular fluid volume. Early manifestations of hyponatremia include muscle cramps, weakness, and fatigue from its effects on muscle cells. Gastrointestinal function is affected, causing anorexia, nausea and vomiting, abdominal cramping, and diarrhea.

As sodium levels continue to decrease, the brain and nervous system are affected by cellular edema. Neurologic mani-

TABLE 10–5 **Causes and Manifestations of Sodium Imbalances**

| IMBALANCE | POSSIBLE CAUSES | MANIFESTATIONS |
|---|---|---|
| **Hyponatremia**<br>Serum sodium < 135 mEq/L<br>Critical value < 120 mEq/L<br>*Other Lab Values*<br>Serum osmolality < 280 mOsm/kg<br>Critical value < 250 mOsm/kg | ■ Excess sodium loss through kidneys, GI tract, or skin<br>■ Water gains related to renal disease, heart failure, or cirrhosis of the liver<br>■ SIADH<br>■ Excessive hypotonic IV fluids | ■ Anorexia, nausea, vomiting, abdominal cramping, and diarrhea<br>■ Headache<br>■ Altered mental status<br>■ Muscle cramps, weakness, and tremors<br>■ Seizures and coma |
| **Hypernatremia**<br>Serum sodium > 145 mEq/L<br>Critical value > 160 mEq/L<br>*Other Lab Values*<br>Serum osmolality > 295 mOsm/kg<br>Critical value > 325 mOsm/kg | ■ Altered thirst<br>■ Inability to respond to thirst sensation or obtain water<br>■ Profuse sweating<br>■ Diarrhea<br>■ Diabetes insipidus<br>■ Oral electrolyte solutions or hyperosmolar tube-feeding formulas<br>■ Excess IV fluids such as normal saline, 3% or 5% sodium chloride, or sodium bicarbonate | ■ Thirst<br>■ Increased temperature<br>■ Dry, sticky mucous membranes<br>■ Restlessness<br>■ Weakness<br>■ Altered mental status<br>■ Decreasing level of consciousness<br>■ Muscle twitching<br>■ Seizures |

festations progress rapidly when the serum sodium level falls below 120 mEq/L and include headache, depression, dulled sensorium, personality changes, irritability, lethargy, hyperreflexia, muscle twitching, and tremors. If serum sodium falls to very low levels, convulsions and coma are likely to occur.

When hyponatremia is associated with decreased ECF volume, the manifestations are those of hypovolemia (*hypotonic dehydration*). In hyponatremia associated with fluid volume excess, manifestations include those of hypervolemia.

## INTERDISCIPLINARY CARE

Interdisciplinary management of hyponatremia focuses on restoring normal blood volume and serum sodium levels.

### Diagnosis

The following laboratory tests may be ordered.

- *Serum sodium* and *osmolality* are decreased in hyponatremia (serum sodium < 135 mEq/L; serum osmolality < 275 mOsm/kg).
- A *24-hour urine specimen* is obtained to evaluate sodium excretion. In conditions associated with normal or increased extracellular volume (such as SIADH), urinary sodium is increased; in conditions resulting from losses of isotonic fluids (e.g., sweating, diarrhea, vomiting, and third-space fluid accumulation), by contrast, urinary sodium is decreased.

### Medications

When both sodium and water have been lost (hyponatremia with hypovolemia), sodium-containing fluids are given to replace both water and sodium. These fluids may be given by mouth, nasogastric tube, or intravenously. Isotonic Ringer's solution or isotonic saline (0.9% NaCl) solution may be administered. Cautious administration of intravenous 3% or 5% NaCl solution may be necessary in clients who have very low plasma sodium levels (110 to 115 mEq/L).

Loop diuretics are administered to clients who have hyponatremia with normal or excess ECF volume. Loop diuretics promote an isotonic diuresis and fluid volume loss without hyponatremia (see page 210). Thiazide diuretics are avoided because they cause a relatively greater sodium loss in relation to water loss.

In addition, drugs to treat the underlying cause of hyponatremia may be administered.

### Fluid and Dietary Management

If hyponatremia is mild, increasing the intake of foods high in sodium may restore normal sodium balance (see Box 10–4). Fluids often are restricted to help reduce ECF volume and correct hyponatremia (see Box 10–3).

## NURSING CARE

Nursing care of the client with hyponatremia focuses on identifying clients at risk and managing problems resulting from the systemic effects of the disorder.

### Health Promotion

People at risk for mild hyponatremia include those who participate in activities that increase fluid loss through excessive perspiration (diaphoresis) and then replace those losses by drinking large amounts of water. This includes athletes, people who do heavy labor in high environmental temperatures, and older adults living in non–air-conditioned settings during hot weather. Teach the following to clients who are at risk:

- Manifestations of mild hyponatremia, including nausea, abdominal cramps, and muscle weakness
- The importance of drinking liquids containing sodium and other electrolytes at frequent intervals when perspiring heavily, when environmental temperatures are high, and/or if watery diarrhea persists for several days.

### Assessment

Assessment data related to hyponatremia include the following:

- *Health history:* Current manifestations, including nausea and vomiting, abdominal discomfort, muscle weakness, headache, other symptoms; duration of symptoms and any precipitating factors such as heavy perspiration, vomiting, or diarrhea; chronic diseases such as heart or renal failure, cirrhosis of the liver, or endocrine disorders; current medications.
- *Physical assessment:* Mental status and level of consciousness; vital signs including orthostatic vitals and peripheral pulses; presence of edema or weight gain.
- *Diagnostic tests:* Serum sodium and osmolality; serum potassium.

### Nursing Diagnoses and Interventions

#### Risk for Imbalanced Fluid Volume

Because of its role in maintaining fluid balance, sodium imbalances often are accompanied by water imbalances. In addition, treatment of hyponatremia can affect the client's fluid balance.

- Monitor intake and output, weigh daily, and calculate 24-hour fluid balance. *Fluid excess or deficit may occur with hyponatremia.*

**PRACTICE ALERT**

Carefully monitor clients receiving sodium-containing intravenous solutions for signs of hypervolemia (increased blood pressure and CVP, tachypnea, tachycardia, gallop rhythm [$S_3$ and/or $S_4$ heart sounds], shortness of breath, crackles). Hypertonic saline solutions can lead to hypervolemia, particularly in clients with cardiovascular or renal disease.

- Use an intravenous flow control device to administer hypertonic saline (3% and 5% NaCl) solutions; carefully monitor flow rate and response. *Hypertonic solutions can increase the risk of pulmonary and cerebral edema due to water retention. Careful monitoring is vital to prevent these complications and possible permanent damage.*
- If fluids are restricted, explain the reason for the restriction, the amount of fluid allowed, and how to calculate fluid intake. *Teaching increases the client's sense of control and compliance.*

For additional nursing interventions that may apply to the client with hyponatremia, review the discussions of fluid volume deficit and fluid volume excess.

### Risk for Ineffective Cerebral Tissue Perfusion

The client with severe hyponatremia experiences fluid shifts that cause an increase in intracellular fluid volume. This can cause brain cells to swell, increasing pressure within the cranial vault.

- Monitor serum electrolytes and serum osmolality. Report abnormal results to the care provider. *As serum sodium levels fall, the manifestations and neurologic effects of hyponatremia become increasingly severe.*
- Assess for neurologic changes, such as lethargy, altered level of consciousness, confusion, and convulsions. Monitor mental status and orientation. Compare baseline data with continuing assessments. *Serum sodium levels of 115 to 120 mEq/L can cause headache, lethargy, and decreased responsiveness; sodium levels less than 110 to 115 mEq/L may cause seizures and coma.*
- Assess muscle strength and tone, and deep tendon reflexes. *Increasing muscle weakness and decreased deep tendon reflexes are manifestations of increasing hyponatremia.*

**PRACTICE ALERT**

Maintain a quiet environment, and institute seizure precautions in clients with severe hyponatremia. Severe hyponatremia can lead to seizures. A quiet environment reduces neurologic stimulation. Safety precautions, such as ensuring that side rails are up and having an airway readily available, reduce risk of injury from seizure.

## Community-Based Care

Teaching for home care focuses on the underlying cause of the sodium deficit and often on prevention. Teach clients who have experienced hyponatremia and those who are at risk for developing hyponatremia about the following:

- Manifestations of mild and more severe hyponatremia to report to the primary care provider
- The importance of regular serum electrolyte monitoring if taking a potent diuretic or on a low-sodium diet
- Types of foods and fluids to replace sodium orally if dietary sodium is not restricted.

## The Client with Hypernatremia

Hypernatremia is a serum sodium level greater than 145 mEq/L. It may develop when sodium is gained in excess of water, or when water is lost in excess of sodium. Either fluid volume deficit or fluid volume excess often accompany hypernatremia.

### Pathophysiology

Two regulatory mechanisms protect the body from hypernatremia: (1) Excess sodium in ECF stimulates the release of ADH so more water is retained by the kidneys; and (2) the thirst mechanism is stimulated to increase the intake of water (Metheny, 2000). These two factors increase extracellular water, diluting the excess sodium and restoring normal levels. Because of the effectiveness of these mechanisms, hypernatremia almost never occurs in clients who have an intact thirst mechanism and access to water.

Water deprivation is a cause of hypernatremia in clients who are unable to respond to thirst due to altered mental status or physical disability. Excess water loss may occur with watery diarrhea or increased insensible losses (due to fever, hyperventilation, excessive perspiration, or massive burns). Unless water is adequately replaced, clients with diabetes insipidus (see Chapter 19 ∞) also may develop hypernatremia. Excess sodium intake can result from ingestion of excess salt or hypertonic intravenous solutions. Clients who experience near-drowning in seawater are at risk for hypernatremia, as are clients with heatstroke.

### Manifestations

Hypernatremia (also known as *hypertonic dehydration*) causes hyperosmolality of ECF. As a result, water is drawn out of cells, leading to cellular dehydration. The most serious effects of cellular dehydration are seen in the brain. As brain cells contract, neurologic manifestations develop. The brain itself shrinks, causing mechanical traction on cerebral vessels. These vessels may tear and bleed. Although the brain rapidly adapts to hyperosmolality to minimize the water loss, acute hypernatremia can cause widespread cerebral vascular bleeding (Metheny, 2000).

Thirst is the first manifestation of hypernatremia. If thirst is not relieved, the primary manifestations relate to altered neurologic function (see Table 10–5). Initial lethargy, weakness, and irritability can progress to seizures, coma, and death in severe hypernatremia. Both the severity of the sodium excess and the rapidity of its onset affect the manifestations of hypernatremia.

## INTERDISCIPLINARY CARE

Treatment of hypernatremia depends on its cause. Hypernatremia is corrected slowly (over a 48-hour period) to avoid development of cerebral edema secondary to a shift of water into the brain cells.

### Diagnosis

The following laboratory and diagnostic tests may be ordered:

- *Serum sodium levels* are greater than 145 mEq/L in hypernatremia.
- *Serum osmolality* is greater than 295 mOsm/kg in hypernatremia.
- The *water deprivation test* may be conducted to identify diabetes insipidus. Water and all other fluids are withheld for a specified period of time. During this time, urine specimens are obtained for osmolality and specific gravity. No change in these values supports the diagnosis of diabetes insipidus.

### Medications

The principal treatment for hypernatremia is oral or intravenous water replacement. Hypotonic intravenous fluids such as 0.45% NaCl solution or 5% dextrose in water (which is isotonic when administered, but provides pure water when the glucose is metabolized) may be administered to correct the water deficit. Diuretics may also be given to increase sodium excretion.

## NURSING CARE

The primary focus of nursing care related to hypernatremia is prevention. Measures to prevent hypernatremia include identifying risk factors, teaching clients and caregivers, monitoring laboratory test results, and working with the interdisciplinary team to reduce the potential for hypernatremia.

### Health Promotion

Clients at risk for hypernatremia, as well as their care providers, need teaching to prevent this electrolyte disorder. Instruct caregivers of debilitated clients who are unable to perceive thirst or unable to respond to it to offer fluids at regular intervals. If the client is unable to maintain adequate fluid intake, contact the primary care provider about an alternate route for fluid intake (e.g., a feeding tube). Teach care providers the importance of providing adequate water for clients receiving tube feedings (many of which are hypertonic).

### Assessment

Assessment data related to hypernatremia include the following:

- *Health history:* Duration of symptoms and any precipitating factors such as water deprivation, increased water loss due to heavy perspiration, temperature or rapid breathing, diarrhea, excess salt intake, or diabetes insipidus; current medications; perception of thirst.
- *Physical assessment:* Vital signs including temperature; mucous membranes; altered mental status or level of consciousness; manifestations of fluid volume excess or fluid volume deficit.
- *Diagnostic tests:* Monitor serum sodium and osmolality, serum potassium.

### Nursing Diagnoses and Interventions

#### Risk for Injury

Mental status and brain function may be affected by hypernatremia itself or by rapid correction of the condition that leads to cerebral edema. In either case, closely monitor the client and take precautions to reduce risk of injury.

- Monitor and maintain fluid replacement to within the prescribed limits. Monitor serum sodium levels and osmolality; report rapid changes to the care provider. *Rapid water replacement or rapid changes in serum sodium or osmolality can cause fluid shifts within the brain, increasing the risk of bleeding or cerebral edema.*
- Monitor neurologic function, including mental status, level of consciousness, and other manifestations such as headache, nausea, vomiting, elevated blood pressure, and decreased pulse rate. *Both hypernatremia and rapid correction of hypernatremia affect the brain and brain function. Careful monitoring is vital to detect changes in mental status that may indicate cerebral bleeding or edema.*
- Institute safety precautions as necessary: Keep the bed in its lowest position, side rails up and padded, and an airway at bedside. *Clients with sodium disorders are at risk for injury due to seizure activity and changes in mental status.*
- Keep clocks, calendars, and familiar objects at bedside. Orient to time, place, and circumstances as needed. Allow significant others to remain with the client as much as possible. *An unfamiliar environment and altered thought processes can further increase the client's risk for injury. Significant others provide a sense of security and reduce the client's anxiety.*

### Community-Based Care

When preparing the client who has experienced hypernatremia for home care, discuss the following topics:

- The importance of responding to thirst and consuming adequate fluids (If the client is dependent on a caregiver, stress the importance of regularly offering fluids to the caregiver.)
- If prescribed, guidelines for following a low-sodium diet (see Box 10–5)
- Use and effects (intended and unintended) of any prescribed diuretic or other medication
- The importance of following a schedule for regular monitoring of serum electrolyte levels and reporting manifestations of imbalance to care provider.

## POTASSIUM IMBALANCE

Potassium, the primary intracellular cation, plays a vital role in cell metabolism, and cardiac and neuromuscular function. The normal serum (ECF) potassium level is 3.5 to 5.0 mEq/L.

### Overview of Normal Potassium Balance

Most potassium in the body is found within the cells (ICF), which have a concentration of 140 to 150 mEq/L. This significant difference in the potassium concentrations of ICF and ECF helps maintain the resting membrane potential of nerve and muscle cells; either a deficit or an excess of potassium can adversely affect neuromuscular and cardiac function. The higher intracellular potassium concentration is maintained by the sodium-potassium pump.

To maintain its balance, potassium must be replaced daily. Normally, potassium is supplied in food. Virtually all foods contain potassium, although some foods and fluids are richer sources of this element than others (Box 10–6).

The kidneys eliminate potassium very efficiently; even when potassium intake is stopped, the kidneys continue to excrete it. Because the kidneys do not conserve potassium well, significant amounts may be lost through this route. However, because the kidneys are the principal organs involved in the elimination of potassium, renal failure can lead to potentially serious elevations of serum potassium.

Aldosterone helps regulate potassium elimination by the kidneys. An increased potassium concentration in ECF stimulates aldosterone production by the adrenal gland. The kidneys respond to aldosterone by increasing potassium excretion. Changes in aldosterone secretion can profoundly affect the serum potassium level.

Normally only small amounts of potassium are lost in the feces, but substantial amounts may be lost from the gastrointestinal

**BOX 10–6 Foods High in Potassium**

**Fruits**
- Apricots
- Avocados
- Bananas
- Cantaloupe
- Dates
- Oranges
- Raisins

**Vegetables and Vegetable Juices**
- Carrots
- Cauliflower
- Mushrooms
- Peas
- Potatoes
- Spinach
- Tomatoes
- V-8 Juice

**Meats and Fish**
- Beef
- Chicken
- Kidney
- Liver
- Lobster
- Pork loin
- Tuna
- Turkey
- Salmon

**Milk Products**
- Buttermilk
- Chocolate milk
- Evaporated milk
- Low-fat yogurt
- Milk

tract with diarrhea or through drainage from an ileostomy (a permanent opening into the small bowel).

Potassium constantly shifts into and out of the cells. This movement between ICF and ECF can significantly affect the serum potassium level. For example, potassium shifts into or out of the cells in response to changes in hydrogen ion concentration (pH, discussed later in this chapter) as the body strives to maintain a stable acid–base balance.

The significant difference between intracellular and extracellular potassium concentrations is vital to the resting membrane potential of cells. Resting membrane potential, in turn, is necessary for transmitting nerve impulses. Potassium imbalances affect transmission and conduction of nerve impulses, maintenance of normal cardiac rhythms, and contraction of skeletal and smooth muscle (McCance & Huether, 2006).

As the primary intracellular cation, potassium plays a major role in regulating the osmolality of ICF, and it is involved in metabolic processes. Potassium is necessary for the storage of glycogen in skeletal muscle cells. Table 10–6 summarizes potassium imbalances, their causes, and manifestations.

## The Client with Hypokalemia

Hypokalemia is an abnormally low serum potassium (less than 3.5 mEq/L). It usually results from excess potassium loss, although hospitalized clients may be at risk for hypokalemia because of inadequate potassium intake.

### Pathophysiology

Excess potassium may be lost through the kidneys or the gastrointestinal tract. These losses deplete total potassium stores in the body.

- Excess potassium loss through the kidneys often is secondary to drugs such as potassium-wasting diuretics, corticosteroids, amphotericin B, and large doses of some antibiotics. Hyperaldosteronism, a condition in which the adrenal glands secrete excess aldosterone, also causes excess elimination of potassium through the kidneys. Glucosuria and osmotic diuresis (e.g., associated with diabetes mellitus) also cause potassium wasting through the kidneys (Metheny, 2000).

**TABLE 10–6 Causes and Manifestations of Potassium Imbalances**

| IMBALANCE | CAUSES | MANIFESTATIONS |
|---|---|---|
| **Hypokalemia**<br>Serum potassium < 3.5 mEq/L<br>Critical value < 2.5 mEq/L | ■ Excess GI losses: vomiting, diarrhea, ileostomy drainage<br>■ Renal losses: diuretics, hyperaldosteronism<br>■ Inadequate intake<br>■ Shift into cells: alkalosis, rapid tissue repair | Cardiovascular<br>■ Dysrhythmias<br>■ ECG changes<br>Gastrointestinal<br>■ Nausea and vomiting<br>■ Anorexia<br>■ Decreased bowel sounds<br>■ Ileus<br>Musculoskeletal<br>■ Muscle weakness<br>■ Leg cramps |
| **Hyperkalemia**<br>Serum potassium > 5.0 mEq/L<br>Critical value > 6.5 mEq/L | ■ Renal failure<br>■ Potassium-sparing diuretics<br>■ Adrenal insufficiency<br>■ Excess potassium intake (e.g., excess potassium replacement)<br>■ Aged blood<br>■ Shift out of cells: cell and tissue damage, acidosis | Cardiovascular<br>■ Tall, peaked T waves, widened QRS<br>■ Dysrhythmias<br>■ Cardiac arrest<br>Gastrointestinal<br>■ Nausea and vomiting<br>■ Abdominal cramping<br>■ Diarrhea<br>Neuromuscular<br>■ Muscle weakness<br>■ Paresthesias<br>■ Flaccid paralysis |

- Gastrointestinal losses of potassium result from severe vomiting, gastric suction, or loss of intestinal fluids through diarrhea or ileostomy drainage.

Potassium intake may be inadequate in clients who are unable or unwilling to eat for prolonged periods. Hospitalized clients are at risk, especially those on extended parenteral fluid therapy with solutions that do not contain potassium. Clients with anorexia nervosa or alcoholism may develop hypokalemia due to both inadequate intake and loss of potassium through vomiting, diarrhea, or laxative or diuretic use.

A *relative* loss of potassium occurs when potassium shifts from ECF into the cells. This usually is due to loss of hydrogen ion and alkalosis, although it also may occur during periods of rapid tissue repair (e.g., following a burn or trauma), in the presence of excess insulin (insulin promotes potassium entry into skeletal muscle and liver cells), during acute stress, or because of hypothermia. In these instances, the total body stores of potassium remain adequate.

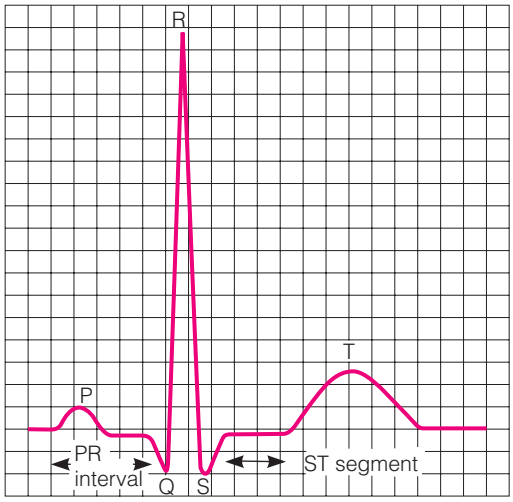
## Manifestations

Hypokalemia affects the transmission of nerve impulses, interfering with the contractility of smooth, skeletal, and cardiac muscle, as well as the regulation and transmission of cardiac impulses.

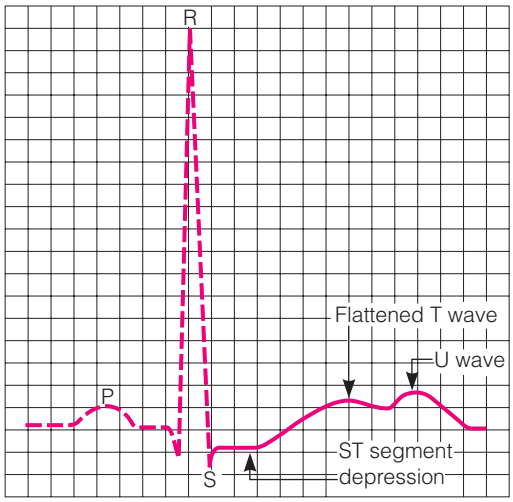
- Characteristic electrocardiogram (ECG) changes of hypokalemia include flattened or inverted T waves, the development of U waves, and a depressed ST segment (Figure 10–11 ■). The most serious cardiac effect is an increased risk of atrial and ventricular *dysrhythmias* (abnormal rhythms). Hypokalemia increases the risk for digitalis toxicity in clients receiving this drug used to treat heart failure (see Chapter 32 ∞).
- Hypokalemia affects both the resting membrane potential and intracellular enzymes in skeletal and smooth muscle cells. This causes skeletal muscle weakness and slowed peristalsis of the gastrointestinal tract. Muscles of the lower extremities are affected first, then the trunk and upper extremities. This effect of hypokalemia is magnified when serum calcium levels are above normal.
- Carbohydrate metabolism is affected by hypokalemia. Insulin secretion is suppressed, as is the synthesis of glycogen in skeletal muscle and the liver.

Hypokalemia also can affect kidney function, particularly the ability to concentrate urine. Severe hypokalemia can lead to *rhabdomyolysis*, a condition in which muscle fibers disintegrate, releasing myoglobin to be excreted in the urine.

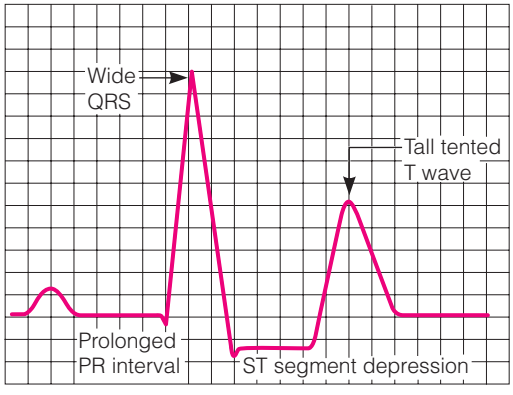
Manifestations of hypokalemia are more pronounced when potassium losses occur acutely. When hypokalemia develops gradually, potassium shifts out of the cells, helping maintain the ratio of intracellular to extracellular potassium. As a result, the neuromuscular manifestations of hypokalemia are less severe. The *Multisystem Effects of Hypokalemia* are summarized on the following page.



(a) Normal ECG



(b) ECG in hypokalemia



(c) ECG in hyperkalemia

**Figure 10–11** ■ The effects of changes in potassium levels on the electrocardiogram (ECG). *A,* Normal ECG; *B,* ECG in hypokalemia; *C,* ECG in hyperkalemia.

## Diagnosis

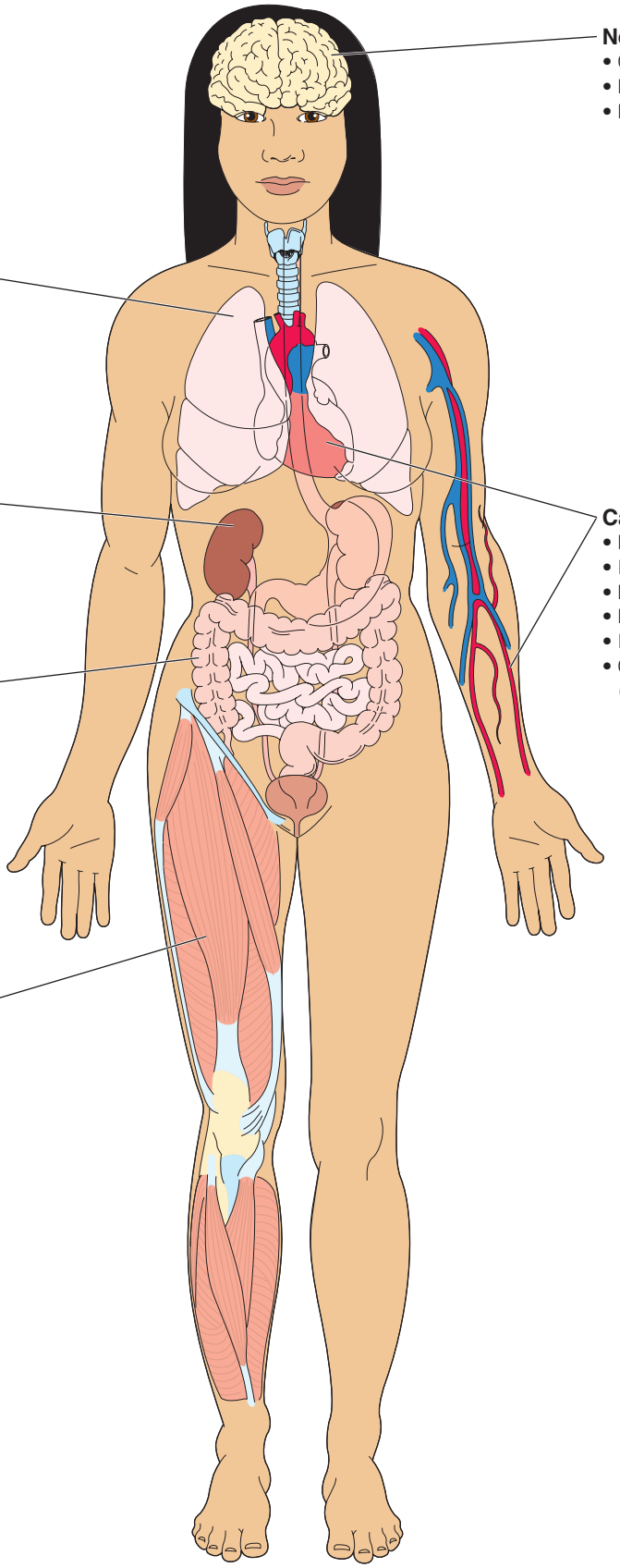
The following laboratory and diagnostic tests may be ordered:

- *Serum potassium* ($K^+$) is used to monitor potassium levels in clients who are at risk for or who are being treated for hypokalemia. A serum $K^+$ of 3.0 to 3.5 mEq/L is considered

# INTERDISCIPLINARY CARE

The management of hypokalemia focuses on prevention and treatment of a deficiency.

# MULTISYSTEM EFFECTS **of Hypokalemia**



**Neurologic**
- Confusion
- Depression
- Lethargy

**Respiratory**
- Respiratory arrest (severe hypokalemia)

**Urinary**
- Dilute urine
- Polyuria
- Polydipsia

**Cardiovascular**
- Dysrhythmias
- Irregular pulse
- Postural hypotension
- ECG (conduction) abnormalities
- Increased risk of digitalis toxicity
- Cardiac arrest (severe hypokalemia)

**Gastrointestinal**
- Nausea and vomiting
- Anorexia
- Diarrhea
- Decreased bowel sounds
- Ileus

**Musculoskeletal**
- Fatigue
- Leg cramps
- Muscle weakness
- Poor muscle tone
- Paresthesias, paralysis

mild hypokalemia. Moderate hypokalemia is defined as a serum $K^+$ of 2.5 to 3.0 mEq/L, and severe hypokalemia as a serum $K^+$ of less than 2.5 mEq/L (Metheny, 2000).

- *Arterial blood gases (ABGs)* are measured to determine acid–base status. An increased pH (alkalosis) often is associated with hypokalemia. (See Table 10–10 later in this chapter for normal ABG values.)
- *Renal function studies*, such as *serum creatinine* and *blood urea nitrogen (BUN),* may be ordered to evaluate for potential causes or effects of hypokalemia.
- *ECG recordings* are obtained to evaluate the effects of hypokalemia on the cardiac conduction system.

### Medications

Oral and/or parenteral potassium supplements are given to prevent and, as needed, treat hypokalemia. To prevent hypokalemia in the client taking nothing by mouth, 40 mEq of potassium chloride per day is added to intravenous fluids. The dose used to treat hypokalemia includes the daily maintenance requirement, replacement of ongoing losses (e.g., gastric suction), and additional potassium to correct the existing deficit. Several days of therapy may be required. Commonly prescribed potassium supplements, their actions, and nursing implications are described in the Medication Administration box below.

### Nutrition

A diet high in potassium-rich foods is recommended for clients at risk for developing hypokalemia or to supplement drug therapy (see Box 10–6).

## NURSING CARE

See the following page for a Nursing Care Plan: A Client with Hypokalemia.

## Health Promotion

When providing general health education, discuss using balanced electrolyte solutions (e.g., Pedialyte or sports drinks) to replace abnormal fluid losses (excess perspiration, vomiting, or severe diarrhea). Discuss the necessity of preventing hypokalemia with clients at risk. Provide diet teaching and refer clients with anorexia nervosa for counseling. Stress the potassium-losing effects of taking diuretics and using laxatives to enhance weight loss. Discuss the potassium-wasting effects of most diuretics with clients taking these drugs, and encourage a diet rich in high-potassium foods, as well as regular monitoring of serum potassium levels.

## Assessment

Assessment data related to hypokalemia include the following:

- *Health history:* Current manifestations, including anorexia, nausea and vomiting, abdominal discomfort, muscle weakness or cramping, other symptoms; duration of symptoms and any precipitating factors such as diuretic use, prolonged vomiting or diarrhea; chronic diseases such as diabetes, hyperaldosteronism, or Cushing's syndrome; current medications.
- *Physical assessment:* Mental status; vital signs including orthostatic vitals, apical and peripheral pulses; bowel sounds, abdominal distention; muscle strength and tone.
- *Diagnostic tests:* Serum electrolytes, $K^+$, $Na^+$, and $Ca^{2+}$ in particular, arterial pH and other ABG results, renal function tests (creatinine and BUN), ECG changes.

## Nursing Diagnoses and Interventions

The effects of hypokalemia on cardiac impulse transmission and cardiac and skeletal muscle function are the highest priority nursing care focus.

### MEDICATION ADMINISTRATION — Hypokalemia

**POTASSIUM SOURCES**

**Potassium acetate (Tri-K)**
**Potassium bicarbonate (K + Care ET)**
**Potassium citrate (K-Lyte)**
**Potassium chloride (K-Lease, Micro-K 10, Apo-K)**
**Potassium gluconate (Kaon Elixir, Royonate)**

Potassium is rapidly absorbed from the gastrointestinal tract; potassium chloride is the agent of choice, because low chloride often accompanies low potassium. Potassium is used to prevent and/or treat hypokalemia (e.g., with parenteral nutrition and potassium-wasting diuretics, and prophylactically after major surgery).

**Nursing Responsibilities**

- When giving oral forms of potassium:
  a. Dilute or dissolve effervescent, soluble, or liquid potassium in fruit or vegetable juice or cold water.
  b. Chill to increase palatability.
  c. Give with food to minimize GI effects.
- When giving parenteral forms of potassium:
  a. Administer slowly.
  b. Do *not* administer undiluted.
  c. Assess injection site frequently for signs of pain and inflammation.
  d. Use an infusion control device.
- Assess for abdominal pain, distention, gastrointestinal bleeding; if present, do not administer medication. Notify healthcare provider.
- Monitor fluid intake and output.
- Assess for manifestations of hyperkalemia: weakness, feeling of heaviness in legs, mental confusion, hypotension, cardiac dysrhythmias, changes in ECG, increased serum potassium levels.

**Health Education for the Client and Family**

- Do not take potassium supplements if you are also taking a potassium-sparing diuretic.
- When parenteral potassium is discontinued, eat potassium-rich foods.
- Do not chew enteric-coated tablets or allow them to dissolve in the mouth; this may affect the potency and action of the medications.
- Take potassium supplements with meals.
- Do not use salt substitutes when taking potassium (most salt substitutes are potassium based).

## NURSING CARE PLAN A Client with Hypokalemia

Rose Ortiz is a 72-year-old widow who lives alone, although close to her daughter's home. Ms. Ortiz has mild heart failure and is being treated with digoxin (Lanoxin) 0.125 mg, furosemide (Lasix) 40 mg PO daily, and a mildly restricted sodium diet (2 g daily). For the last several weeks, Ms. Ortiz has complained that she feels weak and sometimes faint, light-headed, and dizzy. Serum electrolyte tests ordered by her physician reveal a potassium level of 2.4 mEq/L. Potassium chloride solution (Kaochlor 10%, 20 mEq/15 mL) PO twice daily is prescribed, and Ms. Ortiz is referred to Nancy Walters, RN, for follow-up care.

### ASSESSMENT

Ms. Ortiz's health history reveals that she has rigidly adhered to her sodium-restricted diet and has been compliant in taking her prescribed medications, with the exception of occasionally taking an additional "water pill" when her ankles swell. She takes a laxative every evening to ensure a daily bowel movement. She states that she is reluctant to take the potassium chloride the doctor has ordered because her neighbor complains that his potassium supplement upsets his stomach. Physical assessment findings included T 98.4, P 70, R 20, and BP 138/84. Muscle strength in her upper extremities is normal and equal; lower extremity strength is weak but equal. Sensation is normal.

### DIAGNOSES

- *Risk for Injury* related to muscle weakness
- *Risk for Ineffective Health Maintenance* related to lack of knowledge about how diuretic therapy and laxative use affect potassium levels

### EXPECTED OUTCOMES

- Maintain potassium level within normal limits (3.5 to 5.0 mEq/L).
- Regain normal muscle strength.
- Remain free of injury.
- Verbalize understanding of the effects of diuretic therapy and laxatives on potassium levels.
- Identify measures to avoid gastrointestinal irritation when taking oral potassium.
- Identify potassium-rich foods.

### PLANNING AND IMPLEMENTATION

- Explain need to use caution when ambulating, particularly when climbing or descending stairs.
- Discuss side effects of furosemide, and explain how taking additional tablets may have contributed to hypokalemia.
- Discuss alternative measures to prevent constipation without using laxatives on a regular basis (e.g., high-fiber diet, adequate fluid intake).
- Explain purpose of the prescribed potassium and its role in reversing muscle weakness.
- Teach to take potassium supplement after breakfast and supper, diluted in 4 oz of juice or water, and to sip it slowly over a 5- to 10-minute period. Advise to call if gastric irritation occurs.
- Discuss dietary sources of potassium; provide a list of potassium-rich foods.

### EVALUATION

On a follow-up visit 1 week later, Ms. Ortiz states that her muscle weakness, dizziness, and other symptoms have resolved. She is taking the prescribed drugs as directed and is using laxatives only two or three times a week. Ms. Ortiz reports that she has increased her intake of potassium-rich foods and fluids and of high-fiber foods. Her potassium level is within normal limits.

### CRITICAL THINKING IN THE NURSING PROCESS

1. What is the pathophysiologic basis for Ms. Ortiz's muscle weakness and dizziness?
2. How might the chronic overuse of laxatives contribute to hypokalemia?
3. Describe the interaction of digitalis, diuretics, and potassium.
4. Develop a plan of care for Ms. Ortiz for the nursing diagnosis of *Perceived Constipation.*

***See Evaluating Your Response in Appendix C.***

## Decreased Cardiac Output

Hypokalemia affects the strength of cardiac contractions and can lead to dysrhythmias that further impair cardiac output. Hypokalemia also alters the response to cardiac drugs, such as digitalis and the antidysrhythmics.

- Monitor serum potassium levels, particularly in clients at risk for hypokalemia (those with excess losses due to drug therapy, gastrointestinal losses, or who are unable to consume a normal diet). Report abnormal levels to the care provider. *Potassium must be replaced daily, because the body is unable to conserve it. Either lack of intake or abnormal losses of potassium in the urine or gastric fluids can lead to hypokalemia.*
- Monitor vital signs, including orthostatic vitals and peripheral pulses. *As cardiac output falls, the pulse becomes weak and thready. Orthostatic hypotension may be noted with decreased cardiac output.*

### PRACTICE ALERT

Place clients with severe hypokalemia on a cardiac monitor. Closely monitor cardiac rhythm and observe for characteristic ECG changes of hypokalemia (ST segment depression, flattened T waves, and U waves). Report rhythm changes and treat as indicated. Severe hypokalemia can cause life-threatening dysrhythmias.

- Monitor clients taking digitalis for toxicity. Monitor response to antidysrhythmic drugs. *Hypokalemia potentiates digitalis effects and increases resistance to certain antidysrhythmics.*
- Dilute intravenous potassium and administer using an electronic infusion device. In general, potassium is given no faster than 10 to 20 mEq/h. Closely monitor intravenous flow rate and response to potassium replacement. *Rapid potassium administration is dangerous and can lead to hyperkalemia and cardiac arrest.*

**PRACTICE ALERT**
Never administer undiluted potassium directly into the vein.

### Activity Intolerance

Muscle cramping and weakness are common early manifestations of hypokalemia. The lower extremities are usually affected initially. This muscle weakness can cause the client to fatigue easily, particularly with activity.

- Monitor skeletal muscle strength and tone, which are affected by moderate hypokalemia. *Increasing weakness, paresthesias, or paralysis of muscles or progression of affected muscles to include the upper extremities or trunk can indicate a further drop in serum potassium levels.*
- Monitor respiratory rate, depth, and effort; heart rate and rhythm; and blood pressure at rest and following activity. *Tachypnea, dyspnea, tachycardia, and/or a change in blood pressure may indicate decreasing ability to tolerate activities. Report changes to the care provider.*
- Assist with self-care activities as needed. *Increasing muscle weakness can lead to fatigue and affect the ability to meet self-care needs.*

### Risk for Imbalanced Fluid Volume

- Maintain accurate intake and output records. *Gastrointestinal fluid losses can lead to significant potassium losses.*
- Monitor bowel sounds and abdominal distention. *Hypokalemia affects smooth muscle function and can lead to slowed peristalsis and paralytic ileus.*

### Acute Pain

Discomfort is common when intravenous potassium chloride at a concentration of more than 40 mEq/L is given into a peripheral vein.

- When possible, administer intravenous KCl through a central line. *The rapid blood flow through central veins dilutes the KCl solution, decreasing discomfort.*
- Spread the total daily dose of KCl over 24 hours to minimize the concentration of intravenous solutions. *High concentrations of KCl are irritating to vein walls, particularly if inflammation is present.*
- Discuss with the physician using a small amount of lidocaine prior to or with the infusion. *Both a lidocaine bolus given at the infusion site and a small amount of lidocaine in the intravenous infusion have been shown to at least partially relieve discomfort associated with concentrated potassium solutions* (Metheny, 2000).

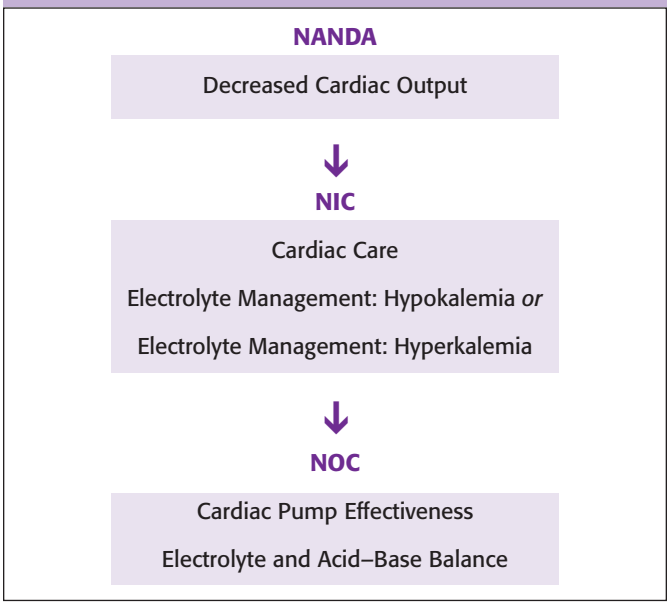
## Using NANDA, NIC, and NOC

Chart 10–2 shows links between NANDA nursing diagnoses, NIC, and NOC for a client with a potassium imbalance.

## Community-Based Care

The focus in preparing the client with or at risk for hypokalemia is prevention. Discharge planning focuses on teaching self-care practices. Include the following topics when preparing the client and family for home care.

NANDA, NIC, AND NOC LINKAGES
CHART 10–2 **The Client with Potassium Imbalance**




Data from *NANDA's Nursing Diagnoses: Definitions & classification 2005–2006* by NANDA International (2003), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

- Recommended diet, including a list of potassium-rich foods
- Prescribed medications and potassium supplements, their use, and desired and unintended effects
- Using salt substitutes (if recommended) to increase potassium intake; avoiding substitutes if taking a potassium supplement or potassium-sparing diuretic
- Manifestations of potassium imbalance (hypokalemia or hyperkalemia) to report to healthcare provider
- Recommendations for monitoring serum potassium levels
- If taking digitalis, manifestations of digitalis toxicity to report to healthcare provider
- Managing gastrointestinal disorders that cause potassium loss (vomiting, diarrhea, ileostomy drainage) to prevent hypokalemia.

## The Client with Hyperkalemia

Hyperkalemia is an abnormally high serum potassium (greater than 5 mEq/L). Hyperkalemia can result from inadequate excretion of potassium, excessively high intake of potassium, or a shift of potassium from the intracellular to the extracellular space. *Pseudohyperkalemia* (an erroneously high serum potassium reading) can occur if the blood sample hemolyzes, releasing potassium from blood cells, before it is analyzed. Hyperkalemia affects neuromuscular and cardiac function.

### Pathophysiology

Impaired renal excretion of potassium is a primary cause of hyperkalemia. Untreated renal failure, adrenal insufficiency (e.g., Addison's disease or inadequate aldosterone production), and medications (such as potassium-sparing diuretics,

the antimicrobial drug trimethoprim, and some NSAIDs) impair potassium excretion by the kidneys.

In clients with normal renal excretion of potassium, excess oral potassium (e.g., by supplement or use of salt substitutes) rarely leads to hyperkalemia. Rapid intravenous administration of potassium or transfusion of aged blood can lead to hyperkalemia.

A shift of potassium ions from the intracellular space can occur in acidosis, with severe tissue trauma, during chemotherapy, and due to starvation. In acidosis, excess hydrogen ions enter the cells, displacing potassium and causing it to shift into the extracellular space. The extent of this shift is greater with metabolic acidosis than with respiratory acidosis (see "Acid–Base Disorders" later in this chapter).

Hyperkalemia alters the cell membrane potential, affecting the heart, skeletal muscle function, and the gastrointestinal tract. The most harmful consequence of hyperkalemia is its effect on cardiac function. The cardiac conduction system is affected first, with slowing of the heart rate, possible heart blocks, and prolonged depolarization. ECG changes include peaked T waves, a prolonged PR interval, and widening of the QRS complex (see Figure 10–11). Ventricular dysrhythmias develop, and cardiac arrest may occur. Severe hyperkalemia decreases the strength of myocardial contractions.

Skeletal muscles become weak, and paralysis may occur with very high serum potassium levels. Hyperkalemia causes smooth muscle hyperactivity, leading to gastrointestinal disturbances.

The seriousness of hyperkalemia is based on the serum potassium ($K^+$) level and ECG changes.

- *Mild hyperkalemia:* serum $K^+$ between 5 and 6.5 mEq/L; ECG changes limited to peaked T wave
- *Moderate hyperkalemia:* serum $K^+$ between 6.5 and 8 mEq/L; ECG changes limited to peaked T wave
- *Severe hyperkalemia:* serum $K^+$ greater than 8 mEq/L; ECG shows absent P waves and widened QRS pattern.

### Manifestations

The manifestations of hyperkalemia result from its effects on the heart, skeletal, and smooth muscles. Early manifestations include diarrhea, colic (abdominal cramping), anxiety, paresthesias, irritability, and muscle tremors and twitching. As serum potassium levels increase, muscle weakness develops, progressing to flaccid paralysis. The lower extremities are affected first, progressing to the trunk and upper extremities. The heart rate may be slow (bradycardia) and irregular. The ECG shows T-wave changes and, at high serum potassium levels, widening of the QRS complex and absence of P waves.

## INTERDISCIPLINARY CARE

The management of hyperkalemia focuses on returning the serum potassium level to normal by treating the underlying cause and avoiding additional potassium intake. The choice of therapy for existing hyperkalemia is based on the severity of the hyperkalemia.

### Diagnosis

The following laboratory and diagnostic tests may be ordered:

- *Serum electrolytes* show a serum potassium level greater than 5.0 mEq/L. Low calcium and sodium levels may increase the effects of hyperkalemia; therefore, these electrolytes are usually measured as well.
- *ABGs* are measured to determine if acidosis is present.
- An *ECG* is obtained and *continuous ECG monitoring* is instituted to evaluate the effects of hyperkalemia on cardiac conduction and rhythm.

### Medications

Medications are administered to lower the serum potassium and to stabilize the conduction system of the heart. For moderate to severe hyperkalemia, calcium gluconate is given intravenously to counter the effects of hyperkalemia on the cardiac conduction system. While the effect of calcium gluconate lasts only for 1 hour, it allows time to initiate measures to lower serum potassium levels. To rapidly lower these levels, regular insulin and 50 g of glucose are administered. Insulin and glucose promote potassium uptake by the cells, shifting potassium out of ECF. In some cases, a $ß_2$-agonist such as albuterol may be given by nebulizer to temporarily push potassium into the cells. Sodium bicarbonate may be given to treat acidosis. As the pH returns toward normal, hydrogen ions are released from the cells and potassium returns into the cells.

To remove potassium from the body, sodium polystyrene sulfonate (Kayexalate), a resin that binds potassium in the gastrointestinal tract, may be administered orally or rectally. If renal function is normal, diuretics such as furosemide are given to promote potassium excretion. Commonly prescribed drugs, their actions, and nursing implications are listed in the Medication Administration box on the following page.

### Dialysis

When renal function is severely limited, either peritoneal dialysis or hemodialysis may be implemented to remove excess potassium. These measures are invasive and typically used only when other measures are ineffective. See Chapter 29 ∞ for more information about dialysis.

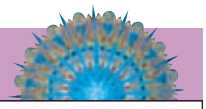
## NURSING CARE

Nursing care focuses related to hyperkalemia include identifying clients at risk, preventing hyperkalemia, and addressing problems resulting from the systemic effects of hyperkalemia. A Nursing Care Plan: A Client with Hyperkalemia is found on page 226.

## Health Promotion

Clients at the greatest risk for developing hyperkalemia include those taking potassium supplements (prescribed or over-the-counter), using potassium-sparing diuretics or salt substitutes, and experiencing renal failure. Athletes participating in competition sports such as body building and using anabolic steroids, muscle-building compounds, or "energy drinks" also may be at risk for hyperkalemia.

MEDICATION ADMINISTRATION **Hyperkalemia**



### DIURETICS

Potassium-wasting diuretics, such as furosemide (Lasix), may be used to enhance renal excretion of potassium.

#### Nursing Responsibilities

- Monitor serum electrolytes.
- Monitor and record weight at regular intervals under standard conditions (same time of day, balanced scale, same clothing).
- Monitor intake and output.

### INSULIN, HYPERTONIC DEXTROSE, AND SODIUM BICARBONATE

Insulin, hypertonic dextrose (10% to 50%), and sodium bicarbonate are used in the emergency treatment of moderate to severe hyperkalemia. Insulin promotes the movement of potassium into the cell, and glucose prevents hypoglycemia. The onset of action of insulin and hypertonic dextrose occurs within 30 minutes and is effective for approximately 4 to 6 hours.

Sodium bicarbonate elevates the serum pH; potassium is moved into the cell in exchange for hydrogen ion. Sodium bicarbonate is particularly useful in the client with metabolic acidosis. Onset of effects occurs within 15 to 30 minutes and is effective for approximately 2 hours.

#### Nursing Responsibilities

- Administer intravenous insulin and dextrose over prescribed interval of time using an infusion pump.
- Administer sodium bicarbonate as prescribed. It may be administered as an intravenous bolus or added to a dextrose-in-water solution and given by infusion.
- In clients receiving sodium bicarbonate, monitor for sodium overload, particularly in clients with hypernatremia, heart failure, and renal failure.
- Monitor the ECG pattern closely.
- Monitor serum electrolytes ($K^+$, $Na^+$, $Ca^{2+}$, $Mg^{2+}$) frequently during treatment.

### CALCIUM GLUCONATE AND CALCIUM CHLORIDE

Intravenous calcium gluconate or calcium chloride is used as a temporary emergency measure to counteract the toxic effects of potassium on myocardial conduction and function.

#### Nursing Responsibilities

- Closely monitor the ECG of the client receiving intravenous calcium, particularly for bradycardia.
- Calcium should be used cautiously in clients receiving digitalis, because calcium increases the cardiotonic effects of digitalis and may precipitate digitalis toxicity, leading to dysrhythmias.

### SODIUM POLYSTYRENE SULFONATE (KAYEXALATE) AND SORBITOL

Sodium polystyrene sulfonate (Kayexalate) is used to treat moderate or severe hyperkalemia. Categorized as a cation exchange resin, Kayexalate exchanges sodium or calcium for potassium in the large intestine. Sorbitol is given with Kayexalate to promote bowel elimination. Kayexalate and sorbitol may be administered orally, through a nasogastric tube, or rectally as a retention enema. The usual dosage is 20 g three or four times a day with 20 mL of 70% sorbitol solution.

#### Nursing Responsibilities

- Because Kayexalate contains sodium, monitor clients with heart failure and edema closely for water retention.
- Monitor serum electrolytes ($K^+$, $Na^+$, $Ca^{2+}$, $Mg^{2+}$) frequently during therapy.
- Restrict sodium intake in clients who are unable to tolerate increased sodium load (e.g., those with CHF or hypertension).

Teach all clients to carefully read food and dietary supplement labels. Discuss the importance of taking prescribed potassium supplements as ordered, and not increasing the dose unless prescribed by the care provider. Advise clients taking a potassium supplement or potassium-sparing diuretic to avoid salt substitutes, which usually contain potassium. Discuss the importance of maintaining an adequate fluid intake (unless a fluid restriction has been prescribed) to maintain renal function to eliminate potassium from the body.

## Assessment

Assessment data related to hyperkalemia include the following:

- *Health history:* Current manifestations, including numbness and tingling, nausea and vomiting, abdominal cramping, muscle weakness, palpitations; duration of symptoms and any precipitating factors such as use of salt substitutes, potassium supplements, or reduced urine output; chronic diseases such as renal failure or endocrine disorders; current medications.
- *Physical assessment:* Apical and peripheral pulses; bowel sounds; muscle strength in upper and lower extremities; ECG pattern.
- *Diagnostic tests:* Serum electrolytes, potassium, sodium, and calcium in particular; ABGs; digitalis levels; ECG.

## Nursing Diagnoses and Interventions

The effects of excess potassium on the electrical conduction and contractility of the heart are the highest priority for nursing care, particularly when the serum potassium level is 6.5 mEq/L or higher.

### Risk for Decreased Cardiac Output

Hyperkalemia affects depolarization of the atria and ventricles of the heart. Severe hyperkalemia can cause dysrhythmias with ventricular fibrillation and cardiac arrest. The cardiac effects of hyperkalemia are more pronounced when the serum potassium level rises rapidly. Low serum sodium and calcium levels, high serum magnesium levels, and acidosis contribute to the adverse effects of hyperkalemia on the heart muscle.

**PRACTICE ALERT**

Monitor the ECG pattern for development of peaked, narrow T waves, prolongation of the PR interval, depression of the ST segment, widened QRS interval, and loss of the P wave. Notify the physician of changes. Progressive ECG changes from a peaked T wave to loss of the P wave and widening of the QRS complex indicate an increasing risk of dysrhythmias and cardiac arrest.

## NURSING CARE PLAN A Client with Hyperkalemia

Montigue Longacre, a 51-year-old African-American male, has end-stage renal failure. He arrives at the emergency clinic complaining of shortness of breath on exertion and extreme weakness.

### ASSESSMENT

Mr. Longacre tells the nurse, Janet Allen, RN, that he normally receives dialysis three times a week. He missed his last treatment, however, to attend his father's funeral. During the last several days, he has eaten a number of fresh oranges he received as a gift. Physical assessment findings include T 99.2, P 100, R 28, BP 168/96, 2+ pretibial edema, and a 6-lb (3.6-kg) weight gain since his last hemodialysis treatment 4 days ago. Laboratory and diagnostic tests show the following abnormal results.

- $K^+$ 6.5 mEq/L (normal 3.5 to 5 mEq/L)
- BUN 118 mg/dL (normal 7 to 18 mg/dL)
- Creatinine 14 mg/dL (normal 0.7 to 1.3 mg/dL)
- $HCO_3^-$ 17 mEq/L (normal 22 to 26 mEq/L)
- Peaked T wave noted on ECG.

Mr. Longacre is placed on continuous ECG monitoring, and the physician prescribes hemodialysis. As an interim measure to lower the serum potassium, the physician prescribes $D_{50}W$ (25 g of dextrose), one ampule, to be administered intravenously with 10 units of regular insulin over 30 minutes.

### DIAGNOSES

- *Activity Intolerance* related to skeletal muscle weakness
- *Risk for Decreased Cardiac Output* related to hyperkalemia
- *Risk for Ineffective Health Maintenance* related to inadequate knowledge of recommended diet
- *Excess Fluid Volume* related to renal failure

### EXPECTED OUTCOMES

- Gradually resume usual physical activities.
- Maintain serum potassium level within normal range.
- Verbalize causes of hyperkalemia, the importance of hemodialysis treatments as scheduled, and the role of diet in preventing hyperkalemia.

### PLANNING AND IMPLEMENTATION

- Monitor intake and output.
- Monitor serum potassium and ECG closely during treatment.
- Teach causes of hyperkalemia and the relationship between hemodialysis and hyperkalemia.
- Discuss the importance of avoiding foods high in potassium to prevent or control hyperkalemia.

### EVALUATION

Following emergency treatment and hemodialysis, Mr. Longacre's ECG and serum potassium level have returned to normal. His muscle strength has returned to near normal, and he verbalizes an understanding of his prescribed hemodialysis regimen. Janet Allen provides verbal and written information about hyperkalemia, the importance of complying with the hemodialysis regimen, and the importance of limiting intake of dietary sources of potassium in renal failure. She also furnishes a list of foods high in potassium and cautions against using potassium-containing salt substitutes and nonprescription drugs.

### CRITICAL THINKING IN THE NURSING PROCESS

1. What information given by Mr. Longacre indicated that he might be experiencing hyperkalemia?
2. Why was continuous ECG monitoring instituted as an emergency measure?
3. What additional emergency measures might have been instituted if Mr. Longacre's serum potassium level had been 8.5 mEq/L and his ECG had showed changes in impulse conduction?
4. Develop a care plan for Mr. Longacre for the nursing diagnosis of *Anxiety.*

*See Evaluating Your Response in Appendix C.*

- Closely monitor the response to intravenous calcium gluconate, particularly in clients taking digitalis. *Calcium increases the risk of digitalis toxicity.*

## Risk for Activity Intolerance

Both hypokalemia (low serum potassium levels) and hyperkalemia (high serum potassium levels) affect neuromuscular activity and the function of cardiac, smooth, and skeletal muscles. Hyperkalemia can cause muscle weakness and even paralysis.

- Monitor skeletal muscle strength and tone. *Increasing weakness, muscle paralysis, or progression of affected muscles to affect the upper extremities or trunk can indicate increasing serum potassium levels.*
- Monitor respiratory rate and depth. Regularly assess lung sounds. *Muscle weakness due to hyperkalemia can impair ventilation. In addition, medications such as sodium bicarbonate or sodium polystyrene sulfonate can cause fluid retention and pulmonary edema in clients with preexisting cardiovascular disease.*
- Assist with self-care activities as needed. *Increasing muscle weakness can lead to fatigue and affect the ability to meet self-care needs.*

## Risk for Imbalanced Fluid Volume

Renal failure is a major cause of hyperkalemia. Clients with renal failure also are at risk for fluid retention and other electrolyte imbalances.

- Closely monitor serum potassium, BUN, and serum creatinine. Notify the physician if serum potassium level is greater than 5 mEq/L, or if serum creatinine and BUN levels are increasing. *Serum creatinine and BUN are the primary indicators of renal function. Levels of these substances rise rapidly in acute renal failure, more slowly in chronic renal failure* (see Chapter 29 ∞).
- Maintain accurate intake and output records. Report an imbalance of 24-hour totals and/or urine output less than 30 mL/hour. *Oliguria (scant urine) or anuria (no urine output) may indicate renal failure and an increased risk for hyperkalemia and fluid volume excess.*

- Monitor clients receiving sodium bicarbonate for fluid volume excess. *Increased sodium from injection of a hypertonic sodium bicarbonate solution can cause a shift of water into the extracellular space.*
- Monitor clients receiving cation exchange resins and sorbitol for fluid volume excess. *The resin exchanges potassium for sodium or calcium in the bowel. Excessive sodium and water retention may occur.*

## Community-Based Care

Preventing future episodes of hyperkalemia is the focus when preparing the client for home care. Include the family, a significant other, or a caregiver when teaching the following topics:

- Recommended diet and any restrictions including salt substitutes and foods high in potassium
- Medications to be avoided, including over-the-counter and fitness supplements
- Follow-up appointments for lab work and evaluation.

# CALCIUM IMBALANCE

Calcium is one of the most abundant ions in the body. The normal adult total serum calcium concentration is 8.5 to 10.0 mg/dL.

## Overview of Normal Calcium Balance

Calcium is obtained from dietary sources, although only about 20% of the calcium ingested is absorbed into the blood. The remainder is excreted in feces. Extracellular calcium is excreted by the kidneys. Approximately 99% of the total calcium in the body is bound to phosphorus to form the minerals in bones and teeth. The remaining 1% is in extracellular fluid. About half of this extracellular calcium is ionized (free); it is this ionized calcium that is physiologically active. The remaining extracellular calcium is bound to protein or other ions.

Ionized calcium is essential to a number of processes:

- Stabilizing cell membranes
- Regulating muscle contraction and relaxation
- Maintaining cardiac function
- Blood clotting.

Serum calcium levels are regulated by the interaction of three hormones: parathyroid hormone (PTH), calcitonin, and calcitriol (a metabolite of vitamin D). When serum calcium levels fall, the parathyroid glands secrete PTH, which mobilizes skeletal calcium stores, increases calcium absorption in the intestines, and promotes calcium reabsorption by the kidneys (Figure 10–12 ■). Calcitriol facilitates this process by stimulating calcium release from the bones, absorption in the intestines, and reabsorption by the kidneys. Calcitonin is secreted by the thyroid gland in response to high serum calcium levels. Its effect on serum calcium levels is the opposite of PTH: It inhibits the movement of calcium out of bone, reduces intestinal absorption of calcium, and promotes calcium excretion by the kidneys.

Serum calcium levels are also affected by acid–base balance. When hydrogen ion concentration falls and the pH rises (alkalosis), more calcium is bound to protein. While the total serum calcium remains unchanged, less calcium is available in the ionized, active form. Conversely, when hydrogen ion concentration increases and the pH falls (acidosis), calcium is released from protein, making more ionized calcium available.

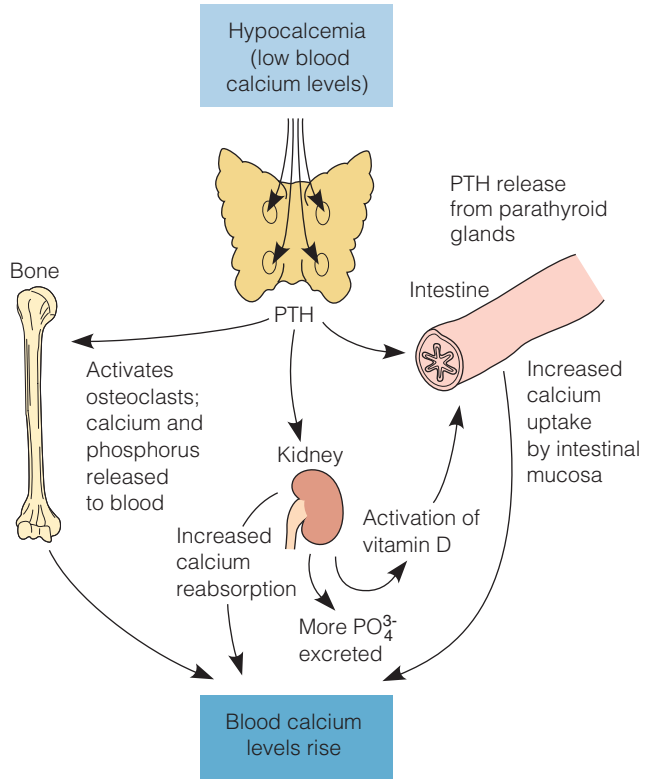



**Figure 10–12** ■ Low calcium levels (hypocalcemia) trigger the release of parathyroid hormone (PTH), increasing calcium ion levels through stimulation of bones, kidneys, and intestines.

Finally, the total amount of calcium in blood plasma fluctuates with plasma protein levels, particularly the albumin level. As the albumin level falls, the total amount of plasma calcium declines. Table 10–7 summarizes the causes and manifestations of calcium imbalances.

## The Client with Hypocalcemia

Hypocalcemia is a total serum calcium level of less than 8.5 mg/dL. Hypocalcemia can result from decreased total body calcium stores or low levels of extracellular calcium with normal amounts of calcium stored in bone. The systemic effects of hypocalcemia are caused by decreased levels of ionized calcium in extracellular fluid.

### Risk Factors

Certain populations of people are at greater risk for hypocalcemia: people who have had a parathyroidectomy (removal of the parathyroid glands), older adults (especially women), people with lactose intolerance, and those with alcoholism. Older adults often consume less milk and milk products (good sources of calcium) and may have less exposure to the sun (a source of vitamin D). Older adults also may be less active, promoting calcium loss from bones. They are more likely to be

**TABLE 10–7 Causes and Manifestations of Calcium Imbalances**

| IMBALANCE | CAUSES | MANIFESTATIONS |
|---|---|---|
| **Hypocalcemia**<br>Serum calcium < 8.5 mg/dL or 4.3 mEq/L<br>Critical value < 6.0 mg/dL | ■ Parathyroidectomy or neck surgery<br>■ Acute pancreatitis<br>■ Inadequate dietary intake<br>■ Lack of sun exposure<br>■ Lack of weight-bearing exercise<br>■ Drugs: loop diuretics, calcitonin<br>■ Hypomagnesemia, alcohol abuse<br>■ Acute renal failure with hyperphosphatemia | Neuromuscular<br>■ Tetany<br>  ■ Paresthesias<br>  ■ Muscle spasms<br>  ■ Positive Chvostek's sign<br>  ■ Positive Trousseau's sign<br>  ■ Laryngospasm<br>  ■ Seizures<br>■ Anxiety, confusion, psychoses<br>Cardiovascular<br>■ Decreased cardiac output<br>■ Hypotension<br>■ Dysrhythmias<br>Gastrointestinal<br>■ Abdominal cramping<br>■ Diarrhea |
| **Hypercalcemia**<br>Serum calcium >10 mg/dL or 5.3 mEq/L<br>Critical value >13.0 mg/dL | ■ Hyperparathyroidism<br>■ Some cancers<br>■ Prolonged immobilization<br>■ Paget's disease<br>■ Excess milk or antacid intake<br>■ Chronic renal failure with associated hyperparathyroidism | Neuromuscular<br>■ Muscle weakness, fatigue<br>■ Decreased deep tendon reflexes<br>Behavioral<br>■ Personality changes<br>■ Altered mental status<br>■ Decreasing level of consciousness<br>Gastrointestinal<br>■ Abdominal pain<br>■ Constipation<br>■ Anorexia, nausea, vomiting<br>Cardiovascular<br>■ Dysrhythmias<br>■ Hypertension<br>Renal<br>■ Polyuria, thirst |

taking drugs that interfere with calcium absorption or promote calcium excretion (e.g., furosemide). Older women are at particular risk after menopause because of reduced estrogen levels. Intolerance to lactose (found in milk and milk products) causes diarrhea and often limits the intake of milk and milk products, leading to possible calcium deficiency. Ethanol, or drinking alcohol, has a direct effect on calcium balance, reduces its intestinal absorption, and interferes with other processes involved in regulating serum calcium levels.

## Pathophysiology

Common causes of hypocalcemia are hypoparathyroidism (see Chapter 19 ∞) resulting from surgery (parathyroidectomy, thyroidectomy, radical neck dissection) and acute pancreatitis. In the client who has undergone surgery, symptoms of hypocalcemia usually occur within the first 24 to 48 hours, but may be delayed.

**PRACTICE ALERT**

Carefully monitor clients who have undergone neck surgery for manifestations of hypocalcemia. Check serum calcium levels, and report changes to the care provider.

Additional causes of hypocalcemia include other electrolyte imbalances (such as hypomagnesemia or hyperphosphatemia), alkalosis, malabsorption disorders that interfere with calcium absorption in the bowel, and inadequate vitamin D (due to lack of sun exposure or malabsorption). Hyperphosphatemia often occurs in acute renal failure, with reciprocal hypocalcemia. Massive transfusion of banked blood also can lead to hypocalcemia. Citrate is added to blood to prevent clotting and as a preservative. When blood is administered faster than the liver can metabolize the citrate, it can bind with calcium, temporarily removing ionized calcium from circulation. Many drugs increase the risk for hypocalcemia, including loop diuretics (such as furosemide), anticonvulsants (such as phenytoin and phenobarbital), phosphates (including phosphate enemas), and drugs that lower serum magnesium levels (such as cisplatin and gentamicin) (Metheny, 2000).

Extracellular calcium acts to stabilize neuromuscular cell membranes. This effect is reduced in hypocalcemia, increasing neuromuscular irritability. The threshold of excitation of sensory nerve fibers is lowered as well, leading to paresthesias (altered sensation). The nervous system becomes more excitable, and muscle spasms develop. In the heart, this change in cell

membranes can lead to dysrhythmias such as ventricular tachycardia and cardiac arrest. Hypocalcemia decreases the contractility of cardiac muscle fibers, leading to decreased cardiac output.

### Manifestations and Complications

The most serious manifestations of hypocalcemia are **tetany** (tonic muscular spasms) and convulsions. Numbness and tingling around the mouth (circumoral) and in the hands and feet develop. Muscle spasms of the face and extremities occur, and deep tendon reflexes become hyperactive. Chvostek's sign, contraction of the facial muscles produced by tapping the facial nerve in front of the ear (Figure 10–13A ■), and Trousseau's sign, carpal spasm induced by inflating a blood pressure cuff on the upper arm to above systolic blood pressure for 2 to 5 minutes (Figure 10–13B), indicate increased neuromuscular excitability in clients without obvious symptoms.

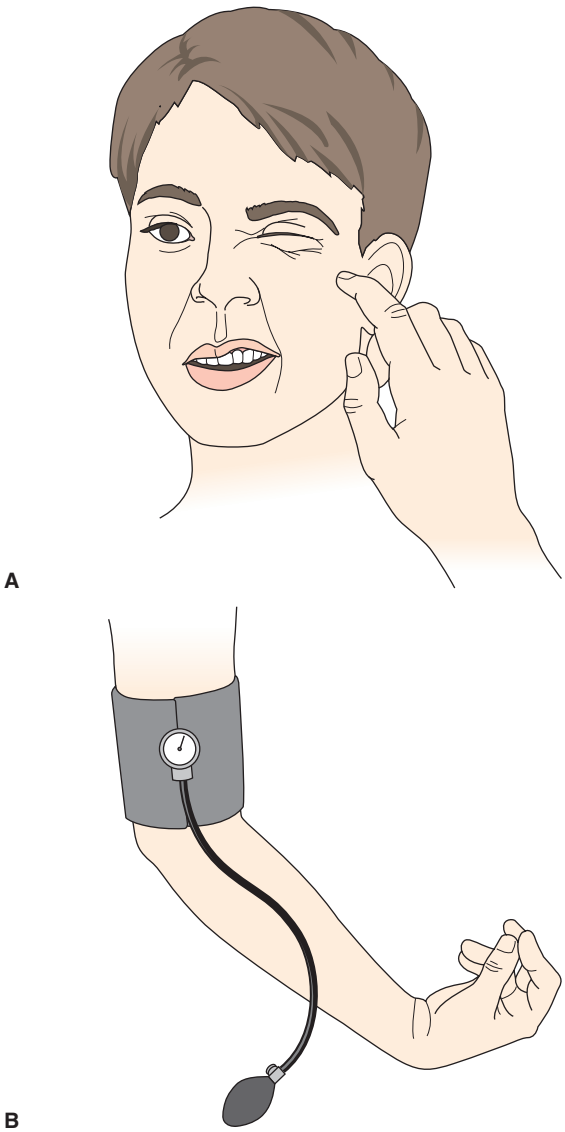


**Figure 10–13** ■ *A,* Positive Chvostek's sign. *B,* Positive Trousseau's sign.

Tetany can cause bronchial muscle spasms, simulating an asthma attack, and visceral muscle spasms, producing acute abdominal pain. Cardiovascular manifestations include hypotension, possible bradycardia (slow heart rate), and ventricular dysrhythmias.

Serious complications of hypocalcemia include airway obstruction and possible respiratory arrest from laryngospasm, ventricular dysrhythmias and cardiac arrest, heart failure, and convulsions.

## INTERDISCIPLINARY CARE

Management of hypocalcemia is directed toward restoring normal calcium balance and correcting the underlying cause.

### Diagnosis

The following laboratory and diagnostic tests may be ordered when hypocalcemia is known or suspected. Measurements include:

- *Total serum calcium*, the amount of ionized (active) calcium available, usually is estimated. In critically ill clients, however, *ionized calcium* may be directly measured using ion-selective electrodes. Direct measurement of ionized calcium requires special handling of the blood specimen, including placing the specimen on ice and analyzing it immediately.
- *Serum albumin,* because the albumin level affects serum calcium results. When the albumin level is low (hypoalbuminemia), the amount of ionized calcium may remain normal even though the total calcium level is low.
- *Serum magnesium,* because hypocalcemia is often associated with hypomagnesemia (serum magnesium < 1.6 mg/dL). In this case, normal magnesium levels must be restored to correct the hypocalcemia.
- *Serum phosphate;* hyperphosphatemia (serum phosphate > 4.5 mg/dL) can lead to hypocalcemia because of the inverse relationship between phosphorus and calcium (as phosphate levels rise, calcium levels fall).
- *Parathyroid hormone (PTH),* to identify the possible diagnoses of hyperparathyroidism.
- An *ECG,* to evaluate the effects of hypocalcemia on the heart, such as a prolonged ST segment.

### Medications

Hypocalcemia is treated with oral or intravenous calcium. The client with severe hypocalcemia is treated with intravenous calcium to prevent life-threatening problems such as airway obstruction. The most common intravenous calcium preparations include calcium chloride and calcium gluconate. Although calcium chloride contains more elemental calcium than calcium gluconate, it also is more irritating to the veins and may cause venous sclerosis (hardening of the vein walls) if given into a peripheral vein. Intravenous calcium preparations can cause necrosis and sloughing of tissue if they extravasate into subcutaneous tissue. Rapid drug administration can lead to bradycardia and possible cardiac arrest due to overcorrection of hypocalcemia with resulting hypercalcemia. See the Medication Administration box on the next page for further information about calcium administration.

MEDICATION ADMINISTRATION **Calcium Salts**

**CALCIUM SALTS**

**Calcium carbonate (BioCal, Calsam, Caltrate, OsCal, Tums, others)**
**Calcium chloride**
**Calcium citrate (Citrical)**
**Calcium glubionate**
**Calcium gluceptate**
**Calcium gluconate (Calcinate)**
**Calcium lactate**

Calcium salts are given to increase calcium levels when there is a deficit (a total body deficit or inadequate levels of extracellular calcium). Calcium is necessary to maintain bone structure and for multiple physiologic processes including neuromuscular and cardiac function as well as blood coagulation. In the presence of vitamin D, calcium is well absorbed from the gastrointestinal tract. Severe hypocalcemia is treated with intravenous calcium preparations.

**Nursing Responsibilities**

Oral calcium salts:
- Administer 1 to 1.5 hours after meals and at bedtime.
- Give calcium tablets with a full glass of water.

Intravenous calcium salts:
- Assess IV site for patency. Do not administer calcium if there is a risk of leakage into the tissues.
- May be given by slow IV push (dilute with sterile normal saline for injection prior to administering) or added to compatible parenteral fluids such as NS, lactated Ringer's solution, or $D_5W$.
- Administer into the largest available vein; use a central line if available.
- Do not administer with bicarbonate or phosphate.
- Continuously monitor ECG when administering IV calcium to clients taking digitalis due to increased risk of digitalis toxicity.
- Frequently monitor serum calcium levels and response to therapy.

**Health Education for the Client and Family**

- Take calcium tablets with a full glass of water 1 to 2 hours after meals. Do not take with food or milk. If possible, do not take within 1 to 2 hours of other medications.
- Maintain adequate vitamin D intake through diet or exposure to the sun to promote calcium absorption.
- Calcium carbonate can cause constipation. Eat a high-fiber diet and maintain a generous fluid intake to prevent constipation.

Oral calcium preparations (calcium carbonate, calcium gluconate, or calcium lactate) are used to treat chronic, asymptomatic hypocalcemia. Calcium supplements may be combined with vitamin D, or vitamin D may be given alone to increase gastrointestinal absorption of calcium.

### Nutrition

A diet high in calcium-rich foods may be recommended for clients with chronic hypocalcemia or with low total body stores of calcium. Box 10–7 lists foods that are high in calcium.

## NURSING CARE

### Health Promotion

Because of the large stores of calcium in bones, most healthy adults have a very low risk of developing hypocalcemia. A deficit of total body calcium is often associated with aging, however, increasing the risk of osteoporosis, fractures, and disability. Women have a higher risk for developing osteoporosis than men due to lower bone density and hormonal influences.

**BOX 10–7 Foods High in Calcium**

- Cottage cheese
- Cheese
- Milk
- Cream
- Yogurt
- Ice cream
- Molasses
- Canned sardines and salmon
- Rhubarb
- Broccoli
- Collard greens
- Soy flour
- Spinach
- Tofu

Teach women of all ages the importance of maintaining adequate calcium intake through diet and, as needed, calcium supplements. Stress the relationship between weight-bearing exercise and bone density, and encourage women to engage in a regular aerobic and weight-training exercise regime. Discuss hormone replacement therapy and its potential benefits during and after menopause. See Chapter 42 ∞ for more information about osteoporosis.

### Assessment

Assessment data related to hypocalcemia include the following:
- *Health history:* Current manifestations, including numbness and tingling around mouth and of hands and feet, abdominal pain, shortness of breath; acute or chronic diseases such as pancreatitis, liver or kidney disease; current medications.
- *Physical assessment:* Muscle spasms; deep tendon reflexes; Chvostek's sign and Trousseau's sign; respiratory rate and depth; vital signs and apical pulse; heart rate and rhythm; presence of convulsions.
- *Diagnostic tests:* Serum electrolytes (calcium, magnesium, phosphate, and potassium in particular), serum albumin, thyroid and parathyroid hormone levels; ECG.

### Nursing Diagnoses and Interventions

The effect of hypocalcemia on neuromuscular irritability, with the risk for muscle spasm and convulsions, is the highest priority for nursing care of the client.

#### Risk for Injury

The client with hypocalcemia is at risk for injury from possible laryngospasm, cardiac dysrhythmias, or convulsions. In addition, too rapid administration of intravenous calcium or ex-

travasation of the medication into subcutaneous tissues can lead to injury.

- Frequently monitor airway and respiratory status. Report changes such as respiratory **stridor** (a high-pitched, harsh inspiratory sound indicative of upper airway obstruction) or increased respiratory rate or effort to the physician. *These changes may indicate laryngeal spasm due to tetany.*

**PRACTICE ALERT**
Laryngeal spasm is a respiratory emergency, requiring immediate intervention to maintain ventilation and gas exchange.

- Monitor cardiovascular status including heart rate and rhythm, blood pressure, and peripheral pulses. *Hypocalcemia decreases myocardial contractility, causing reduced cardiac output and hypotension. It also can cause bradycardia or ventricular dysrhythmias. Cardiac arrest may occur in severe hypocalcemia.*
- Continuously monitor ECG in clients receiving intravenous calcium preparations, especially if the client also is taking digitalis. *Rapid administration of calcium salts can lead to hypercalcemia and cardiac dysrhythmias. Calcium administration increases the risk of digitalis toxicity and resultant dysrhythmias.*
- Provide a quiet environment. Institute seizure precautions such as raising the side rails and keeping an airway at bedside. *A quiet environment reduces central nervous system stimuli and the risk of convulsions in the client with tetany.*

## Community-Based Care

In preparing the client with hypocalcemia for discharge and home care, consider the circumstances leading to low serum calcium levels. Discuss risk factors for hypocalcemia specific to the client, and provide information about managing these risk factors to avoid future episodes of hypocalcemia. Teach about prescribed medications, including calcium supplements. Provide a list of foods high in calcium, as well as sources of vitamin D if recommended. Discuss symptoms to report to the care provider, and stress the importance of follow-up care as scheduled.

## The Client with Hypercalcemia

Hypercalcemia is a serum calcium value greater than 10.0 mg/dL. Excess ionized calcium in ECF can have serious widespread effects.

### Pathophysiology

Hypercalcemia usually results from increased resorption of calcium from the bones. The two most common causes of bone resorption are hyperparathyroidism and malignancies. In hyperparathyroidism, excess PTH is produced. This causes calcium to be released from bones, as well as increased calcium absorption in the intestines and retention of calcium by the kidneys. Hypercalcemia is a common complication of malignancies. It may develop as a result of bone destruction by the tumor or due to hormone-like substances produced by the tumor itself. Prolonged immobility and lack of weight bearing also cause increased resorption of bone with calcium release into extracellular fluids. Self-limiting hypercalcemia also may follow successful kidney transplant. Levels of parathyroid hormone may be altered in chronic renal failure, leading to increased serum calcium levels.

Increased intestinal absorption of calcium also can lead to hypercalcemia. This may result from excess vitamin D, overuse of calcium-containing antacids, or excessive milk ingestion. Renal failure and some drugs such as thiazide diuretics and lithium can interfere with elimination of calcium by the kidneys, causing high serum calcium levels.

The effects of hypercalcemia largely depend on the degree of serum calcium elevation and the length of time over which it develops. In general, higher serum calcium levels are associated with more serious effects. Calcium has a stabilizing effect on the neuromuscular junction; hypercalcemia decreases neuromuscular excitability, leading to muscle weakness and depressed deep tendon reflexes. Gastrointestinal motility is reduced as well. In the heart, calcium exerts an effect similar to digitalis (see Chapter 32 ∞), strengthening contractions and reducing the heart rate. Hypercalcemia affects the conduction system of the heart, leading to bradycardia and heart blocks. The ability of the kidneys to concentrate urine is impaired by hypercalcemia, causing excess sodium and water loss and increased thirst.

Extremely high serum calcium levels affect mental status. This is thought to be due to increased calcium in cerebrospinal fluid. Behavioral effects range from personality changes to confusion, impaired memory, and acute psychoses.

### Manifestations and Complications

Manifestations of hypercalcemia relate to its effects on neuromuscular activity, the central nervous system, the cardiovascular system, and the kidneys. Decreased neuromuscular excitability causes muscle weakness and fatigue, as well as gastrointestinal manifestations such as anorexia, nausea, vomiting, and constipation. Central nervous system (CNS) effects may include confusion, lethargy, behavior or personality changes, and coma. Cardiovascular effects include dysrhythmias, ECG changes, and possible hypertension. Hypercalcemia causes polyuria and, as a result, increased thirst.

Complications of hypercalcemia can affect several different organ systems. Peptic ulcer disease may develop due to increased gastric acid secretion. Pancreatitis can occur as a result of calcium deposits in pancreatic ducts. Excess calcium can precipitate out of urine to form kidney stones. Hypercalcemic crisis, an acute increase in the serum calcium level, can lead to cardiac arrest.

## INTERDISCIPLINARY CARE

The management of hypercalcemia focuses on correcting the underlying cause and reducing the serum calcium level. Treatment is particularly important in clients who have one or more of the following: serum calcium levels greater than 12 mg/dL, overt symptoms of hypercalcemia, compromised renal function, and inability to maintain an adequate fluid intake.

### Diagnosis

The laboratory and diagnostic tests that may be ordered and the resultant findings are as follows:

- *Serum electrolytes* show a total serum calcium greater than 10.0 mg/dL.
- *Serum PTH levels* are measured to identify or rule out hyperparathyroidism as the cause of hypercalcemia.
- *ECG* changes in hypercalcemia include a shortened QT interval, shortened and depressed ST segment, and widened T wave. Bradycardia or heart block may be identified on the ECG.
- *Bone density scans* may be done to monitor bone resorption and the effects of treatment measures on mineralization of bone.

### Medications

Measures to promote calcium elimination by the kidneys and reduce calcium resorption from bone are used to treat hypercalcemia. In acute hypercalcemia, intravenous fluids are given (see "Fluid Management" section that follows) with a loop diuretic such as furosemide to promote elimination of excess calcium. Calcitonin, which promotes the uptake of calcium into bones, also may be used to rapidly lower serum calcium levels.

A number of drugs that inhibit bone resorption are available. The bisphosphonates (pamidronate and etidronate) are commonly used to treat hypercalcemia associated with malignancies. These drugs also are used to prevent and treat osteoporosis. Nursing implications for calcitonin and bisphosphonate drugs are presented in the Medication Administration boxes in Chapter 42 ∞. When a bisphosphonate drug is ineffective to correct hypercalcemia, mithramycin, a chemotherapeutic agent, may be used.

Rapid reversal of hypercalcemia in emergency situations may be accomplished by intravenous administration of sodium phosphate or potassium phosphate. Calcium binds to phosphate, thus decreasing serum calcium levels. Paradoxically, complications of this therapy can include fatal hypocalcemia resulting from binding of the ionized calcium and soft tissue calcifications.

Other drug therapies include the use of intravenous plicamycin (Mithracin) to inhibit bone resorption. Glucocorticoids (cortisone), which compete with vitamin D, and a low-calcium diet may be prescribed to decrease gastrointestinal absorption of calcium, inhibit bone resorption, and to increase urinary calcium excretion. Also, calcitonin may be prescribed to decrease skeletal mobilization of calcium and phosphorus and to increase renal output of calcium and phosphorus. See Chapter 19 ∞ for more information about and nursing implications of glucocorticoid therapy.

### Fluid Management

Intravenous fluids, usually isotonic saline, are administered to clients with severe hypercalcemia to restore vascular volume and promote renal excretion of calcium. Isotonic saline is used because sodium excretion is accompanied by calcium excretion. Careful assessment of cardiovascular and renal function is done prior to fluid therapy; the client is carefully monitored for evidence of fluid overload during treatment.

## NURSING CARE

### Health Promotion

Identify and monitor clients at risk for hypercalcemia. Promote mobility in clients when possible. Assist hospitalized clients to ambulate as soon as possible. In the home setting, discuss the benefits of regular weight-bearing activity with clients, families, and caregivers. Encourage a generous fluid intake of up to 3 to 4 quarts per day. Encourage clients at risk to limit their intake of milk and milk products, as well as calcium-containing antacids and supplements. In addition, clients with prolonged immobility or hypercalcemia are encouraged to consume fluids that increase the acidity of urine (which inhibits calcium stone formation), such as cranberry or prune juice.

### Assessment

Assessment data related to hypercalcemia include the following:

- *Health history:* Current manifestations, including weakness or fatigue, abdominal discomfort, nausea or vomiting, increased urination and thirst; changes in memory or thinking; duration of symptoms and any risk factors such as excess intake of milk or calcium products, prolonged immobility, malignancy, renal failure, or endocrine disorders; current medications.
- *Physical assessment:* Mental status and level of consciousness; vital signs including apical pulse; bowel sounds; muscle strength of upper and lower extremities; deep tendon reflexes.
- *Diagnostic tests:* Serum electrolytes, urinary calcium, ECG, and cardiac rhythm monitoring.

**PRACTICE ALERT**

Remember, calcium has a stabilizing or sedative effect on neuromuscular transmission. Therefore:

Hypocalcemia → Increased neuromuscular excitability, muscle twitching, spasms, and possible tetany

Hypercalcemia → Decreased neuromuscular excitability, muscle weakness, and fatigue

### Nursing Diagnoses and Interventions

#### Risk for Injury

Clients with hypercalcemia are at risk for injury due to changes in mental status, the effects of hypercalcemia on muscle strength, and loss of calcium from bones.

- Institute safety precautions if confusion or other changes in mental status are noted. *Changes in mental status may impair judgment and the client's ability to maintain own safety.*

**PRACTICE ALERT**

Monitor cardiac rate and rhythm, treating and/or reporting dysrhythmias as indicated. Prepare for possible cardiac arrest; keep emergency resuscitation equipment readily available. Hypercalcemia can cause bradycardia, various heart blocks, and cardiac arrest. Immediate treatment may be necessary to preserve life.

- Observe for manifestations of digitalis toxicity, including vision changes, anorexia, and changes in heart rate and rhythm. Monitor serum digitalis levels. *Hypercalcemia increases the risk of digitalis toxicity.*
- Promote fluid intake (oral and/or intravenous) to keep the client well hydrated and maintain dilute urine. Encourage fluids such as prune or cranberry juice to help maintain acidic urine. *Acidic, dilute urine reduces the risk of calcium salts precipitating out to form kidney stones.*
- If excess bone resorption has occurred, use caution when turning, positioning, transferring, or ambulating. *Bones that have lost excess calcium may fracture with minimal stress or trauma (pathologic fractures).*

### Risk for Excess Fluid Volume

Large amounts of isotonic intravenous fluid often are administered to help correct acute hypercalcemia, leading to a risk for hypervolemia. Clients with preexisting cardiac or renal disease are at particular risk.

- Closely monitor intake and output. *A loop diuretic such as furosemide may be necessary if urinary output does not keep up with fluid administration.*
- Frequently assess vital signs, respiratory status, and heart sounds. *Increasing pulse rate, dyspnea, adventitious lung sounds, and an $S_3$ on auscultation of the heart may indicate excess fluid volume and potential heart failure.*
- Place in semi-Fowler's to Fowler's position. *Elevating the head of the bed improves lung expansion and reduces the work of breathing.*
- Administer diuretics as ordered, monitoring response. *Loop diuretics may be ordered to help eliminate excess fluid and calcium.*

## Community-Based Care

Discuss the following topics when preparing the client for discharge:

- Avoid excess intake of calcium-rich foods and antacids.
- Use prescribed drugs to prevent excess calcium resorption. Discuss their dose, use, and desired and possible adverse effects.
- Increase fluid intake to 3 to 4 quarts per day; increase the intake of acid ash foods (meats, fish, poultry, eggs, cranberries, plums, prunes); increase dietary fiber and fluid intake to prevent constipation.
- Maintain weight-bearing physical activity to prevent hypercalcemia.
- Report early manifestations of hypercalcemia to care provider.
- Follow recommended schedule for monitoring serum electrolyte levels.

# MAGNESIUM IMBALANCE

Only about 1% of the magnesium in the body is in extracellular fluid; the rest is found within the cells and in bone. The normal serum concentration of magnesium ranges from 1.6 to 2.6 mg/dL (1.3 to 2.1 mEq/L).

## Overview of Normal Magnesium Balance

Magnesium is obtained through the diet (it is plentiful in green vegetables, grains, nuts, meats, and seafood) and excreted by the kidneys. Magnesium is vital to many intracellular processes, including enzyme reactions and synthesis of proteins and nucleic acids. Magnesium exerts a sedative effect on the neuromuscular junction, decreasing acetylcholine release. It is an essential ion for neuromuscular transmission and cardiovascular function. The physiologic effects of magnesium are affected by both potassium and calcium levels. Approximately 65% of extracellular magnesium is ionized; the remainder is bound to protein. Table 10–8 summarizes common causes and manifestations of magnesium imbalances.

## The Client with Hypomagnesemia

Hypomagnesemia is a magnesium level of less than 1.6 mg/dL. It is a common problem, particularly in critically ill clients. Hypomagnesemia may be caused by deficient magnesium intake, excessive losses, or a shift between the intracellular and extracellular compartments.

### Risk Factors

Loss of gastrointestinal fluids, particularly from diarrhea, an ileostomy, or intestinal fistula is a major risk factor for hypomagnesemia. Disruption of nutrient absorption in the small intestine also increases the risk. Chronic alcoholism is the most common cause of deficient magnesium levels in the United States (Metheny, 2000). Multiple factors associated with alcoholism contribute to hypomagnesemia: deficient nutrient intake, increased gastrointestinal losses, impaired absorption, and increased renal excretion. Other risk factors for hypomagnesemia include:

- Protein-calorie malnutrition or starvation
- Endocrine disorders including diabetic ketoacidosis
- Drugs such as loop or thiazide diuretics, aminoglycoside antibiotics, amphotericin B, and cyclosporine
- Rapid administration of citrated blood (banked blood)
- Kidney disease.

### Pathophysiology

Magnesium deficiency usually occurs along with low serum potassium and calcium levels. The effects of hypomagnesemia relate not only to the magnesium deficiency but also to hypokalemia and hypocalcemia.

Hypomagnesemia causes increased neuromuscular excitability, with muscle weakness and tremors. The accompanying hypocalcemia contributes to this effect. In the central nervous system, this increased neural excitability can lead to seizures and changes in mental status.

Deficient intracellular magnesium in the myocardium increases the risk of cardiac dysrhythmias and sudden death. Hypokalemia increases this risk. Hypomagnesemia also increases the risk of digitalis toxicity. Chronic hypomagnesemia may contribute to hypertension, probably due to increased vasoconstriction.

**TABLE 10–8 Causes and Manifestations of Magnesium Imbalances**

| IMBALANCE | CAUSES | MANIFESTATIONS |
|---|---|---|
| **Hypomagnesemia**<br>Serum magnesium < 1.6 mg/dL<br>Critical value < 1 mg/dL | ■ Chronic alcoholism<br>■ GI losses: intestinal suction, diarrhea, ileostomy<br>■ Impaired absorption<br>■ Inadequate replacement<br>■ Increased excretion: drugs, renal disease, osmotic diuresis | Neuromuscular<br>■ Muscle weakness, tremors<br>■ Tetany, seizures<br>Gastrointestinal<br>■ Dysphagia<br>■ Anorexia, nausea, vomiting, diarrhea<br>Cardiovascular<br>■ Tachycardia<br>■ Dysrhythmias<br>■ Hypertension<br>CNS<br>■ Mood and personality changes<br>■ Paresthesias |
| **Hypermagnesemia**<br>Serum magnesium > 2.6 mg/dL or 2.1 mEq/L<br>Critical value > 4.7 mg/dL | ■ Renal insufficiency or failure<br>■ Excess intake of antacids, laxatives<br>■ Excess magnesium administration | Neuromuscular<br>■ Muscle weakness<br>■ Depressed deep tendon reflexes<br>Gastrointestinal<br>■ Nausea and vomiting<br>Cardiovascular<br>■ Hypotension<br>■ Bradycardia<br>■ Cardiac arrest<br>CNS<br>■ Respiratory depression<br>■ Coma |

### Manifestations and Complications

Neuromuscular manifestations of hypomagnesemia include tremors, hyperreactive reflexes, positive Chvostek's and Trousseau's signs (see Figure 10–13), tetany, paresthesias, and seizures. CNS effects include confusion, mood changes (apathy, depression, agitation), hallucinations, and possible psychoses.

An increased heart rate and ventricular dysrhythmias are common, especially when hypokalemia is present or the client is taking digitalis. Cardiac arrest and sudden death may occur. Gastrointestinal manifestations include nausea, vomiting, anorexia, diarrhea, and abdominal distention.

**BOX 10–8 Foods High in Magnesium**

- Green, leafy vegetables
- Seafood
- Meat
- Wheat bran
- Milk
- Legumes
- Bananas
- Oranges
- Grapefruit
- Chocolate
- Molasses
- Coconut
- Refined sugar

## INTERDISCIPLINARY CARE

Hypomagnesemia is diagnosed by measuring serum electrolyte levels. The ECG shows a prolonged PR interval, widened QRS complex, and depression of the ST segment with T-wave inversion.

Treatment is directed toward prevention and identification of an existing deficiency. Magnesium is added to intravenous total parenteral nutrition solutions to prevent hypomagnesemia.

In clients able to eat, a mild deficiency may be corrected by increasing the intake of foods rich in magnesium (Box 10–8), or with oral magnesium supplements. Oral magnesium supplements may cause diarrhea, however, limiting their use.

Clients with manifestations of hypomagnesemia are treated with parenteral magnesium sulfate. Treatment is continued for several days to restore intracellular magnesium levels. Magnesium may be given intravenously or by deep intramuscular injection. Renal function is evaluated prior to administration, and serum magnesium levels are monitored during treatment. The intravenous route is used for severe magnesium deficiency or if neurologic changes or cardiac dysrhythmias are present. See the Medication Administration box on the next page for the nursing implications of parenteral magnesium sulfate.

## NURSING CARE

### Health Promotion

Discuss the importance of maintaining adequate magnesium intake through a well-balanced diet, particularly with clients at risk (people with alcoholism, malabsorption, or bowel surgery). Many hospitalized clients are at risk for hypomagnesemia due to protein-calorie malnutrition and other disorders. Monitor serum magnesium levels, reporting changes to the healthcare provider.

MEDICATION ADMINISTRATION **Magnesium Sulfate**

Magnesium sulfate is used to prevent or treat hypomagnesemia. It also is used as an anticonvulsant in severe eclampsia or preeclampsia. It may be given intravenously or by intramuscular injection.

**Nursing Responsibilities**

- Assess serum magnesium levels and renal function tests (BUN and serum creatinine) prior to administering. Notify the care provider if magnesium levels are above normal limits or renal function is impaired.
- Frequently monitor neurologic status and deep tendon reflexes during therapy. Withhold magnesium and notify the care provider if deep tendon reflexes are hypoactive or absent.
- Monitor intake and output.
- Administer IM doses deep into the ventral or dorsal gluteal sites.
- Intravenous magnesium sulfate may be given by direct IV push or by continuous infusion.

**Health Education for the Client and Family**

Explain purpose and duration of treatment. Discuss reason for frequent neurologic and reflex assessments.

## Assessment

In addition to asking questions related to risk factors for hypomagnesemia, use the guidelines for assessing clients with hypokalemia and hypocalcemia for subjective and objective assessment data. Monitor diagnostic studies such as serum electrolytes, serum albumin levels, and the ECG.

## Nursing Diagnoses and Interventions

Nursing care for clients with hypomagnesemia focuses on careful monitoring of manifestations and responses to treatment, promoting safety, client and family teaching, and administering prescribed medications.

### Risk for Injury

- Monitor serum electrolytes, including magnesium, potassium, and calcium. *Magnesium deficiency often is accompanied by deficiencies of potassium and calcium.*
- Monitor gastrointestinal function, including bowel sounds and abdominal distention. *Hypomagnesemia reduces gastrointestinal motility.*
- Initiate cardiac monitoring, reporting and treating (as indicated) ECG changes and dysrhythmias. In clients receiving digitalis, monitor for digitalis toxicity. *Low magnesium levels can precipitate ventricular dysrhythmias, including lethal dysrhythmias such as ventricular fibrillation.*
- Assess deep tendon reflexes frequently during intravenous magnesium infusions and prior to each intramuscular dose. *Depressed tendon reflexes indicate a high serum magnesium level.*
- Maintain a quiet, darkened environment. Institute seizure precautions. *Increased neuromuscular and CNS irritability can lead to seizures. A quiet, dark environment reduces stimuli.*

## Community-Based Care

Prior to discharge, instruct the client to increase dietary intake of foods high in magnesium and provide information about magnesium supplements. In addition, if alcohol abuse has precipitated a magnesium deficit, discuss alcohol treatment options, including inpatient treatment and support groups such as Alcoholics Anonymous, Al-Anon, and/or Al-a-Teen.

## The Client with Hypermagnesemia

Hypermagnesemia is a serum magnesium level greater than 2.6 mg/dL. It is much less common than hypomagnesemia. Hypermagnesemia can develop in renal failure, particularly if magnesium is administered parenterally or orally (e.g., magnesium-containing antacids or laxatives). Older adults are at risk for hypermagnesemia as renal function declines with aging and they are more likely to use over-the-counter laxatives and other preparations that contain magnesium.

### Pathophysiology and Manifestations

Elevated serum magnesium levels interfere with neuromuscular transmission and depress the central nervous system. Hypermagnesemia also affects the cardiovascular system, potentially causing hypotension, flushing, sweating, and bradydysrhythmias.

Predictable manifestations occur with increasing serum magnesium levels. With lower levels, nausea and vomiting, hypotension, facial flushing, sweating, and a feeling of warmth occur. As levels increase, signs of CNS depression appear (weakness, lethargy, drowsiness, weak or absent deep tendon reflexes). Marked elevations cause respiratory depression, coma, and compromised cardiac function (ECG changes, bradycardia, heart block, and cardiac arrest).

## INTERDISCIPLINARY CARE

The management of hypermagnesemia focuses on identifying and treating the underlying cause. All medications or compounds containing magnesium (such as antacids, intravenous solutions, or enemas) are withheld. In the client with renal failure, hemodialysis or peritoneal dialysis is instituted to remove the excess magnesium.

Calcium gluconate is administered intravenously to reverse the neuromuscular and cardiac effects of hypermagnesemia. The client may require mechanical ventilation to support respiratory function, and a pacemaker to maintain adequate cardiac output.

## NURSING CARE

Nursing care includes instituting measures to prevent and identify hypermagnesemia in clients at risk, monitoring for

critical effects of hypermagnesemia, and providing measures to ensure the client's safety. Consider the following nursing diagnoses for the client with hypermagnesemia:

- *Decreased Cardiac Output* related to altered myocardial conduction
- *Risk for Ineffective Breathing Pattern* related to respiratory depression
- *Risk for Injury* related to muscle weakness and altered level of consciousness
- *Risk for Ineffective Health Maintenance* related to lack of knowledge about use of magnesium-containing supplements, antacids, laxatives, and enemas.

## Community-Based Care

Discharge teaching and planning focus on instructions to avoid magnesium-containing medications, including antacids, mineral supplements, cathartics, and enemas (Box 10–9).

**BOX 10–9 Medications Containing Magnesium**

**Antacids**
- Gelusil
- Maalox No.1
- Maalox Plus
- Riopan
- Milk of Magnesia
- Mylanta
- Di-Gel
- Gaviscon

**Laxatives**
- Milk of Magnesia
- Magnesium oxide
- Haley's M-O
- Magnesium citrate
- Epsom salts

# PHOSPHATE IMBALANCE

Although most phosphate (85%) is found in bones, it is the primary intracellular anion. About 14% is in intracellular fluid, and the remainder (1%) is in extracellular fluid. The normal serum phosphate (or phosphorus) level in adults is 2.5 to 4.5 mg/dL. Phosphorus levels vary with age, gender, and diet.

## Overview of Normal Phosphate Balance

Phosphate is essential to intracellular processes such as the production of ATP, the fuel that supports muscle contraction, nerve cell transmission, and electrolyte transport. Phosphate is vital for red blood cell function and oxygen delivery to tissues; nervous system and muscle function; and the metabolism of fats, carbohydrates, and protein. It also assists in maintaining acid–base balance.

Phosphorus is ingested in the diet, absorbed in the jejunum, and primarily excreted by the kidneys. When phosphate intake is low, the kidneys conserve phosphorus, excreting less. An inverse relationship exists between phosphate and calcium levels: When one increases, the other decreases. Regulatory mechanisms for calcium levels (parathyroid hormone, calcitonin, and vitamin D) also influence phosphate levels. The causes and manifestations of phosphate imbalances are summarized in Table 10–9.

## The Client with Hypophosphatemia

Hypophosphatemia is a serum phosphorus of less than 2.5 mg/dL. Low serum phosphate levels may indicate a total body deficit of phosphate or a shift of phosphate into the intracellular space, the most common cause of hypophosphatemia. Decreased gastrointestinal absorption of phosphate or increased renal excretion of phosphate also can cause low phosphate levels. Hypophosphatemia often is *iatrogenic,* that is, related to treatment. Selected causes of hypophosphatemia include the following:

- *Refeeding syndrome* can develop when malnourished clients are started on enteral or total parenteral nutrition. Glucose in the formula or solution stimulates insulin release, which promotes the entry of glucose and phosphate into the cells, depleting extracellular phosphate levels.
- Medications frequently contribute to hypophosphatemia, including intravenous glucose solutions, antacids (aluminum- or magnesium-based antacids bind with phosphate), anabolic steroids, and diuretics.

**TABLE 10–9 Causes and Manifestations of Phosphate Imbalances**

| IMBALANCE | CAUSES | MANIFESTATIONS |
|---|---|---|
| **Hypophosphatemia**<br>Serum phosphorus < 2.5 mg/dL<br>Critical value < 1 mg/dL | ■ Shift of phosphorus into cells<br>■ IV glucose administration<br>■ Total parenteral nutrition without phosphorus<br>■ Aluminum- or magnesium-based antacids<br>■ Diuretic therapy<br>■ Alcoholism | ■ Paresthesias<br>■ Muscle weakness<br>■ Muscle pain and tenderness<br>■ Confusion, decreasing LOC<br>■ Seizures<br>■ Bone pain, osteomalacia<br>■ Anorexia, dysphagia<br>■ Decreased bowel sounds<br>■ Possible acute respiratory failure |
| **Hyperphosphatemia**<br>Serum phosphate > 4.5 mg/dL<br>Critical value > 90 mg/dL | ■ Renal failure<br>■ Chemotherapy<br>■ Muscle tissue trauma<br>■ Sepsis<br>■ Severe hypothermia<br>■ Heat stroke | ■ Circumoral and peripheral paresthesias<br>■ Muscle spasms<br>■ Tetany<br>■ Soft tissue calcification |

- Alcoholism affects both the intake and absorption of phosphate.
- Hyperventilation and respiratory alkalosis cause phosphate to shift out of extracellular fluids into the intracellular space.
- Other causes include diabetic ketoacidosis with excess phosphate loss in the urine, stress responses, and extensive burns.

### Pathophysiology and Manifestations

Most effects of hypophosphatemia result from depletion of ATP and impaired oxygen delivery to the cells due to a deficiency of the red blood cell enzyme 2,3-DPG. Severe hypophosphatemia affects virtually every major organ system:

- *Central nervous system:* Reduced oxygen and ATP synthesis in the brain causes neurologic manifestations such as irritability, apprehension, weakness, paresthesias, lack of coordination, confusion, seizures, and coma.
- *Hematologic:* Oxygen delivery to the cells is reduced. Hemolytic anemia (excessive RBC destruction) may develop due to lack of ATP in red blood cells.
- *Musculoskeletal:* Decreased ATP causes muscle weakness and release of creatinine phosphokinase (CPK, a muscle enzyme); acute rhabdomyolysis (muscle cell breakdown) can develop. Muscle cell destruction, in turn, can lead to acute renal failure as myoglobin, a muscle cell protein, exerts a toxic effect on the kidney tubule.
- *Respiratory:* Chest muscle weakness can interfere with effective ventilation, leading to respiratory failure.
- *Cardiovascular:* Hypophosphatemia decreases myocardial contractility; decreased oxygenation of the heart muscle can cause chest pain and dysrhythmias.
- *Gastrointestinal:* Anorexia can occur, as well as dysphagia (difficulty swallowing), nausea and vomiting, decreased bowel sounds, and possible ileus due to reduced gastrointestinal motility.

## INTERDISCIPLINARY CARE

Treatment for hypophosphatemia is directed at prevention, treating the underlying cause of the disorder, and replacing phosphate. An improved diet and oral phosphate supplement (such as Neutra-Phos or Neutra-Phos K capsules) may restore normal phosphate levels in clients with a mild to moderate deficiency. Intravenous phosphate (sodium phosphate or potassium phosphate) is given when serum phosphate levels are less than 1 mg/dL. Oral phosphate supplements are then continued for up to 1 week to restore intracellular phosphate levels (Metheny, 2000).

## NURSING CARE

Nurses can be instrumental in identifying clients at risk for phosphate deficiency and preventing it from developing. Nurses should closely monitor serum electrolyte values in clients at risk, including those who are malnourished, receiving intravenous glucose solutions or total parenteral nutrition, or being treated with diuretic therapy or antacids that bind with phosphate. Nursing diagnoses that may be appropriate for the client with hypophosphatemia include:

- *Impaired Physical Mobility* related to muscle weakness and poor coordination
- *Ineffective Breathing Pattern* related to weakened muscles of respiration
- *Decreased Cardiac Output* related to reduced myocardial contractility
- *Risk for Injury* related to muscle weakness and altered mental status.

## Community-Based Care

In preparing for discharge, teach the client and family about the causes and manifestations of hypophosphatemia. Discuss the importance of avoiding phosphorus-binding antacids, unless prescribed. Stress a well-balanced diet to maintain an adequate intake of phosphate.

# The Client with Hyperphosphatemia

Hyperphosphatemia is a serum phosphate level greater than 4.5 mg/dL. As with other electrolyte imbalances, it may be the result of impaired phosphate excretion, excess intake, or a shift of phosphate from the intracellular space into extracellular fluids.

- Acute or chronic renal failure is the primary cause of impaired phosphate excretion.
- Rapid administration of phosphate-containing solutions can increase serum phosphate levels. This can include phosphate enemas. In addition, excess vitamin D increases phosphate absorption and can lead to hyperphosphatemia in clients with impaired renal function.
- A shift of phosphate from the intracellular to extracellular space can occur during chemotherapy, due to sepsis or hypothermia, or because of extensive trauma or heat stroke.
- Because phosphate levels are affected by serum calcium concentrations, disruption of the mechanisms that regulate calcium levels (e.g., hypoparathyroidism, hyperthyroidism, or vitamin D intoxication) can lead to hyperphosphatemia.

### Pathophysiology and Manifestations

Excessive serum phosphate levels cause few specific symptoms. The effects of high serum phosphate levels on nerves and muscles (muscle cramps and pain, paresthesias, tingling around the mouth, muscle spasms, tetany) are more the result of hypocalcemia that develops secondary to an elevated serum phosphorus level. The phosphate in the serum combines with ionized calcium, and the ionized serum calcium level falls.

Calcification of soft tissues can occur with high phosphate levels. Phosphates bind with calcium to precipitate in soft tissues such as the kidneys and other organs. Soft tissue calcification can impair the function of affected organs.

## INTERDISCIPLINARY CARE

Treatment of the underlying disorder often corrects hyperphosphatemia. When this is not feasible, phosphate-containing drugs are eliminated and intake of phosphate-rich foods such as organ meats and milk and milk products is restricted. Agents that bind with phosphate in the gastrointestinal tract (such as calcium-containing antacids) may be prescribed. If renal

function is adequate, intravenous normal saline may be given to promote renal excretion of phosphate. Dialysis may be necessary to reduce phosphate levels in clients with renal failure.

## NURSING CARE

When providing nursing care for the client with hyperphosphatemia, monitor the client for laboratory data revealing an excess of phosphorus and a deficit of calcium, as well as the signs of hypocalcemia.

## Community-Based Care

Discuss the risk of hyperphosphatemia related to using phosphate preparations as laxatives or enemas, particularly with clients who have other risk factors for the disorder. When preparing the client for discharge, teach about the use of phosphate-binding preparations as ordered and dietary phosphate restrictions.

# ACID–BASE DISORDERS

MEDIALINK Acid–Base Balance Animation

Homeostasis and optimal cellular function require maintenance of the hydrogen ion ($H^+$) concentration of body fluids within a relatively narrow range. Hydrogen ions determine the relative acidity of body fluids. **Acids** release hydrogen ions in solution; **bases** (or **alkalis**) accept hydrogen ions in solution. The hydrogen ion concentration of a solution is measured as its pH. The relationship between hydrogen ion concentration and pH is inverse; that is, as hydrogen ion concentration increases, the pH falls, and the solution becomes more acidic. As hydrogen ion concentration falls, the pH rises, and the solution becomes more alkaline or basic. The pH of body fluids is slightly basic, with the normal pH ranging from 7.35 to 7.45 (a pH of 7 is neutral).

## REGULATION OF ACID–BASE BALANCE

A number of mechanisms work together to maintain the pH of the body within this normal range. Metabolic processes in the body continuously produce acids, which fall into two categories: volatile acids and nonvolatile acids. **Volatile acids** can be eliminated from the body as a gas. Carbonic acid ($H_2CO_3$) is the only volatile acid produced in the body. It dissociates (separates) into carbon dioxide ($CO_2$) and water ($H_2O$); the carbon dioxide is then eliminated from the body through the lungs. All other acids produced in the body are *nonvolatile acids* that must be metabolized or excreted from the body in fluid. Lactic acid, hydrochloric acid, phosphoric acid, and sulfuric acid are examples of nonvolatile acids. Most acids and bases in the body are weak; that is, they neither release nor accept a significant amount of hydrogen ion.

Three systems work together in the body to maintain the pH despite continuous acid production: buffers, the respiratory system, and the renal system.

### Buffer Systems

**Buffers** are substances that prevent major changes in pH by removing or releasing hydrogen ions. When excess acid is present in body fluid, buffers bind with hydrogen ions to minimize the change in pH. If body fluids become too basic or alkaline, buffers release hydrogen ions, restoring the pH. Although buffers act within a fraction of a second, their capacity to maintain pH is limited. The major buffer systems of the body are the bicarbonate-carbonic acid buffer system, phosphate buffer system, and protein buffers.

The bicarbonate-carbonic acid buffer system can be illustrated by the following equation:

$$CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^-$$

Bicarbonate ($HCO_3^-$) is a weak base; when an acid is added to the system, the hydrogen ion in the acid combines with bicarbonate, and the pH changes only slightly. Carbonic acid ($H_2CO_3$) is a weak acid produced when carbon dioxide dissolves in water. If a base is added to the system, it combines with carbonic acid, and the pH remains within the normal range. Although the amounts of bicarbonate and carbonic acid in the body vary to a certain extent, as long as a ratio of 20 parts bicarbonate ($HCO_3^-$) to 1 part carbonic acid ($H_2CO_3$) is maintained, the pH remains within the 7.35 to 7.45 range (Figure 10–14 ■).

The normal serum bicarbonate level is 24 mEq/L, and that of carbonic acid is 1.2 mEq/L. Thus, the ratio of bicarbonate to carbonic acid is 20:1. It is this ratio that maintains the pH within the normal range. Adding a strong acid to extracellular fluid depletes bicarbonate, changing the 20:1 ratio and causing the pH to drop below 7.35. This is known as **acidosis**. Addition of a strong base depletes carbonic acid as it combines with the base. The 20:1 ratio again is disrupted and the pH rises above 7.45, a condition known as **alkalosis**.

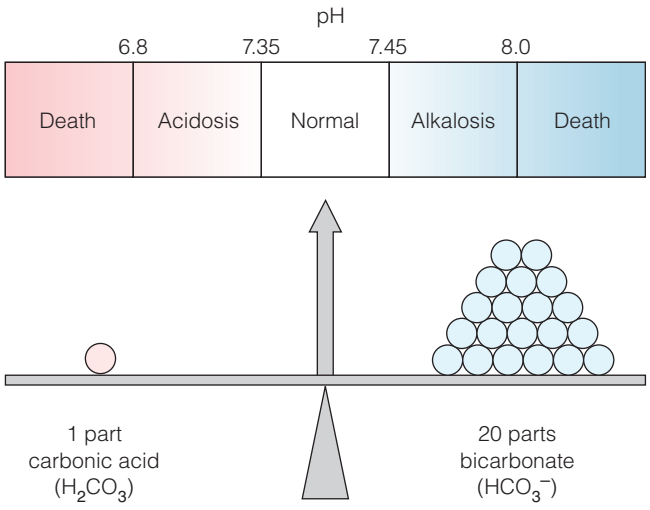



**Figure 10–14** ■ The normal ratio of bicarbonate to carbonic acid is 20:1. As long as this ratio is maintained, the pH remains within the normal range of 7.35 to 7.45.

Intracellular and plasma proteins also serve as buffers. Plasma proteins contribute to buffering of extracellular fluids. Proteins in intracellular fluid provide extensive buffering for organic acids produced by cellular metabolism. In red blood cells, hemoglobin acts as a buffer for hydrogen ion when carbonic acid dissociates. Inorganic phosphates also serve as extracellular buffers, although their roles are not as important as the bicarbonate-carbonic acid buffer system. Phosphates are, however, important intracellular buffers, helping to maintain a stable pH within the cells.

## Respiratory System

The respiratory system (and the respiratory center of the brain) regulates carbonic acid in the body by eliminating or retaining carbon dioxide. Carbon dioxide is a potential acid; when combined with water, it forms carbonic acid (see previous equation), a volatile acid. Acute increases in either carbon dioxide or hydrogen ions in the blood stimulate the respiratory center in the brain. As a result, both the rate and depth of respiration increase. The increased rate and depth of lung ventilation eliminate carbon dioxide from the body, and carbonic acid levels fall, bringing the pH to a more normal range. Although this compensation for increased hydrogen ion concentration occurs within minutes, it becomes less effective over time. Clients with chronic lung disease may have consistently high carbon dioxide levels in their blood.

Alkalosis, by contrast, depresses the respiratory center. Both the rate and depth of respiration decrease, and carbon dioxide is retained. The retained carbon dioxide then combines with water to restore carbonic acid levels and bring the pH back within the normal range.

## Renal System

The renal system is responsible for the long-term regulation of acid–base balance in the body. Excess nonvolatile acids produced during metabolism normally are eliminated by the kidneys. The kidneys also regulate bicarbonate levels in extracellular fluid by regenerating bicarbonate ions as well as reabsorbing them in the renal tubules. Although the kidneys respond more slowly to changes in pH (over hours to days), they can generate bicarbonate and selectively excrete or retain hydrogen ions as needed. In acidosis, when excess hydrogen ion is present and the pH falls, the kidneys excrete hydrogen ions and retain bicarbonate. In alkalosis, the kidneys retain hydrogen ions and excrete bicarbonate to restore acid–base balance.

## Assessment of Acid–Base Balance

Acid–base balance is evaluated primarily by measuring arterial blood gases.

**PRACTICE ALERT**

Arteries are high-pressure vessels in contrast to veins. Obtaining an arterial blood sample requires specialized training. It may be done by a registered nurse, respiratory therapist, or laboratory technician who has been trained in drawing ABGs. Apply firm pressure to the puncture site for 2 to 5 minutes after the needle is withdrawn to prevent bleeding into the surrounding tissues.

Arterial blood is used because it reflects acid–base balance throughout the entire body better than venous blood. Arterial blood also provides information about the effectiveness of the lungs in oxygenating blood. The elements measured are pH, the $Paco_2$, the $Pao_2$, and bicarbonate level.

**PRACTICE ALERT**

You will see the abbreviations $Paco_2$ and $Pao_2$ used interchangeably with $Pco_2$ and $Po_2$. The *P* stands for partial pressure: the pressure exerted by the gas dissolved in the blood. The *a* indicates that the sample is arterial blood. Because these measurements rarely are done on venous blood, the *a* often is deleted from the abbreviation.

The **$Paco_2$** measures the pressure exerted by dissolved carbon dioxide in the blood. The $Paco_2$ reflects the respiratory component of acid–base regulation and balance. The $Paco_2$ is regulated by the lungs. The normal value is 35 to 45 mmHg. A $Paco_2$ of less than 35 mmHg is known as *hypocapnia;* a $Paco_2$ greater than 45 mmHg is *hypercapnia.*

The **$Pao_2$** is a measure of the pressure exerted by oxygen that is dissolved in the plasma. Only about 3% of oxygen in the blood is transported in solution; most is combined with hemoglobin. However, it is the dissolved oxygen that is available to the cells for metabolism. As dissolved oxygen diffuses out of plasma into the tissues, more is released from hemoglobin. The normal value for $Pao_2$ is 80 to 100 mmHg. A $Pao_2$ of less than 80 mmHg is indicative of *hypoxemia.* The $Pao_2$ is valuable for evaluating respiratory function, but is not used as a primary measurement in determining acid–base status.

The **serum bicarbonate** ($HCO_3^-$) reflects the renal regulation of acid–base balance. It is often called the metabolic component of arterial blood gases. The normal $HCO_3^-$ value is 22 to 26 mEq/L.

The **base excess (BE)** is a calculated value also known as *buffer base capacity*. The base excess measures substances that can accept or combine with hydrogen ion. It reflects the degree of acid–base imbalance by indicating the status of the body's total buffering capacity. It represents the amount of acid or base that must be added to a blood sample to achieve a pH of 7.4. This is essentially a measure of increased or decreased bicarbonate. The normal value for base excess for arterial blood is −3.0 to +3.0. Normal ABG values are summarized in Table 10–10.

ABGs are analyzed to identify acid–base disorders and their probable cause, to determine the extent of the imbalance, and to monitor treatment. When analyzing ABG results, it is important to use a systematic approach. First evaluate each individual measurement, then look at the interrelationships to determine the client's acid–base status (Box 10–10).

# ACID–BASE IMBALANCE

Acid–base imbalances fall into two major categories: acidosis and alkalosis. Acidosis occurs when the hydrogen ion concentration increases above normal (pH below 7.35). Alkalosis

**TABLE 10–10 Normal Arterial Blood Gas Values**

| VALUE | NORMAL RANGE | SIGNIFICANCE |
|---|---|---|
| pH | 7.35 to 7.45 | Reflects hydrogen ion ($H^+$) concentration<br>■ < 7.35 = acidosis<br>■ > 7.45 = alkalosis |
| $Paco_2$ | 35 to 45 mmHg | Partial pressure of carbon dioxide ($CO_2$) in arterial blood<br>■ < 35 mmHg = hypocapnia<br>■ > 45 mmHg = hypercapnia |
| $Pao_2$ | 80 to 100 mmHg | Partial pressure of oxygen ($O_2$) in arterial blood<br>■ < 80 mmHg = hypoxemia |
| $HCO_3^-$ | 22 to 26 mEq/L | Bicarbonate concentration in plasma |
| BE | −3 to +3 | Base excess; a measure of buffering capacity |

occurs when the hydrogen ion concentration falls below normal (pH above 7.45).

Acid–base imbalances are further classified as *metabolic* or *respiratory* disorders. In metabolic disorders, the primary change is in the concentration of bicarbonate. In metabolic acidosis, the amount of bicarbonate is decreased in relation to the amount of acid in the body (Figure 10–15A ■). It can develop as a result of abnormal bicarbonate losses or because of excess nonvolatile acids in the body. The pH falls below 7.35 and the bicarbonate concentration is less than 22 mEq/L. Metabolic alkalosis, by contrast, occurs when there is an excess of bicarbonate in relation to the amount of hydrogen ion (Figure 10–15B). The pH is above 7.45 and the bicarbonate concentration is greater than 26 mEq/L.

In respiratory disorders, the primary change is in the concentration of carbonic acid. *Respiratory acidosis* occurs when carbon dioxide is retained, increasing the amount of carbonic acid in the body (Figure 10–16A ■). As a result, the pH falls to less than 7.35, and the $Paco_2$ is greater than 45 mmHg. When too much carbon dioxide is "blown off," carbonic acid levels fall and *respiratory alkalosis* develops (Figure 10–16B). The pH rises to above 7.45 and the $Paco_2$ is less than 35 mmHg.

Acid–base disorders are further defined as *primary* (simple) and *mixed.* Primary disorders usually are due to one cause. For example, respiratory failure often causes respiratory acidosis due to retained carbon dioxide; renal failure usually causes metabolic acidosis due to retained hydrogen ion and impaired

**BOX 10–10 Interpreting Arterial Blood Gases**

1. Look at the pH.
   - pH < 7.35 = acidosis
   - pH > 7.45 = alkalosis
2. Look at the $Paco_2$.
   - $Paco_2$ < 35 mmHg = hypocapnia; more carbon dioxide is being exhaled than normal
   - $Paco_2$ > 45 mmHg = hypercapnia; carbon dioxide is being retained
3. Evaluate the pH–$Paco_2$ relationship for a possible respiratory problem.
   - If the pH is < 7.35 (acidosis) and the $Paco_2$ is > 45 mmHg (hypercapnia), retained carbon dioxide is causing increased $H^+$ concentration and *respiratory acidosis*.
   - If the pH is > 7.45 (alkalosis) and the $Paco_2$ is < 35 mmHg (hypocapnia), low carbon dioxide levels and decreased $H^+$ concentration are causing *respiratory alkalosis*.
4. Look at the bicarbonate.
   - If the $HCO_3^-$ is < 22 mEq/L, bicarbonate levels are lower than normal.
   - If the $HCO_3^-$ is > 26 mEq/L, bicarbonate levels are higher than normal.
5. Evaluate the pH, $HCO_3^-$, and BE for a possible metabolic problem.
   - If the pH is < 7.35 (acidosis), the $HCO_3^-$ is < 22 mEq/L, and the BE is < −3 mEq/L, then low bicarbonate levels and high $H^+$ concentrations are causing *metabolic acidosis*.
   - If the pH is > 7.45 (alkalosis), the $HCO_3^-$ is > 26 mEq/L, and the BE is > +3 mEq/L, then high bicarbonate levels are causing *metabolic alkalosis*.
6. Look for compensation.
   - *Renal compensation:*
     - In respiratory acidosis (pH < 7.35, $Paco_2$ > 45 mmHg), the kidneys retain $HCO_3^-$ to buffer the excess acid, so the $HCO_3^-$ is > 26 mEq/L.
     - In respiratory alkalosis (pH > 7.45, $Paco_2$ < 35 mmHg), the kidneys excrete $HO_3^-$ to minimize the alkalosis, so the $HCO_3^-$ is < 22 mEq/L.
   - *Respiratory compensation*
     - In metabolic acidosis (pH < 7.35, $HCO_3^-$ < 22 mEq/L), the rate and depth of respirations increase, increasing carbon dioxide elimination, so the $PaCo_2$ is < 35 mmHg.
     - In metabolic alkalosis (pH > 7.45, $HCO_3^-$ > 26 mEq/L), respirations slow, carbon dioxide is retained, so the $Paco_2$ is > 45 mmHg.
7. Evaluate oxygenation.
   - $Pao_2$ < 80 mmHg = hypoxemia; possible hypoventilation
   - $Pao_2$ > 100 mmHg = hyperventilation

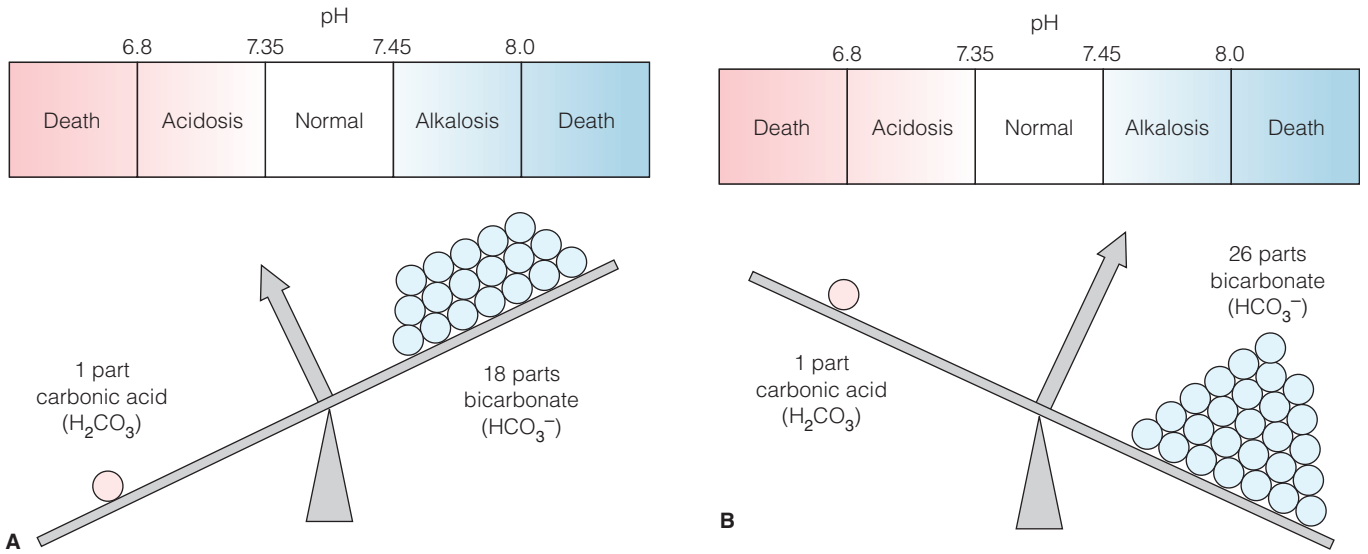



**Figure 10–15** ■ Metabolic acid–base imbalances. *A,* Metabolic acidosis. *B,* Metabolic alkalosis.

bicarbonate production. Table 10–11 summarizes primary acid–base imbalances with common causes of each. Mixed disorders occur from combinations of respiratory and metabolic disturbances. For example, a client in cardiac arrest develops a mixed respiratory and metabolic acidosis due to lack of ventilation (and retained $CO_2$) and hypoxia of body tissues that leads to anaerobic metabolism and acid by-products (excess nonvolatile acids).

## FAST FACTS

- Simple acid–base imbalances are more commonly seen than mixed imbalances. Common causes of simple acid–base imbalances include:
  - Diabetic ketoacidosis (metabolic acidosis)
  - Chronic obstructive lung disease (respiratory acidosis)
  - Anxiety-related (psychogenic) hyperventilation (respiratory alkalosis)
- Critically ill clients are at higher risk for mixed acid–base imbalances.

## Compensation

With primary acid–base disorders, compensatory changes in the other part of the regulatory system occur to restore a normal pH and homeostasis. In metabolic acid–base disorders, the change in pH affects the rate and depth of respirations. This, in turn, affects carbon dioxide elimination and the $Paco_2$, helping restore the carbonic acid to bicarbonate ratio. The kidneys compensate for simple respiratory imbalances. The change in pH affects both bicarbonate conservation and hydrogen ion elimination (Table 10–12).

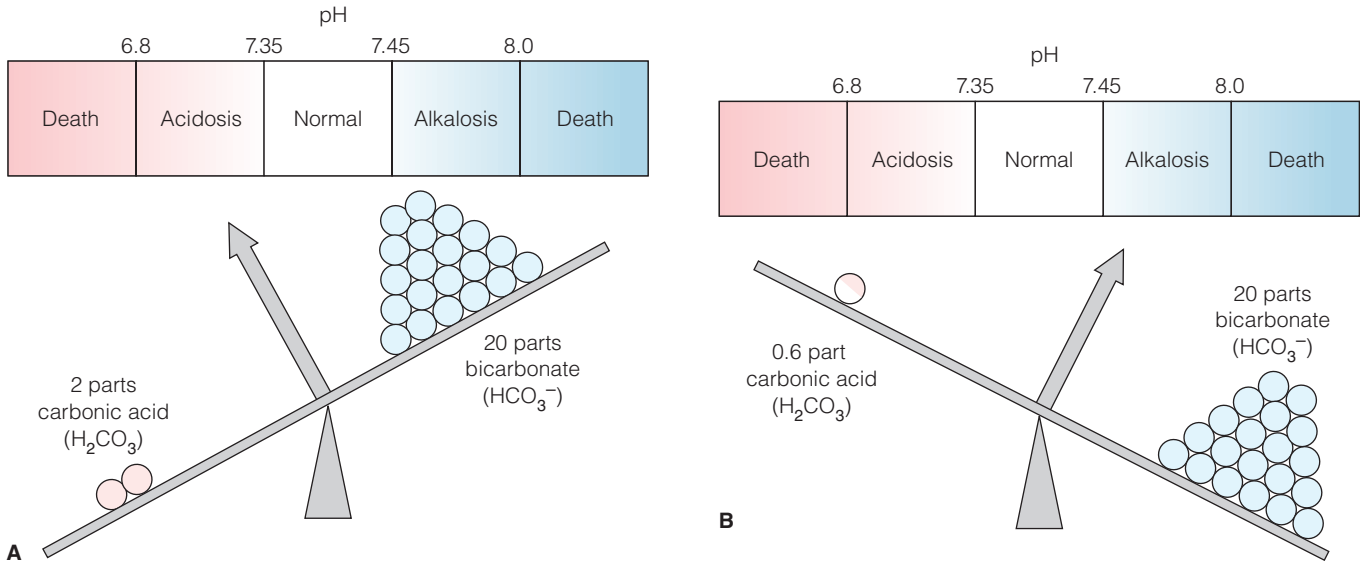



**Figure 10–16** ■ Respiratory acid–base imbalances. *A,* Respiratory acidosis. *B,* Respiratory alkalosis.

**TABLE 10–11 Common Causes of Primary Acid–Base Imbalances**

| IMBALANCE | COMMON CAUSES |
|---|---|
| Metabolic acidosis<br>pH < 7.35<br>$HCO_3^-$ < 22 mEq/L<br>Critical values<br>pH < 7.20<br>$HCO_3^-$ < 10 mEq/L | ↑ Acid production<br>■ Lactic acidosis<br>■ Ketoacidosis related to diabetes, starvation, or alcoholism<br>■ Salicylate toxicity<br>↓ Acid excretion<br>■ Renal failure<br>↑ Bicarbonate loss<br>■ Diarrhea, ileostomy drainage, intestinal fistula<br>■ Biliary or pancreatic fistulas<br>↑ Chloride<br>■ Sodium chloride IV solutions<br>■ Renal tubular acidosis<br>■ Carbonic anhydrase inhibitors |
| Metabolic alkalosis<br>pH > 7.45<br>$HCO_3^-$ > 26 mEq/L<br>Critical values<br>pH > 7.60<br>$HCO_3^-$ > 40 mEq/L | ↑ Acid loss or excretion<br>■ Vomiting, gastric suction<br>■ Hypokalemia<br>↑ Bicarbonate<br>■ Alkali ingestion (bicarbonate of soda)<br>■ Excess bicarbonate administration |
| Respiratory acidosis<br>pH < 7.35<br>$Paco_2$ >45 mm Hg<br>Critical values<br>pH < 7.2<br>$Paco_2$ > 77 mmHg | Acute respiratory acidosis<br>■ Acute respiratory conditions (pulmonary edema, pneumonia, acute asthma)<br>■ Opiate overdose<br>■ Foreign body aspiration<br>■ Chest trauma<br>Chronic respiratory acidosis<br>■ Chronic respiratory conditions (COPD, cystic fibrosis)<br>■ Multiple sclerosis, other neuromuscular diseases<br>■ Stroke |
| Respiratory alkalosis<br>pH > 7.45<br>$Paco_2$ < 35 mm Hg<br>Critical values<br>pH > 7.60<br>$Paco_2$ < 20 mmHg | ■ Anxiety-induced hyperventilation (e.g., anxiety)<br>■ Fever<br>■ Early salicylate intoxication<br>■ Hyperventilation with mechanical ventilator |

Compensatory changes in respirations occur within minutes of a change in pH. These changes, however, become less effective over time. The renal response takes longer to restore the pH, but is a more effective long-term mechanism. If the pH is restored to within normal limits, the disorder is said to be *fully compensated.* When these changes are reflected in ABG values but the pH remains outside normal limits, the disorder is said to be *partially compensated.*

## The Client with Metabolic Acidosis

Metabolic acidosis (bicarbonate deficit) is characterized by a low pH (<7.35) and a low bicarbonate (<22 mEq/L). It may be caused by excess acid in the body or loss of bicarbonate from the body. When metabolic acidosis develops, the respiratory system attempts to return the pH to normal by increasing the rate and depth of respirations. Carbon dioxide elimination increases, and the $Paco_2$ falls (<35 mmHg).

### Risk Factors

Metabolic acidosis rarely is a primary disorder; it usually develops during the course of another disease:

- *Acute lactic acidosis* usually results from tissue hypoxia due to shock or cardiac arrest.
- Clients with type 1 diabetes mellitus are at risk for developing *diabetic ketoacidosis*. (See Chapter 20 ∞ for more information about diabetes and its complications.)
- *Acute* or *chronic renal failure* impairs the excretion of metabolic acids.
- Diarrhea, intestinal suction, or abdominal fistulas increase the risk for excess *bicarbonate loss.*

Other common causes of metabolic acidosis are listed in Table 10–11.

### Pathophysiology

Three basic mechanisms that can cause metabolic acidosis are:

- Accumulation of metabolic acids
- Excess loss of bicarbonate
- An increase in chloride levels.

An accumulation of metabolic acids can result from excess acid production or impaired elimination of metabolic acids by the kidney. Lactic acidosis develops due to tissue hypoxia and a shift to anaerobic metabolism by the cells. Lactate and hy-

**TABLE 10–12 Compensation for Simple Acid–Base Imbalances**

| PRIMARY DISORDER | CAUSE | COMPENSATION | EFFECT ON ABGS |
|---|---|---|---|
| Metabolic acidosis | Excess nonvolatile acids; bicarbonate deficiency | Rate and depth of respirations increase, eliminating additional $CO_2$ | ↓pH<br>↓$HCO_3^-$<br>↓ $Paco_2$ |
| Metabolic alkalosis | Bicarbonate excess | Rate and depth of respirations decrease, retaining $CO_2$ | ↑ pH<br>↑ $HCO_3^-$<br>↑ $Paco_2$ |
| Respiratory acidosis | Retained $CO_2$ and excess carbonic acid | Kidneys conserve bicarbonate to restore carbonic acid:bicarbonate ratio of 1:20 | ↓ pH<br>↑ $Paco_2$<br>↑ $HCO_3^-$ |
| Respiratory alkalosis | Loss of $CO_2$ and deficient carbonic acid | Kidneys excrete bicarbonate and conserve $H^+$ to restore carbonic acid:bicarbonate ratio | ↑ pH<br>↓ $Paco_2$<br>↓ $HCO_3^-$ |

drogen ions are produced, forming lactic acid. Both oxygen and glucose are necessary for normal cell metabolism. When intracellular glucose is inadequate due to starvation or a lack of insulin to move it into cells, the body breaks down fatty tissue to meet its metabolic needs. In this process, fatty acids are released, which are converted to ketones; ketoacidosis develops. Aspirin (acetylsalicylic acid) breaks down into salicylic acid in the body. Substances such as aspirin, methanol (wood alcohol), and ethylene (contained in antifreeze and solvents) cause a toxic increase in body acids by either breaking down into acid products (salicylic acid) or stimulating metabolic acid production (Porth, 2005). Renal failure impairs the body's ability to excrete excess hydrogen ions and form bicarbonate.

Excess metabolic acids increase the hydrogen ion concentration of body fluids. The excess acid is buffered by bicarbonate, leading to what is known as a high **anion gap** acidosis (Box 10–11).

The pancreas secretes bicarbonate-rich fluid into the small intestine. Intestinal suction, severe diarrhea, ileostomy drainage, or fistulas can lead to excess losses of bicarbonate. Hyperchloremic acidosis can develop when excess chloride solutions (such as NaCl or ammonium chloride) are infused, causing a rise in chloride concentrations. It also may be related to renal disease or administration of carbonic anhydrate inhibitor diuretics. The anion gap remains normal in metabolic acidosis due to bicarbonate loss or excess chloride.

Acidosis depresses cell membrane excitability, affecting neuromuscular function. It also increases the amount of free calcium in ECF by interfering with protein binding. Severe acidosis (pH of 7.0 or less) depresses myocardial contractility, leading to a fall in cardiac output. If kidney function is normal, acid excretion and ammonia production increase to eliminate excess hydrogen ions.

Acid–base imbalances also affect electrolyte balance. In acidosis, potassium is retained as the kidney excretes excess

**BOX 10–11 Unraveling the Anion Gap**

Calculation of the anion gap can help identify the underlying mechanism in metabolic acidosis if it is unclear.

The number of cations (positively charged ions) and anions (negatively charged ions) in ECF normally is equal (refer to Figure 10–2). Not all of these ions, however, are measured in laboratory testing (e.g., organic acids and proteins). The anion gap is calculated by subtracting the sum of two measured anions, chloride and bicarbonate, from the concentration of the major cation, sodium (see figure). The normal anion gap is 8 to 12 mEq/L.

Excess acids in ECF are buffered by bicarbonate, reducing serum bicarbonate levels and the total measured concentration of anions. This increases the anion gap (B in figure). When bicarbonate is lost from the body or chloride levels increase, however, the anion gap remains within normal limits (C in figure). This occurs because an increase or decrease in one of these negatively charged ions causes a corresponding change in the other to maintain balance (e.g., ↓ $HCO_3^-$ ↔ ↑ $Cl^-$), and there is no change in the amount of unmeasured anions.

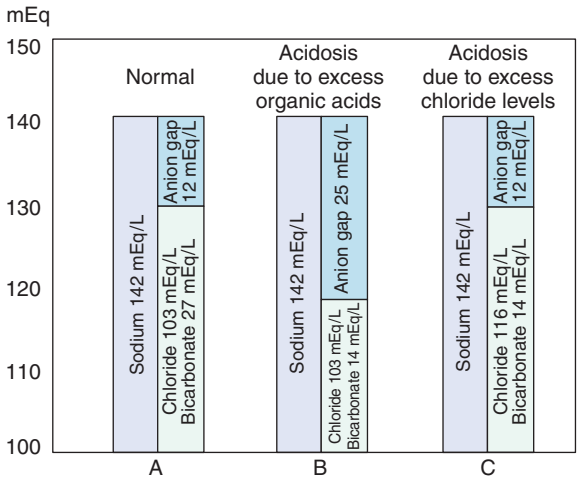



Illustration of the anion gap in metabolic acidosis. *A*, Normal anion gap. *B*, High anion gap caused by excess acids. *C*, Normal anion gap with hyperchloremia.

hydrogen ion. Excess hydrogen ions also enter the cells, displacing potassium from the intracellular space to maintain the balance of cations and anions within the cells. The effect of both processes is to increase serum potassium levels. Also in acidosis, calcium is released from its bonds with plasma proteins, increasing the amount of ionized (free) calcium in the blood. Magnesium levels may fall in acidosis.

### Manifestations

Metabolic acidosis affects the function of many body systems. Its general manifestations include weakness and fatigue, headache, and general malaise. Gastrointestinal function is affected, causing anorexia, nausea, vomiting, and abdominal pain. The level of consciousness declines, leading to stupor and coma. Cardiac dysrhythmias develop, and cardiac arrest may occur. The skin is often warm and flushed. Skeletal problems may develop in chronic acidosis, as calcium and phosphate are released from the bones. Manifestations of compensatory mechanisms are seen. The respirations are deep and rapid, known as **Kussmaul's respirations**. The client may complain of shortness of breath or dyspnea. See the Manifestations box below.

## INTERDISCIPLINARY CARE

Management of metabolic acidosis focuses on treating the underlying cause of the disorder and correcting the acid–base imbalance.

### Diagnosis

The following laboratory and diagnostic tests may be ordered.

- *ABGs* generally show a pH of less than 7.35 and a bicarbonate level of less than 22 mEq/L. A compensatory decrease in $Paco_2$ to less than 35 mmHg is usually present.
- *Serum electrolytes* demonstrate elevated serum potassium levels and possible low magnesium levels. The total calcium may remain unchanged, although more physiologically active ionized calcium is available. Sodium, chloride, and bicarbonate levels are used to calculate the anion gap.
- The *ECG* may show changes that reflect both the acidosis (particularly when severe) and the accompanying hyperkalemia.
- Other diagnostic studies such as the blood glucose and renal function studies may be ordered to identify the underlying cause of metabolic acidosis.

**MANIFESTATIONS of Metabolic Acidosis**

- Anorexia
- Nausea and vomiting
- Abdominal pain
- Weakness
- Fatigue
- General malaise
- Decreasing levels of consciousness
- Dysrhythmias
- Bradycardia
- Warm, flushed skin
- Hyperventilation (Kussmaul's respirations)

### Medications

An alkalinizing solution such as bicarbonate may be given if the pH is less than 7.2 to reduce the effects of the acidosis on cardiac function. Sodium bicarbonate is the most commonly used alkalinizing solution; others include lactate, citrate, and acetate solutions (which are metabolized to bicarbonate). Alkalinizing solutions are given intravenously for severe acute metabolic acidosis. In chronic metabolic acidosis, the oral route is used.

The client treated with bicarbonate must be carefully monitored. Rapid correction of the acidosis may lead to metabolic alkalosis and hypokalemia. Hypernatremia and hyperosmolality may develop as well, leading to water retention and fluid overload.

**PRACTICE ALERT**

As metabolic acidosis is corrected, potassium shifts back into the intracellular space. This can lead to hypokalemia and cardiac dysrhythmias. Carefully monitor serum potassium levels during treatment.

Treatment for diabetic ketoacidosis includes intravenous insulin and fluid replacement (see Chapter 20 ∞ for the treatment of diabetic ketoacidosis). Alcoholic ketoacidosis is treated with saline solutions and glucose. Treatment for lactic acidosis from decreased tissue perfusion (e.g., shock or cardiac arrest) focuses on correcting the underlying problem and improving tissue perfusion. Clients with chronic renal failure and mild or moderate metabolic acidosis may or may not require treatment, depending on the pH and bicarbonate levels. When metabolic acidosis is due to diarrhea, treatment includes correcting the underlying cause and providing fluid and electrolyte replacement.

## NURSING CARE

Nurses frequently provide care for clients with metabolic acidosis, although the focus of care often is the disorder underlying the acidosis (e.g., diabetes mellitus, renal failure) rather than the acidosis itself. For this reason, it is vital for the nurse to be aware of the effects of the acidosis and its implications for nursing care.

### Health Promotion

To promote health in clients at risk for metabolic acidosis, discuss management of their underlying disease process (e.g., type 1 diabetes or renal failure) to prevent complications such as diabetic ketoacidosis and metabolic acidosis. Because early manifestations of metabolic acidosis (e.g., fatigue, general malaise, anorexia, nausea, abdominal pain) resemble those of common viral disorders such as "the flu," stress the importance of promptly seeking treatment if these symptoms develop.

### Assessment

Assessment data related to metabolic acidosis include the following:

- *Health history:* Current manifestations, including anorexia, nausea, vomiting, abdominal discomfort, fatigue, lethargy,

other symptoms; duration of symptoms and any precipitating factors such as diarrhea, ingestion of a toxin such as aspirin, methanol, or ethylene; chronic diseases such as diabetes or renal failure, cirrhosis of the liver, or endocrine disorders; current medications.
- *Physical assessment:* Mental status and level of consciousness; vital signs including respiratory rate and depth; apical and peripheral pulses; skin color and temperature; abdominal contour and distention; bowel sounds; urine output.
- *Diagnostic tests:* ABGs, serum electrolytes, tests for underlying disorders.

## Nursing Diagnoses and Interventions

Nursing management of clients with metabolic acidosis often focuses on the primary disorder (e.g., diabetic ketoacidosis or renal failure); however, the acidosis itself has effects that must be attended to when providing care.

### Decreased Cardiac Output

Metabolic acidosis affects cardiac output by decreasing myocardial contractility, slowing the heart rate, and increasing the risk for dysrhythmias. The accompanying hyperkalemia increases the risk for decreased cardiac output as well (see earlier discussion about hyperkalemia).
- Monitor vital signs, including peripheral pulses and capillary refill. *Hypotension, diminished pulse strength, and slowed capillary refill may indicate decreased cardiac output and impaired tissue perfusion. Poor tissue perfusion can increase the risk for lactic acidosis.*
- Monitor the ECG pattern for dysrhythmias and changes characteristic of hyperkalemia. Notify the physician of changes. *Progressive ECG changes such as widening of the QRS complex indicate an increasing risk of dysrhythmias and cardiac arrest. Dysrhythmias further decrease cardiac output, possibly intensifying the degree of acidosis.*
- Monitor laboratory values, including ABGs, serum electrolytes, and renal function studies (serum creatinine and BUN). *Frequent monitoring of laboratory values allows evaluation of the effectiveness of treatment as well as early identification of potential problems.*

### Risk for Excess Fluid Volume

Administering bicarbonate to correct acidosis increases the risk for hypernatremia, hyperosmolality, and fluid volume excess.
- Monitor and maintain fluid replacement as ordered. Monitor serum sodium levels and osmolality. *Bicarbonate administration can cause hypernatremia and hyperosmolality, leading to water retention.*
- Monitor heart and lung sounds, CVP, and respiratory status. *Increasing dyspnea, adventitious lung sounds, a third heart sound ($S_3$) due to the volume of blood flow through the heart, and high CVP readings are indicative of hypervolemia and should be reported to the care provider.*
- Assess for edema, particularly in the back, sacral, and periorbital areas. *Initially, edema affects dependent tissues—the back and sacrum in clients who are bedridden. Periorbital edema indicates more generalized edema.*
- Assess urine output hourly. Maintain accurate intake and output records. Note urine output less than 30 mL/hour or a positive fluid balance on 24-hour total intake and output calculations. *Heart failure and inadequate renal perfusion may lead to decreased urine output.*
- Obtain daily weights using consistent conditions. *Daily weights are an accurate indicator of fluid balance.*
- Administer prescribed diuretics as ordered, monitoring the client's response to therapy. *Loop or high-ceiling diuretics such as furosemide can lead to further electrolyte imbalances, especially hypokalemia. This is a significant risk like that seen during correction of metabolic acidosis.*

### Risk for Injury

Mental status and brain function are affected by acidosis, increasing the risk for injury.
- Monitor neurologic function, including mental status, level of consciousness, and muscle strength. *As the pH falls, mental functioning declines, leading to confusion, stupor, and a decreasing level of consciousness.*
- Institute safety precautions as necessary: Keep the bed in its lowest position, side rails raised. *These measures help protect the client from injury resulting from confusion or disorientation.*
- Keep clocks, calendars, and familiar objects at bedside. Orient to time, place, and circumstances as needed. Allow significant others to remain with the client as much as possible. *An unfamiliar environment and altered thought processes can further increase the risk for injury. Significant others provide a sense of security and reduce anxiety.*

Nursing care also includes measures to treat the underlying disorder, such as diabetic ketoacidosis. Refer to the chapters on diabetes (Chapter 20 ∞) and renal failure (Chapter 29 ∞) for specific interventions.

## Community-Based Care

Discharge planning and teaching focus on the underlying cause of the imbalance. The client who has developed ketoacidosis as a result of diabetes mellitus, starvation, or alcoholism needs interventions and teaching to prevent future episodes of acidosis. Diet, medication management, and alcohol dependency treatment are vital teaching areas. When metabolic acidosis is related to renal failure, the client should be referred for management of the renal failure itself. Clients who have experienced diarrhea or excess ileostomy drainage leading to bicarbonate loss need information about appropriate diarrhea treatment strategies and when to call their primary care provider.

# The Client with Metabolic Alkalosis

Metabolic alkalosis (bicarbonate excess) is characterized by a high pH (>7.45) and a high bicarbonate (>26 mEq/L). It may be caused by loss of acid or excess bicarbonate in the body. When metabolic alkalosis develops, the respiratory system attempts to return the pH to normal by slowing the respiratory rate. Carbon dioxide is retained, and the $Paco_2$ increases (>45 mmHg).

### Risk Factors

As is the case with other acid–base imbalances, metabolic alkalosis rarely occurs as a primary disorder. Risk factors include hospitalization, hypokalemia, and treatment with alkalinizing solutions (e.g., bicarbonate).

### Pathophysiology

Hydrogen ions may be lost via gastric secretions, through the kidneys, or because of a shift of $H^+$ into the cells. Metabolic alkalosis due to loss of hydrogen ions usually occurs because of vomiting or gastric suction. Gastric secretions are highly acidic (pH 1 to 3). When these are lost through vomiting or gastric suction, the alkalinity of body fluids increases. This increased alkalinity results both from the loss of acid and selective retention of bicarbonate by the kidneys as chloride is depleted. (Chloride is the major anion in ECF; when it is lost, bicarbonate is retained as a replacement anion.)

Increased renal excretion of hydrogen ions can be prompted by hypokalemia as the kidneys try to conserve potassium, excreting hydrogen ion instead. Hypokalemia contributes to metabolic alkalosis in another way as well. When potassium shifts out of cells to maintain extracellular potassium levels, hydrogen ions shift into the cells to maintain the balance between cations and anions within the cell.

Excess bicarbonate usually occurs as a result of ingesting antacids that contain bicarbonate (such as soda bicarbonate or Alka-Seltzer) or overzealous administration of bicarbonate to treat metabolic acidosis. Common causes of metabolic alkalosis are summarized in Table 10–11.

In alkalosis, more calcium combines with serum proteins, reducing the amount of ionized (physiologically active) calcium in the blood. This accounts for many of the common manifestations of metabolic alkalosis. Alkalosis also affects potassium balance: Hypokalemia not only can cause metabolic alkalosis (see above), but it also can result from metabolic alkalosis. Hydrogen ions shift out of the intracellular space to help restore the pH, prompting more potassium to enter the cells and depleting ECF potassium. The high pH depresses the respiratory system as the body retains carbon dioxide to restore the carbonic acid to bicarbonate ratio.

### Manifestations and Complications

Manifestations of metabolic alkalosis (see the box below) occur as a result of decreased calcium ionization and are similar to those of hypocalcemia, including numbness and tingling around the mouth, fingers, and toes; dizziness; Trousseau's sign; and muscle spasm. As the respiratory system compensates for metabolic alkalosis, respirations are depressed and respiratory failure with hypoxemia and respiratory acidosis may develop.

**MANIFESTATIONS of Metabolic Alkalosis**

- Confusion
- Decreasing level of consciousness
- Hyperreflexia
- Tetany
- Dysrhythmias
- Hypotension
- Seizures
- Respiratory failure

## INTERDISCIPLINARY CARE

Interdisciplinary management of metabolic alkalosis focuses on diagnosing and correcting the underlying cause.

### Diagnosis

The following laboratory and diagnostic tests may be ordered.

- *ABGs* show a pH greater than 7.45 and bicarbonate level greater than 26 mEq/L. With compensatory hypoventilation, carbon dioxide is retained, and the $Paco_2$ is greater than 45 mmHg.
- *Serum electrolytes* often demonstrate decreased serum potassium (<3.5 mEq/L) and decreased chloride (<95 mEq/L) levels. The serum bicarbonate level is high. Although the total serum calcium may be normal, the ionized fraction of calcium is low.
- *Urine pH* may be low (pH 1 to 3) if metabolic acidosis is caused by hypokalemia. The kidneys selectively retain potassium and excrete hydrogen ion to restore ECF potassium levels. Urinary chloride levels may be normal or greater than 250 mEq/24 hours.
- The *ECG pattern* shows changes similar to those seen with hypokalemia. These changes may be due to hypokalemia or to the alkalosis.

### Medications

Treatment of metabolic alkalosis includes restoring normal fluid volume and administering potassium chloride and sodium chloride solution. The potassium restores serum and intracellular potassium levels, allowing the kidneys to more effectively conserve hydrogen ions. Chloride promotes renal excretion of bicarbonate. Sodium chloride solutions restore fluid volume deficits that can contribute to metabolic alkalosis. In severe alkalosis, an acidifying solution such as dilute hydrochloric acid or ammonium chloride may be administered. In addition, drugs may be used to treat the underlying cause of the alkalosis.

## NURSING CARE

### Health Promotion

Health promotion activities focus on teaching clients the risks of using sodium bicarbonate as an antacid to relieve heartburn or gastric distress. Stress the availability of other effective antacid preparations and the need to seek medical evaluation for persistent gastric symptoms.

In the hospital setting, carefully monitor laboratory values for clients at risk for developing metabolic alkalosis, particularly clients undergoing continuous gastric suction.

### Assessment

Focused assessment data related to metabolic alkalosis include the following:

- *Health history:* Current manifestations, such as numbness and tingling, muscle spasms, dizziness, other symptoms; duration of symptoms and any precipitating factors such as bicarbonate ingestion, vomiting, diuretic therapy, or endocrine disorders; current medications.
- *Physical assessment:* Vital signs including apical pulse and rate and depth of respirations; muscle strength; deep tendon reflexes.
- *Diagnostic tests:* ABGs, serum electrolytes.

## Nursing Diagnoses and Interventions

As with metabolic acidosis, nursing care of the client with metabolic alkalosis often focuses on intervening for client responses to the primary problem, rather than the alkalosis itself. However, the risk for impaired gas exchange is a priority problem, especially with severe metabolic alkalosis.

### Risk for Impaired Gas Exchange

Respiratory compensation for metabolic alkalosis depresses the respiratory rate and reduces the depth of breathing to promote carbon dioxide retention. As a result, the client is at risk for impaired gas exchange, especially in the presence of underlying lung disease.

- Monitor respiratory rate, depth, and effort. Monitor oxygen saturation continuously, reporting an oxygen saturation level of less than 95% (or as ordered). *The depressed respiratory drive associated with metabolic alkalosis can lead to hypoxemia and impaired oxygenation of tissues. Oxygen saturation levels of less than 90% indicate significant oxygenation problems.*
- Assess skin color; note and report cyanosis around the mouth. *Central cyanosis, seen around the mouth and oral mucous membranes, indicates significant hypoxia.*
- Monitor mental status and level of consciousness (LOC). Report decreasing LOC or behavior changes such as restlessness, agitation, or confusion. *Changes in mental status or behavior may be early signs of hypoxia.*
- Place in semi-Fowler's or Fowler's position as tolerated. *Elevating the head of the bed facilitates alveolar ventilation and gas exchange.*
- Schedule nursing care activities to allow rest periods. *The client who is hypoxemic has limited energy reserves, necessitating frequent rest and limited activities.*
- Administer oxygen as ordered or necessary to maintain oxygen saturation levels. *Supplemental oxygen can help maintain blood and tissue oxygenation despite depressed respirations.*

### Deficient Fluid Volume

Clients with metabolic alkalosis often have an accompanying fluid volume deficit.

**PRACTICE ALERT**

Assess intake and output accurately, monitoring fluid balance. In acute situations, hourly intake and output may be indicated. Urine output of less than 30 mL/hour indicates inadequate tissue perfusion, inadequate renal perfusion, and an increased risk for acute renal failure.

- Assess vital signs, CVP, and peripheral pulse volume at least every 4 hours. *Hypotension, tachycardia, low CVP, and weak, easily obliterated peripheral pulses indicate hypovolemia.*
- Weigh daily under standard conditions (time of day, clothing, and scale). *Rapid weight changes accurately reflect fluid balance.*
- Administer intravenous fluids as prescribed using an electronic infusion pump. Monitor for indicators of fluid overload if rapid fluid replacement is ordered: dyspnea, tachypnea, tachycardia, increased CVP, jugular vein distension, and edema. *Rapid fluid replacement may lead to hypervolemia, resulting in pulmonary edema and cardiac failure, particularly in clients with compromised cardiac and renal function.*
- Monitor serum electrolytes, osmolality, and ABG values. *Rehydration and administration of potassium chloride will affect both acid–base and fluid and electrolyte balance. Careful monitoring is important to identify changes.*

## Community-Based Care

When preparing the client with metabolic alkalosis for discharge, consider the cause of the alkalosis and any underlying factors. For example, provide teaching about the following:

- Using appropriate antacids for heartburn and gastric distress
- Using potassium supplements as ordered or eating high-potassium foods to avoid hypokalemia if taking a potassium-wasting diuretic or if aldosterone production is impaired
- Contacting the primary care provider if uncontrolled or extended vomiting develops.

# The Client with Respiratory Acidosis

Respiratory acidosis is caused by an excess of dissolved carbon dioxide, or carbonic acid. It is characterized by a pH less than 7.35 and a $Paco_2$ greater than 45 mmHg. Respiratory acidosis may be either acute or chronic. In chronic respiratory acidosis, the bicarbonate is higher than 26 mEq/L as the kidneys compensate by retaining bicarbonate.

## Risk Factors

Acute or chronic lung disease (e.g., pneumonia or chronic obstructive pulmonary disease [COPD]) is the primary risk factor for respiratory acidosis. Other conditions that depress or interfere with ventilation, such as excess narcotic analgesics, airway obstruction, or neuromuscular disease, also are risk factors for respiratory acidosis. Selected causes of respiratory acidosis are listed in Table 10–11.

## Pathophysiology

Both acute and chronic respiratory acidosis result from carbon dioxide retention caused by alveolar hypoventilation. Hypoxemia (low oxygen in the arterial blood) frequently accompanies respiratory acidosis.

***ACUTE RESPIRATORY ACIDOSIS*** Acute respiratory acidosis occurs due to a sudden failure of ventilation. Chest trauma, aspiration of a foreign body, acute pneumonia, and overdoses of narcotic or sedative medications can lead to this condition. Because acute respiratory acidosis occurs with the sudden onset of hypoventilation—for example, with cardiac arrest—the

$Paco_2$ rises rapidly and the pH falls markedly. A pH of 7 or lower can occur within minutes (Metheny, 2000). The serum bicarbonate level initially is unchanged because the compensatory response of the kidneys occurs over hours to days.

Hypercapnia (increased carbon dioxide levels) affects neurologic function and the cardiovascular system. Carbon dioxide rapidly crosses the blood–brain barrier. Cerebral blood vessels dilate and, if the condition continues, intracranial pressure increases and *papilledema* (swelling and inflammation of the optic nerve where it enters the retina) develops (Porth, 2005). Peripheral vasodilation also occurs, and the pulse rate increases to maintain cardiac output.

***CHRONIC RESPIRATORY ACIDOSIS*** Chronic respiratory acidosis is associated with chronic respiratory or neuromuscular conditions such as COPD, asthma, cystic fibrosis, or multiple sclerosis. These conditions affect alveolar ventilation because of airway obstruction, structural changes in the lung, or limited chest wall expansion. Most clients with chronic respiratory acidosis have COPD with chronic bronchitis and emphysema. (See Chapter 39 ∞ for more information about COPD.) In chronic respiratory acidosis, the $Paco_2$ increases over time and remains elevated. The kidneys retain bicarbonate, increasing bicarbonate levels, and the pH often remains close to the normal range.

The acute effects of hypercapnia may not develop because carbon dioxide levels rise gradually, allowing compensatory changes to occur. When carbon dioxide levels are chronically elevated, the respiratory center becomes less sensitive to the gas as a stimulant of the respiratory drive. The $Pao_2$ provides the primary stimulus for respirations. Clients with chronic respiratory acidosis are at risk for developing *carbon dioxide narcosis,* with manifestations of acute respiratory acidosis, if the respiratory center is suppressed by administering excess supplemental oxygen.

**PRACTICE ALERT**

Carefully monitor neurologic and respiratory status in clients with chronic respiratory acidosis who are receiving oxygen therapy. Immediately report a decreasing LOC or depressed respirations.

### Manifestations

The manifestations of acute and chronic respiratory acidosis differ. In acute respiratory acidosis, the rapid rise in $Paco_2$ levels causes manifestations of hypercapnia. Cerebral vasodilation causes manifestations such as headache, blurred vision, irritability, and mental cloudiness. If the condition continues, the level of consciousness progressively decreases. Rapid and dramatic changes in ABGs can lead to unconsciousness and ventricular fibrillation, a potentially lethal cardiac dysrhythmia. The skin of the client with acute respiratory acidosis may be warm and flushed, and the pulse rate is elevated.

The manifestations of chronic respiratory acidosis include weakness and a dull headache. Sleep disturbances, daytime sleepiness, impaired memory, and personality changes also may be manifestations of chronic respiratory acidosis (see the box on this page).

**MANIFESTATIONS of Respiratory Acidosis**

**Acute Respiratory Acidosis**

- Headache
- Warm, flushed skin
- Blurred vision
- Irritability, altered mental status
- Decreasing level of consciousness
- Cardiac arrest

**Chronic Respiratory Acidosis**

- Weakness
- Dull headache
- Sleep disturbances with daytime sleepiness
- Impaired memory
- Personality changes

## INTERDISCIPLINARY CARE

Clients with acute respiratory failure usually require treatment in the emergency department or intensive care unit. The focus is on restoring adequate ventilation and gas exchange. Hypoxemia often accompanies acute respiratory acidosis, so oxygen is administered as well. Supplemental oxygen is administered with caution to clients with chronic respiratory acidosis.

### Diagnosis

The following laboratory and diagnostic tests may be ordered.

- *ABGs* show a pH of less than 7.35 and a $Paco_2$ of more than 45 mmHg. In acute respiratory acidosis, the bicarbonate level is initially within normal range but increases to greater than 26 mEq/L if the condition persists. In chronic respiratory acidosis, both the $Paco_2$ and the $HCO_3^-$ may be significantly elevated.
- *Serum electrolytes* may show hypochloremia (chloride level < 98 mEq/L) in chronic respiratory acidosis.
- *Pulmonary function tests* may be done to determine if chronic lung disease is the cause of the respiratory acidosis. These studies would not be done during the acute period, however.

Additional diagnostic tests may be done to identify the underlying cause of the respiratory acidosis. *Chest x-ray* and *sputum studies* (cytology and culture) may be ordered to identify an acute or chronic lung disorder. If drug overdose is suspected, *serum levels* of the drug may be obtained.

### Medications

Bronchodilator drugs may be administered to open the airways and antibiotics prescribed to treat respiratory infections. If excess narcotics or anesthetic has caused acute respiratory acidosis, drugs to reverse their effects (such as naloxone) may be given.

### Respiratory Support

Treatment of respiratory acidosis, either acute or chronic, focuses on improving alveolar ventilation and gas exchange. Clients with severe respiratory acidosis and hypoxemia may require intubation and mechanical ventilation (see Chapter 39 ∞ for more infor-

mation about these procedures). The $PaCO_2$ level is lowered slowly to avoid complications such as cardiac dysrhythmias and decreased cerebral perfusion. In clients with chronic respiratory acidosis, oxygen is administered cautiously to avoid carbon dioxide narcosis.

Pulmonary hygiene measures, such as breathing treatments or percussion and drainage, may be instituted. Adequate hydration is important to promote removal of respiratory secretions.

## NURSING CARE

See the Nursing Care Plan: A Client with Acute Respiratory Acidosis below.

## Health Promotion

Health promotion activities related to respiratory acidosis focus on identifying, monitoring, and teaching clients at risk. Carefully monitor clients receiving anesthesia, narcotic analgesics, or sedatives for signs of respiratory depression. Monitor the response of clients with a history of chronic lung disease to oxygen therapy. Teach clients who have an identified risk for respiratory acidosis (such as people using narcotic analgesia for cancer pain and people with chronic lung disease) and their families about early manifestations of respiratory depression and acidosis, and instruct them to immediately contact their care provider if manifestations develop.

## Assessment

Assessment data related to respiratory acidosis include the following:

- *Health history:* Current manifestations, including headache, irritability or lethargy, difficulty thinking, blurred vision, and other symptoms; duration of symptoms and any precipitating factors such as drug use or respiratory infection; chronic diseases such as cystic fibrosis or COPD; current medications.
- *Physical assessment:* Mental status and level of consciousness; vital signs; skin color and temperature; rate and depth of respirations, pulmonary excursion, lung sounds; examination of optic fundus for possible papilledema.
- *Diagnostic tests:* ABGs, serum electrolytes; white blood cell count (indicator of infection), sputum culture results, serum drug and toxicology results.

## Nursing Diagnoses and Interventions

Restoring effective alveolar ventilation and gas exchange is the priority of interdisciplinary and nursing care for clients with respiratory acidosis.

### NURSING CARE PLAN A Client with Acute Respiratory Acidosis

Marlene Hitz, age 76, is eating lunch with her friends when she suddenly begins to choke and is unable to breathe. After several minutes of trying, an attendant at the senior center successfully dislodges some meat caught in Ms. Hitz's throat using the Heimlich maneuver. Ms. Hitz is taken by ambulance to the emergency department for follow-up because she was apneic for 3 to 4 minutes, her respirations are shallow, and she is disoriented.

#### ASSESSMENT

Ms. Hitz is placed in an observation room. Oxygen is started at 4 L/min per nasal cannula. David Love, the nurse admitting Ms. Hitz, makes the following assessments: T 98.2, P 102, R 36 and shallow, BP 146/92. Skin is warm and dry. Alert but restless and not oriented to time or place; she responds slowly to questions. Stat ABGs are drawn, a chest x-ray is done, and $D_5$ 1/2 NS is started intravenously at 50 mL/h.

The chest x-ray shows no abnormality. ABG results are pH 7.38 (normal: 7.35 to 7.45), $PaCO_2$ 48 mmHg (normal: 35 to 45 mmHg), $PaO_2$ 92 mmHg (normal: 80 to 100 mmHg), and $HCO_3^-$ 24 mEq/L (normal: 22 to 26 mEq/L).

#### DIAGNOSES

- *Impaired Gas Exchange* related to temporary airway obstruction
- *Anxiety* related to emergency hospital admission
- *Risk for Injury* related to confusion

#### EXPECTED OUTCOMES

- Regain normal gas exchange and ABG values.
- Be oriented to time, place, and person.
- Regain baseline mental status.
- Remain free of injury.

#### PLANNING AND IMPLEMENTATION

- Monitor ABGs, to be redrawn in 2 hours.
- Monitor vital signs and respiratory status (including oxygen saturation) every 15 minutes for the first hour then every hour.
- Assess color of skin, nail beds, and oral mucous membranes every hour.
- Assess mental status and orientation every hour.
- Monitor anxiety level as evidenced by restlessness and agitation.
- Maintain a calm, quiet environment.
- Provide reorientation and explain all activities.
- Keep side rails in place, and place call bell within reach.

#### EVALUATION

Ms. Hitz remains in the emergency department for 6 hours. Her ABGs are still abnormal, and David Love now notes the presence of respiratory crackles and wheezes. She is less anxious and responds appropriately when asked who and where she is. Because she has not regained normal gas exchange, Ms. Hitz is admitted to the hospital for continued observation and treatment.

#### CRITICAL THINKING IN THE NURSING PROCESS

1. Describe the pathophysiologic process that leads to acute respiratory acidosis in Ms. Hitz.
2. Describe the effect of acidosis on mental function.
3. What teaching would you provide to Ms. Hitz to prevent future episodes of choking?

*See Evaluating Your Response in Appendix C.*

### Impaired Gas Exchange

- Frequently assess respiratory status, including rate, depth, effort, and oxygen saturation levels. *Decreasing respiratory rate and effort along with decreasing oxygen saturation levels may signal worsening respiratory failure and respiratory acidosis.*

**PRACTICE ALERT**
Frequently assess level of consciousness. A decline in LOC may indicate increasing hypercapnia and the need for increasing ventilatory support (such as intubation and mechanical ventilation).

- Promptly evaluate and report ABG results to the physician and respiratory therapist. *Rapid changes in carbon dioxide or oxygen levels may necessitate modification of the treatment plan to prevent complications of overcorrection of respiratory acidosis.*
- Place in semi-Fowler's to Fowler's position as tolerated. *Elevating the head of the bed promotes lung expansion and gas exchange.*
- Administer oxygen as ordered. Carefully monitor response. Reduce the oxygen flow rate or percentage and immediately report increasing somnolence. *Supplemental oxygen can suppress the respiratory drive in clients with chronic respiratory acidosis.*

### Ineffective Airway Clearance

- Frequently auscultate breath sounds (whether on or off a mechanical ventilator). *Increasing adventitious sounds or decreasing breath sounds (faint or absent) may indicate worsening airway clearance due to obstruction or fatigue.*
- Encourage the client with chronic respiratory acidosis to use pursed-lip breathing. *Pursed-lip breathing helps maintain open airways throughout exhalation, promoting carbon dioxide elimination.*
- Frequently reposition and encourage ambulation as tolerated. *Repositioning, sitting at the bedside, and ambulation promote airway clearance and lung expansion.*
- Encourage fluid intake of up to 3000 mL per day as tolerated or allowed. *Fluids help liquefy secretions and hydrate respiratory mucous membranes, promoting airway clearance.*
- Administer medications such as inhaled bronchodilators as ordered. *Inhaled bronchodilators help relieve bronchial spasm, dilating airways.*
- Provide percussion, vibration, and postural drainage as ordered. *Pulmonary hygiene measures such as these help loosen respiratory secretions so they can be coughed out of airways.*

## Using NANDA, NIC, and NOC

Chart 10–3 shows links between NANDA nursing diagnoses, NIC, and NOC when caring for a client with respiratory acidosis.

## Community-Based Care

Planning and teaching for home care focuses on the problem that caused the client to develop respiratory acidosis. The client who developed acute respiratory acidosis as a result of acute pneumonia or chest trauma may only require teaching to prevent future problems. If acute respiratory acidosis occurred secondarily to a narcotic overdose, determine if the drug was prescribed for pain or if it was an illicit street drug. Provide teaching to the client who requires narcotic medication on a continuing basis. Refer the client using illicit drugs to a substance abuse counselor, treatment center, or Narcotics Anonymous as appropriate.

For clients with chronic lung disease, discuss ways to avoid future episodes of acute respiratory failure. Encourage the client to be immunized against pneumococcal pneumonia and influenza. Discuss ways to avoid acute respiratory infections and measures to take when respiratory status is further compromised.

NANDA, NIC, AND NOC LINKAGES
CHART 10–3 **The Client with Respiratory Acidosis**

**NANDA**

Ineffective Airway Clearance

↓

**NIC**

Acid–Base Management: Respiratory Acidosis
Airway Management
Cough Enhancement
Respiratory Monitoring
Ventilation Assistance

↓

**NOC**

Electrolyte and Acid–Base Balance
Respiratory Status: Airway Patency
Respiratory Status: Ventilation

Data from *NANDA's Nursing Diagnoses: Definitions & classification 2005–2006* by NANDA International (2003), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

## The Client with Respiratory Alkalosis

Respiratory alkalosis is characterized by a pH greater than 7.45 and a $PaCO_2$ of less than 35 mmHg. It is always caused by hyperventilation leading to a carbon dioxide deficit.

### Risk Factors

Anxiety with hyperventilation is the most common cause of respiratory alkalosis; therefore, anxiety disorders increase the risk for this acid–base imbalance. In the client who is critically ill, mechanical ventilation is a risk factor for respiratory alkalosis.

### Pathophysiology

In acute respiratory alkalosis, the pH rises rapidly as the $PaCO_2$ falls. Because the kidneys are unable to rapidly adapt to the change in pH, the bicarbonate level remains within normal lim-

its. Anxiety-based hyperventilation is the most common cause of acute respiratory alkalosis. Other physiologic causes of hyperventilation include high fever, hypoxia, gram-negative bacteremia, and thyrotoxicosis. Early salicylate intoxication (aspirin overdose), encephalitis, and high progesterone levels in pregnancy directly stimulate the respiratory center, potentially leading to hyperventilation and respiratory alkalosis. Hyperventilation also can occur during anesthesia or mechanical ventilation if the rate and tidal volume (depth) of ventilations is excessive.

If hyperventilation continues, the kidneys compensate by eliminating bicarbonate to restore the bicarbonate to carbonic acid ratio. The bicarbonate level is lower than normal in chronic respiratory alkalosis, and the pH may be close to the normal range.

Alkalosis increases binding of extracellular calcium to albumin, reducing ionized calcium levels. As a result, neuromuscular excitability increases and manifestations similar to hypocalcemia develop. Low carbon dioxide levels in the blood cause vasoconstriction of cerebral vessels, increasing the neurologic manifestations of the disorder.

### Manifestations

The manifestations of respiratory alkalosis include lightheadedness, a feeling of panic and difficulty concentrating, circumoral and distal extremity paresthesias, tremors, and positive Chvostek's and Trousseau's signs. The client also may experience tinnitus, a sensation of chest tightness, and palpitations (cardiac dysrhythmias). Seizures and loss of consciousness may occur. (See the Manifestations box on this page.)

## INTERDISCIPLINARY CARE

Management of respiratory alkalosis focuses on correcting the imbalance and treating the underlying cause.

### Diagnosis

*ABGs* generally show a pH greater than 7.45 and a $PaCO_2$ of less than 35 mmHg. In chronic hyperventilation, there is a compensatory decrease in serum bicarbonate to less than 22 mEq/L and the pH may be near normal.

### Medications

A sedative or antianxiety agent may be necessary to relieve anxiety and restore a normal breathing pattern. Additional drugs to correct underlying problems other than anxiety-induced hyperventilation may be ordered.

### Respiratory Therapy

The usual treatment for anxiety-related respiratory alkalosis involves instructing the client to breathe more slowly and having the client breathe into a paper bag or rebreather mask. This allows rebreathing of exhaled carbon dioxide, increasing $PaCO_2$ levels and reducing the pH. If excessive ventilation by a mechanical ventilator is the cause of respiratory alkalosis, ventilator settings are adjusted to reduce the respiratory rate and tidal volume as indicated. When hypoxia is the underlying cause of hyperventilation, oxygen is administered.

### MANIFESTATIONS of Respiratory Alkalosis

- Dizziness
- Numbness and tingling around mouth, hands, and feet
- Palpitations
- Dyspnea
- Chest tightness
- Anxiety/panic
- Tremors
- Tetany
- Seizures, loss of consciousness

## NURSING CARE

### Health Promotion

Identify clients at risk in the hospital (e.g., clients on mechanical ventilation or who have a fever or infection), and monitor assessment data and ABGs to identify early manifestations of hyperventilation and respiratory alkalosis.

### Assessment, Diagnoses, and Interventions

#### Ineffective Breathing Pattern

The usual cause of hyperventilation and respiratory alkalosis is psychologic, although physiologic disorders also can lead to hyperventilation. It is important to not only address the hyperventilation, but also to identify the underlying cause.

- Assess respiratory rate, depth, and ease. Monitor vital signs (including temperature) and skin color. *Assessment data can help identify the underlying cause, such as a fever or hypoxia.*
- Obtain subjective assessment data such as circumstances leading up to the current situation, current health and recent illnesses or medication use, and current manifestations. *Subjective data provide cues to the cause and circumstances of the hyperventilation response.*
- Reassure the client that he or she is not experiencing a heart attack and that symptoms will resolve when breathing returns to normal. *Manifestations of hyperventilation and respiratory alkalosis such as dyspnea, chest tightness or pain, and palpitations can mimic those of a heart attack.*
- Instruct the client to maintain eye contact and breathe with you to slow the respiratory rate. *These measures help to make the client aware of respirations and provide a sense of support and control* (Ackley & Ladwig, 2006).
- Have the client breathe into a paper bag. *This allows the client to rebreathe exhaled carbon dioxide, increasing the $PaCO_2$ and decreasing the pH.*
- Protect the client from injury. *If hyperventilation continues to the point at which the client loses consciousness, respirations will return to normal, as will acid–base balance.*
- If the client has experienced repeated episodes of hyperventilation or has a chronic anxiety disorder, refer for counseling. *Counseling can help the client develop alternative strategies for dealing with anxiety.*

## Community-Based Care

Planning and teaching for home care is directed toward the underlying cause of hyperventilation. If anxiety precipitated the episode, discuss anxiety management strategies with the client. Refer the client and family to a counselor if appropriate. Teach the client to identify a hyperventilation reaction, and how to breathe into a paper bag to manage it at home.

## EXPLORE MEDIALINK

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Audio Glossary
NCLEX-RN® Review

***Animations***

*Acid–Base Balance*
*Fluid Balance*
*Furosemide*
*Membrane Transport*

**COMPANION WEBSITE www.prenhall.com/lemone**

Audio Glossary
NCLEX-RN® Review
Care Plan Activities
*Fluid Volume Deficit*
*Hypocalcemia*
Case Studies
*Third Spacing*
*Hypernatremia*
MediaLink Applications
*Metabolic Acidosis and Type 1 Diabetes*
*Alterations in Electrolytes, Medications, Fluid Volumes*
Links to Resources

## CHAPTER HIGHLIGHTS

- The volume and composition of body fluid is normally maintained by a balance of fluid and electrolyte intake; elimination of water, electrolytes, and acids by the kidneys; and hormonal influences. Change in any of these factors can lead to a fluid, electrolyte, or acid–base imbalance that adversely impacts health.
- Fluid, electrolyte, and acid–base imbalances can affect all body systems, especially the cardiovascular system, the central nervous system, and the transmission of nerve impulses. Conversely, primary disorders of the respiratory, renal, cardiovascular, endocrine, or other body systems can lead to an imbalance of fluids, electrolytes, or acid–base status.
- Fluid and sodium imbalances commonly are related; both affect serum osmolality.
- Potassium imbalances are commonly seen in clients with acute or chronic illnesses. Both hypokalemia and hyperkalemia affect cardiac conduction and function. Carefully monitor cardiac rhythm and status in clients with very low or very high potassium levels.
- Calcium imbalances primarily affect neuromuscular transmission: Hypocalcemia increases neuromuscular irritability; hypercalcemia depresses neuromuscular transmission. Magnesium imbalances have a similar effect.
- Acid–base imbalances may be caused by either metabolic or respiratory problems. Simple acid–base imbalances (respiratory or metabolic acidosis or alkalosis) are more commonly seen than mixed imbalances.
- Buffers, lungs, and kidneys work together to maintain acid–base balance in the body. Buffers respond to changes almost immediately; the lungs respond within minutes; the kidneys, however, require hours to days to restore normal acid–base balance.
- The lungs compensate for metabolic acid–base imbalances by excreting or retaining carbon dioxide. This is accomplished by increasing or decreasing the rate and depth of respirations.
- The kidneys compensate for respiratory acid–base imbalances by producing and retaining or excreting bicarbonate, and by retaining or excreting hydrogen ions.
- Careful monitoring of respiratory and cardiovascular status, mental status, neuromuscular function, and laboratory values is an important nursing responsibility for all clients with fluid, electrolyte, or acid–base imbalances.

## TEST YOURSELF NCLEX-RN® REVIEW

1 A client is admitted to the emergency department with hypovolemia. Which intravenous solution would the nurse anticipate administering?
**1.** Ringer's solution
**2.** 10% dextrose in water
**3.** 3% sodium chloride
**4.** 0.45% sodium chloride

2 When assessing a client with fluid volume deficit, the nurse would expect to find:
**1.** increased pulse rate and blood pressure.
**2.** dyspnea and respiratory crackles.
**3.** headache and muscle cramps.
**4.** orthostatic hypotension and flat neck veins.

3 The nurse caring for a client with acute hypernatremia includes which of the following in the plan of care? (Select all that apply.)
**1.** Conduct frequent neurologic checks.
**2.** Restrict fluids to 1500 mL per day.
**3.** Orient to time, place, and person frequently.
**4.** Maintain intravenous access.
**e.** Limit length of visits.

4 Laboratory results for a client show a serum potassium level of 2.2 mEq/L. Which of the following nursing actions is of highest priority for this client?
**1.** Keep the client on bed rest.
**2.** Initiate cardiac monitoring.
**3.** Start oxygen at 2 L/min.
**4.** Initiate seizure precautions.

5 The nurse evaluates teaching about calcium supplement therapy as effective when the client states that she will take her calcium tablets:
**1.** all at one time in the morning.
**2.** with meals.
**3.** as needed for tremulousness.
**4.** with a full glass of water.

6 A client who is known to be an alcoholic presents with confusion, hallucinations, and a positive Chvostek's sign. Which medication(s) should the nurse anticipate administering?
**1.** magnesium sulfate
**2.** calcium chloride
**3.** insulin and glucose
**4.** sodium bicarbonate

7 Arterial blood gas results for a client show pH 7.21, $PaO_2$ 98 mmHg, $PaCO_2$ 32 mmHg, and $HCO_3^-$ 17 mEq/L. The nurse correctly interprets these values as indicative of which of the following acid–base imbalances?
**1.** metabolic acidosis
**2.** metabolic alkalosis
**3.** respiratory acidosis
**4.** respiratory alkalosis

8 A client is admitted with a suspected heroin overdose and a respiratory rate of 5 to 6 per minute. Which of the following assessment data would the nurse anticipate? (Select all that apply.)
**1.** pH 7.29
**2.** alert and oriented
**3.** $PaCO_2$ 54 mmHg
**4.** $HCO_3^-$ 32 mEq/L
**e.** skin warm and flushed

9 The nurse caring for a client undergoing several days of gastric decompression recognizes that the client is at risk for which of the following acid–base imbalances?
**1.** metabolic acidosis
**2.** metabolic alkalosis
**3.** respiratory acidosis
**4.** respiratory alkalosis

10 A client undergoing mechanical ventilation following a severe chest wall injury and flail chest complains of chest tightness, anxiety, and feeling as though she cannot get enough air. She is afraid she is having a heart attack. The nurse should first
**1.** administer prescribed analgesic.
**2.** contact respiratory therapy to evaluate ventilator settings.
**3.** obtain arterial blood gases.
**4.** notify the physician.

*See Test Yourself answers in Appendix C.*

## BIBLIOGRAPHY


Ackley, B. J., & Ladwig, G. B. (2006). *Nursing diagnosis hand book: A guide to planning care* (7th ed.). St. Louis: Mosby.

Astle, S. M. (2005). Restoring electrolyte balance. *RN, 68*(5), 34–40.

Beers, M. H., & Berkow, R. (Eds.). (2004). *The Merck manual of diagnosis and therapy* (17th ed.). Merck & Co., Inc. Internet edition provided by Medical Services, USMEDSA, USHH.

Bennett, J. A., Tomas, V., & Riegel, B. (2004). Unrecognized chronic dehydration in older adults: Examining prevalence rate and risk factors. *Journal of Gerontological Nursing, 30*(11), 22–28, 52–53.

Burger, C. M. (2004a). Emergency. Hyperkalemia: Averting crisis with early recognition and intervention. *American Journal of Nursing, 104*(11), 61–65.

Burger, C. M. (2004b). Emergency. Hyperkalemia: When serum $K^+$ is not okay. *American Journal of Nursing, 104*(10), 66–70.

Call-Schmidt, T. (2001). Interpreting lab results: A primer. *MEDSURG Nursing, 10*(4), 179–184.

Carmichael, K. A., & Alper, B. S. (2004). State consult. Hypercalcemia. *Clinical Advisor, 7*(12), 67–69.

Copstead, L. C., & Banasik, J. L. (2005). *Pathophysiology* (3rd ed.). St. Louis, MO: Elsevier/Saunders.

Danner, K. (2000). Acid/base balance: Making sense of pH. *CIN PLUS* (On-line serial), *3*(1), 3.

Dochterman, J., & Bulechek, G. (2004). *Nursing interventions classification (NIC)* (4th ed.). St. Louis, MO: Mosby.

Edwards, S. (2001). Regulation of water, sodium and potassium: Implications for practice. *Nursing Standard, 15*(22), 36–44.

Edwards, S. L. (2005). Maintaining calcium balance: Physiology and implications. *Nursing Times, 101*(19), 58–61.

Elgart, H. N. (2004). Assessment of fluids and electrolytes. *AACN Clinical Issues: Advanced Practice in Acute and Critical Care, 15*(4), 607–621.

Hayes, D. D. (2004). Calcium in the balance. *Nursing Made Incredibly Easy!, 2*(2), 46–53.

Holman, C., Roberts, S., & Nicol, M. (2005). Promoting adequate hydration in older people. *Nursing Older People, 17*(4), 31–32.

Hughes, E. (2004). Principles of post-operative patient care. *Nursing Standard, 19*, 43–51, 53.

Johnson, A. L., & Criddle, L. M. (2004). Pass the salt: Indications for and implications of using hypertonic saline. *Critical Care Nurse, 24*(5), 36–38, 40–44, 46+.

Kasper, D. L., Braunwald, E., Fauci, A. S., Hauser, S. L., Longo, D. L., & Jameson, J. L. (Eds.). (2005). *Harrison's principles of internal medicine* (16th ed.). New York: McGraw-Hill.

Krueger, D., & Tasota, F. J. (2003). Eye on diagnostics. Keeping an eye on calcium levels. *Nursing, 33*(6), 68, 70.

Larson, K. (2003). Home health care. Fluid balance in the elderly: Assessment and intervention—Important role in community health and home care nursing. *Geriatric Nursing, 24*(5), 306–309.

Lehne, R. A. (2004). *Pharmacology for nursing care* (5th ed.). Philadelphia: W. B. Saunders.

McCance, K. L., & Huether, S. E. (2006). *Pathophysiology: The biologic basis for disease in adults and children* (5th ed.). St. Louis, MO: Mosby.

Metheny, N. M. (2000). *Fluid and electrolyte balance: Nursing considerations* (4th ed.). Philadelphia: Lippincott.

Mitchell, D. (2004). Consult stat. Here's an easy way to make sense of IV fluid therapy. *RN, 67*(10), 65–67, 39.

Moorhead, S., Johnson, M., & Maas, M. (2004). *Nursing outcomes classification (NOC)* (3rd ed.). St. Louis, MO: Mosby.

NANDA International. (2005). *Nursing diagnoses: Definitions and classification 2005–2006.* Philadelphia: Author.

Oh, H., Suh, Y., Hwang, S., & Seo, W. (2005). Effects of nasogastric tube feeding on serum sodium, potassium and glucose levels. *Journal of Nursing Scholarship, 37*(2), 141–147.

O'Keeffe, S. (2005). Clinician's guide to interpreting arterial blood gases (ABG's). *Clinical Times, 2*(3), 3.

Porth, C. M. (2005). *Pathophysiology: Concepts of altered health states* (7th ed.). Philadelphia: Lippincott.

Pruitt, W. C., & Jacobs, M. (2004). Interpreting arterial blood gases: Easy as ABC. *Nursing, 34*(8), 50–53.

Simpson, H. (2004). Respiratory nursing. Interpretation of arterial blood gases: A clinical guide for nurses. *British Journal of Nursing, 13*(9), 522–528.

Stanner, S. (2005). Preventing undernutrition and dehydration in older people. *Nursing & Residential Care, 7*(1), 17–19.

Suhayda, R., & Walton, J. C. (2002). Preventing and managing dehydration. *MEDSURG Nursing, 11*(6), 267–278.

Sweeney, J. (2005a). Clinical queries. What causes hyponatremia? *Nursing, 35*(6), 18.

Sweeney, J. (2005b). Clinical queries. What causes sudden hypokalemia? *Nursing, 35*(4), 12.

Tierney, L. M., McPhee, S. J., & Papadakis, M. A. (2005). *Current medical diagnosis and treatment* (44th ed.). New York: Lange Medical Books/McGraw-Hill.

Urden, L. D., Stacy, K. M., & Lough, M. E. (2006). *Thelan's critical care nursing: Diagnosis and management* (5th ed.). St. Louis, MO: Mosby.

van Wissen, K., & Breton, C. (2004). Perioperative influences on fluid distribution. *MEDSURG Nursing, 13*(5), 304–311.

Whitney, E. N., & Rolfes, S. R. (2002). *Understanding nutrition* (9th ed.). Belmont, CA: Wadsworth.

Wilkinson, J. M. (2005). *Nursing diagnosis handbook with NIC interventions and NOC outcomes* (8th ed.). Upper Saddle River, NJ: Prentice Hall Health.

Wise, L. C., Mersch, J., Racioppi, J., Crosier, J., & Thompson, C. (2000). Evaluating the reliability and utility of cumulative intake and output. *Journal of Nursing Care Quality, 14*(3), 37–42.

Woodrow, P. (2003). Assessing fluid balance in older people: Fluid replacement. *Nursing Older People, 14*(10), 29–30.

Woodrow, P. (2004). Continuing professional development. Arterial blood gas analysis. *Nursing Standard, 18*(21), 45–52, 54–55.

Yucha, C. (2004). Renal regulation of acid–base balance. *Nephrology Nursing Journal, 31*(2), 201–208.

Zembrzuski, C. (2004). Try this: Best practices in nursing care to older adults. Nutrition and hydration. *MEDSURG Nursing, 13*(1), 60–61.