16

Fluid, Electrolyte, and Acid-Base Imbalances

Mariann M. Harding

Water is the driving force in nature.

Leonardo da Vinci

http://evolve.elsevier.com/Lewis/medsurg/

LEARNING OUTCOMES

1. Describe the composition of the major body fluid compartments.
2. Define processes involved in the regulation of movement of water and electrolytes between the body fluid compartments.
3. Describe the etiology, laboratory diagnostic findings, clinical manifestations, and nursing and interprofessional management of the following disorders:
   a. Extracellular fluid volume imbalances: fluid volume deficit and fluid volume excess
   b. Sodium imbalances: hypernatremia and hyponatremia
   c. Potassium imbalances: hyperkalemia and hypokalemia
   d. Magnesium imbalances: hypermagnesemia and hypomagnesemia
   e. Calcium imbalances: hypercalcemia and hypocalcemia
   f. Phosphate imbalances: hyperphosphatemia and hypophosphatemia
4. Identify the processes involved in maintaining acid-base balance.
5. Discuss the etiology, laboratory diagnostic findings, clinical manifestations, and nursing and interprofessional management of the following acid-base imbalances: metabolic acidosis, metabolic alkalosis, respiratory acidosis, and respiratory alkalosis.
6. Describe the composition of and indications for common IV fluid solutions.
7. Discuss the types and nursing management of commonly used central venous access devices.

KEY TERMS

acidosis, p. 287
active transport, p. 272
alkalosis, p. 287
anions, p. 271
buffers, p. 287
cations, p. 271
central venous access devices (CVADs), p. 294
electrolytes, p. 271
fluid spacing, p. 274
hydrostatic pressure, p. 273
hypertonic, p. 273
hypotonic, p. 273
isotonic, p. 273
oncotic pressure, p. 273
osmolality, p. 272
osmosis, p. 272
osmotic pressure, p. 272

HOMEOSTASIS

Body fluids and electrolytes play an important role in maintaining *homeostasis,* the body's stable internal environment. Body fluids are in constant motion transporting nutrients, electrolytes, and oxygen to cells and carrying waste products away from cells. The body uses a number of adaptive responses to keep the composition and volume of fluids and electrolytes within narrow limits to maintain homeostasis and promote health.

Many diseases and their treatments affect fluid and electrolyte balance. For example, a patient with metastatic colon cancer may develop hypercalcemia because of bone destruction from tumor invasion. Chemotherapy used to treat the cancer may result in nausea and vomiting and, subsequently, dehydration and acid-base imbalances. When correcting dehydration with IV fluids, the patient requires close monitoring to prevent fluid overload.

It is important to anticipate the potential for fluid and electrolyte imbalances associated with certain disorders and medical therapies, recognize the signs and symptoms of imbalances, and intervene with the appropriate action. This chapter describes the (1) normal control of fluids, electrolytes, and acid-base balance; (2) conditions that disrupt homeostasis and resulting manifestations; and (3) actions that the health care provider and you can take to manage fluid, electrolyte, and acid-base imbalances and restore homeostasis.

WATER CONTENT OF THE BODY

The body is composed primarily of water. It accounts for about 50% to 60% of body weight in the adult. Water content varies with body mass, gender, and age (Fig. 16-1). Lean body mass has a higher percentage of water, while adipose tissue has a lesser percentage of water. So, the more fat present in the body, the less the total water content. Women generally have a lower percentage of body water because they tend to have less lean body mass than men. Older adults also tend to have less lean body mass, resulting in a lower percentage of body water when compared to younger adults. In older adults, body water content

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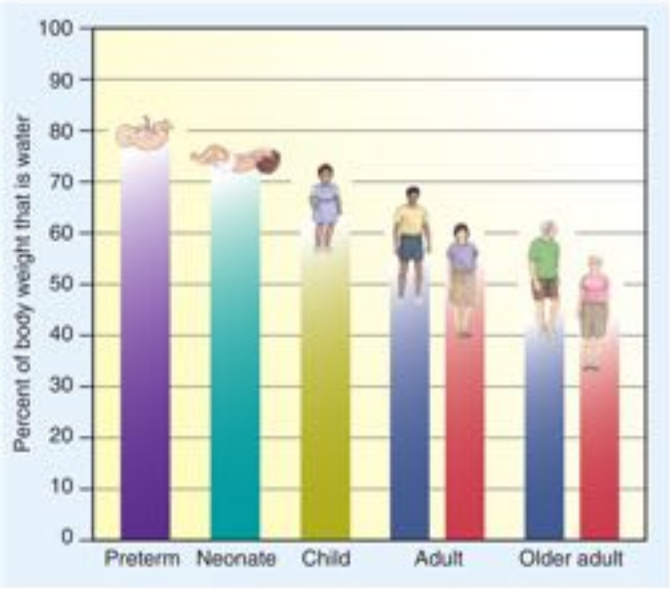

FIG. 16-1 Body water over the life span.

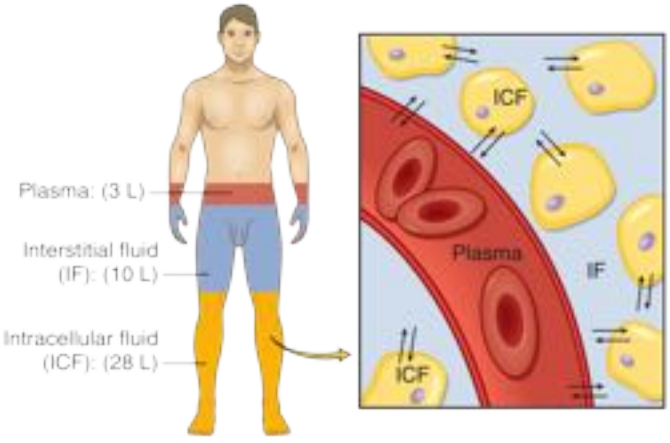

FIG. 16-2 Relative volumes of three body fluids. Values represent fluid distribution in a young male adult.

averages 45% to 50% of body weight. This places them at a higher risk for fluid-related problems than young adults.

Body Fluid Compartments

The two fluid compartments in the body are the *intracellular space* (inside the cells) and the *extracellular space* (outside the cells) (Fig. 16-2). About two thirds of the body water is located within cells and is termed *intracellular fluid* (ICF). ICF makes up about 40% of body weight of an adult. This means a 70-kg young man would have about 42 L of water, with about 28 L of that water located within his cells.

The fluid in the extracellular space is *extracellular fluid* (ECF). The two main compartments containing ECF are the *interstitial fluid*, or the fluid in the spaces between cells, and the intravascular fluid or *plasma*, the liquid part of blood. Other ECF compartments include lymph and *transcellular fluids*. Transcellular fluids include cerebrospinal fluid; fluid in the gastrointestinal (GI) tract and joint spaces; and pleural, peritoneal, intraocular, and pericardial fluid. ECF makes up about one third of the body water; this amounts to about 14 L in a 70-kg man. About one third of ECF is in the intravascular space as plasma (3 L in a 70-kg man), and two thirds is in the interstitial space (10 L in a 70-kg man). The fluid in the transcellular spaces totals about 1 L at any given time.

Calculation of Fluid Gain or Loss

One liter of water weighs 2.2 lb (1 kg). Body weight change, especially sudden change, is an excellent indicator of overall fluid volume loss or gain. For example, if a patient drinks 240 mL (8 oz) of fluid, weight gain will be 0.5 lb (0.23 kg). A patient receiving diuretic therapy who loses 4.4 lb (2 kg) in 24 hours has experienced a fluid loss of about 2 L. An adult patient who is fasting may lose 1 to 2 lb/day. A weight loss exceeding this is likely due to loss of body fluid.

ELECTROLYTES

Electrolytes are substances whose molecules dissociate, or split, into ions when placed in water. *Ions* are electrically charged particles. Cations are positively charged ions. Examples include sodium (Na^+), potassium (K^+), calcium (Ca^{2+}), and magnesium (Mg^{2+}) ions. Anions are negatively charged ions. Examples include bicarbonate (HCO_3^-), chloride (Cl^-), and phosphate (PO_4^{3-}) ions. Most proteins bear a negative charge and are thus anions.

Measurement of Electrolytes

The concentration of electrolytes in body fluids is expressed in milliequivalents (mEq) per liter. Since electrolytes are active chemicals, it is useful to express their concentration according to their chemical activity, or the number of electrolytes able to combine chemically. Ions combine milliequivalent for milliequivalent. For example, 1 mEq (1 mmol) of sodium combines with 1 mEq (1 mmol) of chloride.

Electrolyte Composition of Fluid Compartments

Electrolyte composition varies between ECF and ICF. The overall concentration of electrolytes is nearly the same in the two compartments. However, concentrations of specific ions differ greatly (Fig. 16-3). In ECF the main cation is sodium, with small amounts of potassium, calcium, and magnesium. The primary ECF anion is chloride, with small amounts of bicarbonate, sulfate, and phosphate anions.

In ICF the most prevalent cation is potassium, with small amounts of magnesium and sodium. The prevalent ICF anion is phosphate, with some protein and a small amount of bicarbonate. See Table 16-1 for normal serum electrolyte values.

MECHANISMS CONTROLLING FLUID AND ELECTROLYTE MOVEMENT

The movement of electrolytes and water between ICF and ECF to maintain homeostasis involves many different processes, including simple diffusion, facilitated diffusion, and active transport. Water moves as driven by two forces: hydrostatic pressure and osmotic pressure.

Diffusion

Diffusion is the movement of molecules from an area of high concentration to low concentration (Fig. 16-4). Net movement of molecules stops when the concentrations are equal in both areas. It occurs in liquids, gases, and solids. Simple diffusion requires no external energy.

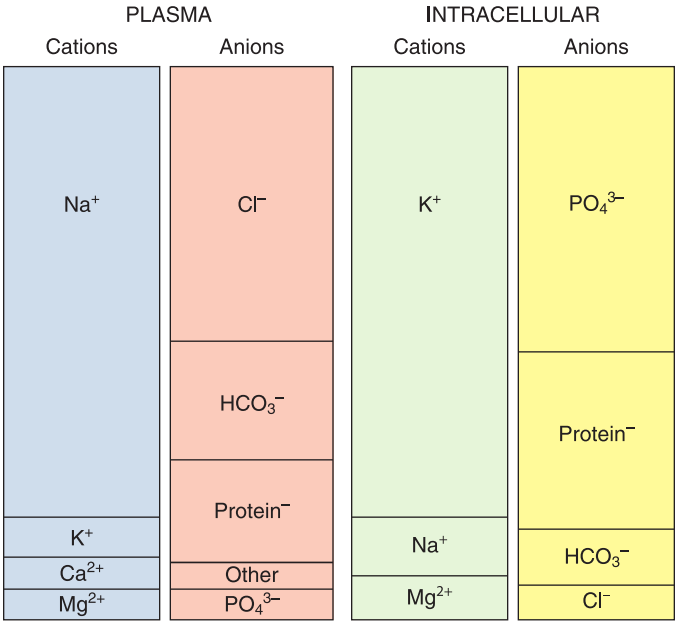

FIG. 16-3 The relative concentrations of the major cations and anions in the intracellular space and the plasma.

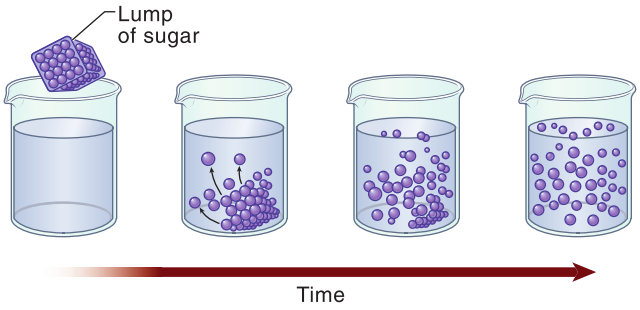

FIG. 16-4 Diffusion is the movement of molecules from an area of high concentration to an area of low concentration. Eventually the sugar molecules are evenly distributed.

TABLE 16-1 Normal Serum Electrolyte Values

Electrolyte	Reference Interval
Anions	
Bicarbonate (HCO_3^-)	22-26 mEq/L (22-26 mmol/L)
Chloride (Cl^-)	96-106 mEq/L (96-106 mmol/L)
Phosphate (PO_4^{3-})*	2.4-4.4 mg/dL (0.78-1.42 mmol/L)
Cations	
Potassium (K^+)	3.5-5.0 mEq/L (3.5-5.0 mmol/L)
Magnesium (Mg^{2+})	1.5-2.5 mEq/L (0.75-1.25 mmol/L)
Sodium (Na^+)	135-145 mEq/L (135-145 mmol/L)
Calcium (Ca^{2+}) (total)	8.6-10.2 mg/dL (2.15-2.55 mmol/L)
Calcium (ionized)	4.6-5.3 mg/dL (1.16-1.32 mmol/L)

*The majority of the phosphorus (P) in the body is found as phosphate (PO_4^{3-}). The terms are used interchangeably in this text.

Facilitated Diffusion

Facilitated diffusion involves the use of a protein carrier in the cell membrane. The protein carrier combines with a molecule, especially one too large to pass easily through the cell membrane, and assists in moving the molecule across the membrane from an area of high to low concentration. Like simple diffusion, facilitated diffusion is passive and requires no energy. An example of facilitated diffusion is glucose transport into the cell. The large glucose molecule must combine with a carrier molecule to be able to cross the cell membrane and enter most cells.

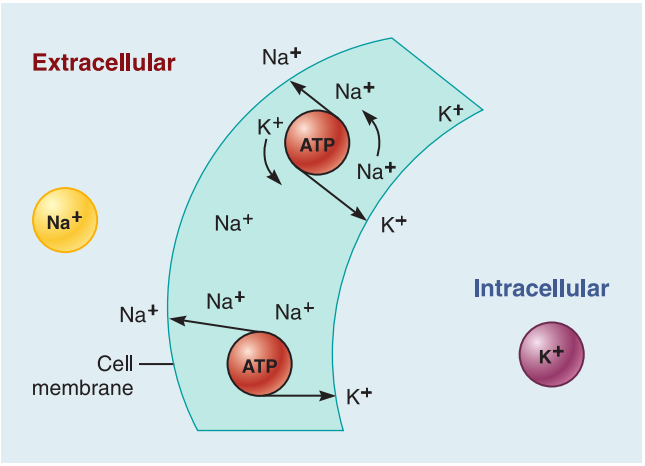

FIG. 16-5 Sodium-potassium pump. As sodium *(Na^+)* diffuses into the cell and potassium *(K^+)* diffuses out of the cell, an active transport system supplied with energy delivers Na^+ back to the extracellular compartment and K^+ to the intracellular compartment. *ATP,* Adenosine triphosphate.

Active Transport

Active transport is a process in which molecules move against the concentration gradient. External energy is required for this process. An example is the sodium-potassium pump. The concentrations of sodium and potassium differ greatly intracellularly and extracellularly (Fig. 16-3). To maintain this concentration difference, the cell uses active transport to move sodium out of the cell and potassium into the cell (Fig. 16-5). The energy source for this movement is adenosine triphosphate (ATP), which is made in the cell's mitochondria.

Osmosis

Osmosis is the movement of water "down" a concentration gradient, that is, from a region of low solute concentration to one of high solute concentration, across a semipermeable membrane. Osmosis requires no outside energy sources. It stops when the concentration differences disappear or when hydrostatic pressure builds and opposes any further movement of water. Imagine a chamber with two compartments separated by a semipermeable membrane, one that allows only the movement of water (Fig. 16-6). If you add albumin to one side, water will move from the less concentrated side (has more water) to the more concentrated side of the chamber water (has less water) until the concentrations are equal.

Whenever dissolved substances are contained in a space with a semipermeable membrane, they can pull water into the space by osmosis. The concentration of the solution determines the strength of the osmotic pull. The higher the concentration, the greater a solution's pull, or **osmotic pressure.** Osmotic pressure is measured in milliosmoles (mOsm). It may be expressed as either fluid osmolarity or fluid osmolality. Although you will often see the terms *osmolarity* and *osmolality* used interchangeably, they are different measurements. *Osmolarity* measures the total milliosmoles per liter of solution, or the concentration of molecules per volume of solution (mOsm/L). **Osmolality**

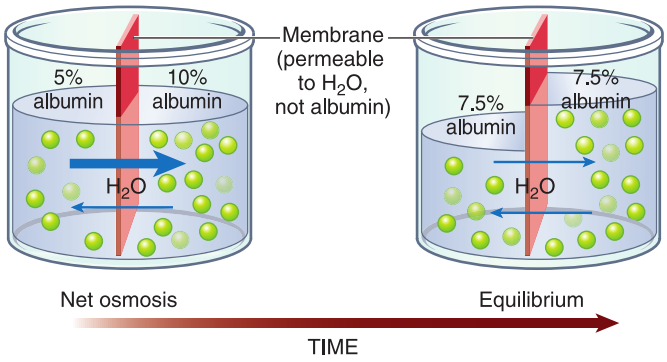

FIG. 16-6 Osmosis is the process of water movement through a semipermeable membrane from an area of low solute concentration to an area of high solute concentration.

measures the number of milliosmoles per kilogram of water, or the concentration of molecules per weight of water. Osmolality is the preferred measure to evaluate the concentration of plasma, urine, and body fluids.[1]

Measurement of Osmolality. Osmolality is nearly the same in the various body fluid spaces. Therefore measuring or estimating plasma osmolality is a useful way to assess the state of the body's water balance. Calculate the plasma osmolality using the following formula.[2]

$$\text{Plasma osmolality} = (2 \times \text{Na}) + \left(\frac{\text{BUN}}{2.8}\right) + \left(\frac{\text{Glucose}}{18}\right)$$

Normal plasma osmolality is between 280 and 295 mOsm/kg. A value greater than 295 mOsm/kg indicates that the concentration of solute is too great or the water content is too little. This condition is termed *water deficit.* A value less than 275 mOsm/kg indicates too little solute for the amount of water or too much water for the amount of solute. This condition is termed *water excess.* Both conditions are clinically significant.

Osmolality of urine can range from 100 to 1300 mOsm/kg. Urine osmolality depends on fluid intake, the amount of antidiuretic hormone (ADH) in circulation, and the renal response to ADH.

Osmotic Movement of Fluids. The osmolality of the fluid surrounding cells affects them. Fluids with the same osmolality as the cell interior are termed **isotonic.** Normally, ECF and ICF are isotonic to one another, so no net movement of water occurs.

Changes in the osmolality of ECF alter the volume of cells. Solutions in which the solutes are less concentrated than in the cells are termed **hypotonic** (hypoosmolar). If a cell is surrounded by hypotonic fluid, water moves into the cell, causing it to swell and possibly to burst. Fluids with solutes more concentrated than in cells, or an increased osmolality, are termed **hypertonic** (hyperosmolar). If hypertonic fluid surrounds a cell, water leaves the cell to dilute ECF; the cell shrinks and may eventually die (Fig. 16-7).

Hydrostatic Pressure

Hydrostatic pressure is the force of fluid in a compartment pushing against a cell membrane or vessel wall. In the blood vessels, hydrostatic pressure is the BP generated by the contraction of the heart. Hydrostatic pressure in the vascular system gradually decreases as the blood moves through the arteries until it is about 30 mm Hg in the capillary bed. At the capillary

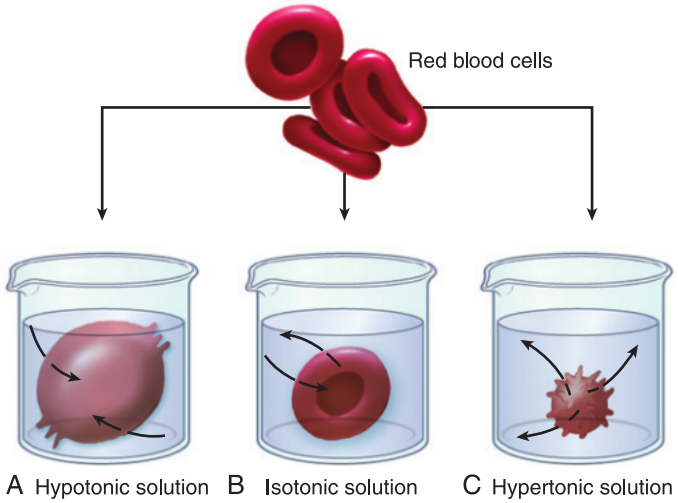

FIG. 16-7 Effects of water status on red blood cells. **A,** Hypotonic solution (H_2O excess) results in cellular swelling. **B,** Isotonic solution (normal H_2O balance) results in no change. **C,** Hypertonic solution (H_2O deficit) results in cellular shrinking.

level, hydrostatic pressure is the major force that pushes water out of the vascular system and into the interstitial space.

Oncotic Pressure

Oncotic pressure (colloidal osmotic pressure) is the osmotic pressure caused by plasma colloids in solution. The major colloids in the vascular system contributing to osmotic pressure are proteins, such as albumin. Plasma has substantial amounts of protein, while the interstitial space has very little. The plasma protein molecules attract water, pulling fluid from the tissue space to the vascular space. Under normal conditions, plasma oncotic pressure is about 25 mm Hg. The small amount of protein found in the interstitial space exerts an oncotic pressure of about 1 mm Hg.

FLUID MOVEMENT IN CAPILLARIES

As plasma flows through the capillary bed, four factors determine if fluid moves out of the capillary and into the interstitial space or if fluid moves back into the capillary from the interstitial space. The amount and direction of movement are determined by the interaction of (1) capillary hydrostatic pressure, (2) plasma oncotic pressure, (3) interstitial hydrostatic pressure, and (4) interstitial oncotic pressure.

Capillary hydrostatic pressure and interstitial oncotic pressure move water out of the capillaries. Plasma oncotic pressure and interstitial hydrostatic pressure move fluid into the capillaries. At the arterial end of the capillary, capillary hydrostatic pressure exceeds plasma oncotic pressure, and fluid moves into the interstitial space. At the venous end of the capillary, the capillary hydrostatic pressure is lower than plasma oncotic pressure, drawing fluid back into the capillary by the oncotic pressure created by plasma proteins (Fig. 16-8).

Fluid Shifts

If capillary or interstitial pressures change, fluid may abnormally shift from one compartment to another, resulting in edema or dehydration.

Shifts of Plasma to Interstitial Fluid. *Edema,* an accumulation of fluid in the interstitial space, occurs if venous hydrostatic

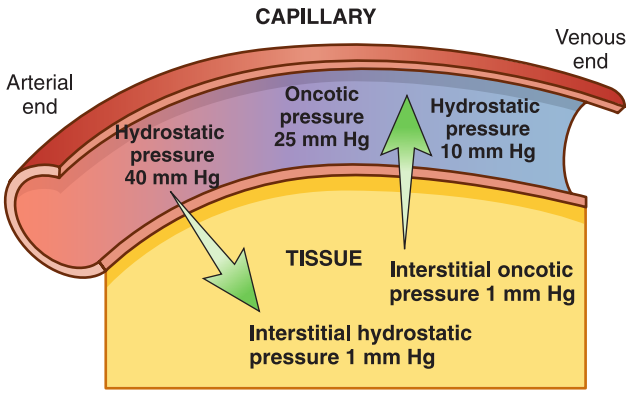

FIG. 16-8 Dynamics of fluid exchange between a capillary and tissue. An equilibrium exists between forces filtering fluid out of the capillary and forces absorbing fluid back into the capillary. Note that the hydrostatic pressure is greater at the arterial end of the capillary than at the venous end. The net effect of pressures at the arterial end of the capillary causes a movement of fluid into the tissue. At the venous end of the capillary, there is net movement of fluid back into the capillary.

pressure rises, plasma oncotic pressure decreases, or interstitial oncotic pressure rises. Edema may also develop if an obstruction of lymphatic outflow causes decreased removal of interstitial fluid.

Elevation of Venous Hydrostatic Pressure. Increasing the pressure at the venous end of the capillary inhibits fluid movement back into the capillary, which results in edema. Causes of increased venous pressure include fluid overload, heart failure, liver failure, obstruction of venous return to the heart (e.g., tourniquets, restrictive clothing, venous thrombosis), and venous insufficiency (e.g., varicose veins).

Decrease in Plasma Oncotic Pressure. Fluid remains in the interstitial space if the plasma oncotic pressure is too low to draw fluid back into the capillary. Low plasma protein content decreases oncotic pressure. This can result from excessive protein loss (renal disorders), deficient protein synthesis (liver disease), and deficient protein intake (malnutrition).

Elevation of Interstitial Oncotic Pressure. Trauma, burns, and inflammation can damage capillary walls and allow plasma proteins to accumulate in the interstitial space. This increases interstitial oncotic pressure, draws fluid into the interstitial space, and holds it there.

Shifts of Interstitial Fluid to Plasma. An increase in the plasma osmotic or oncotic pressure draws fluid into the plasma from the interstitial space. This could happen with administration of colloids, dextran, mannitol, or hypertonic solutions. Increasing the tissue hydrostatic pressure is another way of causing a shift of fluid into plasma. Wearing elastic compression gradient stockings or hose to decrease peripheral edema is a therapeutic application of this effect.

FLUID SPACING

Fluid spacing is a term used to describe the distribution of body water. *First spacing* describes the normal distribution of fluid in ICF and ECF compartments. *Second spacing* refers to an abnormal accumulation of interstitial fluid (i.e., edema). *Third spacing* occurs when excess fluid collects in the nonfunctional area between cells. This fluid is trapped where it is difficult or impossible for it to move back into the cells or blood vessels. Third spacing occurs with ascites; fluid leaking into the abdominal cavity with peritonitis or pancreatitis; and edema associated with burns, trauma, or sepsis.

TABLE 16-2 Normal Fluid Balance in the Adult

Intake	
Fluids	1200 mL
Solid food	1000 mL
Water from oxidation	300 mL
Total	2500 mL
Output	
Insensible loss (skin and lungs)	900 mL
In feces	100 mL
Urine	1500 mL
Total	2500 mL

REGULATION OF WATER BALANCE

A number of factors are involved in maintaining the finely tuned balance among water intake, use, and excretion. For proper fluid balance, an average healthy adult requires a daily water intake between 2000 and 3000 mL (Table 16-2). This amount replaces what is lost from the body in urinary output and insensible losses. Oral fluid intake accounts for most of the water intake. Water intake also includes water from food metabolism and water present in solid foods.

Insensible water loss, which is invisible vaporization from the lungs and skin, assists in regulating body temperature. Accelerated body metabolism, which occurs with increased body temperature and exercise, increases the amount of water lost and may result in the need for additional water replacement.

Do not confuse water loss through the skin with the vaporization of water excreted by sweat glands. Insensible perspiration causes only water loss. Excessive sweating *(sensible perspiration)* caused by exercise, fever, or high environmental temperatures may lead to large losses of water and electrolytes.

Hypothalamic-Pituitary Regulation

Water ingestion equals water loss in the individual who has free access to water, intact thirst and ADH mechanism, and normally functioning kidneys. A body fluid deficit or increase in plasma osmolality activates osmoreceptors in the hypothalamus, which stimulate thirst and the release of ADH from the posterior pituitary gland. ADH acts on the distal tubules and collecting ducts in the kidney by making them more permeable to water. The result is increased water reabsorption from the tubular filtrate into the blood and decreased excretion in the urine. Because ADH is only able to regulate how much water the body holds onto, an intact thirst mechanism is our main protection against developing dehydration or hyperosmolality. Thirst causes us to increase the amount of water we drink. Together these result in increased free water in the body, decreasing plasma osmolality and restoring fluid volume.

Many factors influence ADH secretion and thirst. Decreased BP, nausea, pain, hypoglycemia, and hypoxemia stimulate ADH release. In the postoperative patient, the stress response to surgery and receiving analgesics and anesthesia cause ADH release and decreased osmolality. The unconscious or cognitively impaired patient is at increased risk for fluid deficit and hyperosmolality because of an inability to express thirst and act

on it. A dry mouth will cause a person to drink, even when there is no body water deficit.

Renal Regulation

The primary function of the kidneys is to regulate fluid and electrolyte balance by adjusting urine volume and the excretion of most electrolytes (see Chapter 44). The kidneys filter the total plasma volume many times each day. In the average adult, the kidneys reabsorb 99% of this filtrate, producing around 1.5 L of urine per day. Under the influence of ADH, aldosterone, and other hormones, selective reabsorption and secretion of water and electrolytes in the renal tubules results in urine that is greatly different in composition and concentration from plasma. This process helps maintain normal plasma osmolality, electrolyte balance, blood volume, and acid-base balance.

With severely impaired renal function, the kidneys cannot maintain fluid and electrolyte balance. This condition results in edema, potassium and phosphorus retention, acidosis, and other electrolyte imbalances (see Chapter 46).

Adrenal Cortical Regulation

Glucocorticoids and mineralocorticoids secreted by the adrenal cortex help regulate water and electrolyte balance. The glucocorticoids (e.g., cortisol) primarily have an antiinflammatory effect and increase serum glucose levels, while the mineralocorticoids (e.g., aldosterone) enhance sodium retention and potassium excretion (Fig. 16-9). When sodium is reabsorbed, water follows because of osmotic changes.

Aldosterone is a mineralocorticoid with strong sodium-retaining and potassium-excreting capabilities. Decreased renal perfusion or decreased sodium in the distal portion of the renal tubule activates the renin-angiotensin-aldosterone system (RAAS), resulting in aldosterone secretion. In addition to the RAAS, increased serum potassium, decreased serum sodium, and adrenocorticotropic hormone (ACTH) stimulate aldosterone secretion. Aldosterone increases sodium and water reabsorption in the renal distal tubules, decreasing plasma osmolality and restoring fluid volume.

Cortisol is the most abundant glucocorticoid. In large doses, cortisol has both glucocorticoid (glucose-elevating and antiinflammatory) and mineralocorticoid (sodium-retention) effects. Normally cortisol secretion is in a diurnal or circadian pattern. Increased cortisol secretion occurs in response to physical and psychologic stress. This affects many body functions, including fluid and electrolyte balance (Fig. 16-10).

Cardiac Regulation

Natriuretic peptides, including atrial natriuretic peptide (ANP) and b-type natriuretic peptide (BNP), are hormones produced by cardiomyocytes. They are produced in response to increased atrial pressure (increased volume, such as occurs in heart failure) and high serum sodium levels. They are natural antagonists to the RAAS and suppress secretion of aldosterone, renin, and ADH, and the action of angiotensin II. In the renal tubules, peptides promote excretion of sodium and water, decreasing blood volume and BP.

Gastrointestinal Regulation

In addition to oral intake, the GI tract normally secretes around 8000 mL of digestive fluids each day. The GI tract normally reabsorbs most of this fluid, with only a small amount eliminated in feces. This is why diarrhea and vomiting, which prevent GI reabsorption of secreted fluid, can lead to significant fluid and electrolyte loss.

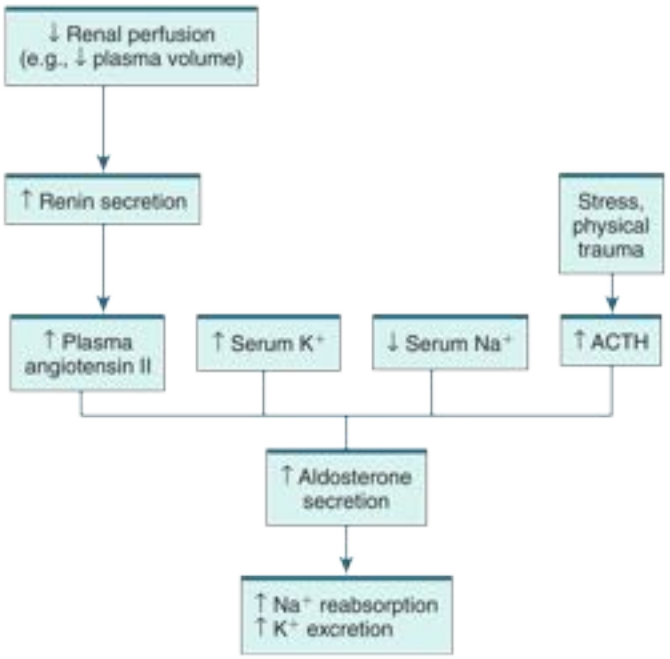

FIG. 16-9 Factors affecting aldosterone secretion. *ACTH*, Adrenocorticotropic hormone.

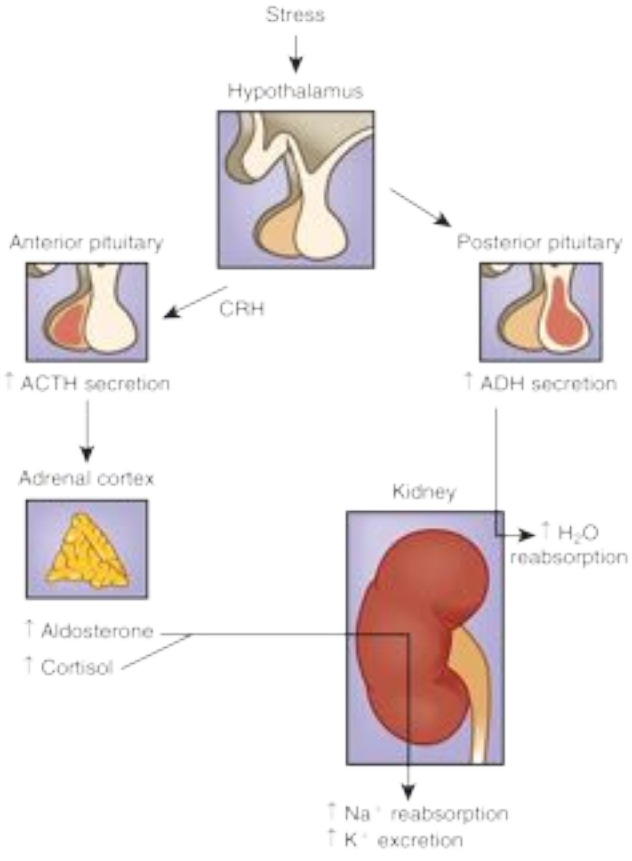

FIG. 16-10 Effects of stress on fluid and electrolyte balance. *ACTH*, Adrenocorticotropic hormone; *ADH*, antidiuretic hormone; *CRH*, corticotropin-releasing hormone.

Gerontologic Considerations: Fluid and Electrolytes

The older adult experiences normal physiologic changes that increase susceptibility to fluid and electrolyte imbalances. Structural changes to the kidneys and a decrease in the renal blood flow lead to decreased glomerular filtration rate and loss of the ability to concentrate urine and conserve water. Hormonal changes include a decrease in renin and aldosterone and an increase in ADH and ANP. The loss of subcutaneous tissue and thinning of the dermis lead to increased moisture lost through the skin and an inability to respond quickly to heat or cold.

Do not automatically attribute older patients' fluid and electrolyte problems to the natural processes of aging. Throughout this chapter are suggestions for adapting your assessment and nursing interventions when caring for the older adult.

FLUID VOLUME IMBALANCES

Fluid and electrolyte imbalances occur to some degree in most patients with a major illness or injury because illness disrupts normal homeostatic mechanisms. Illness or disease directly causes some fluid and electrolyte imbalances (e.g., burns, heart failure). Other times therapeutic measures (e.g., colonoscopy preparation, diuretics) cause or contribute to imbalances. Perioperative patients are at risk for developing fluid and electrolyte imbalances because of fluid restrictions, blood or fluid loss, and the stress of surgery.

Fluid and electrolyte imbalances are commonly classified as *deficits* or *excesses*. Although each imbalance is discussed separately in this chapter, it is common for more than one imbalance to occur in the same patient. For example, a patient with prolonged nasogastric suction will lose sodium, potassium, hydrogen, and chloride ions. These imbalances may result in sodium and potassium deficiencies, a fluid volume deficit, and metabolic alkalosis caused by the loss of HCl acid.

ECF volume deficit *(hypovolemia)* and ECF volume excess *(hypervolemia)* are common clinical conditions. ECF volume imbalances are usually accompanied by one or more electrolyte imbalances, particularly changes in the serum sodium level.

FLUID VOLUME DEFICIT

Fluid volume deficit can occur with abnormal loss of body fluids (e.g., diarrhea, vomiting, hemorrhage, polyuria), inadequate fluid intake, or a plasma to interstitial fluid shift. Though often used interchangeably, *fluid volume deficit* and *dehydration* are not the same. *Dehydration* refers to loss of pure water alone without a corresponding loss of sodium. Table 16-3 lists causes and clinical manifestations of fluid volume deficit.

Interprofessional Care

Managing fluid volume deficit involves correcting the underlying cause and replacing both water and any needed electrolytes. Replacement therapy depends on the severity and type of volume loss. In mild losses, oral rehydration may be used. If the deficit is more severe, volume is replaced with blood products or balanced IV solutions, such as isotonic (0.9%) sodium chloride or lactated Ringer's solution. The choice of fluid depends on the cause and patient's electrolyte status. For rapid volume replacement, 0.9% sodium chloride is preferred. Blood is administered when volume loss is due to blood loss.

TABLE 16-3 Extracellular Fluid Imbalances: Causes and Manifestations

ECF Volume Deficit	ECF Volume Excess
Causes	
• ↑ Insensible water loss or perspiration (high fever, heatstroke) • Diabetes insipidus • Osmotic diuresis • Hemorrhage • GI losses: vomiting, NG suction, diarrhea, fistula drainage • Overuse of diuretics • Inadequate fluid intake • Third-space fluid shifts: burns, pancreatitis	• Excessive isotonic or hypotonic IV fluids • Heart failure • Renal failure • Primary polydipsia • SIADH • Cushing syndrome • Long-term use of corticosteroids
Manifestations	
• Restlessness, drowsiness, lethargy, confusion • Thirst, dry mucous membranes • Cold clammy skin • Decreased skin turgor, ↓ capillary refill • Postural hypotension, ↑ pulse, ↓ CVP • ↓ Urine output, concentrated urine • ↑ Respiratory rate • Weakness, dizziness • Weight loss • Seizures, coma	• Headache, confusion, lethargy • Peripheral edema • Jugular venous distention • S3 heart sound • Bounding pulse, ↑ BP, ↑ CVP • Polyuria (with normal renal function) • Dyspnea, crackles, pulmonary edema • Muscle spasms • Weight gain • Seizures, coma

CVP, Central venous pressure; *ECF,* extracellular fluid; *SIADH,* syndrome of inappropriate antidiuretic hormone.

FLUID VOLUME EXCESS

Fluid volume excess may result from excess intake of fluids, abnormal retention of fluids (e.g., heart failure, renal failure), or a shift of fluid from interstitial fluid into plasma fluid. Weight gain is the most consistent manifestation of fluid volume excess. Table 16-3 lists causes and manifestations of fluid volume excess.

Interprofessional Care

Managing fluid volume excess involves treating the underlying cause and removing fluid without producing abnormal changes in the electrolyte composition or osmolality of ECF. Diuretics and fluid restriction are the primary forms of therapy. Some patients also need sodium restrictions. If the fluid excess leads to ascites or pleural effusion, an abdominal paracentesis or thoracentesis may be necessary.

❖ NURSING MANAGEMENT: FLUID VOLUME IMBALANCES

◆ Nursing Diagnoses

Nursing diagnoses and collaborative problems for the patient with a fluid imbalance include, but are not limited to, the following:

ECF volume deficit:

- Deficient fluid volume *related to* excess ECF losses or decreased fluid intake
- Decreased cardiac output *related to* excess ECF losses or decreased fluid intake

- Risk for impaired oral mucous membrane *related to* fluid volume deficit
- Potential complication: hypovolemic shock

ECF volume excess:

- Excess fluid volume *related to* increased water and/or sodium retention
- Impaired gas exchange *related to* water retention leading to pulmonary edema
- Risk for impaired skin integrity *related to* edema
- Activity intolerance *related to* increased water retention, fatigue, and weakness
- Disturbed body image *related to* altered body appearance secondary to edema
- Potential complications: pulmonary edema, ascites

Nursing Implementation

Daily Weights. Daily weights are the most accurate measure of volume status. An increase of 1 kg (2.2 lb) is equal to 1000 mL (1 L) of fluid retention, provided the person has maintained usual dietary intake or has not been on NPO status. Obtain the weight under standardized conditions. Weigh the patient at the same time every day, wearing the same garments and on the same carefully calibrated scale. Remove excess bedding and empty all drainage bags before the weighing. If items are present that are not there every day, such as bulky dressings or tubes, note this along with the weight.

Intake and Output. Intake and output records provide valuable information about fluid and electrolyte problems. An accurately recorded intake and output will identify sources of excessive intake or fluid losses. Intake should include oral, IV, and tube feedings and retained irrigants. Output includes urine, excess perspiration, wound or tube drainage, vomitus, and diarrhea. Estimate fluid loss from wounds and perspiration. Note the amount and color of the urine and measure the urine specific gravity. Readings greater than 1.025 indicate concentrated urine, while readings less than 1.010 indicate dilute urine.

Laboratory Findings. Monitor laboratory results when available and calculate the serum osmolality. The patient with a fluid volume deficit often has increased BUN, sodium, and hematocrit levels with increased plasma and urine osmolality. With fluid volume excess, the patient will have decreased BUN, sodium, and hematocrit levels with decreased plasma and urine osmolality.

Cardiovascular Care. Monitor vital signs and perform a thorough cardiovascular assessment as needed. Changes in BP, central venous pressure, pulse force, and jugular venous distention reflect ECF volume imbalances. In fluid volume excess, the pulse is full, bounding, and not easily obliterated. Increased volume causes distended neck veins (jugular venous distention), increased central venous pressure, and high BP. Auscultate heart sounds, being alert for the presence of an S3.

In mild to moderate fluid volume deficit, sympathetic nervous system compensation increases the heart rate and results in peripheral vasoconstriction in an effort to maintain BP within normal limits. Pulses may be weak and thready. Assess for orthostatic changes. A change in position from lying to sitting or standing may elicit a decrease in BP or a further increase in the heart rate (orthostatic hypotension). In more severe deficits, hypotension may be present.

Respiratory Care. Monitor pulse oximetry and auscultate lung sounds as needed. ECF excess can result in pulmonary congestion and pulmonary edema, as increased hydrostatic pressure in the pulmonary vessels forces fluid into the alveoli. The patient will experience shortness of breath and moist crackles on auscultation. The patient with ECF deficit will demonstrate an increased respiratory rate because of decreased tissue perfusion and resultant hypoxia. Administer oxygen as ordered.

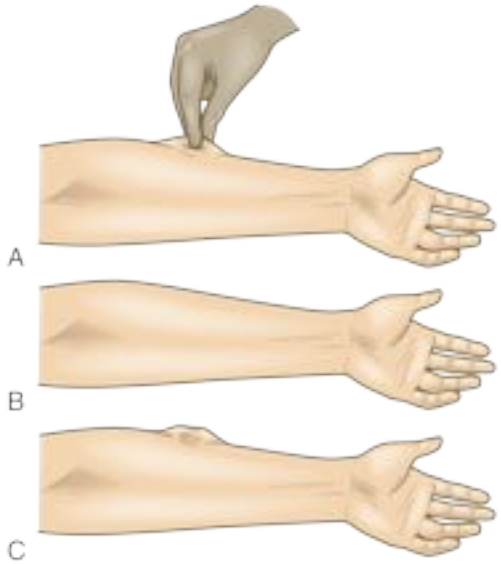

FIG. 16-11 Assessment of skin turgor. **A** and **B**, When normal skin is pinched, it resumes shape in seconds. **C**, If the skin remains wrinkled for 20 to 30 seconds, the patient has poor skin turgor.

Patient Safety. The patient with fluid volume deficit is at risk for falls because of orthostatic hypotension, muscle weakness, and changes in level of consciousness. Assess level of consciousness, gait, and muscle strength. Implement fall precautions. If orthostatic hypotension is present, teach the patient to change positions slowly when rising from a bed or chair. Place alarm monitors on patients who are confused and try to get out of bed without assistance.

Skin Care. Examine the skin for turgor and mobility. Normally a fold of skin, when pinched, will readily move and, on release, rapidly return to its former position. In ECF volume deficit, skin turgor is diminished, and there is a lag in the pinched skinfold's return to its original state (referred to as *tenting*). Skin areas over the sternum, abdomen, and anterior forearm are the usual sites for evaluation of tissue turgor (Fig. 16-11). In older people, decreased skin turgor is less predictive of fluid deficit because of the loss of tissue elasticity.[1]

In mild fluid deficits, the skin may appear warm, dry, and wrinkled. These signs may be difficult to evaluate in the older adult because the patient's skin may be normally dry, wrinkled, and nonelastic. In more severe deficits, the skin may be cool and moist if there is vasoconstriction to compensate for the decreased fluid volume. Oral mucous membranes will be dry, the tongue may be furrowed, and the individual often complains of thirst. Routine oral care is critical for the comfort of a patient who is dehydrated or on a fluid restriction.

Edematous skin may feel cool because of fluid accumulation and a decrease in blood flow secondary to the pressure of the fluid. The fluid can stretch the skin, causing it to feel taut and hard. Assess edema by pressing with a thumb or forefinger over the edematous area. A grading scale is used to standardize the description if an indentation (ranging from 1+ [slight edema; 2-mm indentation] to 4+ [pitting edema; 8-mm indentation]) remains when pressure is released. Evaluate for edema in areas

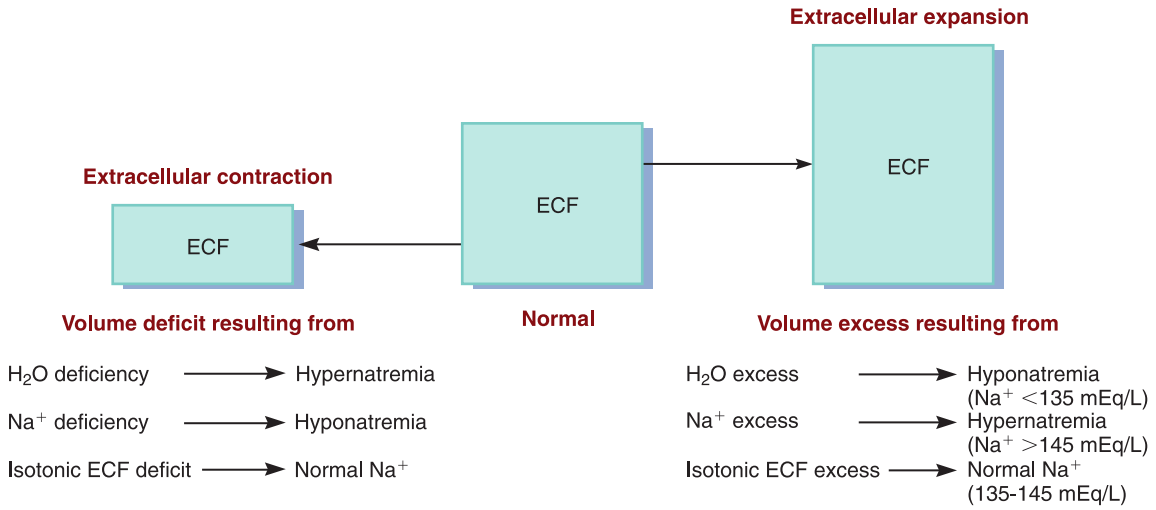

FIG. 16-12 Differential assessment of extracellular fluid (ECF) volume.

where soft tissues overlie a bone, particularly the tibia, fibula, and sacrum.

Good skin care is important. Protect tissues from extremes of heat and cold, prolonged pressure, and trauma. Frequent skin care and changes in position will prevent skin breakdown. Elevate edematous extremities to promote venous return and fluid reabsorption. Dehydrated skin needs frequent care without the use of soap. Applying moisturizing creams or oils increases moisture retention and stimulates circulation.

◆ **Fluid Therapy.** Administer IV fluids as ordered. Carefully monitor the rates of infusion of IV fluid solutions, especially when large volumes of fluid are being given. This is especially true in patients with cardiac, renal, or neurologic problems.

In the presence of fluid volume deficit, you can use several interventions to maintain adequate oral intake. Assess the patient's ability to obtain adequate fluids independently, express thirst, and swallow effectively. Fluids should be easily accessible. Provide a variety of fluids that the patient likes. Serve fluids at a temperature preferred by the patient. Offer fluids every 1 to 2 hours and at select times, such as when administering medications. Remind the patient to finish all drinks.

If the patient is choosing to limit intake to decrease nocturia or incontinence, make it easier for the patient to reach the toilet when needed.[3] Assist those with physical limitations, such as arthritis, to open and hold containers. Involve the dietitian, speech therapist, or occupation therapist for help with patients with dysphagia or physical limitations.

SODIUM IMBALANCES

Sodium, the main cation of ECF, plays a major role in maintaining the concentration and volume of ECF and influencing water distribution between ECF and ICF. Sodium has an important role in the generation and transmission of nerve impulses, muscle contractility, and regulation of acid-base balance.

The serum sodium level reflects the ratio of sodium to water, not necessarily the amount of sodium in the body. Changes in the serum sodium level can reflect a primary water imbalance, primary sodium imbalance, or combination of the two. Sodium imbalances are typically associated with imbalances in ECF volume (Figs. 16-12 and 16-13). Because sodium is the primary determinant of ECF osmolality, sodium imbalances are typically associated with parallel changes in osmolality.

The GI tract absorbs sodium from foods. Typically, daily intake of sodium far exceeds the body's daily requirements. Sodium leaves the body through urine, sweat, and feces. The

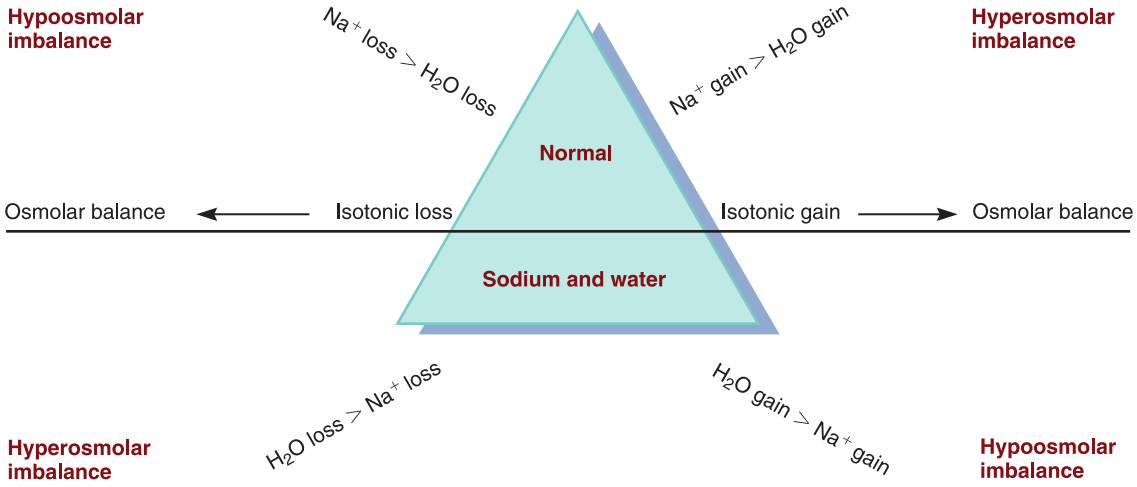

FIG. 16-13 Isotonic gains and losses affect mainly the extracellular fluid (ECF) compartment with little or no water movement into the cells. Hypertonic imbalances cause water to move from inside the cell into ECF to dilute the concentrated sodium, causing cell shrinkage. Hypotonic imbalances cause water to move into the cell, causing cell swelling.

kidneys primarily regulate sodium balance. The kidneys control ECF sodium concentration by excreting or retaining water under the influence of ADH. Aldosterone plays a smaller role in sodium regulation by promoting sodium reabsorption from the renal tubules.

HYPERNATREMIA

Hypernatremia (elevated serum sodium) may occur with inadequate water intake, excess water loss or, rarely, sodium gain. Because sodium is the major determinant of ECF osmolality, hypernatremia causes hyperosmolality. ECF hyperosmolality causes water to move out of the cells to restore equilibrium, leading to cellular dehydration. As discussed earlier, the primary protection against the development of hyperosmolality is thirst. Hypernatremia is not a problem in an alert person who has access to water, can sense thirst, and is able to swallow. Hypernatremia secondary to water deficiency is often the result of an impaired level of consciousness or an inability to obtain fluids.

Several clinical states can produce hypernatremia from water loss (Table 16-4). A deficiency in the synthesis or release of ADH from the posterior pituitary gland (central diabetes insipidus) or a decrease in kidney responsiveness to ADH (nephrogenic diabetes insipidus) can result in profound diuresis, producing a water deficit and hypernatremia. Hyperosmolality with osmotic diuresis can result from hyperglycemia associated with uncontrolled diabetes mellitus or administering concentrated hyperosmolar tube feedings.

Excess sodium intake with inadequate water intake can also lead to hypernatremia. Examples of sodium gain include IV administration of hypertonic saline or sodium bicarbonate, use of sodium-containing drugs, excessive oral intake of sodium (e.g., ingestion of seawater), and *primary aldosteronism* (hypersecretion of aldosterone) caused by a tumor of the adrenal glands.

Clinical Manifestations

The manifestations of hypernatremia are primarily the result of water shifting out of cells into ECF with resultant dehydration and shrinkage of cells (Table 16-4). Dehydration of brain cells results in alterations in mental status, ranging from agitation, restlessness, confusion, and lethargy to coma.[3] If there is any accompanying ECF volume deficit, manifestations such as postural hypotension, tachycardia, and weakness occur.

❖ NURSING AND INTERPROFESSIONAL MANAGEMENT: HYPERNATREMIA

◆ Nursing Diagnoses

Nursing diagnoses and collaborative problems for the patient with hypernatremia include, but are not limited to, the following:

- Risk for electrolyte imbalance *related to* inadequate water intake, excess sodium intake and/or water loss injury *related to* altered sensorium and seizures
- Risk for fluid volume deficit *related to* inadequate water intake and/or water loss
- Risk for injury *related to* altered sensorium and seizures
- Potential complications: seizures and coma

◆ Nursing Implementation

Managing hypernatremia depends on the underlying cause and the patient's volume status. In primary water deficit, fluid replacement is provided either orally or IV with isotonic such as 0.9% sodium chloride.[2] If the problem is sodium excess, expect diluting the high sodium concentration with sodium-free IV fluids, such as 5% dextrose in water, and promoting sodium excretion with diuretics. Dietary sodium intake is often restricted. If the patient has an altered sensorium or is having seizures, initiate seizure precautions.

Monitor serum sodium levels, serum osmolality, and the patient's response to therapy. The serum sodium level should not decrease by more than 8 to 15 mEq/L in an 8-hour period.[2] Quickly reducing levels can cause a rapid shift of water back into the cells, resulting in cerebral edema and neurologic

TABLE 16-4 Sodium Imbalances: Causes and Manifestations

Hypernatremia (Na^+ >145 mEq/L [mmol/L])	Hyponatremia (Na^+ <135 mEq/L [mmol/L])
Causes	
Excessive Sodium Intake	***Excessive Sodium Loss***
• IV fluids: hypertonic NaCl, excessive isotonic NaCl, IV sodium bicarbonate • Hypertonic tube feedings without water supplements • Near-drowning in salt water	• *GI losses:* diarrhea, vomiting, fistulas, NG suction • *Renal losses:* diuretics, adrenal insufficiency, Na^+ wasting renal disease • *Skin losses:* burns, wound drainage
Inadequate Water Intake	***Inadequate Sodium Intake***
• Unconscious or cognitively impaired individuals	• Fasting diets
Excessive Water Loss (↑ sodium concentration)	***Excessive Water Gain (↓ sodium concentration)***
• ↑ Insensible water loss (high fever, heatstroke, prolonged hyperventilation) • Osmotic diuretic therapy • Diarrhea	• Excessive hypotonic IV fluids • Primary polydipsia
Diseases	***Diseases***
• Diabetes insipidus • Primary hyperaldosteronism • Cushing syndrome • Uncontrolled diabetes mellitus	• SIADH • Heart failure • Primary hypoaldosteronism • Cirrhosis
Manifestations	
Hypernatremia With Decreased ECF Volume	***Hyponatremia With Decreased ECF Volume***
• Restlessness, agitation, lethargy, seizures, coma • Intense thirst, dry swollen tongue, sticky mucous membranes • Postural hypotension, ↓ CVP, weight loss, ↑ pulse • Weakness, muscle cramps	• Irritability, apprehension, confusion, dizziness, personality changes, tremors, seizures, coma • Dry mucous membranes • Postural hypotension, ↓ CVP, ↓ jugular venous filling, ↑ pulse, thready pulse • Cold and clammy skin
Hypernatremia With Normal or Increased ECF Volume	***Hyponatremia With Normal or Increased ECF Volume***
• Restlessness, agitation, twitching, seizures, coma • Intense thirst, flushed skin • Weight gain, peripheral and pulmonary edema, ↑ BP, ↑ CVP	• Headache, apathy, confusion, muscle spasms, seizures, coma • Nausea, vomiting, diarrhea, abdominal cramps • Weight gain, ↑ BP, ↑ CVP

CVP, Central venous pressure; *ECF,* extracellular fluid; *SIADH,* syndrome of inappropriate antidiuretic hormone.

complications. This risk is greatest in the patient who developed hypernatremia over several days or longer.

HYPONATREMIA

Hyponatremia (low serum sodium) may result from a loss of sodium-containing fluids, water excess in relation to the amount of sodium (dilutional hyponatremia), or a combination of both (Table 16-4). Hyponatremia is usually associated with ECF hypoosmolality from the excess water. To restore balance, fluid shifts out of the ECF and into the cells, leading to cellular edema.

Common causes of hyponatremia from loss of sodium-rich body fluids include draining wounds, diarrhea, vomiting, and primary adrenal insufficiency. Inappropriate use of sodium-free or hypotonic IV fluids causes hyponatremia from water excess. This may occur in patients after surgery or major trauma or administering fluids to patients with renal failure. Patients with psychiatric disorders may have an excessive water intake. Syndrome of inappropriate antidiuretic hormone secretion (SIADH) results in dilutional hyponatremia caused by abnormal retention of water. See Chapter 49 for a discussion of the causes of SIADH.

Clinical Manifestations

The manifestations of hyponatremia are due to cellular swelling and first appear in the central nervous system (CNS). Mild hyponatremia has minor, nonspecific neurologic symptoms including headache, irritability, and difficulty concentrating. More severe hyponatremia can cause confusion, vomiting, seizures, and even coma. If hyponatremia is severe and develops rapidly, irreversible neurologic damage or death from brain herniation can occur.[4]

NURSING AND INTERPROFESSIONAL MANAGEMENT: HYPONATREMIA

Nursing Diagnoses

Nursing diagnoses and collaborative problems for the patient with hyponatremia include, but are not limited to, the following:

- Risk for electrolyte imbalance *related to* excess sodium loss and/or excess water intake or retention
- Risk for injury *related to* altered sensorium and decreased level of consciousness
- Risk for acute confusion *related to* electrolyte imbalance
- Potential complication: severe neurologic changes

Nursing Implementation

Managing hyponatremia from fluid loss includes replacing fluid using isotonic sodium-containing solutions, encouraging oral intake, and withholding all diuretics.[2] In mild hyponatremia caused by water excess, fluid restriction is often the only treatment. The aim of the restriction is 500 mL less than the previous 24-hour urine output.[4] Loop diuretics and demeclocycline may be given. If hyponatremia is acute or more serious, small amounts of IV hypertonic saline solution (3% sodium chloride) can restore the serum sodium level while the body is returning to a normal water balance.

Vasopressor receptor antagonists (drugs that block the activity of ADH), are used to treat patients who cannot tolerate fluid restrictions or have more severe symptoms.[4] These drugs include conivaptan (Vaprisol) and tolvaptan (Samsca). Conivaptan is given IV to hospitalized patients with severe hyponatremia from water excess. Tolvaptan is an oral preparation used to treat hyponatremia from heart failure and SIADH.

Monitor serum sodium levels and the patient's response to therapy to avoid rapid or overcorrection. The level should not increase by more than 8 to 12 mEq/L in the first 24 hours.[4] Quickly increasing levels of sodium can cause osmotic demyelination syndrome with permanent damage to nerve cells in the brain. An accurate urine output record is essential. The patient may need a urinary catheter placed if unable to assist with monitoring output. If the patient has an altered sensorium or is having seizures, initiate seizure precautions.

POTASSIUM IMBALANCES

Potassium is the major ICF cation, with 98% of the body potassium being intracellular. For example, potassium concentration within muscle cells is around 140 mEq/L; potassium concentration in ECF is 3.5 to 5.0 mEq/L. The sodium-potassium pump in cell membranes maintains this concentration difference by pumping potassium into the cell and sodium out. Insulin helps by stimulating the sodium-potassium pump.

Because the ratio of ECF potassium to ICF potassium is the major factor in the resting membrane potential of nerve and muscle cells, neuromuscular and cardiac function are often affected by potassium imbalances. Potassium is involved with regulating intracellular osmolality and promoting cellular growth. It is required for glycogen to be deposited in muscle and liver cells. Potassium also plays a role in acid-base balance (discussed in the section on Acid-Base Regulation later in this chapter).

Diet is the source of potassium. The typical Western diet contains roughly 50 to 100 mEq of potassium daily, mainly from protein-rich foods and many fruits and vegetables. Many salt substitutes used in low-sodium diets contain substantial potassium. Patients may receive potassium from parenteral sources, including IV fluids; transfusions of stored, hemolyzed blood; and medications (e.g., potassium penicillin).

The kidneys are the primary route for potassium loss, eliminating about 90% of the daily potassium intake. Potassium excretion depends on the serum potassium level, urine output, and renal function. When serum potassium is high, urine potassium excretion increases and when serum levels are low, excretion decreases. Large urine output can cause excess potassium loss. Impaired kidney function can cause potassium retention. There is an inverse relationship between sodium and potassium reabsorption in the kidneys. Factors that cause sodium retention (e.g., low blood volume, hyponatremia, aldosterone secretion) cause potassium excretion.

HYPERKALEMIA

Hyperkalemia (high serum potassium) may result from impaired renal excretion, a shift of potassium from ICF to ECF, a massive intake of potassium, or a combination of these factors (Table 16-5). The most common cause of hyperkalemia is renal failure. Adrenal insufficiency with a subsequent aldosterone deficiency leads to potassium retention. Factors that cause potassium to move from ICF to ECF include acidosis, massive cell destruction (as in burn or crush injury, tumor lysis, severe infections), and intense exercise. In metabolic acidosis, potassium ions shift

TABLE 16-5 Potassium Imbalances: Causes and Manifestations

Hyperkalemia (K^+ >5.0 mEq/L [mmol/L])	Hypokalemia (K^+ <3.5 mEq/L [mmol/L])
Causes	
Excess Potassium Intake	***Potassium Loss***
• Excessive or rapid parenteral administration • Potassium-containing drugs (e.g., potassium penicillin) • Potassium-containing salt substitute	• *GI losses:* diarrhea, vomiting, fistulas, NG suction, ileostomy drainage • *Renal losses:* diuretics, hyperaldosteronism, magnesium depletion • *Skin losses:* diaphoresis • Dialysis
Shift of Potassium Out of Cells	***Shift of Potassium Into Cells***
• Acidosis • Tissue catabolism (e.g., fever, crush injury, sepsis, burns) • Intense exercise • Tumor lysis syndrome	• Increased insulin release (e.g., IV dextrose load) • Insulin therapy (e.g., with diabetic ketoacidosis) • Alkalosis • ↑ Epinephrine (e.g., stress)
Failure to Eliminate Potassium	***Lack of Potassium Intake***
• Renal disease • Adrenal insufficiency • *Medications:* Angiotensin II receptor blockers, ACE inhibitors, heparin, potassium-sparing diuretics, NSAIDs	• Starvation • Diet low in potassium • Failure to include potassium in parenteral fluids if NPO
Clinical Manifestations	
• Fatigue, irritability • Muscle weakness, cramps • Loss of muscle tone • Paresthesias, decreased reflexes • Abdominal cramping, diarrhea, vomiting • Confusion • Irregular pulse • Tetany	• Fatigue • Muscle weakness, leg cramps • Soft, flabby muscles • Paresthesias, decreased reflexes • Constipation, nausea, paralytic ileus • Shallow respirations • Weak, irregular pulse • Hyperglycemia
ECG Changes	***ECG Changes***
• Tall, peaked T wave • Prolonged PR interval • ST segment depression • Widening QRS • Loss of P wave • Ventricular fibrillation • Ventricular standstill	• Flattened T wave • Presence of U wave • ST segment depression • Prolonged QRS • Peaked P wave • Ventricular dysrhythmias • First- and second-degree heart block

ACE, Angiotensin-converting enzyme.

from ICF to ECF in exchange for hydrogen ions moving into the cell.

Digoxin-like drugs and β-adrenergic blockers (e.g., propranolol) can impair entry of potassium into cells, resulting in a higher ECF potassium concentration. Several drugs, such as heparin, potassium-sparing diuretics, angiotensin II receptor blockers (e.g., losartan), and angiotensin-converting enzyme (ACE) inhibitors (e.g., lisinopril), can contribute to hyperkalemia by reducing the kidney's ability to excrete potassium.[5]

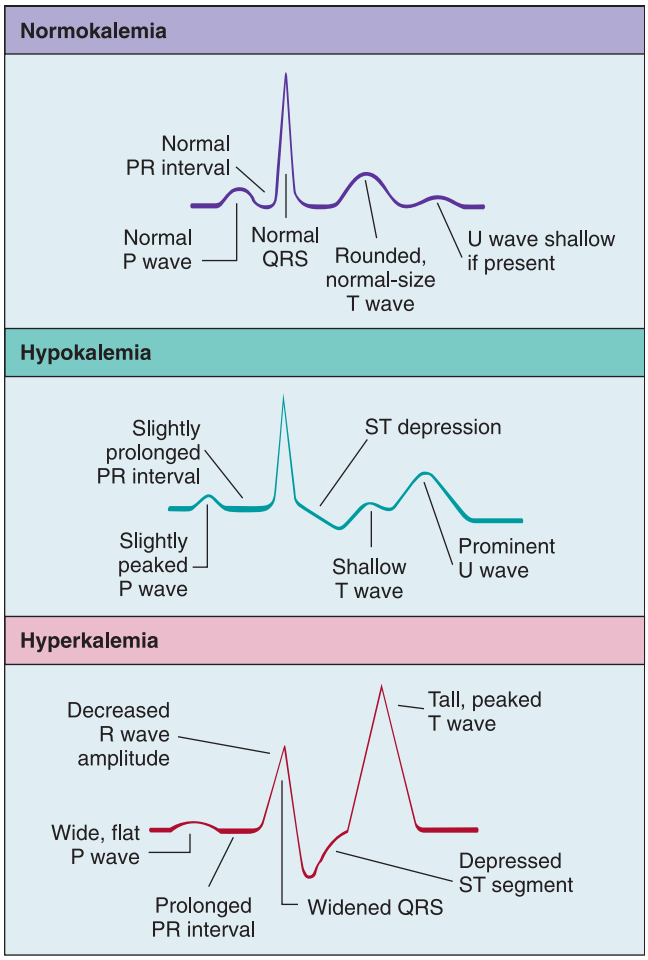

FIG. 16-14 ECG changes associated with alterations in potassium status.

Clinical Manifestations

The increased potassium concentration outside the cell changes the normal ECF and ICF ratio, resulting in increased cell excitability and changes in impulse transmission to the nerves and muscles. The most clinically significant problems are the disturbances in cardiac conduction. The initial finding is tall, peaked T waves. As potassium increases, cardiac depolarization decreases, leading to loss of P waves, a prolonged PR interval, ST segment depression, and widening QRS complex (Fig. 16-14). Heart block, ventricular fibrillation, or cardiac standstill may occur. Elevated potassium may cause failure to capture in a patient who has a pacemaker.[6]

The patient may experience fatigue, confusion, tetany, muscle cramps, paresthesias, and weakness. As potassium increases, loss of muscle tone and weakness or paralysis of other skeletal muscles, including the respiratory muscles can occur, leading to respiratory arrest. Abdominal cramping, vomiting, and diarrhea occur from hyperactivity of gastrointestinal smooth muscles.

❖ NURSING AND INTERPROFESSIONAL MANAGEMENT: HYPERKALEMIA

◆ Nursing Diagnoses

Nursing diagnoses and collaborative problems for the patient with hyperkalemia include, but are not limited to, the following.

- Risk for electrolyte imbalance *related to* excessive retention or cellular release of potassium
- Risk for activity intolerance *related to* muscle weakness
- Risk for injury *related to* muscle weakness and seizures
- Potential complication: dysrhythmias

Nursing Implementation

Management of hyperkalemia consists of the following:

1. Eliminate oral and parenteral potassium intake (Table 46-11).
2. Increase elimination of potassium. This may be accomplished with loop or thiazide diuretics, dialysis, patiromer (Veltassa), and/or sodium polystyrene sulfonate (Kayexalate). Kayexalate, given orally or rectally, is used acutely to bind potassium in the bowel. Each gram of the drug removes roughly 1 mEq of potassium. Patients with renal failure require hemodialysis.[6] Patiromer, given orally, also binds potassium in the GI tract. It takes several hours to days to work and is best for patients with chronic hyperkalemia. Patiromer binds with many other oral drugs and reduces their effectiveness. You should not give it within 6 hours of other drugs.
3. Force potassium from ECF to ICF. A combination of IV regular insulin and a β-adrenergic agonist stimulates the sodium-potassium pump, shifting potassium into cells. Using these drugs together is more effective than using either alone. Both metered dose inhalers and nebulized β-adrenergic agonists (e.g., nebulized albuterol) are equally effective.[5] IV sodium bicarbonate is an option if the patient is acidotic.
4. Stabilize cardiac membranes. IV calcium chloride or calcium gluconate reverse the membrane potential effects of the elevated ECF potassium and restore the electrical gradient. This protects the patient from life-threatening dysrhythmias.[6]

When the potassium elevation is mild and the kidneys are functioning, it may be sufficient to (1) withhold potassium from the diet and IV sources and (2) increase renal potassium elimination by administering fluids and loop or thiazide diuretics. Patients with severe hyperkalemia or symptomatic patients should receive one of the treatments to force potassium into cells.

Provide continuous ECG monitoring of all patients with clinically significant hyperkalemia to detect dysrhythmias and monitor the effects of therapy. The patient experiencing dangerous cardiac dysrhythmias should receive IV calcium immediately. Monitor BP because rapidly administering calcium can cause hypotension. When administering insulin, monitor for hypoglycemia and give glucose as needed.

HYPOKALEMIA

Hypokalemia (low serum potassium) can result from an increased loss of potassium, an increased shift of potassium from ECF to ICF, or rarely from deficient dietary potassium intake. The most common causes of hypokalemia are abnormal losses from either the kidneys or GI tract. GI tract losses are associated with diarrhea, laxative misuse, vomiting, and ileostomy drainage. Renal losses occur when a patient is diuresing or has a low magnesium level. Low plasma magnesium levels stimulate renin and aldosterone release, resulting in potassium excretion.

Among the factors causing potassium to move from ECF to ICF are insulin therapy, especially in conjunction with diabetic ketoacidosis, and β-adrenergic stimulation (catecholamine release in stress, coronary ischemia). Alkalosis can cause a shift of potassium into cells in exchange for hydrogen, lowering potassium in ECF and causing symptomatic hypokalemia.

Clinical Manifestations

Hypokalemia alters the resting membrane potential, resulting in hyperpolarization (an increased negative charge within the cell) and impaired muscle contraction. Therefore the manifestations of hypokalemia involve changes in cardiac and muscle function (Table 16-5).

The most serious clinical problems are cardiac changes, including impaired repolarization, resulting in a flattened T wave, depressed ST segment, and the presence of a U wave. The P waves peak and the QRS complex is prolonged (Fig. 16-14). There is an increased incidence of heart block and potentially lethal ventricular dysrhythmias.

As with hyperkalemia, skeletal muscle weakness and paresthesias may occur. Severe hypokalemia can cause paralysis. This usually involves the extremities but can involve the respiratory muscles, leading to shallow respirations and respiratory arrest. Alterations in smooth muscle function may lead to decreased GI motility (e.g., constipation, paralytic ileus). Finally, hypokalemia impairs insulin secretion, leading to glucose intolerance and hyperglycemia.

NURSING AND INTERPROFESSIONAL MANAGEMENT: HYPOKALEMIA

Nursing Diagnoses

Nursing diagnoses and collaborative problems for the patient with hypokalemia include, but are not limited to, the following:

- Risk for electrolyte imbalance *related to* excess potassium loss
- Risk for activity intolerance *related to* muscle weakness
- Risk for injury *related to* muscle weakness and hyporeflexia
- Potential complication: dysrhythmias

Nursing Implementation

Managing hypokalemia consists of oral or IV potassium chloride (KCl) supplements and increased dietary intake of potassium. Consuming potassium-rich foods can usually correct mild hypokalemia. Clinically significant hypokalemia requires administering oral or IV KCl.

SAFETY ALERT Administering IV KCl

- IV KCl must always be diluted and never given in concentrated amounts.
- Never give KCl via IV push or as a bolus.
- Invert IV bags containing KCl several times to ensure even distribution in the bag.
- Do not add KCl to a hanging IV bag to prevent giving a bolus dose.

IV KCl infusion rates should not exceed 10 mEq/hr unless the patient is in a critical care setting with continuous ECG monitoring and central line access for administration.[5] IV KCl must be given by infusion pump to ensure correct administration rate. Because KCl is irritating to the vein, assess IV sites at least hourly for phlebitis and infiltration. Infiltration can cause necrosis and sloughing of the surrounding tissue.

TABLE 16-6 Patient & Caregiver Teaching

Prevention of Hypokalemia

Include the following instructions when teaching at-risk patients how to prevent hypokalemia:

1. For all patients at risk:
   - Report the signs and symptoms of hypokalemia (Table 16-5) to the HCP.
   - Have serum potassium levels checked regularly.
   - Regularly include foods high in potassium in your diet (see Table 46-11).
   - Consume alcohol in moderation only.
   - Avoid consuming large amounts of licorice.
2. For patients taking oral potassium supplements:
   - Take the medication as prescribed to prevent overdosing.
   - Take the supplement with a full glass of water. Do not crush or chew tablets.

Patients who are critically ill and those at risk for hypokalemia should have continuous ECG monitoring to detect cardiac changes. Monitor serum potassium levels and urine output as appropriate.

CHECK YOUR PRACTICE

You are taking care of a 78-yr-old female patient with heart failure. She is taking furosemide (Lasix) and digitalis.

- What should you be alert for in a patient who takes both of these drugs?

KCl is usually given only if the urine output is at least 0.5 mL/kg of body weight per hour. Because patients on digoxin therapy have an increased risk of toxicity if their serum potassium level is low, monitor the patient for digitalis toxicity.

Teach patients ways to prevent hypokalemia (Table 16-6). Patients at risk for hypokalemia should have regular serum potassium levels monitored. Teach the patient taking digitalis to report signs and symptoms of digoxin toxicity immediately to the HCP.

CALCIUM IMBALANCES

Calcium is necessary for many metabolic processes. It is the major cation in bones and teeth. Calcium plays a role in blood clotting, transmission of nerve impulses, myocardial contractions, and muscle contractions. The source of calcium is dietary intake. Calcium absorption requires the active form of vitamin D. Vitamin D is obtained from foods or made in the skin by the action of sunlight on cholesterol.

The total body content of calcium is about 1200 g. The bones contain 99% of the body's calcium; the remainder is in plasma and body cells. Of the calcium in plasma, 50% is bound to plasma proteins, primarily albumin; 40% is in a free or ionized form, and the remainder is found bound with phosphate, citrate, or carbonate. The ionized or free calcium is biologically active. The serum pH influences how much calcium is ionized or bound to albumin. A decreased plasma pH (acidosis) decreases calcium binding to albumin, leading to more ionized calcium. An increased plasma pH (alkalosis) increases calcium binding, leading to decreased ionized calcium.

Serum calcium levels reflect the total level of all forms of plasma calcium. Serum albumin levels affect the interpretation of total calcium levels. Total calcium values increase or decrease directly with serum albumin levels. Ionized calcium levels are measured using special laboratory techniques or calculated using a formula. Albumin levels do not affect ionized calcium levels.

Parathyroid hormone (PTH) and calcitonin regulate calcium levels. Since the bones serve as a readily available store of calcium, the body is usually able to maintain normal serum calcium levels by regulating the movement of calcium into or out of the bone. Low serum calcium levels stimulate the parathyroid glands to produce and release PTH. PTH increases bone resorption (movement of calcium out of bones), increases GI absorption of calcium, and increases renal tubule reabsorption of calcium. High serum calcium levels stimulate the release of calcitonin from the thyroid gland. Calcitonin has the opposite effect of PTH. It lowers the serum calcium level by increasing calcium deposition into bone, increasing renal calcium excretion, and decreasing GI absorption.

HYPERCALCEMIA

Hypercalcemia (high serum calcium) is caused by hyperparathyroidism in about two thirds of persons. Malignancies, especially from hematologic, breast, and lung cancers, cause the remaining third. Malignancies lead to hypercalcemia through bone destruction from tumor invasion or tumor secretion of parathyroid-related proteins, which stimulate calcium release from bones.[7] More rare causes include thiazide diuretic use, prolonged immobilization, and increased calcium intake (e.g., use of calcium-containing antacids).

Excess calcium acts like a sedative, leading to reduced excitability of muscles and nerves. Neurologic manifestations begin with fatigue, lethargy, weakness, and confusion and progress to hallucinations, seizures, and coma. Disturbances in cardiac conduction can lead to dysrhythmias, including heart block and ventricular tachycardia. Table 16-7 lists the causes and manifestations of hypercalcemia.

❖ NURSING AND INTERPROFESSIONAL MANAGEMENT: HYPERCALCEMIA

◆ Nursing Diagnoses

Nursing diagnoses and collaborative problems for the patient with hypercalcemia include, but are not limited to, the following:

- Risk for electrolyte imbalance *related to* excessive bone destruction
- Risk for activity intolerance *related to* generalized muscle weakness
- Risk for injury *related to* neuromuscular and sensorium changes
- Potential complication: dysrhythmias

◆ Nursing Implementation

Management depends on the degree of hypercalcemia, patient's condition, and the underlying cause. Patients with mild hypercalcemia should stop any medications related to hypercalcemia, start a diet low in calcium, increase weight-bearing activity, and maintain adequate hydration. The patient must drink 3000 to 4000 mL of fluid daily to promote the renal excretion of calcium and decrease the chance of kidney stone formation. Fluids that promote urine acidity (cranberry or prune juice) will help to prevent formation of stones.

Managing severe hypercalcemia includes administering saline, a bisphosphonate, and calcitonin. Begin with hydrating

TABLE 16-7 Calcium Imbalances: Causes and Manifestations

Hypercalcemia (Ca^{2+} >10.2 mg/dL [2.55 mmol/L])	Hypocalcemia (Ca^{2+} <8.6 mg/dL [2.15 mmol/L])
Causes	
Increased Total Calcium	***Decreased Total Calcium***
• Hyperparathyroidism • Hematologic malignancy • Malignancies with bone metastasis • Prolonged immobilization • Vitamin A or D overdose • Paget's disease • Adrenal insufficiency • Thyrotoxicosis • Thiazide diuretics • Milk-alkali syndrome • Calcium-containing antacids • *Mycobacterium* infection	• Primary hypoparathyroidism • Renal insufficiency • Acute pancreatitis • Elevated phosphorus • Vitamin D deficiency, malnutrition • Magnesium deficiency • Bisphosphonates • Tumor lysis syndrome • Loop diuretics • Chronic alcoholism • Diarrhea • ↓ Serum albumin
Increased Ionized Calcium	***Decreased Ionized Calcium***
• Acidosis	• Alkalosis • Excess administration of citrated blood
Manifestations	
• Lethargy, weakness, fatigue • Decreased memory • Depressed reflexes • ↑ BP • Confusion, psychosis • Anorexia, nausea, vomiting • Bone pain, fractures • Polyuria, dehydration • Nephrolithiasis • Seizures, coma	• Weakness, fatigue • Depression, irritability, confusion • Hyperreflexia, muscle cramps • ↓ BP • Numbness and tingling in extremities and region around mouth • Chvostek's sign • Trousseau's sign • Laryngeal and bronchial spasms • Tetany, seizures
ECG Changes	***ECG Changes***
• Shortened ST segment • Shortened QT interval • Ventricular dysrhythmias • Increased digitalis effect	• Elongation of ST segment • Prolonged QT interval • Ventricular tachycardia

the patient with IV isotonic saline to maintain a urine output of 100 to 150 mL per hour.[3] IV saline therapy requires careful monitoring. Fluid overload can occur in patients who cannot excrete the excess sodium because of impaired renal function. Bisphosphonates (e.g., pamidronate, zoledronic acid) are the most effective agents in treating hypercalcemia, particularly when caused by a malignancy.[3] They interfere with the activity of osteoclasts, cells that break down bone. Because it takes 2 to 4 days for bisphosphonates to achieve maximum effect, patients receive IM or SC calcitonin for an immediate effect. Calcitonin rapidly increases renal calcium excretion. However, therapy is only effective for a few days and may cause tachycardia. Dialysis is an option in life-threatening situations.

HYPOCALCEMIA

Hypocalcemia (low serum calcium) can result from any condition associated with PTH deficiency. This may occur with surgical removal of a portion of or injury to the parathyroid glands during thyroid or neck surgery or with neck radiation. The patient who receives multiple blood transfusions can become

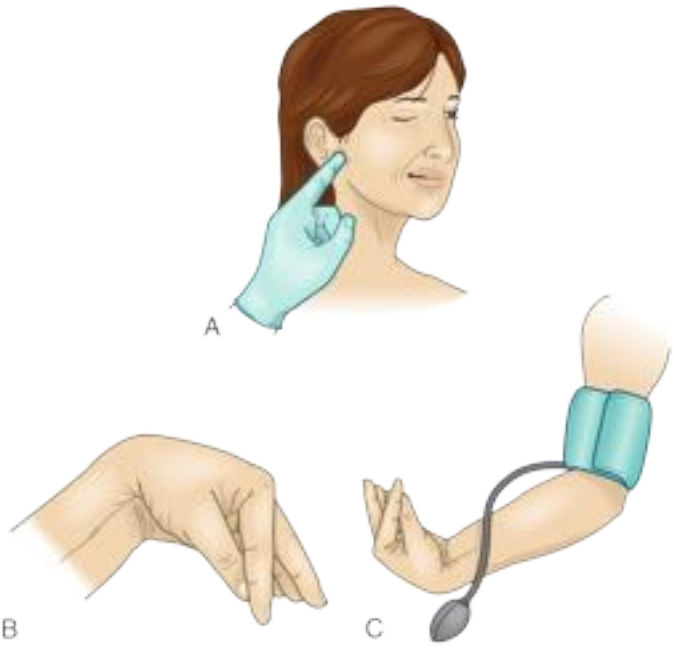

FIG. 16-15 Tests for hypocalcemia. **A,** Chvostek's sign is contraction of facial muscles in response to a light tap over the facial nerve in front of the ear. **B,** Trousseau's sign is a carpal spasm induced by **C,** inflating a BP cuff above the systolic pressure for a few minutes.

hypocalcemic because the citrate used to anticoagulate the blood binds with calcium, decreasing ionized calcium levels. Sudden alkalosis may result in symptomatic hypocalcemia despite a normal total serum calcium level. The high pH increases calcium binding to protein, decreasing the amount of ionized calcium. Table 16-7 lists causes and manifestations of hypocalcemia.

Low ionized calcium levels decrease the threshold for activating the sodium channels that cause cell membrane depolarization. This results in increased nerve excitability and sustained muscle contraction, or *tetany*. Clinical signs of tetany include Chvostek's sign and Trousseau's sign. *Chvostek's sign* is contraction of facial muscles in response to a tap over the facial nerve in front of the ear (Fig. 16-15, *A*). *Trousseau's sign* refers to carpal spasms induced by inflating a BP cuff on the arm (Fig. 16-15, *B* and *C*). When the cuff is inflated above the systolic pressure, carpal spasms occur within 3 minutes if hypocalcemia is present. Other manifestations of tetany are laryngeal stridor, dysphagia, paresthesia, and numbness and tingling around the mouth or in the extremities. Cardiac effects of hypocalcemia include decreased cardiac contractility and ECG changes. A prolonged QT interval may develop into ventricular tachycardia.

❖ NURSING AND INTERPROFESSIONAL MANAGEMENT: HYPOCALCEMIA

◆ Nursing Diagnoses

Nursing diagnoses and collaborative problems for the patient with hypocalcemia include, but are not limited to, the following:

- Risk for electrolyte imbalance *related to* decreased PTH level
- Ineffective breathing pattern *related to* laryngospasm
- Acute pain *related to* sustained muscle contractions
- Risk for injury *related to* tetany and seizures
- Potential complications: fracture, respiratory arrest

Nursing Implementation

Managing hypocalcemia depends on the underlying cause and the presence of symptoms. Treating mild or asymptomatic hypocalcemia involves a diet high in calcium-rich foods and calcium and vitamin D supplementation. Symptomatic hypocalcemia is treated with IV calcium gluconate.[9] Measures to promote CO_2 retention, such as breathing into a paper bag or sedating the patient, can control muscle spasm and other symptoms of tetany until the calcium level is corrected. Patients taking loop diuretics may need to change to thiazide diuretics to decrease urinary calcium excretion. Closely observe any patient who had thyroid or neck surgery in the immediate postoperative period for manifestations of hypocalcemia because of the proximity of the surgery to the parathyroid glands. Adequately treat pain and anxiety because hyperventilation-induced respiratory alkalosis can precipitate hypocalcemic symptoms.

PHOSPHATE IMBALANCES

Phosphorus is the primary anion in ICF and the second most abundant element in the body after calcium. Most phosphorus is in bones and teeth as calcium phosphate. The remaining phosphorus is metabolically active and essential to the function of muscle, red blood cells, and the nervous system. It is involved in the acid-base buffering system; the mitochondrial formation of ATP; cellular uptake and use of glucose; and carbohydrate, protein, and fat metabolism.

PTH maintains serum phosphorus levels and balance. Proper phosphate balance requires adequate renal functioning because the kidneys are the major route of phosphate excretion. When the phosphate level in the glomerular filtrate falls below normal or PTH levels are low, the kidneys reabsorb additional phosphorus. A reciprocal relationship exists between phosphorus and calcium. This means a low serum calcium level stimulates the release of PTH, decreasing reabsorption of phosphorus and lowering phosphorus levels.

HYPERPHOSPHATEMIA

Hyperphosphatemia (high serum phosphate) is common in patients with acute kidney injury or chronic kidney disease, which alters the kidney's ability to excrete phosphate. Other causes include excess phosphate intake from the use of phosphate-containing laxatives or enemas or a shift of phosphate from ICF to ECF. This may occur in patients with tumor lysis syndrome or rhabdomyolysis. Hypoparathyroidism and Vitamin D intoxication cause increased kidney phosphate reabsorption. Table 16-8 summarizes causes and clinical manifestations of hyperphosphatemia.

Hyperphosphatemia is often asymptomatic unless calcium binds with phosphate, leading to manifestations of hypocalcemia.[8] These manifestations include tetany, muscle cramps, paresthesias, and seizures. Long term, increased phosphate levels result in the development of calcified deposits outside of the bones. These calcium deposits can be found in soft tissues such as joints, arteries, skin, corneas, and kidneys and produce organ dysfunction, notably renal failure.

Managing hyperphosphatemia involves identifying and treating the underlying cause. Intake of foods and fluids high in phosphorus (e.g., dairy products) should be restricted. Oral phosphate-binding agents (e.g., calcium carbonate) limit intestinal phosphate absorption and increase phosphate secretion in the intestine. With severe hyperphosphatemia, hemodialysis may be used to rapidly decrease levels. Volume expansion and forced diuresis with a loop diuretic may increase phosphate excretion. If hypocalcemia is present, institute measures to correct calcium levels. See Chapter 46 for more information regarding treating hyperphosphatemia associated with kidney disease.

TABLE 16-8 Phosphate Imbalances: Causes and Manifestations

Hyperphosphatemia (PO_4^{3-} >4.4 mg/dL [1.42 mmol/L])	Hypophosphatemia (PO_4^{3-} <2.4 mg/dL [0.78 mmol/L])
Causes	
• Renal failure • Phosphate enemas (e.g., Fleet Enema) • Excessive ingestion (e.g., phosphate-containing laxatives) • Rhabdomyolysis • Tumor lysis syndrome • Thyrotoxicosis • Hypoparathyroidism • Sickle cell anemia, hemolytic anemia • Hyperthermia	• Malabsorption syndromes • Chronic diarrhea • Malnutrition, vitamin D deficiency • Parenteral nutrition • Chronic alcoholism • Phosphate-binding antacids • Diabetic ketoacidosis • Hyperparathyroidism • Refeeding syndrome • Respiratory alkalosis
Manifestations	
• Hypocalcemia • Numbness and tingling in extremities and region around mouth • Hyperreflexia, muscle cramps • Tetany, seizures • Calcium-phosphate precipitates in skin, soft tissue, cornea, viscera, blood vessels	• CNS depression (confusion, coma) • Muscle weakness, including respiratory muscle weakness • Polyneuropathy, seizures • Cardiac problems (dysrhythmias, heart failure) • Osteomalacia, rickets • Rhabdomyolysis

HYPOPHOSPHATEMIA

Hypophosphatemia (low serum phosphate) can result from decreased intestinal absorption, increased urinary excretion, or from ECF to ICF shifts. Malabsorption, diarrhea, and phosphate-binding antacids lead to decreased absorption. Phosphate shifts occur in respiratory alkalosis, treatment of diabetic ketoacidosis, and refeeding syndrome (reinstitution of nutrition to patients who are severely malnourished). Hypophosphatemia may occur in those who are malnourished or receive parenteral nutrition with inadequate phosphorus replacement. Table 16-8 lists causes and clinical manifestations of hypophosphatemia.

Most of the manifestations of hypophosphatemia result from impaired cellular energy and O_2 delivery due to low levels of cellular ATP and 2,3-diphosphoglycerate (2,3-DPG), an enzyme in RBCs that facilitates O_2 delivery to the tissues. Mild to moderate hypophosphatemia is often asymptomatic. Severe hypophosphatemia may be fatal because of decreased cellular function. Acute manifestations include CNS depression, muscle weakness and pain, respiratory failure, and heart failure. Chronic hypophosphatemia alters bone metabolism, resulting in rickets and osteomalacia.

Managing mild phosphorus deficiency involves increasing oral intake with dairy products or phosphate supplements. Dairy products are better tolerated because phosphate supplements are often associated with adverse GI effects.[10] Symptomatic hypophosphatemia can be fatal and usually requires IV administration of sodium phosphate or potassium

phosphate.[8] Frequent monitoring is necessary during IV therapy as complications include hypocalcemia, hyperkalemia, hypotension, and dysrhythmias.

MAGNESIUM IMBALANCES

Magnesium, the second most abundant intracellular cation, plays an important role in essential cellular processes. It is a cofactor in many enzyme systems, including those responsible for carbohydrate metabolism, DNA and protein synthesis, blood glucose control, and BP regulation. Magnesium is required for the production and use of adenosine triphosphate (ATP), the energy source for the sodium-potassium pump. Muscle contraction and relaxation, normal neurologic function, and neurotransmitter release depend on magnesium.

About 50% to 60% of the body's magnesium is stored in muscle and bone; 30% is in cells. Only 1% is in ECF. The intestines and kidneys regulate magnesium levels. GI absorption increases when magnesium levels are low. The kidney regulates serum magnesium by controlling the amount of magnesium reabsorbed in the ascending loop of Henle and distal tubules.[8]

HYPERMAGNESEMIA

Hypermagnesemia (high serum magnesium level) usually occurs only with increased magnesium intake accompanied by renal insufficiency or failure. A patient with chronic kidney disease who ingests products containing magnesium (e.g., Maalox, milk of magnesia) will have a problem with excess magnesium. Magnesium excess could develop in a pregnant woman receiving magnesium sulfate for the treatment of eclampsia or in patients taking laxatives and antacids that contain magnesium. Table 16-9 lists the causes and manifestations of hypermagnesemia.

Excess magnesium inhibits acetylcholine release at the myoneural junction and calcium movement into cells, impairing nerve and muscle function. Initial manifestations include hypotension, facial flushing, lethargy, urinary retention, nausea, and vomiting. As the serum magnesium level increases, deep tendon reflexes are lost, followed by muscle paralysis and coma. Respiratory and cardiac arrest can occur.

Management begins with avoiding magnesium-containing drugs and limiting diet intake of magnesium-containing foods (e.g., green vegetables, nuts, bananas, oranges, peanut butter, chocolate). If renal function is adequate, increased fluids and diuretics promote urinary excretion. In the patient with impaired renal function, dialysis is required. If hypermagnesemia is symptomatic, giving IV calcium gluconate will oppose the effects of the excess magnesium on cardiac muscle.

TABLE 16-9 Magnesium Imbalances: Causes and Manifestations

Hypermagnesemia (Mg^+ >2.5 mEq/L [1.25 mmol/L])	Hypomagnesemia (Mg^+ <1.5 mEq/L [0.75 mmol/L])
Causes	
• Renal failure	• GI tract fluid losses (e.g., diarrhea, NG suction)
• IV administration of magnesium, especially for treatment of eclampsia	• Chronic alcoholism
• Tumor lysis syndrome	• Malabsorption syndromes
• Hypothyroidism	• Prolonged malnutrition
• Metastatic bone disease	• ↑ Urine output
• Adrenal insufficiency	• Hyperglycemia
• Antacids, laxatives	• Proton pump inhibitor therapy
Manifestations	
• Lethargy, drowsiness	• Confusion
• Muscle weakness	• Muscle cramps
• Urinary retention	• Tremors, seizures
• Nausea, vomiting	• Vertigo
• Diminished deep tendon reflexes	• Hyperactive deep tendon reflexes
• Flushed, warm skin, especially facial	• Chvostek's and Trousseau's signs
• ↓ Pulse, ↓ BP	• ↑ Pulse, ↑ BP, dysrhythmias

HYPOMAGNESEMIA

Hypomagnesemia (low serum magnesium level) occurs in patients with limited magnesium intake or increased gastrointestinal or renal losses. Causes of hypomagnesemia from insufficient food intake include prolonged fasting or starvation and chronic alcoholism. Another potential cause is prolonged parenteral nutrition without magnesium supplementation. Fluid loss from the GI tract, inflammatory bowel disease, and proton pump inhibitors interfere with magnesium absorption. Many diuretics and osmotic diuresis from high glucose levels may cause magnesium loss through increased urinary excretion.[11] Table 16-9 lists the causes and manifestations of hypomagnesemia.

Clinically, hypomagnesemia resembles hypocalcemia. Neuromuscular manifestations are common, such as muscle cramps, tremors, hyperactive deep tendon reflexes, Chvostek's sign, and Trousseau's sign. Neurologic manifestations include confusion, vertigo, and seizures.

Magnesium deficiency can lead to cardiac dysrhythmias, such as torsades de pointes and ventricular fibrillation. Hypomagnesemia is associated with digitalis toxicity.

Managing hypomagnesemia depends on the underlying cause and the patient's symptoms. Mild magnesium deficiencies involve oral supplements and increased dietary intake of foods high in magnesium. If hypomagnesemia is severe or if hypocalcemia is present, IV magnesium (e.g., magnesium sulfate) is given. Monitor vital signs and use an infusion pump, since rapid administration can lead to hypotension and cardiac or respiratory arrest.

ACID-BASE IMBALANCES

The body normally maintains a steady balance between the acids continually produced during normal metabolism and the bases that neutralize and promote the excretion of the acids. Because these acids alter the body's internal environment, their regulation is necessary to maintain homeostasis and acid-base balance. Many health problems may lead to acid-base imbalances. Patients with diabetes mellitus, chronic obstructive pulmonary disease, and kidney disease frequently develop acid-base imbalances.

Remember that an acid-base imbalance is not a disease but a symptom of an underlying health problem. Always consider the possibility of acid-base imbalance in patients with serious illnesses.

pH AND HYDROGEN ION CONCENTRATION

The acidity or alkalinity of a solution depends on its hydrogen ion (H^+) concentration. An increase in H^+ concentration leads

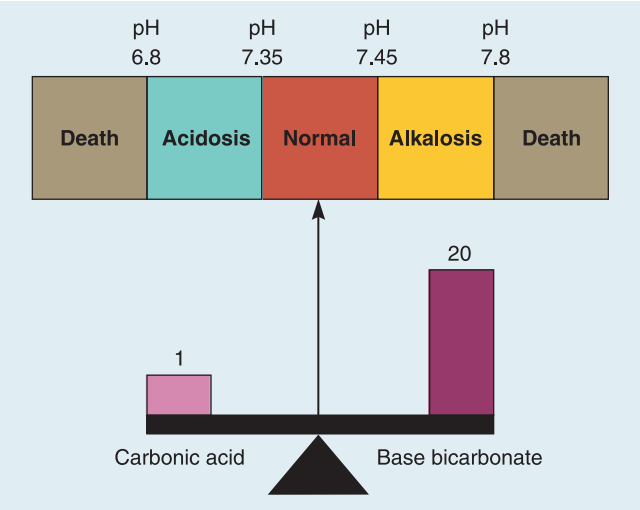

FIG. 16-16 The normal range of plasma pH is 7.35 to 7.45. A normal pH is maintained by a ratio of 1 part carbonic acid to 20 parts bicarbonate.

to acidity; a decrease leads to alkalinity. H^+ concentration is usually expressed as a negative logarithm (symbolized as *pH*). The use of the negative logarithm means that the lower the pH, the higher the H^+ concentration. In contrast to a pH of 7, a pH of 8 represents a 10-fold decrease in H^+ concentration.

The pH of a chemical solution may range from 1 to 14. A solution with a pH of 7 is considered neutral. An acid solution has a pH less than 7, and an alkaline solution has a pH greater than 7. Blood is slightly alkaline and has a normal arterial pH of 7.35 to 7.45. Medically, if the pH drops below 7.35, a person has **acidosis**. If the blood pH is greater than 7.45, the person has **alkalosis** (Fig. 16-16).

ACID-BASE REGULATION

The body uses three mechanisms to regulate acid-base balance and maintain the pH between 7.35 and 7.45. These are the buffer systems, respiratory system, and renal system. Each mechanism reacts at different speeds. Buffers are the fastest, reacting immediately. The respiratory system responds in minutes and reaches maximum effectiveness in hours. The renal response occurs hours to days after a change in pH, but the kidneys can maintain balance indefinitely in patients with chronic imbalances.

Buffer System

Buffering is the primary regulator of acid-base balance. **Buffers** act chemically to change strong acids into weaker ones or bind them to neutralize them. This minimizes the effect of acids on blood pH until their excretion from the body. Buffers can only maintain pH when the respiratory and renal systems function adequately.

All body fluids contain buffers. The major buffer system in ECF is carbonic acid–bicarbonate. Other buffers include phosphate, protein, and hemoglobin. The cell can act as a buffer by shifting H^+ in and out of the cell. When ECF levels of H^+ are increased, H^+ enters the cell in exchange for potassium. This may result in hyperkalemia. Conversely, with decreased H^+ levels, H^+ enters plasma in exchange for potassium. This is why alkalosis can cause hypokalemia.

A buffer consists of a weak acid, which releases H^+ when fluid is too alkaline, or a base and its salt, which takes up H^+ when fluid is too acidic. The carbonic acid–bicarbonate (H_2CO_3/HCO_3^-) buffer system neutralizes hydrochloric (HCl) acid in the following manner:

$$\underset{\text{Strong acid}}{HCl} + \underset{\text{Strong base}}{Na_2CO_3} \rightarrow \underset{\text{Salt}}{NaCl} + \underset{\text{Weak acid}}{H_2CO_3}$$

In this way, combining a strong acid with a strong base prevents the acid from making a large decrease in pH. The carbonic acid is broken down to H_2O and CO_2. The lungs excrete CO_2, either combined with insensible H_2O as carbonic acid, or alone as CO_2.

The phosphate, protein, and hemoglobin buffer systems act in the same way as the bicarbonate system. The main components of the phosphate system are monohydrogen phosphate (HPO_4^{2-}) and dihydrogen phosphate ($H_2PO_4^-$). A phosphate, combined with sodium, can neutralize a strong acid such as HCl, by forming sodium chloride (NaCl) and sodium biphosphate (NaH_2PO_4), a weak acid. If a strong base, such as sodium hydroxide (NaOH), is present, sodium biphosphate (NaH_2PO_4) neutralizes it to a weaker base (Na_2HPO_4) and H_2O.

Intracellular and extracellular proteins can act as acids or bases. Because the chemical structure of amino acids has an acid (the carboxyl group [COOH]) and a base (amine), they can accept H^+ if pH decreases or release H^+ if fluid is too alkaline. Some amino acids have basic radicals (NH_3OH [ammonium hydroxide]), which can dissociate into NH_3^+ (ammonia) and OH^- (hydroxide). The OH^- can combine with H^+ to form H_2O.

Hemoglobin is able to bind with both H^+, forming a weak acid, and CO_2, forming carbaminohemoglobin ($HbCO_2$). $HbCO_2$ dissociates in the lungs, releasing CO_2 for exhalation. Because H^+ can only combine with hemoglobin that has released its oxygen, hemoglobin less saturated with oxygen (venous blood) is a better buffer than saturated hemoglobin (arterial blood). Hemoglobin also assists in controlling pH by shifting chloride in and out of RBCs in exchange for bicarbonate.

The buffer system maintains a 20:1 ratio between HCO_3^- and carbonic acid in ECF. The body's ability to maintain this ratio is important in controlling pH. For example, excess carbonic acid increases the ratio and decreases pH, resulting in acidosis. In response, the body can increase HCO_3^- levels to keep the ratio 20:1 and maintain the pH near normal, a state called *compensation*. For example, a ratio of 40:2 would be present in compensated acid-base balance.

Respiratory System

The lungs help maintain a normal pH by excreting CO_2 and water, which are by-products of cellular metabolism. When released into the circulation, CO_2 enters RBCs and combines with H_2O to form H_2CO_3. This carbonic acid dissociates into H^+ and HCO_3^-. Hemoglobin buffers the free H^+, and the HCO_3^- diffuses into the plasma. This process is reversed in the pulmonary capillaries, forming CO_2 that is then excreted by the lungs. The overall reversible reaction is expressed in the following manner:

$$CO_2 + H_2O \rightarrow H_2CO_3 \rightarrow H_3 + HCO_3^-$$

The amount of CO_2 in the blood directly relates to carbonic acid concentration and subsequently to H^+ concentration. With increased respirations, more CO_2 is expelled and less remains in the blood. This leads to less carbonic acid and less H^+. With decreased respirations, more CO_2 remains in the blood. This leads to increased carbonic acid and more H^+.

The respiratory center in the medulla in the brainstem controls the rate of excretion of CO_2. If increased amounts of CO_2 or H^+ are present, the respiratory center stimulates an increased rate and depth of breathing to "blow off" CO_2 through hyperventilation. If the center senses low H^+ or CO_2 levels, respirations are inhibited and CO_2 retained. If a respiratory problem (e.g., respiratory failure) is the cause of an acid-base imbalance, the respiratory system loses ability to correct a pH alteration. The older adult has an impaired compensatory ability because of decreased respiratory function.

Renal System

Under normal conditions, the body depends on the kidneys to reabsorb and conserve all of the HCO_3^- they filter and excrete a portion of the acid produced by cellular metabolism. The three mechanisms of acid elimination are (1) secretion of small amounts of free hydrogen into the renal tubule, (2) combination of H^+ with ammonia (NH_3) to form ammonium (NH_4^+), and (3) excretion of weak acids.

The kidneys normally excrete acidic urine (average pH is 6). As a compensatory mechanism, the pH of the urine can decrease to 4 or increase to 8. To compensate for acidosis, the kidneys can reabsorb additional HCO_3^- and eliminate excess H^+. This increases the blood pH and decreases the urine pH. If the renal system is the cause of an acid-base imbalance (e.g., renal failure), it loses the ability to correct a pH alteration. In the older adult, the kidneys are less able to compensate for acid load.

ALTERATIONS IN ACID-BASE BALANCE

An acid-base imbalance results when there is an alteration in the ratio of 20:1 between base and acid content. This occurs when a disease or process alters one side of the ratio (e.g., CO_2 retention in pulmonary disease) and the compensatory processes that maintain the other side of the ratio (e.g., increased renal HCO_3^- reabsorption) either fail or are inadequate. The compensatory process may be inadequate because either the pathophysiologic process is overwhelming or there is insufficient time for the compensatory process to work.

Acid-base imbalances are classified as respiratory or metabolic. *Respiratory imbalances* result from the retention or an excess of CO_2 altering carbonic acid concentrations. *Metabolic imbalances* affect the base HCO_3^-. Acidosis occurs with an increase in carbonic acid (respiratory acidosis) or a decrease in HCO_3^- (metabolic acidosis). Alkalosis occurs with a decrease in carbonic acid (respiratory alkalosis) or an increase in HCO_3^- (metabolic alkalosis). Imbalances are further classified as acute or chronic. Chronic imbalances allow greater time for compensatory changes.

Respiratory Acidosis

Respiratory acidosis (carbonic acid excess) occurs whenever the person hypoventilates (Table 16-10). Hypoventilation leads to a buildup of CO_2, resulting in an accumulation of carbonic acid in the blood. Carbonic acid dissociates, releasing H^+ and decreasing pH. If CO_2 is not eliminated from the blood, acidosis results from the accumulation of carbonic acid (Fig. 16-17, *A*).

During acute respiratory acidosis, the renal compensatory mechanisms begin to operate within 24 hours. The kidneys conserve HCO_3^- and secrete increased concentrations of H^+ into the urine. Until the renal mechanisms have an effect, the serum HCO_3^- level will usually be normal, and then it will increase.

TABLE 16-10 Acid-Base Imbalances

Causes	Pathophysiology	Laboratory Findings
Respiratory Acidosis		
• Chronic respiratory disease (e.g., COPD) • Barbiturate or sedative overdose • Chest wall abnormality • Severe pneumonia • Atelectasis • Respiratory muscle weakness • Mechanical hypoventilation • Pulmonary edema	• ↑ CO_2 retention from hypoventilation • Compensatory response is ↑ HCO_3^- retention by kidney	↓ Plasma pH ↑ $PaCO_2$ HCO_3^- normal (uncompensated) ↑ HCO_3^- (compensated) *Sample ABG* Uncompensated: pH 7.31 $PaCO_2$ 54 mm Hg HCO_3^- 25 mEq/L
Respiratory Alkalosis		
• Hyperventilation (e.g., hypoxia, anxiety, fear, pain, exercise, fever) • Stimulated respiratory center (e.g., septicemia, stroke, meningitis, encephalitis, brain injury, salicylate poisoning) • Liver failure • Mechanical hyperventilation	• ↑ CO_2 excretion from hyperventilation • Compensatory response is ↑ HCO_3^- excretion by kidney	↑ Plasma pH ↓ $PaCO_2$ HCO_3^- normal (uncompensated) ↓ HCO_3^- (compensated) *Sample ABG* Uncompensated: pH 7.52 $PaCO_2$ 27 mm Hg HCO_3^- 24 mEq/L
Metabolic Acidosis		
• Diabetic ketoacidosis • Lactic acidosis • Starvation • Diarrhea • Renal tubular acidosis • Renal failure • Gastrointestinal fistulas • Shock	• Gain of fixed acid, inability to excrete acid or loss of base • Compensatory response is ↑ CO_2 excretion by lungs	↓ Plasma pH $PaCO_2$ normal (uncompensated) ↓ $PaCO_2$ (compensated) ↓ HCO_3^- *Sample ABG* Uncompensated: pH 7.29 $PaCO_2$ 38 mm Hg HCO_3^- 18 mEq/L
Metabolic Alkalosis		
• Vomiting • Nasogastric suctioning • Diuretic therapy • Hypokalemia • Excess $NaHCO_3$ intake • Mineralocorticoid use	• Loss of strong acid or gain of base • Compensatory response is ↑ CO_2 retention by lungs	↑ Plasma pH $PaCO_2$ normal (uncompensated) ↑ $PaCO_2$ (compensated) ↑ HCO_3^- *Sample ABG* Uncompensated: pH 7.50 $PaCO_2$ 40 mm Hg HCO_3^- 34 mEq/L

ABG, Arterial blood gas.

Respiratory Alkalosis

Respiratory alkalosis (carbonic acid deficit) occurs with hyperventilation, or an increase in respiratory rate or volume (Table 16-10). The primary cause of respiratory alkalosis is hypoxemia from acute pulmonary disorders (e.g., pneumonia, pulmonary embolus). Hyperventilation can occur as a physiologic response

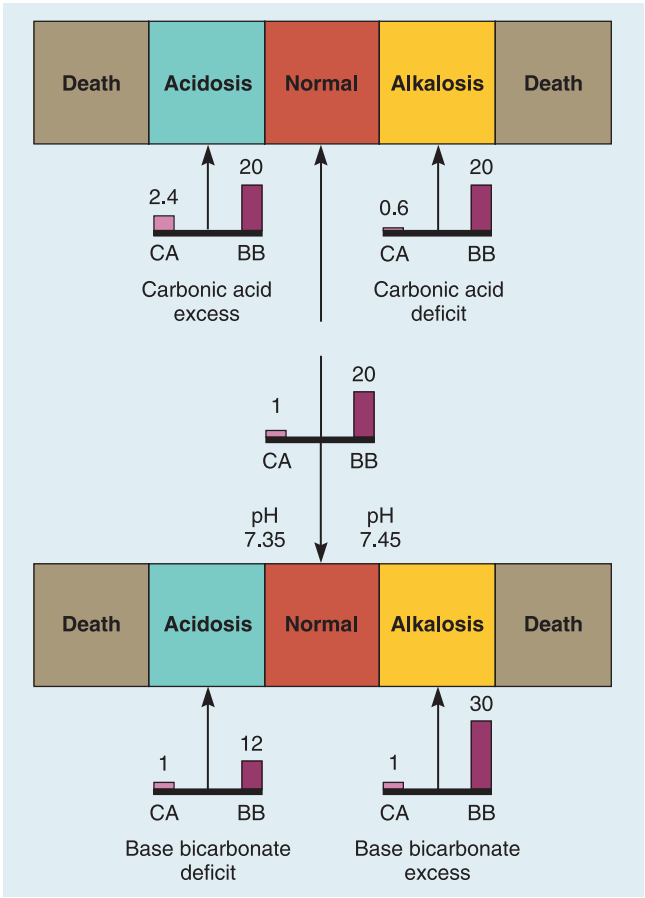

FIG. 16-17 Kinds of acid-base imbalances. **A,** Respiratory imbalances caused by carbonic acid (*CA*) excess and carbonic acid deficit. **B,** Metabolic imbalances caused by base bicarbonate (*BB*) deficit and base bicarbonate excess.

to metabolic acidosis and increased metabolic demands (e.g., fever). Pain, anxiety, and some CNS disorders can increase respirations without a physiologic need. The decrease in the arterial CO_2 level leads to decreased carbonic acid concentration in the blood and an increased pH (Fig. 16-17, *A*).

Compensated respiratory alkalosis is rare. In acute respiratory alkalosis, aggressive treatment of the cause of hypoxemia is essential and usually does not allow time for compensation to occur. Some buffering may occur with shifting of HCO_3^- into cells in exchange for chloride ions (Cl^-). In chronic respiratory alkalosis that occurs with pulmonary fibrosis or CNS disorders, compensation may include renal excretion of HCO_3^-.

Metabolic Acidosis

Metabolic acidosis (base bicarbonate deficit) occurs when an acid other than carbonic acid accumulates in the body or when bicarbonate is lost in body fluids (Table 16-10 and Fig. 16-17, *B*). Ketoacid accumulation in diabetic ketoacidosis and lactic acid accumulation with shock are examples of acid accumulation. Severe diarrhea results in loss of HCO_3^-. In renal disease, the kidneys lose their ability to reabsorb HCO_3^- and secrete H^+. To compensate for metabolic acidosis, the kidneys attempt to excrete additional acid and the lungs increase CO_2 excretion. The patient often develops *Kussmaul respirations* (deep, rapid breathing).

If metabolic acidosis is present, calculating the anion gap will help determine the source of the acidosis. The *anion gap* is the difference between the measured serum cations and anions in ECF. Calculate the anion gap using the following formula:

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

A normal anion gap is 8 to 12 mmol/L. The anion gap increases in metabolic acidosis associated with acid gain (e.g., lactic acidosis, diabetic ketoacidosis) but is normal in metabolic acidosis caused by bicarbonate loss (e.g., diarrhea).

Metabolic Alkalosis

Metabolic alkalosis (base bicarbonate excess) occurs when a loss of acid (e.g., from prolonged vomiting or gastric suction) or a gain in HCO_3^- (e.g., from ingestion of baking soda) occurs (Table 16-10 and Fig. 16-17, *B*). Renal excretion of HCO_3^- occurs in response to metabolic alkalosis. The lung's compensatory response is limited. The respiratory rate decreases in order to increase plasma CO_2. However, once hypoxemia occurs or plasma CO_2 reaches a certain level, stimulation of chemoreceptors increases respirations.

Mixed Acid-Base Disorders

A mixed acid-base disorder occurs when two or more disorders are present at the same time. The pH depends on the type, severity, and acuity of each disorder involved and any compensatory mechanisms at work. Respiratory acidosis combined with metabolic alkalosis (e.g., a patient with atelectasis and NG suction) may result in a near-normal pH, whereas respiratory acidosis combined with metabolic acidosis causes a greater decrease in pH than either disorder alone. An example of a mixed acidosis is a patient in severe shock with poor perfusion and hypoventilation. Mixed alkalosis can occur in a patient hyperventilating because of postoperative pain and losing acid secondary to NG suctioning.

CLINICAL MANIFESTATIONS OF ACID-BASE IMBALANCES

Clinical manifestations of acidosis and alkalosis are summarized in Tables 16-11 and 16-12. In both respiratory and metabolic acidosis, the CNS is depressed. Headache, lethargy, weakness, and confusion develop, leading eventually to coma and death. Compensatory mechanisms also produce specific clinical manifestations. For example, the deep, rapid respirations of a patient with metabolic acidosis occur with respiratory compensation.

In both types of alkalosis, symptoms usually result from an accompanying electrolyte abnormality rather than the alkalosis. Hypocalcemia occurs due to increased calcium binding with albumin, lowering the amount of ionized, active calcium. Therefore, muscle cramping, tingling and numbness of the fingers, and tetany occur. Once HCO_3^- levels are higher than 50 mEq/L, patients may develop seizures, hypoventilation, and coma.[12]

Blood Gas Values

Arterial blood gas (ABG) values provide objective information about a patient's acid-base status, the underlying cause of an imbalance, and the body's ability to regulate pH. Knowing the patient's clinical situation and the extent of renal and respiratory compensation allows you to identify acid-base disorders and the patient's ability to compensate. Blood gas analysis also

TABLE 16-11 Manifestations of Acidosis

Respiratory (↑ $PaCO_2$)	Metabolic (↓ HCO_3^-)
Neurologic	
Lethargy	Lethargy
Confusion	Confusion
Dizziness	Dizziness
Headache	Headache
Coma	Coma
Cardiovascular	
↓ BP	↓ BP
Ventricular fibrillation (related to hyperkalemia from compensation)	Dysrhythmias (related to hyperkalemia from compensation)
Warm, flushed skin	Cold, clammy skin
Gastrointestinal	
No significant findings	Nausea, vomiting, diarrhea, abdominal pain
Neuromuscular	
Seizures	Muscle weakness
Respiratory	
Hypoventilation with hypoxia	Deep, rapid respirations

TABLE 16-12 Manifestations of Alkalosis

Respiratory (↓ $PaCO_2$)	Metabolic (↑ HCO_3^-)
Neurologic	
Dizziness	Irritability
Light-headedness	Lethargy
Confusion	Confusion
Headache	Headache
Cardiovascular	
Tachycardia	Tachycardia
Dysrhythmias (related to hypokalemia from compensation)	Dysrhythmias (related to hypokalemia from compensation)
Gastrointestinal	
Nausea, vomiting, diarrhea	Nausea, vomiting
Epigastric pain	Anorexia
Neuromuscular	
Tetany	Tetany
Numbness	Tremors
Tingling of extremities	Tingling of fingers and toes
Hyperreflexia	Muscle cramps, hypertonic muscles
Seizures	Seizures
Respiratory	
Hyperventilation (lungs are unable to compensate if there is a respiratory problem)	Hypoventilation (compensatory action by lungs)

shows the partial pressure of arterial O_2 (PaO_2) and O_2 saturation. These values help you evaluate the patient's overall oxygenation status and identify hypoxemia.

To interpret the results of an ABG, perform the following six steps:

1. Look at each of the values. If the pH is between 7.35 and 7.45, and the CO_2, HCO_3^-, and PaO_2 are within normal limits, the ABGs are normal. If any value is out of normal, then continue.
2. Determine if the pH is acidotic or alkalotic. Values less than 7.35 are acidotic and values greater than 7.45 are alkalotic. A normal pH may indicate normal acid-base status, compensation is occurring, or a mixed disorder is present.

TABLE 16-13 Normal Arterial Blood Gas Values

Parameter	Reference Interval
pH	7.35-7.45
$PaCO_2$	35-45 mm Hg
Bicarbonate (HCO_3^-)	22-26 mEq/L (mmol/L)
PaO_2†	80-100 mm Hg
SaO_2	>95%
Base excess	±2.0 mEq/L

$PaCO_2$, Partial pressure of CO_2 in arterial blood; *PaO_2*, partial pressure of O_2 in arterial blood; *SaO_2*, arterial O_2 saturation.
†Decreases above sea level and with increasing age.

3. Determine the cause. First, analyze the $PaCO_2$ to see if the patient has respiratory acidosis or alkalosis. Since the lungs control $PaCO_2$, it is the respiratory component of the ABG. Because CO_2 forms carbonic acid when dissolved in blood, high $PaCO_2$ levels decrease pH and indicate respiratory acidosis. Low $PaCO_2$ levels increase pH and indicate respiratory alkalosis. If the pH value moves in the appropriate direction for the $PaCO_2$ change (e.g., ↓ pH with ↑ $PaCO_2$; ↑ pH with ↓ $PaCO_2$), a respiratory problem is the primary disturbance.
4. Analyze the HCO_3^- level to see if the patient has metabolic acidosis or alkalosis. Since the kidneys primarily control HCO_3^-, it is the metabolic component of the ABG. Because HCO_3^- is a base, high levels of HCO_3^- increase pH and result in metabolic alkalosis. Low levels decrease pH and result in metabolic acidosis. If the pH value moves in the appropriate direction for the change (e.g., ↓ pH with ↓ HCO_3^-; ↑ pH with ↑ HCO_3^-), a metabolic problem is the primary disturbance.
5. Determine if the patient is compensating or a mixed disorder is present. Look at the component (CO_2 or HCO_3^-) that is not the cause of the primary disturbance. If the component that does not match the pH is moving in the opposite direction, the body is attempting to compensate. For example, if the pH is slightly acidotic (7.33), the CO_2 is high (55 mm Hg), and the HCO_3^- is high (36 mEq/L), the CO_2 is the parameter that matches the acidotic pH. The patient's underlying acid-base imbalance is respiratory acidosis. The HCO_3^- level is alkalotic. Since this is in the opposite direction of respiratory acidosis, compensation is occurring. If both parameters match the pH, consider the possibility that a combined respiratory or metabolic acidosis or alkalosis is present. For example, if the pH is acidotic (7.28), the CO_2 is high (55 mm Hg), and the HCO_3^- is low (16 mEq/L), the patient's underlying acid-base imbalance is combined respiratory-metabolic acidosis.
6. Assess the PaO_2 and O_2 saturation. If these values are abnormal, hypoxemia is present.

Table 16-13 lists normal blood gas values. Table 16-14 explains how to analyze ABG results. The laboratory findings section of Table 16-10 provides ABG findings of the four major acid-base disturbances. Table 16-15 shows *ROME*, a quick memory device for understanding acid-base imbalances. See Chapter 25 for further discussion of ABGs.

ASSESSMENT OF FLUID, ELECTROLYTE, AND ACID-BASE IMBALANCES

Assessing patients for fluid, electrolyte, and acid-base imbalances is an important part of your nursing practice. In addition

TABLE 16-14 Arterial Blood Gas (ABG) Analysis

ABG Values	Analysis
pH 7.30 $PaCO_2$ 30 mm Hg HCO_3^- 16 mEq/L PaO_2 95 mm Hg on room air	1. The pH, $PaCO_2$, and HCO_3^- are abnormal. pH <7.35 indicates acidosis. 2. $PaCO_2$ is low, indicating respiratory alkalosis. 3. HCO_3^- is low, indicating metabolic acidosis. 4. HCO_3^- matches the pH, indicating metabolic acidosis is the primary problem. 5. The CO_2 is moving in the opposite direction, indicating the lungs are attempting to compensate for the metabolic acidosis. 6. PaO_2 is normal and does not indicate hypoxemia.

Interpretation
This ABG is interpreted as metabolic acidosis with partial compensation.

TABLE 16-15 ROME: Memory Device for Acid-Base Imbalances

For acid-base imbalances a quick memory device (mnemonic) can be used.

In ***Respiratory*** conditions, the pH and the $PaCO_2$ go in ***Opposite*** directions.
- In respiratory alkalosis, the pH is ↑ and the $PaCO_2$ is ↓.
- In respiratory acidosis, the pH is ↓ and the $PaCO_2$ is ↑.

In ***Metabolic*** conditions, the pH and the HCO_3^- go in the same direction (***Equal***). The $PaCO_2$ may also go in the same direction.
- In metabolic alkalosis, pH and HCO_3^- are ↑ and the $PaCO_2$ is ↑ or normal.
- In metabolic acidosis, pH and HCO_3^- are ↓ and the $PaCO_2$ is ↓ or normal.

		pH	$PaCO_2$
Respiratory			
Opposite	Acidosis	↓	↑
	Alkalosis	↑	↓
Metabolic		pH	HCO_3^-
Equivalent	Acidosis	↓	↓
	Alkalosis	↑	↑

to assessing for the clinical manifestations presented earlier in this chapter, obtain subjective and objective data from any patient with suspected fluid, electrolyte, or acid-base imbalances as discussed below.

Subjective Data

Important Health Information

Past Health History. Question the patient about any history of problems involving the kidneys, heart, GI system, or lungs that could affect the present fluid, electrolyte, and acid-base balance. Obtain information about specific diseases such as diabetes mellitus, diabetes insipidus, chronic obstructive pulmonary disease, renal failure, ulcerative colitis, and Crohn's disease. Ask the patient about any prior fluid, electrolyte, or acid-base disorders.

Medications. Assess the patient's current and past use of medications. The ingredients in many drugs, especially over-the-counter drugs, are often hidden sources of sodium, potassium, calcium, magnesium, and other electrolytes. Many prescription drugs, including diuretics, corticosteroids, and electrolyte supplements, can cause fluid and electrolyte imbalances.

Surgery or Other Treatments. Ask the patient about past or present renal dialysis, kidney surgery, or bowel surgery resulting in a temporary or permanent external collecting system such as an ileostomy.

Functional Health Patterns

Health Perception–Health Management Pattern. If the patient is currently experiencing a problem related to fluid, electrolyte, and acid-base balance, obtain a careful description of the illness, including onset, course, and treatment. Question the patient about any recent changes in body weight.

Nutritional-Metabolic Pattern. Ask the patient about diet and any special dietary practices. Weight reduction diets, fad diets, or any eating disorders, such as anorexia or bulimia, can lead to fluid and electrolyte problems. If the patient is on a special diet, such as low sodium or high potassium, assess the ability to adhere to the dietary prescription.

Elimination Pattern. Make note of the patient's usual bowel and bladder habits. Carefully document any deviations from the expected elimination pattern, such as diarrhea, oliguria, polyuria, or incontinence.

Activity-Exercise Pattern. Ask about the patient's exercise pattern and any complaints of excessive perspiration. Determine if the patient is exposed to extremely high temperatures during leisure or work activity. Ask the patient what he or she does to replace fluid and electrolytes lost through excessive perspiration. Assess the patient's activity level to determine any functional problems that could lead to lack of ability to obtain food or fluids.

Cognitive-Perceptual Pattern. Ask about any changes in sensations, such as numbness, tingling, or muscle weakness, which could indicate a fluid and electrolyte problem. Ask the patient and caregiver if there have been any changes in mentation or alertness, such as confusion, memory impairment, or lethargy.

Objective Data

Physical Examination. A complete physical examination is needed because fluid, electrolyte, and acid-base balance affects all body systems. As you assess each system, check for manifestations that you would expect with an imbalance. Common abnormal assessment findings of major body systems offer clues to possible imbalances (Table 16-16).

Laboratory Values. Assessing serum electrolyte values is a good starting point for evaluating fluid and electrolyte balance (Table 16-1). Serum electrolytes can also provide important information about a patient's acid-base balance. Changes in the serum HCO_3^- (often reported as total CO_2 or CO_2 content on an electrolyte panel) indicate metabolic acidosis (low HCO_3^- level) or alkalosis (high HCO_3^- level).

SAFETY ALERT Managing Critical Test Results
- Promptly report critical laboratory values to the HCP.
- Assess the patient and initiate appropriate actions (e.g., ECG monitoring).

Remember serum electrolyte values often provide limited information. They reflect the concentration of that electrolyte in ECF but not necessarily in ICF. For example, most potassium is found in the cells. Changes in serum potassium values may be the result of a true deficit or excess of potassium or reflect the movement of potassium into or out of the cell during acid-base imbalances. An abnormal serum sodium level may reflect a sodium problem or, more likely, a water problem.

Other useful laboratory tests include serum and urine osmolality, serum glucose, blood urea nitrogen, serum creatinine, venous blood gas sampling, urine specific gravity, and urine electrolytes.

TABLE 16-16 Assessment Abnormalities

Fluid and Electrolyte Imbalances

Finding	Possible Etiology
Skin	
Poor skin turgor	Fluid volume deficit
Cold, clammy skin	Fluid volume deficit, Na^+ deficit
Pitting edema	Fluid volume excess
Flushed, dry skin	Na^+ excess, Mg^{2+} excess
Pulse	
Bounding pulse	Fluid volume excess
Rapid, weak, thready pulse	Fluid volume deficit
Weak, irregular, rapid pulse	Severe K^+ deficit, Mg^{2+} deficit
Weak, irregular, slow pulse	Severe K^+ excess, Mg^{2+} excess
Blood Pressure	
Hypotension	Fluid volume deficit, Ca^{2+} deficit, Mg^{2+} excess
Hypertension	Fluid volume excess, Ca^{2+} excess, Mg^{2+} deficit
Respirations	
Rapid respirations	Fluid volume deficit
Shortness of breath	Fluid volume excess
Moist crackles	Fluid volume excess
Restricted airway	Ca^{2+} deficit, PO_4^{3-} excess
Skeletal Muscles	
Muscle cramping	K^+ excess , Ca^{2+} deficit, PO_4^{3-} excess, Mg^{2+} deficit
Muscle weakness	K^+ excess, K^+ deficit, Ca^{2+} excess, PO_4^{3-} deficit, Mg^{2+} excess
Trousseau's sign	Ca^{2+} deficit, Mg^{2+} deficit, PO_4^{3-} excess
Chvostek's sign	Ca^{2+} deficit, Mg^{2+} deficit, PO_4^{3-} excess
Neurologic	
Tremors, seizures	Na^+ excess, K^+ excess, Ca^{2+} deficit, PO_4^{3-} excess, Mg^{2+} deficit
Irritability	Na^+ deficit, K^+ excess, Ca^{2+} deficit, PO_4^{3-} excess
Fatigue	Fluid volume deficit, K^+ deficit, Ca^{2+} excess
Confusion	Fluid volume excess, Na^+ deficit, K^+ excess, K^+ deficit, Ca^{2+} excess, Mg^{2+} excess, Mg^{2+} deficit, PO_4^{3-} deficit
Decreased level of consciousness	Fluid volume deficit, fluid volume excess, Na^+ excess, Na^+ deficit, PO_4^{3-} deficit

ORAL FLUID AND ELECTROLYTE REPLACEMENT

In all cases of fluid, electrolyte, and acid-base imbalances, the primary treatment involves correcting the underlying cause. Oral rehydration solutions containing water, potassium, sodium, and glucose may be used to correct mild fluid and electrolyte deficits. Glucose not only provides calories, but also promotes sodium and water absorption in the small intestine. Commercial oral rehydration solutions are now available for home use. Cola drinks are avoided because they do not contain adequate electrolyte replacement and the sugar content may lead to osmotic diuresis.

IV FLUID AND ELECTROLYTE REPLACEMENT

IV fluid and electrolyte therapy is necessary to treat many different fluid and electrolyte imbalances. Many patients need maintenance IV fluid therapy when they cannot take oral fluids (e.g., during and after surgery). Other patients need corrective or replacement therapy for losses that are ongoing or have already occurred. The amount and type of solution is determined by the normal daily maintenance requirements and by imbalances identified by laboratory results. IV replacement solutions are classified by their concentration or tonicity (Table 16-17). Tonicity is an important factor in determining the appropriate solution to correct imbalances.

Solutions

Hypotonic. A hypotonic solution has more water than electrolytes, with an osmolality of less than 250 mOsm/kg.[13] Infusing a hypotonic solution dilutes ECF, lowering serum osmolality. Osmosis then produces a movement of water from ECF to interstitial spaces and cells, causing cells to swell. After achieving equilibrium, ICF and ECF have the same osmolality. Hypotonic solutions (e.g., 0.45% NaCl) are useful in treating patients with hypernatremia and are a good maintenance fluid because normal daily losses are hypotonic. They are not good for replacement because they can deplete ECF and lower BP. Because hypotonic solutions have the potential to cause cellular swelling, monitor patients for changes in mentation that may indicate cerebral edema.[13]

Although 5% dextrose in water is technically an isotonic solution, the dextrose quickly metabolizes. The net result is the administration of hypotonic free water with equal expansion of ECF and ICF. One liter of a 5% dextrose solution provides 50 g of dextrose, or 170 calories. While this amount of dextrose is not enough to meet caloric requirements, it helps prevent ketosis associated with starvation.

Isotonic. An isotonic solution has a similar concentration of water and electrolytes to plasma, with an osmolality of 250 to 375 mOsm/L.[13] Because of the similarity, administering an isotonic solution expands only ECF, and the fluid does not move into cells. This makes isotonic solutions the ideal fluid replacement for patients with ECF volume deficits. Examples of isotonic solutions include 0.9% NaCl and lactated Ringer's solution.

Isotonic saline (0.9% NaCl) or *normal saline* has a sodium concentration (154 mEq/L) somewhat higher than that of plasma (135 to 145 mEq/L) and a chloride concentration (154 mEq/L) significantly higher than the plasma chloride level (96 to 106 mEq/L). Therefore excessive administration of isotonic saline has the potential to increase sodium and chloride levels. Isotonic saline is used when a patient has experienced both fluid and sodium losses (e.g., diarrhea, vomiting). It is the fluid of choice for replacement.[13]

Lactated Ringer's solution contains sodium, potassium, chloride, calcium, and lactate (the precursor of bicarbonate) in about the same concentrations as ECF. This makes it the ideal fluid in certain situations, such as surgery, burns, or GI fluid losses. Patients with liver dysfunction, hyperkalemia, and severe hypovolemia should not receive lactated Ringer's because they have a decreased ability to convert lactate to bicarbonate.

Hypertonic. A hypertonic solution initially raises the osmolality of ECF and expands volume. The higher osmotic pressure draws water out of the cells into ECF. It is useful in the treatment of hyponatremia and trauma patients with head injury. Hypertonic solutions require frequent monitoring of BP, lung sounds, and serum sodium levels because of the risk for intravascular fluid volume excess.

Although concentrated dextrose and water solutions (10% dextrose or greater) are hypertonic solutions, once the dextrose

TABLE 16-17 Commonly Used Crystalloid Solutions

Solution	Tonicity	mOsm/kg	Contents	Indications and Considerations
Dextrose in Water				
5%	Isotonic, but physiologically hypotonic	278	50 g/L dextrose	• Contains free water only, no electrolytes • Provides 170 cal/L • Used to replace water losses and treat hypernatremia
10%	Hypertonic	556	100 g/L dextrose	• Contains free water only, no electrolytes • Provides 340 cal/L • Used with parenteral nutrition
Saline				
0.45%	Hypotonic	154	77 mEq/L Na^+ 77 mEq/L Cl^-	• Contains free water, Na^+ and Cl^-, no calories • Used to treat hypernatremia and uncontrolled hyperglycemia • Used as a maintenance solution
0.9%	Isotonic	308	154 mEq/L Na^+ 154 mEq/L Cl^-	• Contains Na^+ and Cl^- in excess of plasma levels • Does not contain free water or calories • Used to expand intravascular volume and replace extracellular fluid losses • Only solution that may be administered with blood products • May cause volume overload in patients with cardiac or renal disease
3.0%	Hypertonic	1026	513 mEq/L Na^+ 513 mEq/L Cl^-	• Contains Na^+ and Cl^- in excess of plasma levels • Used to treat symptomatic hyponatremia and trauma patients with head injury • Administer slowly because it may cause volume overload and pulmonary edema
Dextrose in Saline				
5% in 0.25%	Isotonic	355	50 g/L dextrose 34 mEq/L Na^+ 34 mEq/L Cl^-	• Provides Na^+, Cl^-, and free water • Used to replace hypotonic losses and treat hypernatremia • Provides 170 cal/L
5% in 0.45%	Hypertonic	432	50 g/L dextrose 77 mEq/L Na^+ 77 mEq/L Cl^-	• Provides Na^+, Cl^-, and free water • Used as a maintenance solution
5% in 0.9%	Hypertonic	586	50 g/L dextrose 154 mEq/L Na^+ 154 mEq/L Cl^-	• Contains Na^+ and Cl^- in excess of plasma levels • Used to treat metabolic alkalosis and volume deficits in patients with hyponatremia
Multiple Electrolyte Solutions				
Ringer's solution	Isotonic	309	147 mEq/L Na^+ 156 mEq/L Cl^- 4 mEq/L K^+ 4 mEq/L Ca^{2+}	• Similar in composition to plasma except that it has excess Cl^-, no Mg^{2+}, no HCO_3^- • Does not provide free water or calories • Used to expand the intravascular volume and replace extracellular fluid losses
Lactated Ringer's (Hartmann's) solution	Isotonic	273	130 mEq/L Na^+ 109 mEq/L Cl^- 4 mEq/L K^+ 3 mEq/L Ca^{2+} 28 mmol/L lactate	• Similar in composition to normal plasma except does not contain Mg^{2+} • Does not provide free water or calories • Used to treat hypovolemia, burns, and GI fluid losses • Cannot be used in patients with alkalosis or lactic acidosis

is metabolized, the net result is the administration of water. This free water ultimately expands ECF and ICF. The primary use of these solutions is providing calories as part of parenteral nutrition (see Chapter 39). You may administer solutions containing 10% dextrose or less through a peripheral line. You must use a central line to administer solutions with dextrose concentrations greater than 10%.

IV Additives. Additives in basic IV solutions replace specific losses. KCl, $CaCl_2$, $MgSO_4$, and HCO_3^- are common additives. The use of each was described earlier in the discussion of the specific electrolyte deficiencies. Many premixed IV solutions containing specific additives are available. Using these solutions reduces error as the solution contains the correct amount of the electrolyte in the proper volume and type of IV solution.

Colloids. Colloids solutions contain large molecules that increase oncotic pressure and pull fluid into the blood vessels. Because this action restores blood volume, colloids are also called volume expanders or plasma expanders. Colloids include human plasma products (albumin, fresh frozen plasma, blood) and semisynthetics (dextran, starches).

Albumin is available in 5% and 25% solutions. The 5% solution has an albumin concentration similar to plasma and results in plasma volume expansion equal to the volume infused. This makes the 5% concentration ideal for treating hypovolemic patients. In contrast, 25% albumin solution is hypertonic and draws fluid from the interstitial space. This makes it useful in treating patients with burns, hepatic failure, and ascites.

Dextrans are synthetic complex sugar solutions. There are two types: low-molecular-weight dextran (dextran 40) and high-molecular-weight dextran (dextran 70). Because dextran metabolizes slowly, it remains in the vascular system for a prolonged period. It pulls additional fluid into the intravascular space, expanding it by more volume than what is infused. Hydroxylethyl starches (e.g., Hespan) are synthetic colloids that work similarly to dextran to expand plasma volume.

Colloids can lead to circulatory overload because they pull fluid into ECF. Monitor vital signs and urine output frequently and assess for signs and symptoms of fluid volume excess. All colloids affect blood coagulation, with dextran and starches having the strongest anticoagulation effect. Monitor coagulation times and implement any necessary precautions. Fatal anaphylactic reactions have occurred with Hespan and dextran. Monitor the patient closely for a hypersensitivity reaction and discontinue an infusion immediately if a reaction occurs.

If the patient has lost blood, whole blood or packed RBCs are necessary. Packed RBCs have the advantage of giving the patient primarily RBCs rather than RBCs and fluid volume. Although packed RBCs have a decreased plasma volume, they increase the oncotic pressure and pull fluid into the intravascular space. The use of whole blood, with its additional fluid volume, may cause circulatory overload, particularly in patients who are susceptible to complications from excess circulating volume (e.g., heart failure). To prevent fluid volume excess, loop diuretics may be administered with blood and colloids. See Chapter 30 for more information on the administration of blood and blood products.

TEAMWORK & COLLABORATION

IV Therapy

Although many states permit licensed practical nurses (LPNs) to administer IV fluids and drugs, the registered nurse (RN) should administer IV fluids or medications to unstable or critically ill patients.

Role of Nursing Personnel

Registered Nurse (RN)

- Assess patient for clinical manifestations of fluid and electrolyte disturbances.
- Determine if ordered IV therapies are appropriate.
- Choose and insert appropriate IV catheters and infusion devices.
- Administer IV fluids and medications to unstable and critically ill patients.
- Evaluate patient for clinical manifestations of fluid overload or hypovolemia and initiate appropriate changes in IV fluids.
- Evaluate if IV therapies are addressing patient's fluid and electrolyte needs.

Licensed Practical/Vocational Nurse (LPN/LVN)

- Administer IV fluids and medications to stable patients (consider state nurse practice act and agency policy).
- Adjust IV flow rate for stable patients according to HCP orders (consider state nurse practice act and agency policy).
- Insert IV catheters (consider state nurse practice act and agency policy).
- Monitor for clinical manifestations of adverse reactions to IV fluids or medications.

Unlicensed Assistive Personnel (UAP)

- Measure and record oral intake and output.
- Report swelling or redness at IV site or patient complaints of discomfort at IV site to RN.

Role of Other Team Members

Pharmacist

- Determine appropriateness of IV therapies and need for dose adjustments.
- Prepare IV infusions and medications.
- Screen for potential problems, such as compatibility issues.
- Monitor response to therapy.

CENTRAL VENOUS ACCESS DEVICES

Central venous access devices (CVADs) are catheters placed in large blood vessels (e.g., subclavian vein, jugular vein) of people who require frequent or special access to the vascular system. There are three main types of CVADs: centrally inserted catheters, peripherally inserted central catheters (PICCs), and implanted ports.

Advantages of CVADs include immediate access to the central venous system, a reduced need for multiple venipunctures, and decreased risk of extravasation injury. CVADs permit frequent, continuous, rapid, or intermittent administration of fluids and medications. They allow for the administration of drugs that are potential *vesicants* (agents that can cause tissue damage), blood and blood products, and parenteral nutrition. CVADs can provide a means to perform hemodynamic monitoring and obtain venous blood samples. They are useful with patients who have limited peripheral vascular access or who have a projected need for long-term vascular access. CVADs can also be used for injections of radiopaque contrast media. Table 16-18 provides examples of conditions where CVADs are used.

The major disadvantages of CVADs are an increased risk of systemic infection and the invasiveness of the procedure. Extravasation (leakage of fluid) can still occur if there is displacement of or damage to the device.

Centrally Inserted Catheters

The tip of centrally inserted catheters (also called central venous catheters [CVCs]) rests in the distal end of the superior vena cava near its junction with the right atrium (Fig. 16-18). The other end of the catheter exits through a separate incision on the chest or abdominal wall. *Nontunneled catheters* are usually placed in the subclavian or internal jugular vein, more rarely in the femoral vein. They are best for patients with short-term needs in an acute care setting. *Surgically placed tunneled catheters* (e.g., Hickman) are suitable for long-term needs. Tunneling of the catheter through subcutaneous tissue and the synthetic cuff used to anchor the catheter provide stability and decrease infection risk. After the site heals, the catheter does not require a dressing, making it easier for the patient to maintain the site at home.

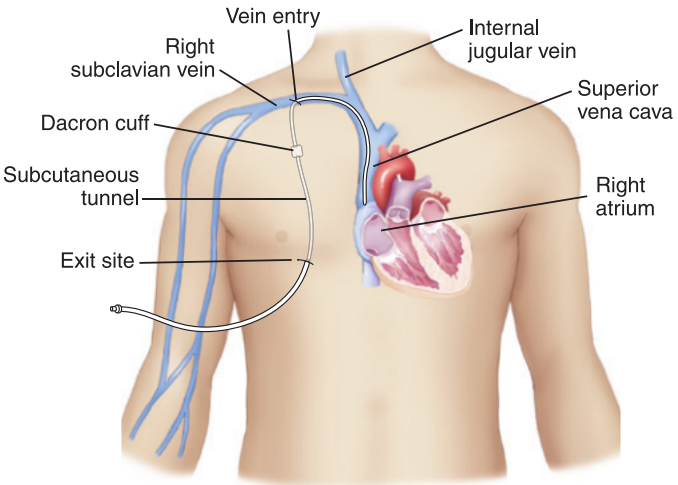

FIG. 16-18 Tunneled central venous catheter. Note tip of the catheter in the superior vena cava.

TABLE 16-18 Indications for Central Venous Access Devices (CVADs)*

Condition	Indications for Use
Medication administration	
• Cancer	• Chemotherapy, infusion of irritating or vesicant medications
• Infection	• Long-term administration of antibiotics
• Pain	• Long-term administration of pain medication
• Drugs at risk for causing phlebitis	• Epoprostenol (Flolan) • Calcium chloride • Potassium chloride • Amiodarone (Cordarone)
Nutritional replacement	• Infusion of parenteral nutrition • Infusion of high percentage dextrose solutions
Blood samples	• Multiple blood draws for diagnostic tests over time
Blood transfusions	• Infusion of blood or blood products
Renal failure	• Perform hemodialysis (especially on an acute basis) or continuous renal replacement therapy
Shock, burns	• Infusion of high volumes of fluid and electrolyte replacement
Hemodynamic monitoring	• Used to measure central venous pressure to assess fluid balance
Heart failure	• Perform ultrafiltration
Autoimmune disorders	• Perform plasmapheresis

*This list is not all-inclusive, and these are examples only.

CVCs are available with single-, double-, triple-, or quad-lumens. Multi-lumen catheters are useful in the critically ill patient because each lumen can be simultaneously used to provide a different therapy. For example, incompatible drugs infuse in separate lumens without mixing while a third lumen provides access for blood sampling.

Peripherally Inserted Central Catheters

Peripherally inserted central catheters (PICCs) are central venous catheters inserted into a vein in the arm. The basilic vein is preferred because of its large diameter (Fig. 16-19). The cephalic, median cubital, or brachial veins are other options. Single-, double-, or triple-lumens are available; double lumens are preferred, because they allow for simultaneous uses. PICCs are used with patients who need vascular access for 1 week to 6 months, but they can be in place for longer periods.

Advantages of a PICC over a CVC are lower infection rate, fewer insertion-related complications, decreased cost, and ability to insert at the bedside or in an outpatient area. PICCs, however, are associated with an increased risk of deep vein thrombosis and phlebitis (Table 16-19). If phlebitis occurs, it usually happens within 7 to 10 days after insertion. Do not use the arm with the PICC to obtain a BP reading or draw blood. When the BP cuff is inflated, the PICC can touch the vein wall, increasing the risk of vein damage and thrombosis.

Implanted Infusion Ports

An implanted infusion port consists of a surgically implanted central venous catheter connected to a reservoir or port (Fig. 16-20, *A*). The catheter tip lies in the desired vein. The port lies in a surgically created subcutaneous pocket on the upper chest or arm. It consists of a titanium or plastic reservoir covered with a self-sealing silicone septum. You access the port by using a special noncoring needle with a deflected tip. This prevents damage to the septum that could make the port useless (Fig. 16-20, *B*).

Drugs are placed in the reservoir either by a direct injection or through injection into an established IV line. The reservoir then slowly releases the medicine into the bloodstream. Implanted ports are good for long-term therapy and have a low risk of infection. The hidden port offers the patient cosmetic advantages and overall has less maintenance than other types of CVADs. Monitor accessed ports for infiltration that can occur if the needle is not in place or dislodges.

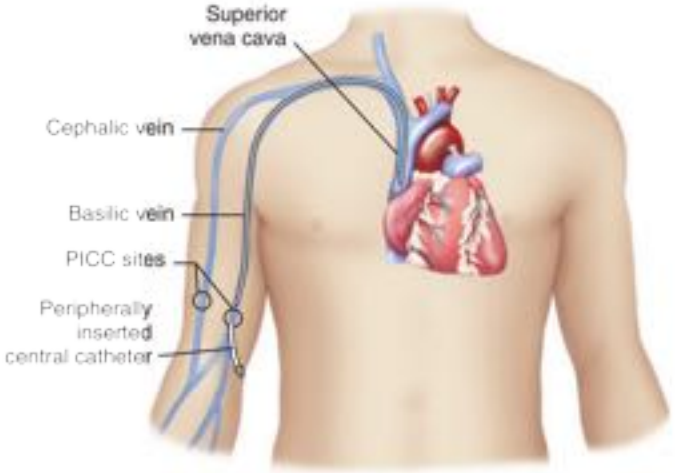

FIG. 16-19 Peripherally inserted central catheter (PICC) can be inserted using the basilic or cephalic vein.

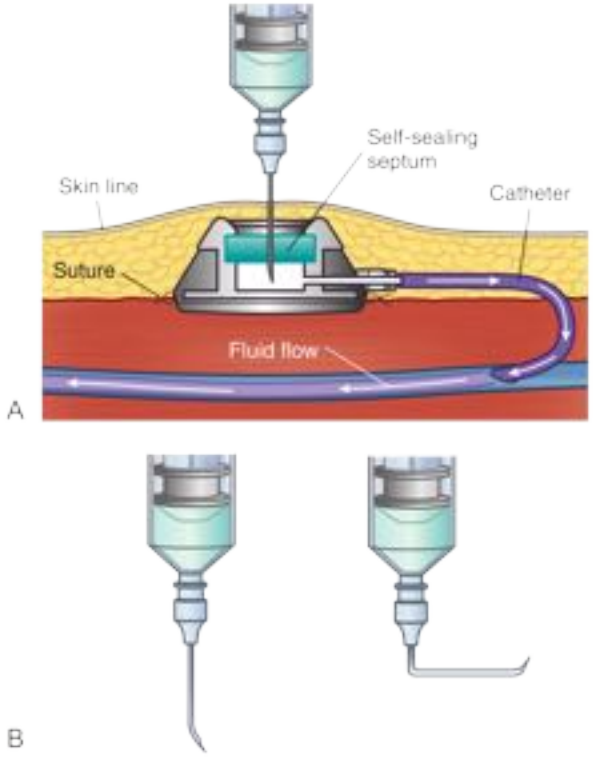

FIG. 16-20 **A,** Cross section of implantable port displaying access of the port with the Huber-point needle. Note the deflected point of the Huber-point needle, which prevents coring of the port's septum. **B,** Two Huber-point needles used to enter the implanted port. The 90-degree needle is used for top-entry ports for continuous infusion.

TABLE 16-19 Complications of Central Venous Access Devices (CVADs)

Possible Cause	Manifestations	Management
Catheter Occlusion		
• Clamped or kinked catheter • Tip against wall of vessel • Thrombosis • Precipitate buildup in lumen	• Sluggish infusion or aspiration • Inability to infuse and/or aspirate	• Instruct patient to change position, raise arm, and cough. • Assess for and alleviate clamping or kinking. • Flush with normal saline using a 10-mL syringe. Do not force flush. • Perform fluoroscopy to determine cause and site. • Instill anticoagulant or thrombolytic agent.
Embolism		
• Catheter breaking • Dislodgment of thrombus • Entry of air into circulation	• Chest pain • Respiratory distress (dyspnea, tachypnea, hypoxia, cyanosis) • Hypotension • Tachycardia	• Administer O_2. • Clamp catheter. • Place patient on left side with head down (air emboli). • Notify physician.
Catheter-Related Infection (local or systemic)		
• Contamination during insertion or use • Migration of organisms along catheter • Immunosuppressed patient	• *Local:* redness, tenderness, purulent drainage, warmth, edema • *Systemic:* fever, chills, malaise	***Local*** • Culture drainage from site. • Apply warm, moist compresses. • Remove catheter if indicated. ***Systemic*** • Take blood cultures. • Give antibiotic therapy. • Give antipyretic therapy. • Remove catheter if indicated.
Pneumothorax		
• Perforation of visceral pleura during insertion	• Decreased or absent breath sounds • Respiratory distress (cyanosis, dyspnea, tachypnea) • Chest pain • Distended unilateral chest	• Administer O_2. • Position in semi-Fowler's position. • Prepare for chest tube insertion.
Catheter Migration		
• Improper suturing • Insertion site trauma • Changes in intrathoracic pressure • Forceful catheter flushing • Spontaneous	• Sluggish infusion or aspiration • Edema of chest or neck during infusion • Patient complaint of gurgling sound in ear • Dysrhythmias • Increased external catheter length	• Perform fluoroscopy to verify position. • Assist with removal and new CVAD placement.

Complications

CVADs always have a potential for complications. Careful monitoring and assessment may assist in early identification of potential complications. Table 16-19 lists common possible complications, potential causes, clinical manifestations, and interventions.

❖ NURSING MANAGEMENT: CENTRAL VENOUS ACCESS DEVICES

Nursing management of CVADs includes assessment, dressing changes and cleansing, injection cap changes, and maintenance of catheter patency. The exact frequency and procedures for these requirements vary by type of CVAD and institution, so it is important to follow your specific institution's policies and procedures. The following section discusses some general guidelines.

Catheter and insertion site assessment includes inspecting the site for redness, edema, warmth, drainage, and tenderness or pain. Observing the catheter for misplacement or slippage is important. Perform a comprehensive pain assessment, particularly noting any complaints of chest or neck discomfort, arm pain, or pain at the insertion site. Do not use a newly placed CVAD until the tip position is verified with a chest x-ray.

Before manipulating a catheter for any reason, perform hand hygiene. Perform dressing changes and cleanse the catheter insertion site using strict sterile technique. Typical dressings include transparent semipermeable dressings or gauze and tape. If the site is bleeding, a gauze dressing may be preferable. Otherwise, transparent dressings are preferred. They allow observation of the site without having to remove the dressing. Transparent dressings may be left in place for up to 1 week. Change any dressing immediately if it becomes damp, loose, or soiled.

Cleanse the skin around the catheter insertion site according to institution policy. A chlorhexidine-based preparation is the cleansing agent of choice. Its effects last longer than either povidone-iodine or isopropyl alcohol, offering improved killing of bacteria.[14] When using chlorhexidine, cleansing the skin with friction is critical to preventing infection. When applying a new dressing, allow the area to air dry completely. Secure the lumen ports to the skin above the dressing site. Document the date and time of the dressing change and initial the dressing.

Disinfect catheter hubs, needleless connectors, and injection ports before accessing the catheter. Use an alcoholic chlorhexidine preparation, 70% alcohol, or povidone-iodine. Change injection caps at regular intervals according to institution policy or if they have damage from excessive punctures. Use strict sterile technique. Teach the patient to turn the head to the opposite side of the CVAD insertion site during cap change. If you cannot clamp the catheter, instruct the patient to lie flat in

bed and perform the Valsalva maneuver whenever the catheter is open to air to prevent an air embolism.

Flushing is one of the most effective ways to maintain catheter patency. It also keeps incompatible drugs or fluids from mixing. Use a normal saline solution in a syringe that has a barrel capacity of 10 mL or more to avoid excess pressure on the catheter. If you feel resistance, do not apply force. This could result in a ruptured catheter or create an embolism if a thrombus is present. Because of the risk of contamination and infection, prefilled syringes or single-dose vials are preferred over multiple-dose vials. If you are not using a positive-pressure valve cap, clamp any unused lines after flushing.

Use the push-pause technique when flushing all catheters. Push-pause creates turbulence within the catheter lumen, promoting the removal of debris that adheres to the catheter lumen and decreasing the chance of occlusion. This technique involves injecting saline with a rapid alternating push-pause motion, instilling 1 to 2 mL with each push on the syringe plunger. If you are using a negative-pressure cap or neutral pressure cap, clamp the catheter while maintaining positive pressure while instilling the last 1 mL of saline. This prevents reflux of blood back into the catheter. If a positive-pressure valve cap is present, it works to prevent the reflux of blood and resultant catheter lumen occlusion. Remove the syringe before clamping the catheter to allow the positive pressure valve to work correctly.

Removal of CVADs

Removing a CVAD is done according to institution policy and the nurse's scope of practice. In many agencies, nurses with demonstrated competency can remove PICCs and nontunneled central venous catheters. The procedure involves removing any sutures and then gently withdrawing the catheter. Instruct the patient to perform the Valsalva maneuver as the last 5 to 10 cm of the catheter is withdrawn. Immediately apply pressure to the site with sterile gauze to prevent air from entering and to control bleeding. Inspect the catheter tip to determine that it is intact. After bleeding has stopped, apply an antiseptic ointment and sterile dressing to the site.

CASE STUDY

Fluid and Electrolyte Imbalance

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Patient Profile

S.S., a 63-year-old white woman with acute lymphocytic leukemia, has been receiving chemotherapy on an outpatient basis. She completed her third treatment 5 days ago and has been experiencing nausea and vomiting for 2 days despite using ondansetron (Zofran). S.S.'s daughter brings her to the hospital, where she is admitted to the medical unit. As the admitting nurse, you perform a thorough assessment.

Subjective Data

- Complains of lethargy, weakness, dizziness, and dry mouth
- States she has been too nauseated to eat or drink anything for 2 days

Objective Data

- Heart rate 110 beats/min, pulse thready
- BP 100/65
- Weight loss of 5 lb since she received her chemotherapy treatment 5 days ago
- Dry oral mucous membranes

Discussion Questions

1. Based on her clinical manifestations, what fluid imbalance does S.S. have?
2. What additional assessment data should you obtain?
3. What are her risk factors for fluid and electrolyte imbalances?
4. You draw blood for a serum chemistry evaluation. What electrolyte imbalances are likely and why?
5. S.S. is at risk for which acid-base imbalance? Describe the changes that would occur in S.S.'s ABGs with this acid-base imbalance. How would the body compensate?
6. ***Teamwork and Collaboration:*** What is the interprofessional team's priority at this time for S.S.?
7. The physician orders dextrose 5% in 0.45% saline to infuse at 100 mL/hr. What type of solution is this, and how will it help S.S.'s fluid imbalance?
8. ***Priority Decision:*** What are the priority nursing interventions for S.S.?
9. ***Evidence-Based Practice:*** S.S. has a double-lumen PICC in her left arm. One lumen is connected to the IV infusion; the other is unused. What is the recommended practice for maintaining the patency of the unused lumen?
10. ***Quality Improvement:*** What outcomes will indicate that interprofessional care was effective?

Answers available at http://evolve.elsevier.com/Lewis/medsurg.

BRIDGE TO NCLEX EXAMINATION

The number of the question corresponds to the same-numbered outcome at the beginning of the chapter.

1. During the postoperative care of a 76-year-old patient, the nurse monitors the patient's intake and output carefully, knowing that the patient is at risk for fluid and electrolyte imbalances primarily because
   - **a.** older adults have an impaired thirst mechanism and need reminding to drink fluids.
   - **b.** water accounts for a greater percentage of body weight in the older adult than in younger adults.
   - **c.** older adults are more likely than younger adults to lose extracellular fluid during surgical procedures.
   - **d.** small losses of fluid are significant because body fluids account for 45% to 50% of body weight in older adults.

2. During administration of a hypertonic IV solution, the mechanism involved in equalizing the fluid concentration between ECF and the cells is
   - **a.** osmosis.
   - **b.** diffusion.
   - **c.** active transport.
   - **d.** facilitated diffusion.

3a. An older woman was admitted to the medical unit with GI bleeding and fluid volume deficit. Clinical manifestations of this problem are (*select all that apply*)
   - **a.** weight loss.
   - **b.** dry oral mucosa.
   - **c.** full bounding pulse.
   - **d.** engorged neck veins.
   - **e.** decreased central venous pressure.

3b. The nursing care for a patient with hyponatremia and fluid volume excess includes
- **a.** fluid restriction.
- **b.** administration of hypotonic IV fluids.
- **c.** administration of a cation-exchange resin.
- **d.** placement of an indwelling urinary catheter.

3c. The nurse should be alert for which manifestations in a patient receiving a loop diuretic?
- **a.** Restlessness and agitation
- **b.** Paresthesias and irritability
- **c.** Weak, irregular pulse and poor muscle tone
- **d.** Increased blood pressure and muscle spasms

3d. Which patient is at *greatest* risk for developing hypermagnesemia?
- **a.** 83-year-old man with lung cancer and hypertension
- **b.** 65-year-old woman with hypertension taking β-adrenergic blockers
- **c.** 42-year-old woman with systemic lupus erythematosus and renal failure
- **d.** 50-year-old man with benign prostatic hyperplasia and a urinary tract infection

3e. It is important for the nurse to assess for which clinical manifestation(s) in a patient who has just undergone a total thyroidectomy (*select all that apply*)?
- **a.** Confusion
- **b.** Weight gain
- **c.** Depressed reflexes
- **d.** Circumoral numbness
- **e.** Positive Chvostek's sign

3f. The nurse expects the long-term treatment of a patient with hyperphosphatemia secondary to renal failure will include
- **a.** fluid restriction.
- **b.** calcium supplements.
- **c.** magnesium supplements.
- **d.** increased intake of dairy products.

4. The lungs act as an acid-base buffer by
- **a.** increasing respiratory rate and depth when CO_2 levels in the blood are high, reducing acid load.
- **b.** increasing respiratory rate and depth when CO_2 levels in the blood are low, reducing base load.
- **c.** decreasing respiratory rate and depth when CO_2 levels in the blood are high, reducing acid load.
- **d.** decreasing respiratory rate and depth when CO_2 levels in the blood are low, increasing acid load.

5. A patient has the following arterial blood gas results: pH 7.52, $PaCO_2$ 30 mm Hg, HCO_3^- 24 mEq/L. The nurse determines that these results indicate
- **a.** metabolic acidosis.
- **b.** metabolic alkalosis.
- **c.** respiratory acidosis.
- **d.** respiratory alkalosis.

6. The typical fluid replacement for the patient with a fluid volume deficit is
- **a.** dextran.
- **b.** 0.45% saline.
- **c.** lactated Ringer's.
- **d.** 5% dextrose in 0.45% saline.

7. The nurse is unable to flush a central venous access device and suspects occlusion. The *best* nursing intervention would be to
- **a.** apply warm moist compresses to the insertion site.
- **b.** attempt to force 10 mL of normal saline into the device.
- **c.** place the patient on the left side with head-down position.
- **d.** instruct the patient to change positions, raise arm, and cough.

1. d, 2. a, 3a. a, b, e, 3b. a, 3c. c, 3d. c, 3e. a, d, e, 3f. b, 4. a, 5. d, 6. c, 7. d

For rationales to these answers and even more NCLEX review questions, visit *http://evolve.elsevier.com/Lewis/medsurg*.

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Review Questions (Online Only)
Key Points
Answer Keys for Questions
- Answer Guidelines for Case Study on p. 297
- Rationales for Bridge to NCLEX Examination Questions
- Answer Guidelines for Managing Care of Multiple Patients Case Study (Section 2) on p. 299

Student Case Study
- Patient With Hyponatremia/Fluid Volume Imbalance

Conceptual Care Map Creator
Audio Glossary
Fluids and Electrolytes Tutorial
Content Updates

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*Evidence-based information for clinical practice.

CASE STUDY

Managing Care of Multiple Patients

You and another RN are assigned to care for the following three patients in the intensive care unit (ICU). There is also a UAP (unlicensed assistive personnel) on duty.

Patients

(©Stockphoto/ Thinkstock)

G.N., a 65-yr-old African American man, was admitted to the ICU after falling out of a tree onto a lit gas grill. He has partial-thickness burns on his face, neck, and upper trunk. He has undergone surgical repair of right tibia and left hip fractures as well as debridement of a severely lacerated right leg. His voice is slightly hoarse and he is coughing up sooty sputum. He is receiving O_2 at 4 L/min via nasal cannula, and his O_2 saturation is 93%. His WBC count is 26,400/μL (26.4 × 10^9/L) with 80% neutrophils (10% bands).

(©Stockphoto/ Thinkstock)

J.N., a 35-yr-old African American woman, was transferred to the ICU from the clinical unit last evening with acute respiratory failure. She was diagnosed 2 days ago with AIDS and *Pneumocystis jiroveci* pneumonia (PCP). Prior to this hospital admission she had consistently refused antiretroviral therapy (ART) because she could not afford it. She was started on oral trimethoprim/sulfamethoxazole (Bactrim) and combination antiretroviral therapy. However, her respiratory distress worsened and she was transferred to the ICU for intubation and mechanical ventilation. She is now receiving IV Bactrim.

(© Kevin Peterson/ Photodisc/ Thinkstock)

S.S., a 63-yr-old white woman with acute lymphocytic leukemia, has been receiving chemotherapy on an outpatient basis. She completed her third treatment 5 days ago and has been experiencing nausea and vomiting for 2 days despite using ondansetron (Zofran). She was initially admitted to the clinical unit but was transferred to the ICU for close monitoring after becoming severely hypotensive overnight. Her most recent blood pressure was 98/50, HR 82 and regular, O_2 saturation 95% on 2L via nasal cannula, RR 22 and regular.

Discussion Questions

1. ***Priority Decision:*** After receiving report, which patient should you see first? Provide rationale.
2. ***Teamwork and Collaboration:*** Which morning tasks should you delegate to the UAP *(select all that apply)*?
   a. Take blood pressure readings on S.S.
   b. Perform respiratory assessment on G.N.
   c. Document strict intake and output on S.S.
   d. Provide oral care around the endotracheal tube for J.N.
   e. Titrate S.S.'s IV infusion rate based on her blood pressure reading.
3. ***Priority and Teamwork and Collaboration:*** When you enter J.N.'s room, the ventilator alarms are going off. The UAP had just finished providing oral care and she tells you that J.N. has become increasingly agitated within the last 5 minutes. Which *initial* action would be *most* appropriate?
   a. Suction J.N.'s endotracheal tube.
   b. Have the UAP stay with J.N. while you obtain IV sedation for her.
   c. Ask the UAP to describe exactly what she did when she was performing oral care.
   d. Assess the ventilator to identify what alarm is going off and to make sure all connections are secure.

Case Study Progression

J.N.'s ventilator tubing became disconnected. You quickly reconnect her tubing, and as she settles down her oxygenation improves. You ask the UAP to obtain vital signs on all your patients while you begin a more thorough assessment of J.N.

4. During your assessment of J.N., you note gray-white patches on the inside of her mouth. You recognize these as *most* likely caused by
   a. *Candida albicans*.
   b. poor oral hygiene.
   c. *Coccidioides immitis*.
   d. irritation from recent mouth care.
5. G.N.'s family asks you why surgery was performed on his leg wound. Your response is based on the knowledge that debridement *(select all that apply)*
   a. is used to remove infected tissue.
   b. is used to remove nonviable tissue.
   c. prepares the wound bed for healing.
   d. can only be accomplished in surgery.
   e. is necessary in any burn wound involving the skin.
6. S.S.'s morning laboratory results reveal a serum potassium level of 2.8 mEq/L. You notify the health care provider and obtain an order for IV potassium. During infusion of the potassium replacement, you prioritize assessment of S.S.'s
   a. bowel function.
   b. cardiac rhythm.
   c. muscle strength.
   d. level of consciousness.
7. ***Management Decision:*** While sitting at the computer charting your patients' morning assessments, you overhear the UAP telling a nursing student that J.N. deserves what she got because HIV is God's way of punishing those who sin. Your *most* appropriate response would be to
   a. report the UAP's actions to your supervisor.
   b. ask the UAP to keep her opinions to herself.
   c. ignore the conversation because it does not affect patient care.
   d. talk to the UAP and student about keeping personal feelings separate from patient care.

Answers available at http://evolve.elsevier.com/Lewis/medsurg.

SECTION 3

Perioperative Care

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I choose to listen to the river for a while, thinking river thoughts, before joining the night and the stars.

Edward Abbey