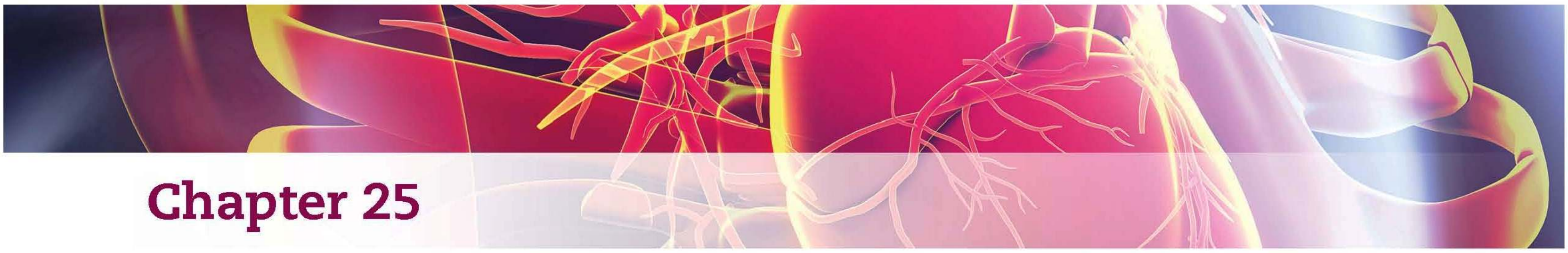

Chapter 25

Drugs for Fluid Balance, Electrolyte, and Acid–Base Disorders

Drugs at a Glance

Learning Outcomes

After reading this chapter, the student should be able to:

1. Describe conditions for which intravenous fluid therapy may be indicated.
2. Explain how changes in the osmolality or tonicity of a fluid can cause water to move between fluid compartments.
3. Compare and contrast the use of crystalloids and colloids in intravenous therapy.
4. Explain the importance of electrolyte balance in the body.
5. Explain the pharmacotherapy of sodium and potassium imbalances.
6. Discuss common causes of alkalosis and acidosis and the medications used to treat these conditions.
7. Describe the nurse's role in the pharmacologic management of fluid balance, electrolyte, and acid–base disorders.
8. For each of the classes listed in Drugs at a Glance, know representative drugs, and explain the mechanism of drug action, primary actions, and important adverse effects.
9. Use the nursing process to care for patients who are receiving pharmacotherapy for fluid balance, electrolyte, and acid–base disorders.

indicates a prototype drug, each of which is featured in a Prototype Drug box.

The volume and composition of fluids in the body must be maintained within narrow limits. Excess fluid volume can lead to hypertension (HTN), congestive heart failure (CHF), or peripheral edema, whereas depletion results in dehydration and perhaps shock. Body fluids must also contain specific amounts of essential ions or electrolytes and be maintained at particular pH values. Accumulation of excess acids or bases can change the pH of body fluids and rapidly result in death if left untreated. This chapter examines drugs used to reverse fluid balance, electrolyte, or acid–base disorders.

FLUID BALANCE

Body fluids travel between compartments, which are separated by semipermeable membranes. Control of water balance in the various compartments is essential to homeostasis. Fluid imbalances are frequent indications for pharmacotherapy.

25.1 Body Fluid Compartments

The greatest bulk of body fluid consists of water, which serves as the universal solvent in which most nutrients, electrolytes, and minerals are dissolved. Water alone is responsible for about 60% of the total body weight in a middle-age adult. A newborn may contain 80% water, whereas an older adult may contain only 40%.

In a simple model, water in the body can be located in one of two places, or compartments. The **intracellular fluid (ICF) compartment,** which contains water that is *inside* cells, accounts for about two thirds of the total body water. The remaining one third of body fluid resides *outside* cells in the **extracellular fluid (ECF) compartment.** The ECF compartment is further divided into two parts: fluid in the plasma, or intravascular space, and fluid in the interstitial spaces between cells. The relationship between these fluid compartments is illustrated in Figure 25.1.

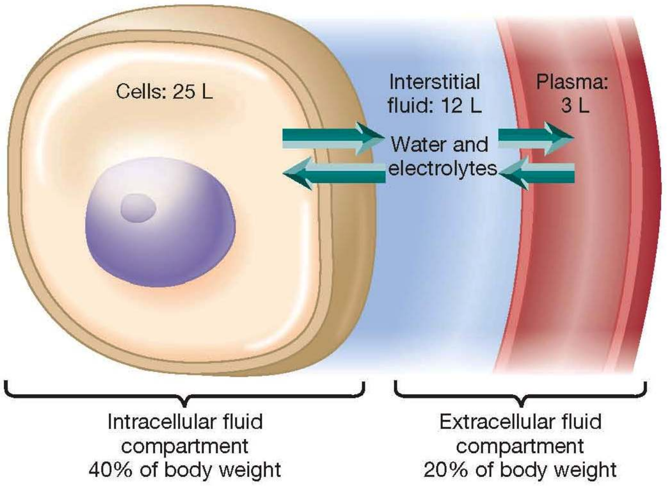

FIGURE 25.1 Major fluid compartments in the body

There is a continuous exchange and mixing of fluids between the various compartments, which are separated by membranes. For example, the plasma membranes of cells separate the ICF from the ECF. The capillary membranes separate plasma from the interstitial fluid. Although water travels freely among the compartments, the movement of large molecules and those with electrical charges is governed by processes of diffusion and active transport. Movement of ions and drugs across membranes is a primary concept of pharmacokinetics (see chapter 4).

25.2 Osmolality, Tonicity, and the Movement of Body Fluids

Osmolality and tonicity are two related terms central to understanding fluid balance in the body. Large changes in the osmolality or tonicity of a body fluid can cause significant shifts in water balance between compartments. Nurses often administer intravenous (IV) fluids to compensate for these changes.

The **osmolality** of a fluid is a measure of the number of dissolved particles, or solutes, in 1 kg (1 L) of water. In most body fluids, three solutes determine the osmolality: sodium, glucose, and urea. Sodium is the greatest contributor to osmolality due to its abundance in most body fluids. The normal osmolality of body fluids ranges from 275 to 295 milliosmols per kilogram (mOsm/kg).

The term **tonicity** is sometimes used interchangeably with osmolality, although they are somewhat different. Tonicity is the ability of a solution to cause a change in water movement across a membrane due to osmotic forces. Whereas osmolality is a laboratory value that can be precisely measured, tonicity is a general term used to describe the *relative* concentration of IV fluids. The tonicity of the plasma is used as the reference point when administering IV solutions: Normal plasma is considered isotonic. Solutions that have the same concentration of solutes as plasma are called *isotonic*. *Hypertonic* solutions contain a greater concentration of solutes than plasma, whereas *hypotonic* solutions have a lesser concentration of solutes than plasma.

Through **osmosis,** water moves from areas of low solute concentration (low osmolality) to areas of high solute concentration (high osmolality). If a *hypertonic* (hyperosmolar) IV solution is administered, the plasma gains more solutes than the interstitial fluid. Water will move, by osmosis, from the interstitial fluid compartment to the plasma compartment. This type of fluid shift removes water from cells and can result in dehydration. Water will move in the opposite direction, from plasma to interstitial fluid, if a *hypotonic* solution is administered. This type of fluid shift could result in hypotension due to movement of water out of the vascular system. Isotonic solutions produce no net fluid shift when infused. These movements are illustrated in Figure 25.2.

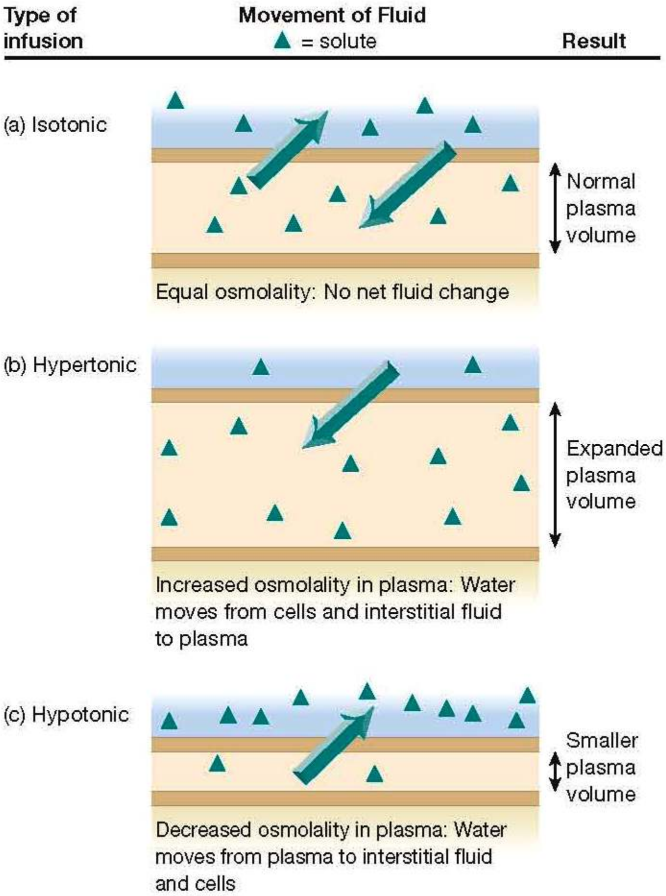

FIGURE 25.2 Movement of fluids and solution tonicity

25.3 Regulation of Fluid Intake and Output

The average adult has a water *intake* of approximately 2,500 mL/day, most of which comes from ingested food and beverages. Water *output* is achieved through the kidneys, lungs, skin, feces, and sweat. To maintain water balance, water intake must equal water output. Net gains or losses of water can be estimated by changes in total body weight.

The most important physiological regulator of fluid intake is the thirst mechanism. The sensation of thirst occurs when osmoreceptors in the hypothalamus sense that the ECF has become hypertonic. Saliva secretion diminishes and the mouth dries, driving the individual to drink liquids. As the ingested water is absorbed, the osmolality of the ECF falls and the thirst center in the hypothalamus is no longer stimulated.

The kidneys are the primary regulators of fluid output. Through activation of the renin–angiotensin–aldosterone system, the hormone aldosterone is secreted by the adrenal cortex. Aldosterone causes the kidneys to retain additional sodium and water in the body, thus increasing the osmolality of the ECF. A second hormone, antidiuretic hormone (ADH), is released by the pituitary gland during periods of high plasma osmolality. ADH acts directly on the distal tubules of the kidney to increase water reabsorption. This increased water in the intravascular space dilutes the plasma, thus lowering its osmolality.

Failure to maintain proper balance between intake and output can result in fluid balance disorders that are indications for pharmacologic intervention. Fluid *deficit* disorders can cause dehydration or shock, which are treated by administering oral or IV fluids. Fluid *excess* disorders are treated with diuretics (see chapter 24). In the treatment of fluid imbalances, the ultimate goal is to diagnose and correct the *cause* of the disorder while administering supporting fluids and medications to stabilize the patient.

FLUID REPLACEMENT AGENTS

Net loss of fluids from the body can result in dehydration and shock. IV fluid therapy is used to maintain blood volume and support blood pressure.

25.4 Intravenous Therapy With Crystalloids and Colloids

When fluid output exceeds fluid intake, volume deficits may result. Shock, dehydration, or electrolyte loss may occur; large deficits are fatal, unless treated. The following are some common reasons for fluid depletion:

- Loss of gastrointestinal (GI) fluids due to vomiting, diarrhea, chronic laxative use, or GI suctioning
- Excessive sweating during hot weather, athletic activity, or prolonged fever
- Severe burns
- Hemorrhage
- Excessive diuresis due to diuretic therapy or uncontrolled diabetic ketoacidosis.

The immediate goal in treating a volume deficit disorder is to replace the depleted fluid. Replacement of depleted fluids should always be conducted in a controlled, stepwise manner because infusing fluids too rapidly can cause fluid overload, pulmonary edema, and cardiovascular stress. In nonacute circumstances, replacement is best achieved by drinking more liquids or by administering fluids via a feeding tube. In acute situations, IV fluid therapy is indicated. Regardless of the route, careful attention must be paid to restoring normal levels of blood elements and electrolytes as well as fluid volume. IV replacement fluids are of two basic types: crystalloids and colloids. The use of blood products in treating volume depletion due to hemorrhage is presented in chapter 29.

Crystalloids

Crystalloids are IV solutions that contain electrolytes and other substances that closely mimic the body's ECF. They are used to replace depleted fluids and to promote urine output. Crystalloid solutions are capable of quickly diffusing

Table 25.1 Selected Crystalloid IV Solutions

Drug	Tonicity
Normal saline (0.9% NaCl)	Isotonic
Hypertonic saline (3% NaCl)	Hypertonic
Hypotonic saline (0.45% NaCl)	Hypotonic
Lactated Ringer's	Isotonic
Plasma-Lyte 148	Isotonic
Plasma-Lyte 56	Hypotonic
DEXTROSE SOLUTIONS	
5% dextrose in water (D_5W)	Isotonic*
5% dextrose in normal saline	Hypertonic
5% dextrose in 0.2% normal saline	Isotonic
5% dextrose in lactated Ringer's	Hypertonic
5% dextrose in Plasma-Lyte 56	Hypertonic

Note: *Because dextrose is metabolized quickly, the solution is sometimes considered hypotonic.

across membranes, thus leaving the plasma and entering the interstitial fluid and ICF. An estimated two thirds of infused crystalloids will distribute in the interstitial space. Isotonic, hypotonic, and hypertonic solutions are available. Sodium is the most common crystalloid added to solutions. Some crystalloids contain dextrose, a form of glucose, commonly in concentrations of 2.5%, 5%, or 10%. Dextrose is added to provide nutritional value: 1 L of 5% dextrose supplies 170 calories. In addition, water is formed during the metabolism of dextrose, enhancing the rehydration of the patient. When dextrose is infused, it is metabolized, and the solution becomes hypotonic. Selected crystalloids are listed in Table 25.1.

Infusion of crystalloids will increase total fluid volume in the body, but the compartment that is most expanded depends on the solute (sodium) concentration of the fluid administered. *Isotonic* crystalloids can expand the circulating intravascular (plasma) fluid volume without causing major fluid shifts between compartments. They are primarily used for hydration and to expand ECF volume. Isotonic crystalloids such as normal saline (NS) are often used to treat fluid loss due to vomiting, diarrhea, or surgical procedures, especially when the blood pressure is low. Because isotonic crystalloids can rapidly expand circulating blood volume, care must be taken not to cause fluid overload in the patient.

Infusion of *hypertonic* crystalloids expands plasma volume by drawing water away from the cells and tissues. These agents are used to relieve cellular edema, especially cerebral edema. When patients are dehydrated and have hypertonic plasma, a solution that is initially hypertonic may be infused, such as D_5 0.45% NS, that matches the tonicity of the plasma. This allows the fluid to enter the vascular compartment without causing a net fluid loss or gain in the cells. As the dextrose is subsequently metabolized, the solution becomes hypotonic. This hypotonic solution then causes water to shift into the intracellular space, relieving the dehydration within the cells. A solution of 3% NS is hypertonic and usually reserved for treating severe hyponatremia. Overtreatment with hypertonic crystalloids such as 3% NS can lead to excessive expansion of the intravascular (plasma) compartment, fluid overload, and hypertension.

Hypotonic crystalloids will cause water to move out of the plasma to the tissues and cells in the *intracellular* compartment; thus, these solutions are not considered efficient plasma volume expanders. Hypotonic crystalloids are indicated for patients with hypernatremia and cellular dehydration. Care must be taken not to cause depletion of the intravascular compartment (hypotension) or too much expansion of the intracellular compartment (peripheral edema). Patients who are dehydrated with *low* blood pressure should be given NS; patients who are dehydrated with *normal* blood pressure should be given a hypotonic solution.

Colloids

Colloids are proteins, starches, or other large molecules that remain in the blood for a long time because they are too large to easily cross the capillary membranes. While circulating, they have the same effect as hypertonic solutions, drawing water molecules from the cells and tissues into the plasma through their ability to increase plasma osmolality and osmotic pressure. Sometimes called *plasma volume expanders*, these solutions are particularly important in treating hypovolemic shock due to burns, hemorrhage, or surgery.

The most commonly used colloid is normal serum albumin, which is featured as a prototype drug for shock in chapter 29. Several colloid products contain dextran, a synthetic polysaccharide. Dextran infusions can double the plasma volume within a few minutes, although its effects last only about 12 hours. Plasma protein fraction is a natural volume expander that contains 83% albumin and 17% plasma globulins. Plasma protein fraction and albumin are also indicated in patients with hypoproteinemia. Hetastarch is a synthetic colloid with properties similar to those of 5% albumin, but with an extended duration of action. Selected colloid solutions are listed in Table 25.2.

Table 25.2 Selected Colloid Solutions (Plasma Volume Expanders)

Drug	Tonicity
5% albumin	Isotonic
Dextran 40 in normal saline	Isotonic
Dextran 40 in D_5W	Isotonic
Dextran 70 in normal saline	Isotonic
Hetastarch 6% in normal saline	Isotonic
Plasma protein fraction	Isotonic

Prototype Drug | Dextran 40 *(Gentran 40, LMD others)*

Therapeutic Class: Plasma volume expander **Pharmacologic Class:** Colloid

Actions and Uses

Dextran 40 is a polysaccharide that is too large to pass through capillary walls. It is similar to dextran 70, except dextran 40 has a lower molecular weight. Dextran 40 acts by raising the osmotic pressure of the blood, thereby causing fluid to move from the interstitial spaces of the tissues to the intravascular space (blood). Given as an IV infusion, it has the capability of expanding plasma volume within minutes after administration. Cardiovascular responses include increased blood pressure, increased cardiac output, and improved venous return to the heart. Dextran 40 is excreted rapidly by the kidneys. Indications include fluid replacement for patients experiencing hypovolemic shock due to hemorrhage, surgery, or severe burns. When given for acute shock, it is infused as rapidly as possible until blood volume is restored.

Dextran 40 also reduces platelet adhesiveness and improves blood flow through its ability to reduce blood viscosity. These properties have led to its use in preventing deep vein thromboses and postoperative pulmonary emboli.

Administration Alerts

- Emergency administration may be given 1.2 to 2.4 g/min.
- Nonemergency administration should be infused no faster than 240 mg/min.
- Discard unused portions once opened because dextran contains no preservatives.
- Pregnancy category C.

PHARMACOKINETICS

Onset	Peak	Duration
Several minutes	Unknown	12–24 h

Adverse Effects

Vital signs should be monitored continuously during dextran 40 infusions to prevent hypertension caused by plasma volume expansion. Signs of fluid overload include tachycardia, peripheral edema, distended neck veins, dyspnea, or cough. A small percentage of patients are allergic to dextran 40, including the possibility of anaphylaxis. The drug should be discontinued immediately if signs of hypersensitivity are suspected.

Contraindications: Dextran 40 is contraindicated in patients with renal failure or severe dehydration. Other contraindications include severe CHF and hypervolemic disorders.

Interactions

Drug–Drug: There are no clinically significant interactions.

Lab Tests: Dextran 40 may prolong bleeding time.

Herbal/Food: Unknown.

Treatment of Overdose: For patients with normal renal function, discontinuation of the infusion will result in reduction of adverse effects. Patients with renal impairment may benefit from the administration of an osmotic diuretic.

ELECTROLYTES

Electrolytes are small charged molecules essential to homeostasis. Too little or too much of an electrolyte can result in serious complications and must be quickly corrected. Table 25.3 describes electrolytes that are important to human physiology.

Table 25.3 Electrolytes Important to Human Physiology

Compound	Formula	Cation	Anion
Calcium chloride	$CaCl_2$	Ca^{2+}	$2Cl^-$
Disodium phosphate	Na_2HPO_4	$2Na^+$	HPO_4^{2-}
Potassium chloride	KCl	K^+	Cl^-
Sodium bicarbonate	$NaHCO_3$	Na^+	HPO_3^-
Sodium chloride	$NaCl$	Na^+	Cl^-
Sodium sulfate	Na_2SO_4	$2Na^+$	SO_4^{2-}

25.5 Physiological Role of Electrolytes

Minerals are inorganic substances needed in very small amounts to maintain homeostasis. Minerals are held together by ionic bonds and dissociate, or ionize, when placed in water. The resulting ions have positive or negative charges and are able to conduct electricity, hence the name **electrolyte.** Positively charged electrolytes are called **cations;** those with a negative charge are **anions.** Electrolyte levels are measured in units of milliequivalents per liter (mEq/L).

Electrolytes are essential to many body functions, including nerve conduction, membrane permeability, muscle contraction, water balance, and bone growth and remodeling. Levels of electrolytes in body fluids are maintained within very narrow ranges, primarily by the kidneys and GI tract. As electrolytes are lost due to normal excretory functions, they must be replaced by adequate intake;

Table 25.4 Electrolyte Imbalances

Ion	Condition	Abnormal Serum Value (mEq/L)	Supportive Treatment*
Calcium	Hypercalcemia	Greater than 11	Hypotonic fluids or calcitonin
	Hypocalcemia	Less than 4	Calcium supplements or vitamin D
Chloride	Hyperchloremia	Greater than 112	Hypotonic fluid
	Hypochloremia	Less than 95	Hypertonic salt solution
Magnesium	Hypermagnesemia	Greater than 4	Hypotonic fluid
	Hypomagnesemia	Less than 0.8	Magnesium supplements
Phosphate	Hyperphosphatemia	Greater than 6	Dietary phosphate restriction
	Hypophosphatemia	Less than 1	Phosphate supplements
Potassium	Hyperkalemia	Greater than 5	Hypotonic fluid, buffers, or dietary potassium restriction
	Hypokalemia	Less than 3.5	Potassium supplements
Sodium	Hypernatremia	Greater than 145	Hypotonic fluid or dietary sodium restriction
	Hyponatremia	Less than 135	Hypertonic salt solution or sodium supplement

Note: *For all electrolyte imbalances, the primary therapeutic goal is to identify and correct the *cause* of the imbalance.

otherwise, electrolyte imbalances will result. Although imbalances can occur with any ion, Na^+, K^+, and Ca^{2+} are of greatest importance. The major body electrolyte imbalance states and their treatments are listed in Table 25.4. Calcium, phosphorous, and magnesium imbalances are discussed in chapter 43; the role of calcium in bone homeostasis is presented in chapter 48.

An electrolyte imbalance is a sign of an underlying medical condition that needs attention. Imbalances are associated with a large number of disorders, with renal impairment being the most common cause. In some cases, drug therapy itself can cause the electrolyte imbalance. For example, aggressive therapy with loop diuretics such as furosemide (Lasix) can rapidly deplete the body of sodium and potassium. The therapeutic goal is to quickly correct the electrolyte imbalance while the underlying condition is being diagnosed and treated. Treatments for electrolyte imbalances depend on the severity of the condition and range from simple adjustments in dietary intake to rapid electrolyte infusions. Serum electrolyte levels must be carefully monitored during therapy to prevent imbalances in the *opposite* direction; levels can change rapidly from hypo-concentrations to hyper-concentrations.

25.6 Pharmacotherapy of Sodium Imbalances

Sodium ion (Na^+) is the most abundant cation in extracellular fluid. Because of sodium's central roles in neuromuscular physiology, acid–base balance, and overall fluid distribution, sodium imbalances can have serious consequences. Although definite sodium monitors or sensors have yet to be discovered in the body, the regulation of sodium balance is well understood.

Sodium balance and water balance are intimately connected. As Na^+ levels increase in a body fluid, solute particles accumulate, and the osmolality increases. Water will move toward this area of relatively high osmolality. In simplest terms, water travels toward or with Na^+. The physiological consequences of this relationship cannot be overstated: As the Na^+ and water content of plasma increases, so does blood volume and blood pressure. Thus, Na^+ movement provides an important link between water retention, blood volume, and blood pressure.

In healthy individuals, the kidney regulates sodium intake to be equal to sodium output. High levels of aldosterone secreted by the adrenal cortex promote Na^+ and water retention by the kidneys as well as K^+ excretion in the urine. Inhibition of aldosterone promotes sodium and water excretion. When a patient ingests high amounts of sodium, aldosterone secretion decreases, thus allowing excess Na^+ to enter the urine. This relationship is illustrated in Figure 25.3.

Hypernatremia

Sodium excess, or **hypernatremia,** occurs when the serum sodium level rises above 145 mEq/L. The most common cause of hypernatremia is decreased Na^+ excretion due to kidney disease. Hypernatremia may also be caused by excessive intake of sodium, either through dietary consumption or by overtreatment with IV fluids containing sodium chloride or sodium bicarbonate. Another cause of hypernatremia is high net water losses, such as occur from inadequate water intake, watery diarrhea, fever, or burns. High doses of corticosteroids or estrogens also promote Na^+ retention.

A high serum sodium level increases the osmolality of the plasma, drawing fluid from interstitial spaces and cells, thus causing cellular dehydration. Manifestations of hypernatremia include thirst, fatigue, weakness, muscle twitching, convulsions, altered mental status, and a decreased level of consciousness. For minor hypernatremia, a low-salt diet may be effective in returning serum sodium to normal levels. In patients with acute hypernatremia, however, the treatment goal is to rapidly return the osmolality of the

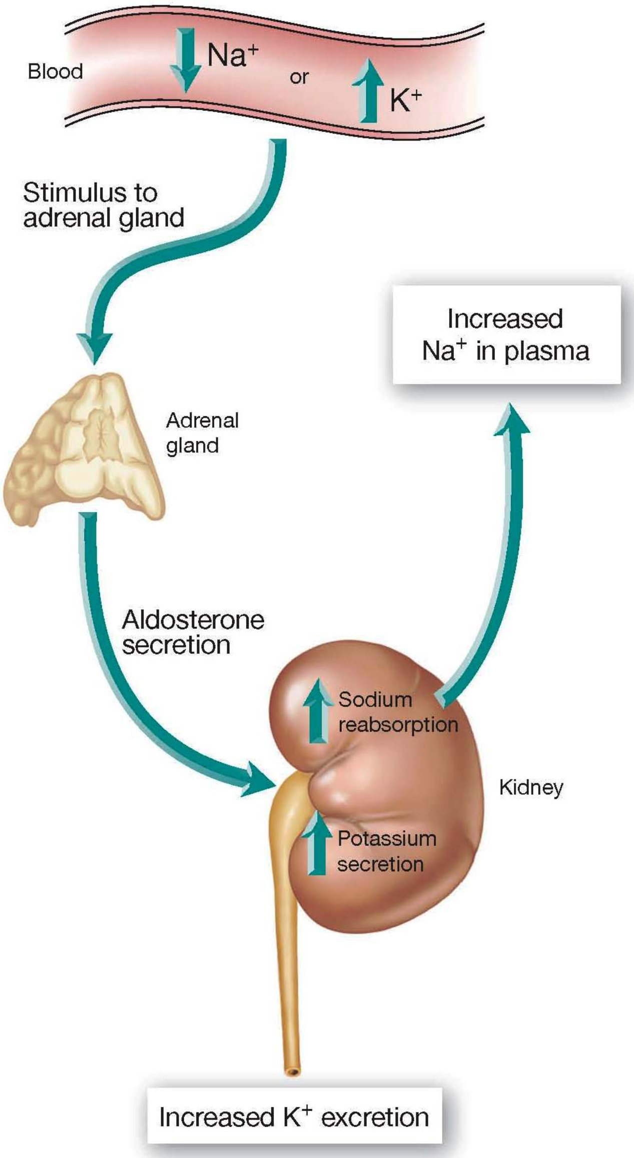

FIGURE 25.3 Renal regulation of sodium and potassium balance

plasma to normal. If the patient is hypovolemic, infusing hypotonic fluids such as 5% dextrose or 0.45% NaCl will increase plasma volume while at the same time reducing plasma osmolality. If the patient is hypervolemic, diuretics may be used to remove Na^+ and fluid from the body.

Hyponatremia

Sodium deficiency, or **hyponatremia,** is a serum sodium level less than 135 mEq/L. Hyponatremia may occur through *excessive dilution* of the plasma, caused by excessive ADH secretion or administration of hypotonic IV solutions. Hyponatremia may also result from *increased sodium loss* due to disorders of the skin, GI tract, or kidneys. Significant loss of sodium by the skin may occur in burn patients and in those experiencing excessive sweating or prolonged fever. GI sodium losses may occur from vomiting, diarrhea, or GI suctioning, and renal Na^+ loss may occur with diuretic use and in certain advanced kidney disorders. Early symptoms of hyponatremia include nausea, vomiting, anorexia, and abdominal cramping. Later signs include altered neurologic function such as confusion, lethargy, convulsions, coma, and muscle twitching or tremors.

Hyponatremia caused by excessive dilution is treated with loop diuretics (see chapter 24). These drugs will cause an isotonic diuresis, thus removing the fluid overload that caused the hyponatremia. Hyponatremia caused by Na^+ loss may be treated with oral or parenteral NaCl or with IV fluids containing salt, such as NS or lactated Ringer's. Tolvaptan (Samsca) is a newer drug approved to quickly raise serum sodium levels in patients experiencing symptoms of hyponatremia. Tolvaptan is a vasopressin (antidiuretic hormone) antagonist that enhances water excretion. As the amount of water in the blood is reduced, the serum sodium concentration increases. Therapy with the drug should only be conducted in a hospital where serum sodium levels can be monitored closely. Treatment is limited to 30 days due to the risk for liver injury.

25.7 Pharmacotherapy of Potassium Imbalances

Potassium ion (K^+), the most abundant intracellular cation, serves important roles in regulating intracellular osmolality and in maintaining acid–base balance. Potassium levels must be carefully balanced between adequate dietary intake and renal excretion. Like Na^+ excretion, K^+ excretion is influenced by the actions of aldosterone on the kidney. In fact, the renal excretion of Na^+ and K^+ ions is closely linked—for every sodium ion that is *reabsorbed,* one potassium ion is *secreted* into the renal tubules. Serum potassium levels must be maintained within narrow limits. Both hyper- and hypokalemia are associated with fatal dysrhythmias and serious neuromuscular disorders.

Hyperkalemia

Hyperkalemia is a serum potassium level greater than 5 mEq/L, which may be caused by high consumption of potassium-rich foods or dietary supplements, particularly when patients are taking potassium-sparing diuretics such as spironolactone (see chapter 24). Excess K^+ may also accumulate when renal excretion is diminished due to kidney pathology. The most serious consequences of hyperkalemia are related to cardiac function: dysrhythmias and heart block. Other symptoms are muscle twitching, fatigue, paresthesias, dyspnea, cramping, and diarrhea.

In mild cases of hyperkalemia, K^+ levels may be returned to normal by restricting primary dietary sources of potassium such as bananas, citrus and dried fruits, peanut butter, broccoli, and green leafy vegetables. If the patient is taking a potassium-sparing diuretic, the dose must be lowered, or a thiazide or loop diuretic must be substituted. In severe cases, serum K^+ levels may be temporarily lowered

Prototype Drug | Sodium Chloride *(NaCl)*

Therapeutic Class: Drug for hyponatremia **Pharmacologic Class:** Electrolyte, sodium supplement

Actions and Uses

Sodium chloride is administered for hyponatremia when serum levels fall below 130 mEq/L. Normal saline consists of 0.9% NaCl, and it is used to treat mild hyponatremia. When serum sodium falls below 115 mEq/L, a highly concentrated 3% NaCl solution may be infused. Other concentrations include 0.45% and 0.22%, and both hypotonic and isotonic solutions are available. For less severe hyponatremia, 1 g tablets are available by the oral (PO) route.

Ophthalmic solutions of NaCl may be used to treat corneal edema, and an over-the-counter (OTC) nasal spray is available to relieve dry, inflamed nasal membranes. In conjunction with oxytocin, 20% NaCl may be used as an abortifacient late in pregnancy when instilled into the amniotic sac.

Administration Alerts

- Pregnancy category C.

Pharmacokinetics

Because sodium ion is a natural electrolyte, it is not possible to obtain accurate pharmacokinetic values.

Adverse Effects

Patients receiving NaCl infusions must be monitored frequently to prevent symptoms of hypernatremia, which include lethargy, confusion, muscle tremor or rigidity, hypotension, and restlessness. Because some of these symptoms are also common to hyponatremia, periodic laboratory assessments must be taken to be certain that sodium values lie within the normal range. When infusing 3% NaCl solutions, nurses should continuously check for signs of pulmonary edema.

Contraindications: This drug should not be administered to patients with hypernatremia, heart failure, or impaired renal function.

Interactions

Drug–Drug: There are no clinically significant drug interactions.

Lab Tests: NaCl increases the serum sodium level.

Herbal/Food: Unknown.

Treatment of Overdose: If fluid accumulation occurs due to excess sodium, diuretics may be administered to reduce pulmonary or peripheral edema.

Community-Oriented Practice

MAINTAINING FLUID BALANCE DURING EXERCISE

Hyponatremia from excessive fluid intake has been noted as a growing problem in athletes, particularly novice athletes who may have heard that they need to "keep drinking" to maintain hydration. Many sports drinks contain some electrolytes but are also high in fructose or other sugars. This creates a hypertonic solution that may paradoxically cause increased water loss. Unless exercise is extreme or prolonged, athletes, especially children, should be encouraged to drink when thirsty and maintain urine at a color of clear yellow, not dark yellow or colorless. Adequate fluid intake to match thirst will help ensure normal hydration and sodium levels and prevent complications such as exercise-associated hyponatremia.

by administering glucose and regular insulin IV, or aerosolized albuterol which cause K^+ to leave the extracellular fluid and enter cells. Calcium gluconate or calcium chloride may be administered to counteract K^+ toxicity to the heart. Sodium bicarbonate is sometimes infused to correct any acidosis that may be concurrent with the hyperkalemia. Excess K^+ may be eliminated by giving polystyrene sulfonate (Kayexalate) PO or rectally. This agent, which is not absorbed, exchanges Na^+ for K^+ as it travels through the intestine. The onset of action is 1 hour, and the dose may be repeated every 4 hours as needed. This drug is given concurrently with a laxative such as sorbitol to promote rapid evacuation of the potassium.

Hypokalemia

Hypokalemia occurs when the serum potassium level falls below 3.5 mEq/L. Hypokalemia is a frequent adverse effect resulting from high doses of loop diuretics such as furosemide (Lasix). In addition, strenuous muscular activity and severe vomiting or diarrhea can result in significant K^+ loss. Because the body does not have large stores of K^+, adequate daily intake is necessary. Neurons and muscle fibers are most sensitive to K^+ loss, and muscle weakness, lethargy, anorexia, dysrhythmias, and cardiac arrest are possible consequences.

Mild hypokalemia is treated by increasing the dietary intake of potassium-rich foods such as dried fruit, nuts, molasses, avocados, lima beans, and bran cereals. If increasing dietary intake is not possible, a large number of oral potassium supplements are available. Liquid preparations are very effective, although many must be diluted with water or fruit juices prior to administration. Extended release (K-Dur 20, Slow-K, Micro-K) and powders (Klor-Con) are also available. Severe deficiencies require doses of parenteral potassium supplements.

Prototype Drug | Potassium Chloride (KCl)

Therapeutic Class: Drug for hypokalemia **Pharmacologic Class:** Electrolyte, potassium supplement

Actions and Uses

Potassium chloride is the drug of choice for preventing or treating hypokalemia. It is also used to treat mild forms of alkalosis. Oral formulations include tablets, powders, and liquids, usually heavily flavored due to the unpleasant taste of the drug. Because potassium supplements can cause peptic ulcers, the drug should be diluted with plenty of water. When given IV, potassium must be administered slowly, since bolus injections can overload the heart and cause cardiac arrest. Because pharmacotherapy with loop or thiazide diuretics is the most common cause of K^+ depletion, patients taking these drugs are usually prescribed oral potassium supplements to prevent hypokalemia.

Administration Alerts

- Always give oral medication while the patient is upright to prevent esophagitis.
- Do not crush tablets or allow the patient to chew tablets.
- Dilute liquid forms before giving PO or through a nasogastric tube.
- Never administer IV push or in concentrated amounts, and do not exceed an IV rate of 10 mEq/h.
- Be extremely careful to avoid extravasation and infiltration.
- Pregnancy category A.

Pharmacokinetics

Because potassium ion is a natural electrolyte, it is not possible to obtain accurate pharmacokinetic values.

Adverse Effects

Nausea and vomiting are common, because potassium chloride irritates the GI mucosa when administered PO. The drug may be taken with meals or antacids to lessen gastric distress. When administered IV, phlebitis and venous irritation can occur. The drug should preferably be administered through a larger vessel to minimize this risk. The most serious adverse effects of potassium chloride are related to the possible accumulation of excess K^+. Hyperkalemia may occur if the patient takes potassium supplements concurrently with potassium-sparing diuretics. Because the kidneys perform more than 90% of the body's potassium excretion, reduced renal function can rapidly lead to hyperkalemia, particularly in patients taking potassium supplements.

Contraindications: Potassium chloride is contraindicated in patients with hyperkalemia, chronic renal failure, systemic acidosis, severe dehydration, extensive tissue breakdown as in severe burns, adrenal insufficiency, or the administration of a potassium-sparing diuretic.

Interactions

Drug–Drug: Potassium supplements interact with potassium-sparing diuretics and angiotensin-converting enzyme (ACE) inhibitors to increase the risk for hyperkalemia.

Lab Tests: Potassium chloride increases the serum potassium level.

Herbal/Food: Unknown.

Treatment of Overdose: When overdose is suspected, potassium-sparing diuretics and all foods and medications containing significant amounts of potassium should be withheld. Treatment includes IV administration of 50% dextrose solution containing 10–20 units of regular insulin. Sodium bicarbonate may be infused to correct acidosis. Polystyrene sulfonate may be administered to enhance potassium elimination.

☑ Check Your Understanding 25.1

What are the two most common causes of electrolyte imbalances? *See appendix C for the answer.*

ACID–BASE IMBALANCE

Acidosis (excess acid) and alkalosis (excess base) are not diseases but are symptoms of an underlying disorder. Acidic and basic agents may be administered to rapidly correct pH imbalances in body fluids, supporting the patient's vital functions while the underlying disease is being treated. The correction of acid–base imbalance is illustrated in Figure 25.4.

25.8 Buffers and the Maintenance of Body pH

The degree of acidity or alkalinity of a solution is measured by its **pH.** A pH of 7.0 is defined as neutral, above 7.0 as basic or alkaline, and below 7.0 as acidic. To maintain homeostasis, the pH of plasma and most body fluids must be kept within the narrow range of 7.35 to 7.45. Nearly all proteins and enzymes in the body function optimally within this narrow range of pH values. A few enzymes, most notably those in the digestive tract, require pH values outside the 7.35 to 7.45 range to function properly.

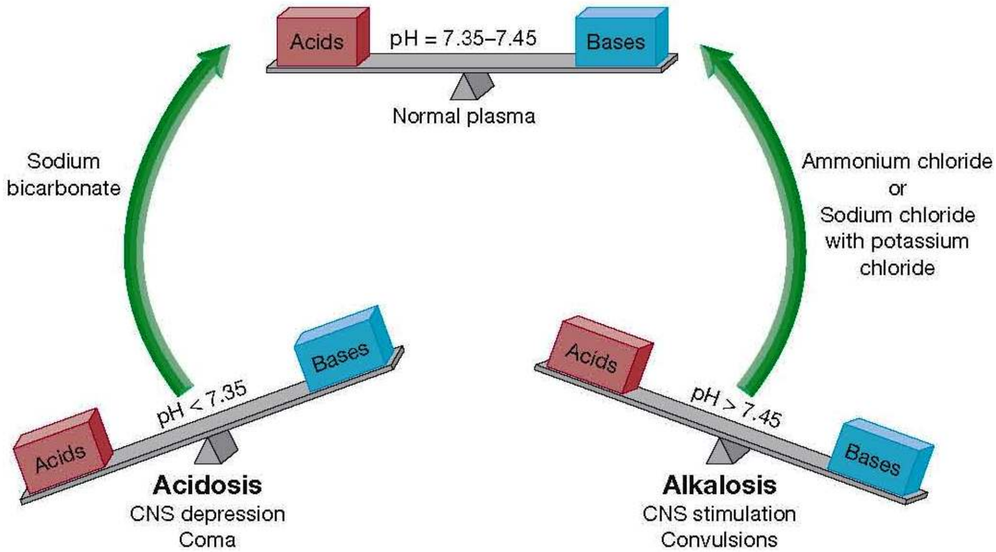

FIGURE 25.4 Acid–base imbalances

Nursing Practice Application

Intravenous Fluid and Electrolyte Replacement Therapy

ASSESSMENT	POTENTIAL NURSING DIAGNOSES*
Baseline assessment prior to administration: • Obtain a complete health history including cardiovascular (including HTN, myocardial infarction [MI]), neurologic (including cerebrovascular accident [CVA] or head injury), burns, endocrine, and hepatic or renal disease. Obtain a drug history including allergies, current prescription and OTC drugs, and herbal preparations. Be alert to possible drug interactions. • Obtain baseline weight and vital signs, level of consciousness (LOC), breath sounds, and urinary output as appropriate. • Evaluate appropriate laboratory findings (e.g., electrolytes, complete blood count [CBC], urine specific gravity and urinalysis, blood urea nitrogen [BUN] and creatinine, total protein and albumin levels, activated partial thromboplastin time (aPTT), antiprothrombin antibodies (aPT), or international normalized ratio (INR), renal and liver function studies).	• *Deficient Fluid Volume* • *Decreased Cardiac Output* • *Fatigue* • *Activity Intolerance* • *Deficient Knowledge* (drug therapy) • *Risk for Falls,* related to hypotension, dizziness associated with adverse effects • *Risk for Injury,* related to hypotension, dizziness associated with adverse effects • *Risk for Deficient Fluid Volume* • *Risk for Excessive Fluid Volume*, related to drug therapy • *Risk for Electrolyte Imbalance* • *Risk for Ineffective Health Management* *NANDA I © 2014
Assessment throughout administration: • Assess for desired therapeutic effects (e.g., electrolyte values return to within normal range, adequate urine output). • Continue monitoring of vital signs, urinary output, and LOC as appropriate. • Assess for and promptly report adverse effects: tachycardia, HTN, dysrhythmias, decreasing LOC, increasing dyspnea, lung congestion, pink-tinged frothy sputum, decreased urinary output, muscle weakness or cramping, or allergic reactions.	

continued

Nursing Practice Application *continued*

IMPLEMENTATION	
Interventions and (Rationales)	**Patient-Centered Care**
Ensuring therapeutic effects: • Continue frequent assessments as described earlier for therapeutic effects. Assist the patient with obtaining fluids and with eating as needed. (Urinary output is within normal limits. Electrolyte balance is restored. **Lifespan:** The older adult, infants, and patients who cannot access fluids or eat by themselves, e.g., post-stroke, are at increased risk for fluid and electrolyte imbalance.)	• Teach the patient to continue to consume enough liquids to remain adequately, but not overly, hydrated. Drinking when thirsty, avoiding alcoholic beverages, maintaining a healthy diet, and ensuring adequate but not excessive salt intake will assist in maintaining normal fluid and electrolyte balance. • Have the patient weigh self daily and record weight along with blood pressure (BP) and pulse measurements as appropriate. • Teach the patient, family, or caregiver how to monitor pulse and BP if needed. Ensure proper use and functioning of any home equipment obtained.
Minimizing adverse effects: • Monitor for signs of fluid volume excess or deficit (e.g., increasing BP [excess], decreasing BP [deficit], tachycardia, changes in quality of pulse [bounding or thready]). Monitor for signs of potential electrolyte imbalance including nausea, vomiting, GI cramping, diarrhea, muscle weakness, cramping or twitching, paresthesias, and irritability. Confusion; decreasing LOC; increasing hypotension or HTN, especially if associated with tachycardia; decreased urine output; and seizures are reported immediately. (Many fluid and electrolyte imbalances have similar symptoms. When assessing the patient for adverse effects, consider past history, drug history, and current condition and medications to correlate symptoms to possible causes.)	• Instruct the patient to report changes in muscle strength or function; numbness and tingling in lips, fingers, arms, or legs; palpitations; dizziness; nausea or vomiting; GI cramping; or decreased urination. • Instruct patients with hypokalemia to consume foods high in potassium: fresh fruits such as strawberries and bananas, dried fruits such as apricots and prunes, vegetables and legumes such as tomatoes, beets, and beans, juices such as orange, grapefruit or prune, and fresh meats. Instruct patients with hyperkalemia to avoid the above foods as well as salt substitutes (which often contain potassium salts), and to consult with a health care provider before taking vitamin and mineral supplements or specialized sports beverages. Licorice should be avoided because it causes potassium loss and sodium retention.
• Frequently monitor CBC, electrolytes, aPTT, and aPT or INR levels. (Crystalloid solutions may cause electrolyte imbalances. Colloid solutions may reduce normal blood coagulation. Frequent monitoring of electrolyte levels while on replacement therapy may be needed to ensure therapeutic effects.)	• Instruct the patient on the need to return periodically for laboratory work.
• Continue to monitor vital signs. Take BP lying, sitting, and standing to detect orthostatic hypotension. **Lifespan:** Be cautious with older adults who are at increased risk for hypotension. (Dehydration and electrolyte imbalances may cause dizziness and hypotension. Orthostatic hypotension may increase the risk of injury.)	• **Safety:** Teach the patient to rise from lying or sitting to standing slowly to avoid dizziness or falls. If dizziness occurs, the patient should sit or lie down and not attempt to stand or walk until the sensation passes. • **Safety:** Instruct the patient to call for assistance prior to getting out of bed or attempting to walk alone, and to avoid driving or other activities requiring mental alertness or physical coordination if dizziness or lightheadedness occurs.
• Weigh the patient daily and report a weight gain or loss of 1 kg (2 lb) or more in a 24-hour period. (Daily weight is an accurate measure of fluid status and takes into account intake, output, and insensible losses. Weight gain or edema may signal excessive fluid volume or electrolyte imbalances.)	• Have the patient weigh self daily, ideally at the same time of day, and record weight along with BP and pulse measurements. Have the patient report significant weight loss or gain. • Teach the patient that excessive heat conditions contribute to excessive sweating and fluid and electrolyte loss, and extra caution is warranted in these conditions.
• **Safety:** Closely monitor for signs and symptoms of allergy if colloids are used. (Colloids may cause allergic and anaphylactic reactions.)	• Instruct the patient to immediately report dyspnea, itching, feelings of throat tightness, palpitations, chest pain or tightening, or headache.
• **Safety:** Closely monitor IV sites when infusing potassium or ammonium. Double-check doses with another nurse before giving. (Potassium and ammonium are irritating to the vessel and phlebitis may result. Potassium is a "high-alert" medication and double-checking doses before administering prevents medication errors.)	• Instruct the patient to report any irritation, pain, redness, or swelling at the IV site or in the arm where the drug is infusing.

Nursing Practice Application *continued*

IMPLEMENTATION

Interventions and (Rationales)	Patient-Centered Care
Patient understanding of drug therapy:	
• Use opportunities during administration of medications and during assessments to provide patient education. (Using time during nursing care helps to optimize and reinforce supportive drug treatment and care.)	• The patient, family, or caregiver should be able to state the reason for the drug; appropriate dose and scheduling; what adverse effects to observe for and when to report; and the anticipated length of medication therapy.
Patient self-administration of drug therapy:	
• When administering the medication, instruct the patient, family, or caregiver in proper self-administration of the drug (e.g., early in the day to prevent disruption of sleep from nocturia). (Proper administration will increase the effectiveness of the drug.)	• The patient, family, or caregiver are able to discuss appropriate dosing and administration needs. • **Lifespan:** Assess swallowing ability before the patient takes potassium chloride or ammonium chloride; they may cause mouth, esophageal, or gastric irritation. • Teach the patient that liquid forms should always be diluted with water or fruit juice and tablets swallowed whole.

See Tables 25.1, 25.2, and 25.4 for a list of the drugs to which these nursing actions apply.

The body generates significant amounts of acid during normal metabolic processes. Without sophisticated means of neutralizing these metabolic acids, the overall pH of body fluids would quickly fall below the normal range. **Buffers** are chemicals that help maintain normal body pH by neutralizing strong acids and bases. The two primary buffers in the body are bicarbonate ions and phosphate ions.

The body uses two mechanisms to remove acid. The carbon dioxide (CO_2) produced during body metabolism is an acid efficiently removed by the lungs during exhalation. The kidneys remove excess acid in the form of hydrogen ion (H^+) by excreting it in the urine. If retained in the body, CO_2 and/or H^+ would lower body pH. Thus, the lungs and the kidneys collaborate in the removal of acids to maintain normal acid–base balance.

25.9 Pharmacotherapy of Acidosis

Acidosis occurs when the pH of the plasma falls below 7.35, which is confirmed by measuring arterial pH, partial pressure of carbon dioxide (P_{CO_2}), and plasma bicarbonate levels. Diagnosis must differentiate between respiratory etiology and metabolic (renal) etiology. Occasionally, the cause has mixed respiratory and metabolic components. The most profound symptoms of acidosis affect the central nervous system (CNS) and include lethargy, confusion, and CNS depression leading to coma. A deep, rapid respiration rate indicates an attempt by the lungs to rid the body of excess acid. Common causes of acidosis are listed in Table 25.5.

In patients with acidosis, the therapeutic goal is to quickly reverse the level of acids in the blood. The preferred treatment for acute acidosis is to administer infusions of sodium bicarbonate. Bicarbonate ion acts as a base to quickly neutralize acids in the blood and other body fluids. The patient must be carefully monitored during infusions because this drug can "overcorrect" the acidosis, causing blood pH to turn alkaline. Sodium citrate, sodium lactate, and sodium acetate are alternative alkaline agents sometimes used in place of bicarbonate.

25.10 Pharmacotherapy of Alkalosis

Alkalosis develops when the plasma pH rises above 7.45. Like acidosis, alkalosis may have either respiratory or metabolic causes, as shown in Table 25.5. Also like acidosis, the CNS is greatly affected. Symptoms of CNS stimulation occur including nervousness, hyperactive reflexes, and convulsions. In metabolic alkalosis, slow, shallow breathing indicates that the body is attempting to compensate by retaining acid and lowering internal pH. Life-threatening dysrhythmias are the most serious adverse effects of alkalosis.

Table 25.5 Causes of Alkalosis and Acidosis

Acidosis	Alkalosis
RESPIRATORY ORIGINS OF ACIDOSIS	**RESPIRATORY ORIGINS OF ALKALOSIS**
Hypoventilation or shallow breathing	Hyperventilation due to asthma, anxiety, or high altitude
Airway constriction	
Damage to respiratory center in medulla	
METABOLIC ORIGINS OF ACIDOSIS	**METABOLIC ORIGINS OF ALKALOSIS**
Severe diarrhea	Constipation for prolonged periods
Kidney failure	Ingestion of excess sodium bicarbonate
Diabetes mellitus	Diuretics that cause potassium depletion
Excess alcohol ingestion	Severe vomiting
Starvation	

Prototype Drug | Sodium Bicarbonate

Therapeutic Class: Drug to treat acidosis or bicarbonate deficiency
Pharmacologic Class: Electrolyte, sodium and bicarbonate supplement

Actions and Uses

Sodium bicarbonate is a drug of choice for correcting metabolic acidosis. After dissociation, the bicarbonate ion directly raises the pH of body fluids. Sodium bicarbonate may be given orally, if acidosis is mild, or IV in cases of acute disease. IV concentrations range from 4.2% to 8.4%. Although sodium bicarbonate also neutralizes gastric acid, it is not used to treat peptic ulcers due to its tendency to cause uncomfortable gastric distention. The oral preparation of sodium bicarbonate is known as *baking soda*.

Sodium bicarbonate may also be used to alkalinize the urine and speed the excretion of acidic substances. This process is useful in the treatment of overdoses of acidic medications such as aspirin and phenobarbital, and as adjunctive therapy for certain chemotherapeutic drugs such as methotrexate.

Sodium bicarbonate may be used in chronic renal failure to neutralize the metabolic acidosis that occurs when the kidneys cannot excrete hydrogen ion. When IV sodium bicarbonate is given, it causes the urine to become more alkaline. Less acid is reabsorbed in the renal tubules, so more acid and acidic medicine is excreted. This process is known as ion trapping.

Administration Alerts

- Do not add oral preparation to calcium-containing solutions.
- Give oral sodium bicarbonate 2 to 3 hours before or after meals and other medications.
- Pregnancy category C.

PHARMACOKINETICS

Onset	Peak	Duration
15 min PO; immediate IV	2 h PO; unknown IV	1–3 h PO; 8–10 min IV

Adverse Effects

Most of the adverse effects of sodium bicarbonate therapy are the result of metabolic alkalosis caused by receiving *too much* bicarbonate ion. Symptoms may include confusion, irritability, slow respiration rate, and vomiting. Simply discontinuing the sodium bicarbonate infusion often reverses these symptoms; however, potassium chloride or ammonium chloride may be administered to reverse acute alkalosis. During sodium bicarbonate infusions, serum electrolytes should be carefully monitored, because sodium levels may give rise to hypernatremia and fluid retention. In addition, high levels of bicarbonate ion passing through the kidney tubules increase K^+ secretion, and hypokalemia is possible.

Contraindications: Patients who are vomiting or have continuous GI suctioning will lose acid and chloride and may be in a state of metabolic alkalosis; therefore, they should not receive sodium bicarbonate. Because of the sodium content of this drug, it should be used cautiously in patients with cardiac disease and renal impairment. Sodium bicarbonate is contraindicated in patients with hypertension, peptic ulcers, diarrhea, or vomiting.

Interactions

Drug–Drug: Sodium bicarbonate may decrease the absorption of ketoconazole and may decrease elimination of dextroamphetamine, ephedrine, pseudoephedrine, and quinidine. The elimination of lithium, salicylates, and tetracyclines may be increased.

Lab Tests: Urinary and serum pH increase with sodium bicarbonate administration. Urinary urobilinogen levels may increase.

Herbal/Food: Chronic use with milk or calcium supplements may cause milk–alkali syndrome, a condition characterized by serious hypercalcemia and possible kidney failure.

Treatment of Overdose: Overdose results in metabolic alkalosis, which is treated by administering acidic agents (see section 25.10).

Treatment of metabolic alkalosis is directed toward addressing the underlying condition that is causing the excess alkali to be retained. In mild cases, alkalosis may be corrected by administering NaCl concurrently with KCl. This combination increases the renal excretion of bicarbonate ion, which indirectly increases the acidity of the blood.

Patients with renal impairment or who have heart failure may not be able to tolerate the increased water load that follows NaCl infusions. For these acute patients, acidifying agents may be used. Hydrochloric acid and ammonium chloride are two drugs that can quickly lower the pH in patients with severe alkalosis.

Patient Safety: Concentrated Electrolyte Solutions

The student nurse is working with a clinical nurse preceptor and asks why they must wait for the pharmacy to deliver IV medications. "Wouldn't it just be faster to mix them ourselves? The patient in room 220 is supposed to have an IV with potassium and the IV is almost out." How should the nurse respond? *See appendix C for the suggested answer.*

Chapter Review

KEY Concepts

The numbered key concepts provide a succinct summary of the important points from the corresponding numbered section within the chapter. If any of these points are not clear, refer to the numbered section within the chapter for review.

25.1 There is a continuous exchange of fluids across membranes separating the intracellular and extracellular fluid compartments. Large molecules and those that are ionized are less able to cross membranes.

25.2 Osmolality refers to the number of dissolved solutes (usually sodium, glucose, or urea) in a body fluid. Changes in the osmolality of body fluids can cause water to move to different compartments.

25.3 Overall fluid balance is achieved through complex mechanisms that regulate fluid intake and output. The greatest contributor to osmolality is sodium, which is controlled by the hormone aldosterone.

25.4 Intravenous fluid therapy using crystalloids and colloids replaces lost fluids. Crystalloids contain electrolytes and are distributed primarily to the interstitial spaces. Colloids are large molecules that stay in the intravascular space to rapidly expand plasma volume.

25.5 Electrolytes are charged inorganic molecules that are essential to nerve conduction, membrane permeability, water balance, and other critical body functions. Imbalances may lead to serious abnormalities.

25.6 Sodium is essential to maintaining osmolality, water balance, and acid–base balance. Hypernatremia may be corrected with hypotonic IV fluids or diuretics. Hyponatremia may be treated with infusions of sodium chloride. Dilutional hyponatremia is treated with diuretics.

25.7 Potassium is essential for proper nerve and muscle function as well as for maintaining acid–base balance. Hyperkalemia may be treated with glucose and insulin or by administration of polystyrene sulfonate. Hypokalemia is corrected with oral or IV potassium supplements.

25.8 Buffers in the body maintain overall pH within narrow limits. The kidneys and lungs work together to remove excess metabolic acid.

25.9 Pharmacotherapy of acidosis, a plasma pH below 7.35, includes the administration of sodium bicarbonate.

25.10 Pharmacotherapy of alkalosis, a plasma pH above 7.45, includes the administration of sodium chloride with potassium chloride. In acute cases, an acidifying agent such as hydrochloric acid or ammonium chloride may be infused.

REVIEW Questions

1. A patient is receiving intravenous sodium bicarbonate for treatment of metabolic acidosis. During this infusion, how will the nurse monitor for therapeutic effect?
    1. Blood urea nitrogen (BUN)
    2. White blood cell counts
    3. Serum pH
    4. Renal function laboratory values
2. Which of the following nursing interventions is most important when caring for a patient receiving dextran 40 (Gentran 40, LMD)?
    1. Assess the patient for deep venous thrombosis.
    2. Observe for signs of fluid overload.
    3. Encourage fluid intake.
    4. Monitor arterial blood gases.
3. The patient's serum sodium value is 152 mEq/L. Which of the following nursing interventions is most appropriate for this patient? (Select all that apply.)
    1. Assess for inadequate water intake or diarrhea.
    2. Administer a 0.45% NaCl intravenous solution.
    3. Hold all doses of glucocorticoids.
    4. Notify the health care provider.
    5. Have the patient drink as much water as possible.
4. A patient is receiving 5% dextrose in water (D_5W). Which of the following statements is correct?
    1. The solution may cause hypoglycemia in the patient who has diabetes.
    2. The solution may be used to dilute mixed intravenous drugs.
    3. The solution is considered a colloid solution.
    4. The solution is used to provide adequate calories for metabolic needs.
5. A patient will be sent home on diuretic therapy and has a prescription for liquid potassium chloride (KCl). What teaching will the nurse provide before the patient goes home?
    1. Do not dilute the solution with water or juice; drink the solution straight.
    2. Increase the use of salt substitutes; they also contain potassium.
    3. Report any weakness, fatigue, or lethargy immediately.
    4. Take the medication immediately before bed to prevent heartburn.
6. The nurse weighs the patient who is on an infusion of lactated Ringer's postoperatively and finds that there has been a weight gain of 1.5 kg since the previous day. What would be the nurse's next highest priority?
    1. Check with the patient to determine whether there have been any dietary changes in the last few days.
    2. Assess the patient for signs of edema and blood pressure for possible hypertension.
    3. Contact dietary to change the patient's diet to reduced sodium.
    4. Request a diuretic from the patient's provider.

PATIENT-FOCUSED Case Study

Sam Monzoni is a 72-year-old man with a history of heart failure. He is assessed in the emergency department after complaining of weakness and palpitations at work. Sam has been taking furosemide (Lasix) and potassium chloride (KCl) at home. His current ECG reveals atrial fibrillation, and serum electrolyte testing reveals a potassium level of 2.5 mEq/L. The health care provider orders an IV solution of 1,000 mL of lactated Ringer's with 40 mEq KCl to infuse over 8 hours.

1. What is the most likely cause of the change in serum potassium level?
2. What factors must the nurse consider to safely administer this drug?
3. What patient teaching should be given before sending this patient home?

CRITICAL THINKING Questions

1. An 18-year-old woman is admitted to the short stay unit for a minor surgical procedure. The nurse starts an IV line in the patient's left forearm and infuses D_5W at 15 mL/h. The patient asks why she needs the IV line since her provider told her that she will be returning home that afternoon. Why was an IV ordered for this patient, and what should the nurse explain to her?

2. A 24-year-old male is brought into the emergency department after collapsing at a local bike race. On admission, his serum sodium level is found to be 112 mEq/L. An IV infusion of 3% sodium chloride is ordered. What must the nurse monitor during this patient's infusion?

See appendix C for answers and rationales for all activities.

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