Vernon is 18 months old. Several days ago he developed vomiting and diarrhea. His parents tried to get him to eat, but he had little appetite. He drank a little water and a few sips of juice, but the next morning he was listless and would not drink anything. The diarrhea continued.

Vernon’s mother brought him to the urgent care center. Vernon is irritable on arrival, and his mother reports that he has been alternately irritable and lethargic. His mucous membranes and tongue appear dry, and skin turgor over the abdomen is slightly decreased. His mother notes that Vernon has had only two wet diapers today and says the urine in his diapers was dark in color. She also reports that he weighed 12 kg (26 lb) at the clinic last week. However, when the nurse weighs him, the scale reads only 11 kg (24 1/2 lb). Vernon is moderately dehydrated. He needs rapid replacement of the proper type of fluids.

What happens inside the body when dehydration occurs? How can a nurse recognize dehydration? What types of fluid does Vernon need? What nursing management is important for his recovery? Why are young children at greater risk for dehydration than adults? What do parents need to be taught to prevent and manage dehydration? This chapter presents information that will enable you to answer these questions.

LEARNING OUTCOMES

After reading this chapter, you will be able to do the following:
1. Describe normal fluid and electrolyte status for children at various ages.
2. Identify regulatory mechanisms for fluid and electrolyte balance.
4. Analyze assessment findings to recognize fluid and electrolyte problems and acid-base imbalance in children.
5. Describe appropriate interventions for children experiencing fluid and electrolyte problems and acid-base imbalance.

KEY TERMS

- acidemia 496 hypotonic fluid 518
- acidosis 497 interstitial fluid 496
- alkalemia 496 intracellular fluid 496
- alkalosis 497 fluid 496
- anion 521 isotonic dehydration
- body fluid 496 intravascular fluid 496
- body surface area 499 isotonic dehydration (or isonatremic dehydration)
- buffer 497
- cation 517
- dehydration 501 isotonic fluid 518
- diffusion 496 Kussmaul respirations 542
- electrolytes 496 oncotic pressure 513
- extracellular fluid 496 osmolality 516
- filtration 496 osmosis 496
- hypernatremia 501 Pco₂ 497
- hypertonic fluid 520 pH 496
- hypertonic dehydration (or hypernatremic dehydration) 501 pitting edema 515
- hypotonic dehydration (or hyponatremic dehydration) 501 Po₂ 497
- saline 512

MediaLink

http://www.prenhall.com/ball

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Focus on
Fluid, Electrolyte, and Acid-Base Balance

ANATOMY AND PHYSIOLOGY

Physiology of Fluid and Electrolyte Balance

Fluid in the body is in a dynamic state. In persons of all ages, fluid continuously leaves the body through the skin, in feces and urine, and during respiration. Much of the human body is composed of water. **Body fluid** is body water that has solutes dissolved in it. Some of the solutes are **electrolytes**, or charged particles (ions). Electrolytes such as sodium (Na⁺), potassium (K⁺), calcium (Ca²⁺), magnesium (Mg²⁺), chloride (Cl⁻), and inorganic phosphorus (Pi) ions must be present in the proper concentrations for cells to function effectively.

In persons of all ages, body fluid is located in several compartments. The two major fluid compartments contain the **intracellular fluid** (fluid inside the cells) and the **extracellular fluid** (fluid outside the cells). The extracellular fluid is made up of **intravascular fluid** (the fluid within the blood vessels) and **interstitial fluid** (the fluid between the cells and outside the blood and lymphatic vessels). Extracellular fluid accounts for about one third of total body water, while intracellular fluid accounts for about two thirds. The concentrations of electrolytes in the fluid differ depending on the fluid compartment. For example, extracellular fluid is rich in sodium ions; intracellular fluid, by contrast, is low in sodium ions but rich in potassium ions (Table 16–1).

Fluid moves between the intravascular and interstitial compartments by a process called **filtration**. Water moves into and out of the cells by the process of **osmosis**. These processes are discussed later in the chapter. Electrolytes move over cell membranes both by **diffusion** of particles from a location of greater to less concentration and by active transport that is effective even against the concentration gradient.

Physiology of Acid-Base Balance

Normal acid-base balance is necessary for proper function of the cells and the body. The number of hydrogen ions (H⁺) present in a fluid determines its acidity. Increasing the hydrogen ion concentration makes a solution more acidic. Because the hydrogen ion concentration in body fluids is very low, acidity is expressed as **pH** (the negative logarithm of the hydrogen ion concentration) rather than as the hydrogen ion concentration itself. The range of possible pH values is 1 to 14. A pH of 7 is neutral. The lower the pH, the more acidic the solution. A pH above 7 is basic or alkaline. The higher the pH, the more basic the solution. Body fluids are normally slightly basic.

The pH of body fluids is regulated carefully to provide a suitable environment for cell function. The pH of the blood influences the pH inside the cells. **Acidemia** is a term that refers to a blood pH below normal levels, whereas **alkalemia** is an increased blood pH. For the enzymes outside the cells to function optimally, the pH must be in the normal range. If the pH inside the cells becomes too high or too low, then the speed of chemical reactions becomes inappropriate for proper cell function. Cell protein function relies on the correct level of hydrogen ions. Thus, acid-base imbalances result in clinical signs and symptoms. In severe cases, they may cause death.

In the course of their normal function, all cells in the body produce acids. Cells produce two kinds of acids: carbonic acid (H₂CO₃) and metabolic (noncarbonic) acid. These acids are released into the extracellular fluid and must be neutralized.

<table>
<thead>
<tr>
<th>Components</th>
<th>Extracellular Fluid (ECF)</th>
<th>Intracellular Fluid (ICF)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na⁺</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>K⁺</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td>Ca²⁺</td>
<td>Low</td>
<td>Low</td>
</tr>
<tr>
<td>Mg²⁺</td>
<td>Low</td>
<td>Low</td>
</tr>
<tr>
<td>Pi</td>
<td>Low</td>
<td>Low</td>
</tr>
<tr>
<td>Cl⁻</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>Proteins</td>
<td>High</td>
<td>Low</td>
</tr>
</tbody>
</table>
or excreted from the body to prevent dangerous accumulation. They can be neutralized to some degree by the buffers in body fluids. Carbonic acid is excreted by the lungs in the form of carbon dioxide and water. Metabolic acids are excreted by the kidneys. Examples of metabolic acids are pyruvic, sulfuric, acetooacetic, lactic, hydrochloric, and beta-hydroxybutyric acids.

**Buffers**

The maintenance of hydrogen ions within the normal range relies heavily on buffers. A **buffer** is a compound that binds hydrogen ions when their concentration rises and releases them when their concentration falls (Figure 16–1➤). Several kinds of buffers are present in the body (Table 16–2). Various body fluids have buffers to meet their special needs. The bicarbonate buffer system neutralizes metabolic acids (Figure 16–2➤); however, it cannot neutralize carbonic acid.

All buffer systems have limits. For example, if there are too many metabolic acids, the bicarbonate buffers become depleted. The acids then accumulate in the body until they are excreted by the kidneys. Clinically, this is seen as a decreased serum bicarbonate concentration and decreased blood pH.

**Role of the Lungs**

The lungs are responsible for excreting excess carbonic acid from the body. A child breathes out carbon dioxide and water, the components of carbonic acid, with each breath. With faster and deeper breaths, more carbonic acid is excreted. Since carbonic acid is converted in the body to carbon dioxide and water by the enzyme carbonic anhydrase, an indirect laboratory measurement of carbonic acid is \( P_{CO_2} \), the partial pressure of carbon dioxide in arterial blood.

Although a child can voluntarily increase or decrease the rate and depth of respirations, they are usually involuntarily controlled. The \( \text{PO}_2 \) (partial pressure of oxygen in arterial blood), \( P_{CO_2} \), and pH of the blood are monitored by chemoreceptors in the hypothalamus of the brain and in the aorta and carotid arteries. The input from the chemoreceptors is combined with other neural input to change breathing according to needs. Rate and depth increase or decrease according to the amount of carbonic acid that needs to be excreted.

If a child has a condition that decreases the excretion of carbonic acid or causes breathing to be too slow or shallow (such as overmedication following surgery), carbonic acid accumulates in the blood. Clinically, this is seen as an increased blood \( P_{CO_2} \) and is a form of respiratory acidosis. The reverse will also be true in the child breathing excessively or deeply. This leads to decreased \( P_{CO_2} \) and respiratory alkalosis.

**Role of the Kidneys**

The kidneys regulate metabolic acids from the body in two ways: They reabsorb filtered bicarbonate to prevent its loss in the urine, and they regenerate bicarbonate when needed to restore balance (Yucha, 2004). Bicarbonate is formed when acids and ammonium combine with extra ions. The blood bicarbonate concentration is an indicator of the amount of metabolic acids present, because bicarbonate is used in buffering the acids. When the concentration is normal, metabolic acids are present in usual amounts (Figure 16–3➤).

In a healthy child, the result of these renal processes is excretion of metabolic acids and maintenance of blood bicarbonate concentration within normal limits. These processes
may take several hours to days to be effective in restoring balance when acidosis occurs. In the child whose kidneys are not producing enough urine, metabolic acids may not be effectively excreted. Accumulation of these acids uses up many of the available bicarbonate buffers, resulting in a decreased serum bicarbonate concentration and metabolic acidosis.

**Role of the Liver**

The liver also plays a role in maintaining acid-base balance by metabolizing protein, which produces hydrogen ions. It also synthesizes proteins needed to maintain osmotic pressures in the fluid compartments.

**PEDIATRIC DIFFERENCES**

Infants and young children differ physiologically from adults in ways that make them vulnerable to fluid, electrolyte, and acid-base imbalances. The percentage of body weight that is composed of water varies with age (Figure 16–4 ➤). The percentage is highest at birth (and higher in premature than in

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### Figure 16–3 ➤

**A**, Recycling of bicarbonate by the kidneys. Bicarbonate ions that are in the blood are filtered into the renal tubules at the glomerulus. In the proximal tubules, bicarbonate ions are reabsorbed into the blood at the same time that hydrogen ions are transported from the blood into the renal tubular fluid. **B**, Secretion and buffering of hydrogen ions in the kidneys. If the urine is too acidic, the cells that line the urinary tract could be damaged. To prevent this problem, hydrogen ions secreted into the distal tubules are neutralized by phosphate buffers or bound to ammonia and excreted in the form of ammonium.

### Figure 16–4 ➤

The major body fluid compartments at various ages. *Extracellular fluid* is composed mainly of vascular fluid (fluid in blood vessels) and interstitial fluid (fluid between the cells and outside the blood and lymphatic vessels). *Intracellular fluid* is that within cells.

full-term infants) and decreases with age (see As Children Grow, Figure 16–5 ➤). Neonates and young infants have a proportionately larger extracellular fluid volume than older children and adults because their brain and skin (both rich in interstitial fluid) occupy a greater proportion of their body weight. Much of our extracellular fluid is exchanged each day. During infancy, there is a high daily fluid requirement with little fluid volume reserve; this makes the infant vulnerable to dehydration. As an infant grows, the proportion of water inside the cells increases, extracellular amount decreases in comparison, and the risk of fluid imbalance begins to decrease.

Infants and children under 2 years of age lose a greater proportion of fluid each day than older children and adults and are thus more dependent on adequate intake. They have a greater amount of skin surface or body surface area (BSA; relationship between height and weight measured in squared meters) and thus have greater insensible water losses through the skin. Because of this large BSA, they are also at greater risk when burned.

In addition, respiratory and metabolic rates are high during early childhood. These factors lead to greater water loss from the lungs and greater water demand to fuel the body’s metabolic processes (Figure 16–6 ➤). Due to these factors, the exercising child dehydrates easily and must consume more fluid during physical activity, particularly during hot weather (Committee on Sports Medicine and Fitness, 2000).

### AS CHILDREN GROW

#### Fluid and Electrolyte Differences

**Newborn**
- 75% Total body water
  - ECF 45%
  - ICF 30%

**Infant**
- 65% Total body water
  - ECF 25%
  - ICF 30–40%

**Child/Adolescent**
- 50% Total body water
  - ECF 10–15%
  - ICF 40%

---

**Figure 16–5 ➤** The newborn and infant have a high percentage of body weight comprised of water, especially extracellular fluid, which is lost from the body easily. Note the small stomach size which limits ability to rehydrate quickly.
When fluid status is compromised, a number of body mechanisms are activated to help restore balance. Several of these mechanisms occur in the kidney. The kidneys conserve water and needed electrolytes while excreting waste products and drug metabolites. In children under 2 years of age, however, the glomeruli, tubules, and nephrons of the kidneys are immature. They are thus unable to conserve or excrete water and solutes effectively (see Chapter 25). Because more water is generally excreted, the infant and young child can become dehydrated quickly or develop electrolyte imbalances. In addition, infants have a weaker transport system for ions and bicarbonate, placing them at greater risk for acidosis and acid-base imbalances. Children under 2 years of age also have difficulty regulating electrolytes such as sodium and calcium. Renal response to high solute loads is slower and less developed, with function improving gradually during the first year of life.

Examples of diagnostic and laboratory tests used to evaluate fluid, electrolyte, and acid-base balance are provided in the accompanying table. Use the guidelines below to perform a nursing assessment for these functions.

### Diagnostic and Laboratory Tests for Fluid, Electrolyte, and Acid-Base Balance

<table>
<thead>
<tr>
<th>Laboratory Test</th>
<th>Purpose</th>
<th>Nursing Implications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial blood gases</td>
<td>Arterial blood can be analyzed for pH, partial pressure of carbon dioxide (Pco2), partial pressure of oxygen (Po2), or serum bicarbonate (HCO3⁻). Levels are analyzed for information about acid-base balance.</td>
<td>Arterial blood gases are commonly obtained from an existing arterial line. Prepare the child and family for the procedure. Obtain necessary supplies and be certain the child is restrained adequately so that the line is safely accessed. Label and transport the sample to the laboratory on ice.</td>
</tr>
<tr>
<td>Serum electrolyte panel</td>
<td>The variety of electrolytes measured in the serum can reflect imbalances in water and electrolyte values. They provide the basis for further assessment and diagnosis of the condition and for the types of fluids needed during management to reestablish balance.</td>
<td>Prepare the child for the blood test. Perform the test in a treatment room. An existing intravenous line may be used for access if it is available.</td>
</tr>
<tr>
<td>Urinary specific gravity</td>
<td>This measure of urine's density is used to assess its concentration. An increasing number indicates higher concentration of molecules, signifying lower levels of hydration.</td>
<td>A very small amount of urine is needed to complete a specific gravity test. Obtain the sample and perform the refractometer test.</td>
</tr>
</tbody>
</table>

### Assessment Guidelines for the Child with a Fluid, Electrolyte, or Acid-Base Alteration

<table>
<thead>
<tr>
<th>Assessment Focus</th>
<th>Assessment Guidelines</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight</td>
<td>• Has weight decreased since last measurement or weight reported by family?</td>
</tr>
<tr>
<td></td>
<td>• If so, how much? What percent of body weight is the weight loss?</td>
</tr>
<tr>
<td>Skin and mucous membranes</td>
<td>• What are the temperature, turgor, and moistness of the skin?</td>
</tr>
<tr>
<td></td>
<td>• Describe moistness of oral mucous membranes.</td>
</tr>
<tr>
<td></td>
<td>• Describe moistness of the eyes and presence of tears.</td>
</tr>
<tr>
<td></td>
<td>• Is edema present in any body parts?</td>
</tr>
<tr>
<td>Cardiovascular and respiratory systems</td>
<td>• What are pulse and blood pressure?</td>
</tr>
<tr>
<td></td>
<td>• Test capillary refill and small-vein filling times.</td>
</tr>
<tr>
<td></td>
<td>• What is the respiratory rate? Is the rate regular?</td>
</tr>
<tr>
<td>Gastrointestinal system</td>
<td>• Does the child have nausea, vomiting, or diarrhea? If so, how often and for how long has it continued?</td>
</tr>
<tr>
<td></td>
<td>• Is the child eating and drinking? How much and what types of foods and fluids?</td>
</tr>
<tr>
<td>Urinary system</td>
<td>• What is the child’s urinary output?</td>
</tr>
<tr>
<td></td>
<td>• What is the urine specific gravity?</td>
</tr>
<tr>
<td>Musculoskeletal system</td>
<td>• Describe muscle tone and symmetry.</td>
</tr>
<tr>
<td>Neurological system</td>
<td>• Describe the child’s state of alertness and any changes observed.</td>
</tr>
<tr>
<td></td>
<td>• What is the level of consciousness?</td>
</tr>
<tr>
<td></td>
<td>• Is the anterior fontanel at the skin surface or does it appear sunken?</td>
</tr>
</tbody>
</table>
A thorough understanding of fluid, electrolyte, and acid-base homeostasis and imbalances is essential when providing nursing care to pediatric patients, like Vernon in the preceding scenario. This chapter presents information about the processes that maintain fluid and electrolyte balance, and describes the common imbalances that may occur in children. It also describes how the body regulates acid-base status and explains the management of acid-base imbalances.

Many health conditions cause changes in body fluids that must be regulated and managed. Sometimes management of fluid status in the home or in a short-term ambulatory facility can prevent more serious illness or hospitalization. Examples of conditions that commonly require fluid, electrolyte, or acid-base balance include gastroenteritis, burns, kidney disorders, oral fluid restriction for surgery, anorexia or bulimia, or dehydration and electrolyte imbalances that can result from athletics in hot weather.

**FLUID VOLUME IMBALANCES**

When fluid excretion and losses are balanced by the proper volume and type of fluid intake, fluid balance will be maintained. If, however, fluid output and intake are not matched, fluid imbalance may occur rapidly. In addition to the immaturity of physiologic processes, many health conditions make young children more vulnerable to fluid deficit. The major types of fluid imbalances are extracellular fluid volume deficit (dehydration), extracellular fluid volume excess, and interstitial fluid volume excess (edema).

**Extracellular Fluid Volume Imbalances**

**Extracellular Fluid Volume Deficit (Dehydration)**

Extracellular fluid volume deficit occurs when there is not enough fluid in the extracellular compartment (vascular and interstitial). Depending on the cause of dehydration, sodium may be at a normal, low, or elevated level. (Hyponatremia and hypernatremia are described later in the chapter, on pages 517–521.) The state of body water deficit is called dehydration. The three major types of dehydration are:

- **Isotonic dehydration** (or **isonatremic dehydration**). This occurs when fluid loss is not balanced by intake and the losses of water and sodium are in proportion. The serum sodium is therefore within normal limits even though the circulating blood volume is lowered. Most of the fluid lost is from the extracellular component. This type of dehydration is commonly manifested in the illnesses of young children through such symptoms as vomiting and diarrhea.

- **Hypotonic dehydration** (or **hyponatremic dehydration**). This occurs when fluid loss is characterized by a proportionately greater loss of sodium than water. Serum sodium is below normal levels. Compensatory fluid shifts occur from the extracellular to intracellular components in an attempt to establish normal proportions, thus leading to even greater extracellular dehydration. Severe and prolonged vomiting and diarrhea, burns, and renal disease can lead to this condition, as well as administration of intravenous fluid without electrolytes in treatment of dehydration.

- **Hypertonic dehydration** (or **hypernatremic dehydration**). This occurs when sodium loss is proportionately less than water loss. Serum sodium is above normal levels. Compensatory fluid shifts occur from the intracellular to extracellular components in an attempt to establish normal proportions. The extracellular component therefore remains fairly normal, delaying the onset of signs and symptoms of dehydration until the condition is quite serious. Neurological symptoms reflecting intracellular imbalance may occur simultaneously with more common symptoms of dehydration. The condition may be caused by health problems such as diabetes insipidus (see Chapter 29) or administration of intravenous fluid or tube feedings with high electrolyte levels.

The body continuously attempts to compensate for fluid and electrolyte imbalance by shifting fluid and electrolytes from one component to another. Therefore, it is rare for
CHAPTER 16
only one type of dehydration to occur; the child's fluid and electrolyte status and symptoms are constantly changing. Ongoing assessment and management will be needed.

ETIOLOGY AND PATHOPHYSIOLOGY Extracellular fluid volume deficit is usually caused by the loss of sodium-containing fluid from the body. The situations that most often cause loss of fluid containing sodium are vomiting, diarrhea, nasogastric suction, hemorrhage, and burns. Vomiting and diarrhea are common manifestations of disease in children throughout the world, and each year up to 5 million children die from dehydration related to diarrhea. About 300 to 500 die annually in the United States from this problem; about 220,000 are hospitalized (9% of pediatric hospitalizations); and about 1.5 million receive care on an outpatient basis (Dale, 2004; Dennehy, 2005; Nager & Wang, 2002).

Another cause of extracellular fluid volume deficit in infants is increased water loss in low-birth-weight infants who are kept under radiant warmers to maintain heat (Figure 16–7➤). Their high BSA puts them at risk of dehydration due to insensible fluid loss through the skin. Less frequently, adrenal insufficiency, accumulation of extracellular fluid in a "third space" such as the peritoneal cavity, and overse use of diuretics may be the cause. The latter etiology is most often seen in bulimic adolescents for weight control (see Chapter 4➤).

Excessive exercise during very hot weather without sufficient fluid replacement can lead to fluid and electrolyte imbalance. Children are more likely than adults to experience imbalance from exercise, because of the physiological differences explained earlier in this chapter. Because children have a larger BSA, they can gain more heat from the environment when it is hot, and lose more when it is cold (Binkley, Beckett, Casa et al., 2002). In addition, the high metabolic rate of children is further increased during exercise so that fluid lost in metabolism is significant. Children may not feel thirsty and so fail to drink even when dehydrated (Committee on Sports Medicine and Fitness, 2000).

Burns involve complex health problems that are described in Chapter 30➤. Burns of the skin usually involve huge loss of body fluids, including water and electrolytes, particularly sodium. Hypotonic dehydration is the type most commonly seen in the initial period after a burn. Serum proteins are also lost, so body fluid is more likely to leak into interstitial spaces, causing edema and further contributing to the fluid deficit. The kidneys decrease urine production because of their decreased blood flow, which leads to lowered urinary output. While the fluid imbalance of burns is therefore very complicated, the first imbalance encountered is often that of dehydration with accompanying hyponatremia.

For burns, gastroenteritis, and other illnesses, initial dehydration in the first 3 days reflects a high loss of extracellular fluid. About 80% of the fluid loss is extracellular, and only about 20% intracellular. However, with time the relationship begins to change, so that in illnesses over 3 days, about 60% of fluid loss is extracellular while 40% is intracellular (Johns Hopkins Hospital, 2005). Because the electrolyte composition of extracellular and intracellular fluids differs (see Table 16–1), electrolyte management will need to be adapted in long-term conditions.

CLINICAL MANIFESTATIONS The signs of dehydration relate to the severity or degree of the body water deficit (Table 16–3). They are a result of both the decreased fluid (e.g., diminished turgor and mucous membrane moisture) and the body’s response to the fluid deficit (e.g., pulse and blood pressure changes). See the Clinical Manifestations of Extracellular Fluid Volume Deficit on the next page. Clinical Manifestations of Exertional Heat Illness are listed on page 504.

Mild dehydration is hard to detect, because children appear alert and have moist mucous membranes. Infants may be irritable and older children are thirsty. In moderate dehydration, the child is often lethargic and sleepy, but there may be periods of

![Figure 16–7➤](image-url) Use of an overhead warmer or phototherapy increases insensible fluid excretion through the skin, thus increasing the fluid intake needed.
restlessness and irritability, especially in infants. Skin turgor is diminished, mucous membranes appear dry, and urine is dark in color and diminished in amount. Pulse rate is usually increased and blood pressure can be normal or low. Vernon, described at the beginning of this chapter, was displaying symptoms of moderate dehydration. His urine output was decreased, and he had lost about 8% of his body weight. What other signs and symptoms of moderate dehydration can you identify in the opening scenario? What additional assessments would you want to perform on Vernon?

Severe dehydration is manifested by increasing lethargy or nonresponsiveness, markedly decreased blood pressure, rapid pulse, poor skin turgor, dry mucous membranes, seizure activity, and markedly decreased or absent urinary output.

<table>
<thead>
<tr>
<th>Clinical Assessment</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percent of body weight lost</td>
<td>Up to 5% (40–50 mL/kg)</td>
<td>6–9% (60–90 mL/kg)</td>
<td>10% or more (100 mL/kg or more)</td>
</tr>
<tr>
<td>Level of consciousness</td>
<td>Alert, restless, thirsty</td>
<td>Irritable or lethargic (infants and very young children); alert, thirsty, restless (older children and adolescents)</td>
<td>Lethargic to comatose (infants and young children); often conscious, apprehensive (older children and adolescents)</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Normal</td>
<td>Normal or low; postural hypotension (older children and adolescents)</td>
<td>Low to undetectable</td>
</tr>
<tr>
<td>Pulse</td>
<td>Normal</td>
<td>Normal or rapid</td>
<td>Tachycardia or bradycardia</td>
</tr>
<tr>
<td>Skin turgor</td>
<td>Normal</td>
<td>Poor</td>
<td>Very poor</td>
</tr>
<tr>
<td>Mucous membranes</td>
<td>Moist</td>
<td>Dry</td>
<td>Parched</td>
</tr>
<tr>
<td>Urine</td>
<td>May appear normal</td>
<td>Decreased output (&lt; 1 mL/kg/hr) dark color; increased specific gravity</td>
<td>Very decreased or absent output</td>
</tr>
<tr>
<td>Thirst</td>
<td>Slightly increased</td>
<td>Moderately increased</td>
<td>Greatly increased unless lethargic</td>
</tr>
<tr>
<td>Fontanel</td>
<td>Normal</td>
<td>Sunken</td>
<td>Sunken</td>
</tr>
<tr>
<td>Extremities</td>
<td>Warm; normal capillary refill</td>
<td>Delayed capillary refill (&gt; 2 sec)</td>
<td>Cool, discolored, delayed capillary refill (&gt; 3–4 sec)</td>
</tr>
<tr>
<td>Respiration</td>
<td>Normal</td>
<td>Normal or rapid</td>
<td>Changing rate and pattern</td>
</tr>
<tr>
<td>Deep</td>
<td>Normal</td>
<td>Slightly sunken, decreased tears</td>
<td>Deeply sunken, absent tears</td>
</tr>
</tbody>
</table>

restlessness and irritability, especially in infants. Skin turgor is diminished, mucous membranes appear dry, and urine is dark in color and diminished in amount. Pulse rate is usually increased and blood pressure can be normal or low. Vernon, described at the beginning of this chapter, was displaying symptoms of moderate dehydration. His urine output was decreased, and he had lost about 8% of his body weight. What other signs and symptoms of moderate dehydration can you identify in the opening scenario? What additional assessments would you want to perform on Vernon?

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<table>
<thead>
<tr>
<th>Etiology</th>
<th>Clinical Manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decreased fluid volume</td>
<td>Weight loss</td>
</tr>
<tr>
<td>Inadequate circulating blood volume to offset the force of gravity when in upright position</td>
<td>Sunken fontanel (infant)</td>
</tr>
<tr>
<td>Inadequate circulation to the brain</td>
<td>Dizziness</td>
</tr>
<tr>
<td>Inadequate circulation to the kidneys</td>
<td>Oliguria</td>
</tr>
<tr>
<td>Cardiac reflex response to decreased intravascular volume</td>
<td>Thready, rapid pulse</td>
</tr>
<tr>
<td>Decreased interstitial fluid volume</td>
<td>Decreased skin turgor</td>
</tr>
</tbody>
</table>
Diagnostic Tests

The diagnosis of dehydration is best accomplished by clinical observations (see Table 16–3). A major observation that provides clues about the degree of dehydration is percent of weight loss. A synthesis of studies on dehydration showed that abnormal capillary refill time, skin turgor, and abnormal respirations were the most useful clinical signs of dehydration to assist in identifying the disorder (Steiner, DeWalt, & Byerly, 2004). The serum electrolyte panel may be helpful in severe and continuing dehydration that is complicated by electrolyte imbalance or acidosis. The tests include serum electrolytes, creatinine, and glucose. Elevated blood urea nitrogen (>17 mg/dL) and low serum bicarbonate (<16 mmol/L) are also useful to identify moderate and severe dehydration (Wathen, MacKenzie, & Bothner, 2004). The results can be used to target the fluid type and amount to best meet the imbalances identified. Urine specific gravity may provide useful information.

Clinical Therapy

Medical management depends on accurate identification of the degree of dehydration. The treatment of extracellular fluid volume deficit is administration of fluid containing sodium. This may be accomplished by oral rehydration therapy or by intravenous fluids.

Oral rehydration therapy has been used for a number of years in developing countries without an accessible supply of intravenous fluids. More recently, the benefits of using this therapy early to prevent severe dehydration and to treat mild and
moderate dehydration in children in developed countries has been recognized. The therapy is successful in treating the dehydration caused by many gastrointestinal illnesses and prevents hospitalization for many infants and young children. It is the treatment of choice for children with diarrhea who have mild to moderate dehydration (King, Glass, Breezer, & Duggan, 2003). Solutions are available commercially that contain water, carbohydrate (glucose), sodium, potassium, chloride, and lactate. Some clinicians allow lactose-free milk, breast milk, or half-strength milk to be given in addition to oral rehydration therapy solution. A WHO/UNICEF solution was developed for use with cholera and is not generally used for diarrhea treatment in the United States, as its sodium and chloride loads are higher than that of other commercial solutions (Box 16–1).

When the child is severely dehydrated, intravenous fluid will be given, often accompanied with oral rehydration. The intravenous fluid is often Ringer’s lactate followed by or accompanied with dilute saline, such as one half or one quarter normal saline. The fluid combination replenishes the extracellular fluid volume and adds solutes to return the body fluid to normal. The child may be hospitalized or treated with intravenous fluids in a short-stay unit until the dehydration is controlled. Once hydration is completed, the child may resume an age-appropriate diet.

### NURSING MANAGEMENT

#### Nursing Assessment and Diagnosis

Weigh the child daily with the same scale and without clothing. Compare to past weights and calculate weight loss. Carefully measure intake and output, urine specific gravity, level of consciousness, pulse rate and quality, skin turgor, mucus membrane moisture, quality and rate of respirations, and blood pressure (Figure 16–8 ➤). Compare the blood pressure when the child is supine with the pressure when the child is sitting with legs hanging down or standing. If the child is dehydrated, the sitting or standing blood pressure will be lower than the supine blood pressure, because blood accumulates in the dependent legs. The nurse will obtain samples of urine and blood as needed for dehydration evaluation. Evaluate the alertness of the child and any signs of lethargy or weakness.

The nursing diagnosis of Deficient Fluid Volume applies to all children who have an extracellular fluid volume deficit. Other diagnoses depend on the severity of the condition and the age of the child. Several nursing diagnoses that might be appropriate for the mildly to severely dehydrated child are included in the accompanying nursing care plans. Additional care of the child with dehydration from gastroenteritis can be found in Chapter 24 ➤. Specific examples of nursing diagnoses include the following:

- Fluid Volume (Deficient) related to fluid volume loss or failure of regulatory mechanisms
- Risk for Ineffective Peripheral Tissue Perfusion related to hypovolemia
- Risk for Injury related to postural hypotension

#### Planning and Implementation

Nursing care of the dehydrated child focuses on preventing dehydration when possible, providing oral rehydration fluids, teaching parents oral rehydration methods, and, if necessary, administering intravenous fluids to restore fluid balance. The accompanying nursing care plans summarize care of the child with mild to severe dehydration.

#### Prevent Dehydration

Nursing care can often prevent dehydration. Carefully monitor temperature probes in radiant warmers and isolettes for newborns to prevent overheating and resulting dehydration. Teach parents to use proper clothing for infants to prevent overheating. Nurses play an important role in educating parents, youth, school personnel, and

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**BOX 16–1**

**ORAL REHYDRATION AND MAINTENANCE FLUIDS FOR MILD AND MODERATE DEHYDRATION**

<table>
<thead>
<tr>
<th>Pedialyte</th>
<th>Nutralyte</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ricelyte</td>
<td>Revital</td>
</tr>
<tr>
<td>Infalyte</td>
<td>Hydralyte</td>
</tr>
<tr>
<td>KaoLectrolyte</td>
<td>Rehydralyte</td>
</tr>
<tr>
<td>Cerealyte</td>
<td>Equalyte</td>
</tr>
<tr>
<td>Lytren</td>
<td>Resol</td>
</tr>
<tr>
<td>Pediatric Oral Maintenance Solution (ORS)</td>
<td>WHO/UNICEF oral rehydration solution</td>
</tr>
</tbody>
</table>
Figure 16–8 ➤ Assessing skin turgor takes skill and practice. A, In moderate dehydration the skin may have a doughy texture and appearance. B, In severe dehydration, “tenting” of the skin is observed. Diminished turgor is most easily assessed in infants or children with little subcutaneous fat; it is more difficult to assess in those with larger amounts of fat. The chest, abdomen, and upper thighs are locations to measure turgor.

### NURSING CARE PLAN The Child with Mild or Moderate Dehydration

<table>
<thead>
<tr>
<th>GOAL</th>
<th>INTERVENTION</th>
<th>RATIONALE</th>
<th>EXPECTED OUTCOME</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Ineffective Management of Therapeutic Regimen related to family knowledge deficit diarrhea and vomiting.</td>
<td><strong>NIC Priority Intervention:</strong> Family Involvement: Facilitate family participation in care of the child.</td>
<td>• Explain how to replace body fluid with an oral rehydration solution. Encourage parents to keep the solution at home and begin use with the first sign of diarrhea.</td>
<td><strong>NOC Suggested Outcome:</strong> Participation: Healthcare Decisions: Personal involvement in selecting healthcare options. Parents are successfully able to treat the child’s diarrhea and vomiting at home. Child is adequately hydrated.</td>
</tr>
<tr>
<td></td>
<td>• Provide verbal and written instructions to parents at each well-child visit.</td>
<td>• Use of an oral rehydration solution can enable successful treatment of vomiting and diarrhea at home.</td>
<td></td>
</tr>
<tr>
<td><strong>NIC Priority Intervention:</strong> Teaching:</td>
<td>• Teach parents to continue the child’s normal diet in addition to providing replacement fluids for diarrhea.</td>
<td>• Diet plus fluid supplementation leads to faster recovery.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Teach parents childhood conditions that commonly lead to dehydration.</td>
<td>• Parents are provided with a reference for late use.</td>
<td></td>
</tr>
<tr>
<td>2. Knowledge Deficient (Parent) related to causes of dehydration</td>
<td><strong>NIC Priority Intervention:</strong> Teaching:</td>
<td><strong>NOC Suggested Outcome:</strong> Knowledge: Extent of understanding conveyed about treatment regimen.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Teach causes of dehydration.</td>
<td>Parents recognize conditions of risk for dehydration in children.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Teach parents to seek care when the child’s vomiting or diarrhea worsens, or the child’s mental alertness changes.</td>
<td>• Severe dehydration may occur if milder forms are not successfully treated.</td>
<td>Parents seek prompt attention for the child’s worsening condition, preventing the development of severe dehydration.</td>
</tr>
</tbody>
</table>
## NURSING CARE PLAN

### The Child with Severe Dehydration

<table>
<thead>
<tr>
<th>GOAL</th>
<th>INTERVENTION</th>
<th>RATIONALE</th>
<th>EXPECTED OUTCOME</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Fluid Volume, Deficient related to excess losses and inadequate intake</td>
<td><strong>NIC Priority Intervention:</strong> Fluid Management: Promote fluid balance.</td>
<td>Frequent assessment of hydration status facilitates rapid intervention and evaluation of the effectiveness of fluid replacement.</td>
<td>The child has signs of normal hydration.</td>
</tr>
<tr>
<td></td>
<td>• Monitor weight daily. Assess intake and output every shift. Assess heart rate, postural blood pressure, skin turgor, small-vein filling time, capillary refill time, fontanel (infant), and urine specific gravity every 4 hours or more frequently as indicated.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Administer intravenous fluids as ordered. Monitor for crackles in dependent portions of the lungs.</td>
<td>Replace fluid lost from the body. Excessive replacement of sodium-containing fluids could cause extracellular fluid volume excess.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Monitor serum sodium concentration daily or more often.</td>
<td>Elevated serum sodium concentration causes brain cell shrinkage and decreased level of consciousness.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Have the child sit before rising from bed and assist to stand slowly.</td>
<td>Slow adjustment to upright posture reduces light-headedness from decreased blood volume.</td>
<td></td>
</tr>
<tr>
<td>2. Risk for injury related to decreased level of consciousness</td>
<td><strong>NIC Priority Intervention:</strong> Fall Prevention: Institute special precautions.</td>
<td>Safety measures protect the child.</td>
<td>The child does not fall or suffer other injury.</td>
</tr>
<tr>
<td></td>
<td>• Raise the side rails of the bed. Ensure that a small child does not become tangled in bedcovers.</td>
<td>Safety measures protect the child.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Monitor level of consciousness every 2–4 hours or more often as indicated.</td>
<td>Frequent assessment provides evidence of the need for safety interventions and of the effectiveness of therapy.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Monitor serum sodium concentration daily or more often.</td>
<td>Elevated serum sodium concentration causes brain cell shrinkage and decreased level of consciousness.</td>
<td></td>
</tr>
<tr>
<td>3. Activity Intolerance related to bed rest/immobility</td>
<td><strong>NIC Priority Intervention:</strong> Activity Therapy: Plan activities to meet child’s developmental needs.</td>
<td>Activities will provide distraction and promote recovery.</td>
<td>The child engages in normal developmental activities and receives adequate rest.</td>
</tr>
<tr>
<td></td>
<td>The child will engage in normal activity for age.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Plan activities appropriate for the age of the child that can be done in bed.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Group nursing interventions to provide time for the child to rest.</td>
<td>The child will require more rest than usual.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Provide assistance during meals and other activities as needed.</td>
<td>Prevention of overexertion will conserve body fluid and promote healing.</td>
<td></td>
</tr>
</tbody>
</table>
coaches about the dangers of heat-related illness. Prevention is key, so that children can exercise safely. Prior to a new exercise regime, perform assessment for risk factors. This includes medical conditions that put the child at high risk, such as cystic fibrosis, diabetes, obesity, or mental retardation. Prior history of heat-related illness or recent change from a cooler to hotter environment increases risk. Long exercise periods increase the stress upon the body. The major nursing interventions are partnering with families and athletic coaches to prevent problems and to recognize and treat them promptly. See Families Want to Know: Preventing Heat-Related Illness. Recognize that heat syndromes can result in death, so prevention, prompt recognition, and treatment are essential.

**Provide Oral Rehydration Fluids**

In mild or moderate dehydration, oral rehydration fluid is the first intervention (see Box 16–1) (Fonseca, Holdgate, & Craig, 2004; Spandorfer, Alessandrini, Joffe, Locatio, & Shaw, 2005). It is given in frequent small amounts; for example, 1 to 3 teaspoons of fluid every 10 to 15 minutes is a useful guideline for starting oral rehydration. For the first 2 to 4 hours of treatment, 50 mL of fluid for each kilogram of the child’s weight should be the target intake. Instruct parents to continue to administer 1 teaspoon every 2 to 3 minutes even if the child vomits, as small amounts of the fluid may still be absorbed. Children are often treated in special sections of emergency departments or outpatient clinics for several hours to begin hydration. Oral or nasogastric tube feedings of oral rehydration are administered while monitoring occurs.

**Teach Parents Oral Rehydration Methods**

Instruct parents about the types of fluids and amounts to be given. See Families Want to Know: Oral Rehydration Therapy Guidelines. Begin teaching parents of all newborns and reinforce teaching at each well-child visit. Advise parents to continue the child’s normal diet in addition to providing the rehydration solution. Cereals, starches, soups, fruits, and vegetables are allowed. Tell parents to avoid simple sugars, which can worsen diarrhea because of osmotic effects, including soft drinks (if used, they should be diluted with equal parts of water), undiluted juice, Jell-O, and sweetened cereal.

**FAMILIES WANT TO KNOW**

**Preventing Heat-Related Illness**

Teach parents, coaches, and youth the following preventive techniques:

- **Precede exercise programs with physical examination designed to identify risks.**
- **Reduce intensity of activity when temperature or humidity is high.**
- **Allow a 10- to 14-day period of acclimation to higher temperatures before reaching usual exercise level.**
- **Ensure hydration before activity begins.**
- **During activity, stop for fluids every 15–20 minutes. Children up to 90 pounds should drink 150 mL (5 ounces) and over 90 pounds should drink 250 mL (9 ounces). A combination of water and sports drink is best.**
- **Recognize low urine volume or dark color as a sign of dehydration.**
- **Wear light-colored, light clothing. Never use rubber clothing designed to promote weight loss through sweating.**
- **Maintain adequate sleep and nutritional status.**

Additional tips for coaches:

- **Weigh all children before and after events to evaluate if weight, and therefore fluids, are maintained.**
- **Be familiar with signs of heat-related illness.**
- **Have cell phones or other mechanisms to call for emergency assistance.**
- **Have at least two adults present at exercise sessions.**
- **Keep adequate fluids and sports drinks readily available.**
- **During all-day practices, allow 2–3 hours rest during the middle of the day with fluids and food provided.**
- **Practice in shade or use fans, if possible.**
- **Obtain and use a wet-bulb globe temperature (WBGT) risk measurement that considers humidity (70% of heat stress), radiation (20% of heat stress), and temperature (10% of heat stress). For WBGT < 75°F, activities are allowed with monitoring for heat-related problems; for 75–79°F, enforce longer rest periods in the shade every 15 minutes; for 79–84°F, limit activities for all children and eliminate activity for those not acclimated; and >85°F, cancel all athletic activity.**
- **Understand symptoms for recognition of all heat-related problems.**
- **Obtain prompt first aid treatment for any heat-related problems.**

Repeated vomiting of large volumes of fluid or a worsening of the child’s condition can indicate the need for intravenous therapy. Teach parents when to seek further medical care. If the child’s condition worsens or does not improve after 4 hours of oral rehydration therapy, parents should contact a healthcare professional.

**Monitor Intravenous Fluid Administration**

The hospitalized child usually requires administration of intravenous fluids. The nurse is usually responsible for starting the intravenous line and administering the prescribed fluids (see Evidence-Based Practice: Intravenous Starts). Be sure that the amount of fluid administered corresponds with the diagnosed dehydration state of the child (Box 16–2).

### Use of Oral Rehydration Therapy

In spite of the recommendation of the American Academy of Pediatrics (Provisional Committee on Quality Improvement, 1996) and other groups to use outpatient oral rehydration therapy (ORT) for mild and moderate dehydration, many healthcare providers continue to hospitalize these children and administer intravenous therapy (Nager & Wang, 2002). Hospitalization is expensive and disruptive for families, while care providers state that it seems easier than keeping a child in an outpatient setting for several hours to institute ORT.

Several analyses of studies performed with children have demonstrated the success and efficacy of ORT (Fonseca et al., 2004). In order to study the possibility of decreased treatment time, a study with 96 children from 3 to 36 months of age randomly assigned the moderately dehydrated children to receive either rapid nasogastric hydration or rapid intravenous (IV) hydration. Both methods were accomplished within 3 hours in an emergency department, and were effective in treating children for dehydration. However, the nasogastric rehydration was significantly less expensive. The authors offer the possibility of rapid rehydration as a cost-effective management technique for moderately dehydrated children (Nager & Wang, 2002). In another study of 18 moderately dehydrated children with gastroenteritis, half were given ORT and half were started on intravenous therapy. The length of treatment in the emergency department was significantly lower for the ORT group than the IV group, and ORT required significantly less staff time. Parents reported greater satisfaction with ORT therapy, and the outcomes for the children were comparable (Atherly-John, Cunningham, & Crain, 2002).

Nurses can support ORT for children with mild or moderate dehydration. Teach parents to keep appropriate fluids at home and to institute the therapy early during vomiting and diarrhea episodes. Monitor children receiving the therapy in outpatient settings, whether by traditional oral therapy or rapid nasogastric hydration.

**ReCalculate fluid needs after first 4 hours and adjust as needed. If the child is not taking increased fluids and other symptoms persist, contact the healthcare provider.**

**When rehydration is complete, resume normal diet.**

### Oral Rehydration Therapy Guidelines

Calculate the specific amounts required for individual children based on the guidelines below and instruct parents in terminology they understand. Provide measuring devices with proper amounts marked.

- Children with diarrhea and no dehydration should be continued on age-appropriate diets.
- For mild dehydration, give 50 mL/kg oral rehydration therapy in the first 4 hours in addition to replacing fluids lost in stool and emesis. (Measure emesis and give 10 mL/kg of fluid for each diarrheal stool.)
- Start slowly, administering 3–5 mL in a small cup or spoon every few minutes. Increase amounts gradually if no vomiting occurs.
- Recommend or provide samples of ORT solutions. Suggest ready to feed or powdered forms for choice by parents.
- For moderate dehydration, give 100 mL/kg oral rehydration therapy in first 4 hours in addition to replacing fluids lost as previously described.
- For severe dehydration, the child is hospitalized and treated with intravenous fluids. When hydrated adequately or concurrently with intravenous hydration, begin oral rehydration therapy with 50–100 mL/kg of fluid in 4 hours and stool replacement as previously described.
- Recalculate fluid needs after first 4 hours and adjust as needed. If the child is not taking increased fluids and otherwise improving by this time, contact the healthcare provider.
- When rehydration is complete, resume normal diet.
**EVIDENCE-BASED PRACTICE**

**Intravenous Starts**

**Problem**

Parents often voice dissatisfaction with the number of intravenous starts to which their young hospitalized children are subjected, as children experience pain and anxiety when intravenous lines are started.

**Evidence**

The Children’s Hospital of Denver created a task force to address problems of venous access. Co-chairs in this collaborative project included a nurse from the neonatal intensive care unit, a general surgeon, and a radiologist. Additional nurses from units using peripheral intravenous lines served as committee members. A tracking tool was developed to collect information on intravenous placements for a 1 month period. Other children’s hospitals were also surveyed for their practices regarding venous access policies and procedures (The Venous Access Task Force, 2002).

**Implications**

Based on the analysis of internal and external data, the task force developed recommendations to (1) develop a group of specially trained nurses to act as resources for other staff nurses, (2) create an algorithm to provide guidelines for decisions about peripheral vs. peripherally inserted central venous catheters vs. central venous catheter insertion, (3) use the specially trained nurses to educate staff members about the new algorithm, and (4) develop a tracking/evaluation mechanism for the new algorithm and trained nurse group.

**Critical Thinking**

This clinical situation is an example of partnership among healthcare specialists in order to improve care for hospitalized children. What were the benefits of having an interdisciplinary group to examine the topic of intravenous starts? How would you suggest that parent satisfaction before and after a program like this be measured? What could be the benefits of having a nursing resource group and an intravenous start guideline to positively influence the hospitalization of young children requiring intravenous infusion for dehydration?

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**BOX 16–2**

**CALCULATION OF INTRAVENOUS FLUID NEEDS**

1. First, calculate the maintenance fluid needs of the child, according to the following guideline:

   **Usual Weight** | **Maintenance Amount**
   -----------------|----------------------
   Up to 10 kg      | 100 mL/kg/24 hr      
   11–20 kg         | 1000 mL + \( \frac{50 \text{ mL/kg for weight above 10 kg}}{24 \text{ hr}} \) 
   >20 kg           | 1500 mL + \( \frac{20 \text{ mL/kg for weight above 20 kg}}{24 \text{ hr}} \) 

   **Example:** Vernon's weight is 12 kg. He needs 1000 mL + \( 50 \times 2 \), or 1100 mL/24 hr for maintenance fluid.

2. Next, calculate replacement fluid for that lost:

   **Example:** Vernon has lost 1 kg (8%) of his body weight. Multiplying the percentage of body weight \( x 10 \) yields the mL/kg/24 hr required:

   \[
   8 \times 10 = 80 \text{ mL/kg/24 hr} \\
   80 \text{ mL/kg } \times 12 \text{ kg} = 960 \text{ mL}
   \]

   Thus, Vernon's replacement fluid needs are 960 mL/24 hr.

   It is also helpful to know that 1 liter of fluid weighs about 1 kilogram. The amount of fluid deficit can be roughly estimated using this formula. In Vernon's case, since he has lost 1 kg of weight, his replacement fluid need is roughly equivalent to 1 L (1000 mL). This is very close to the 960 mL calculated in the previous formula.

3. Finally, calculate continued losses and add to the total maintenance and replacement needs.

---

**SKILLS 9–5:** Administering **IV** Fluids
precautions as necessary. Even when an intravenous infusion is used, the child is started on oral rehydration simultaneously. As more oral fluids are tolerated, the intravenous infusion is decreased.

**Maintain Safety**
The child who is dehydrated is often dizzy and lethargic. Keep side rails up and supervise and assist the child when getting up. Have parents stay at the bedside while the child is being treated in ambulatory units and urge them to maintain safety precautions as they take the child home.

**Discharge Planning and Home Care Teaching**
Prevention of dehydration is the best approach when possible. Encourage breast-feeding because it is associated with a decreased incidence of gastroenteritis. During health promotion and health maintenance visits, encourage all parents to keep oral rehydration fluids at home in case they are needed. Address the need for increasing fluids in hot weather and when the child is exercising. Reinforce safety teaching to decrease incidence of burns, an important cause of dehydration. Teach the signs and symptoms of gastroenteritis. Indications for bringing a child in for care include:

- Under 6 months or weight of < 8 kg
- Other health problems, premature birth
- Fever of 38°C (100.4°F) for infants < 3 months or 39°C (102.2°F) for child 3–36 months
- Visible blood in stool
- Persistent vomiting and substantial volumes of diarrhea
- Report of symptoms indicative of dehydration
- Change in mental status
- Inability of child to take in ORT or of parent to feed ORT (King, Glass, Bresee, & Duggan, 2003).

Prior to discharge from the hospital or outpatient facility after treatment for dehydration, parents need instructions about types of fluids and amounts to encourage. Teach the signs of dehydration (see Table 16–3) so that if the child does not take in adequate fluids, parents can seek help immediately. Instruct them to begin the child’s normal diet once hydration is complete, as determined by adequate urinary output and normal behaviors. Review methods of minimizing the child’s chance of acquiring gastrointestinal infections (e.g., avoiding contact with other children who are infected; using careful handwashing and dishwashing procedures when a child in the home is affected).

Nurses should be alert for children in the community who have other health conditions that predispose them to fluid and electrolyte imbalance. Examples include those with cancer, AIDS, cystic fibrosis, and renal disease. When these children are seen for health promotion and health maintenance visits, or because of a health complication, they should be evaluated for fluid and electrolyte imbalance.

**Evaluation**
Expected outcomes of nursing care for the child with dehydration include the following:

- Water and electrolytes are balanced in intracellular and extracellular compartments.
- Urinary output is within normal limits.
- Adequate fluid intake meets maintenance needs.
- Vital signs are within normal limits.

**Extracellular Fluid Volume Excess**
Extracellular fluid volume excess occurs when there is too much fluid in the extracellular compartment (vascular and interstitial). This imbalance may also be called saline excess or extracellular volume overload. If this disorder occurs by itself (without saline...
disturbance), the serum sodium concentration is normal. There is simply too much extracellular fluid, even though it has a normal concentration.

Infants and children who develop an extracellular fluid volume excess have a condition that causes them to retain saline (sodium and water) or they have been given an overload of sodium-containing isotonic intravenous fluid (Figure 16–9). What conditions cause retention of saline? The hormone aldosterone is secreted by the adrenal cortex. One of its normal functions is to cause the kidneys to retain saline in the body (Figure 16–10). Saline excess can be caused by any condition that results in excessive aldosterone secretion, such as adrenal tumors that secrete aldosterone, congestive heart failure, liver cirrhosis, and chronic renal failure (Figure 16–11). Most glucocorticoid medications (such as prednisone) have a mild saline-retaining effect when taken long term. Intravenous fluid volume regulation is important, especially in young children. Either inaccurate calculation of needed fluid or inadvertent infusion of excess fluids can cause overload.

Because fluid has weight, extracellular fluid volume excess is characterized by weight gain. An overload of fluid in the blood vessels and interstitial spaces can cause clinical manifestations such as bounding pulse, distended neck veins in children (not usually evident in infants), hepatomegaly, dyspnea, orthopnea, and lung crackles. Edema is the sign of overload of the interstitial fluid compartment. In an infant, edema is often generalized. Edema in children with extracellular fluid volume excess occurs in the dependent parts of the body, that is, in the parts closest to the ground. Thus, edema is evident in sacral areas in a child supine in bed. Edema that develops from other causes is described in the next section of this chapter.

Diagnosis of extracellular fluid volume excess is determined by clinical evaluation of weight gain and other manifestations. Serum electrolyte panels aid in diagnosis, and studies of liver or renal function may provide information about the cause of the condition.

Clinical therapy for extracellular fluid volume excess focuses on treating the underlying cause of the disorder in order to reduce the extracellular fluid volume excess. For example, a child who has congestive heart failure is given medications to strengthen the heart’s ability to contract (see Chapter 21). Diuretics may be given to remove fluid from the body, thus reducing the extracellular fluid volume directly.

**NURSING MANAGEMENT**

Rapid weight gain is the most sensitive index of extracellular fluid volume excess. Therefore, daily weighing is an important nursing assessment. Measure the child’s intake and output and weigh the diapers of infants. When treatment is successful, output is greater than intake. Assess the character of the pulse and observe for neck vein distention when the child is sitting (usually visible only in older children). Monitor for signs of pulmonary edema (an indication of severe imbalance) by listening to lung sounds in the dependent lung fields (crackles) and assessing for respiratory distress (rapid respiratory rate, use of accessory muscles of respiration). Observe for edema.

The potential for a child to develop a fluid overload is present whenever an isotonic intravenous solution containing sodium is being administered. Examples of these types of solutions include normal saline (0.9% NaCl), Ringer’s solution, and lactated Ringer’s solution. Therefore, monitor the infusion rate frequently and carefully and use a pump when possible to aid in accurate administration (Figure 16–12).
If an excess of fluid has already developed, administer the medical therapy as prescribed and monitor for any complications of the therapy. For example, many diuretics increase potassium excretion in the urine, an increase that may lead to an abnormally low plasma potassium concentration unless potassium intake is increased. (Refer to the discussion of hypokalemia later in this chapter.) It is also important to monitor for the development of extracellular fluid volume deficit as a result of diuretic therapy.

If edema is present, provide careful skin care and protection for edematous areas. Teach parents how to provide skin care and perform position changes at home. See the following section for additional interventions related to edema.

If a child has a long-term condition such as chronic renal failure that predisposes him or her to extracellular fluid volume excess, a dietary sodium restriction may be prescribed (see Chapter 25 for further details). Teach parents how to manage sodium restriction. Plan low-sodium meals that fit the family’s cultural practices. If the child is old enough to participate, incorporate games into the teaching. If a scale is available, teach parents to take and record an accurate daily weight.

Expected outcomes include electrolyte balance, maintenance of intact skin, and dietary intake as prescribed.

**Interstitial Fluid Volume Excess (Edema)**

Edema is an abnormal increase in the volume of the interstitial fluid. It may be caused by an extracellular fluid volume excess or it may be due to other causes.

The causes of edema are best understood in the context of normal capillary dynamics. Fluid moves between the vascular and interstitial compartment by the process of filtration. Filtration is the net result of forces that tend to move fluid in opposing directions. The strongest forces will determine the direction of fluid movement.

At the capillary level, two forces (blood hydrostatic pressure and interstitial osmotic pressure) tend to move fluid from the capillaries into the interstitial fluid, while two other forces (blood colloid osmotic pressure and interstitial fluid hydrostatic pressure) tend to move fluid in the opposite direction (from the interstitial fluid into the capillaries). The net result of these forces usually moves fluid from the capillaries into the interstitial compartment at the arterial end of the capillaries and fluid from the interstitial compartment back into the capillaries at the venous end of the capillaries. This process brings oxygen and nutrients to the cells and removes carbon dioxide and other waste products.

Edema occurs if the balance of these four forces is altered so that excess fluid either enters or leaves the interstitial compartment (*Pathophysiology Illustrated*, Figure 16–13). This may occur through (1) increased blood hydrostatic pressure, (2) decreased blood colloid osmotic pressure, (3) increased interstitial fluid osmotic pressure, or (4) blocked lymphatic drainage. Various clinical conditions are associated with these altered forces (Box 16–3), as described here.

1. **Increased blood hydrostatic pressure.** When extracellular fluid volume excess occurs, the increased fluid volume in the vascular compartment congests the veins. The pressure against the sides of the capillary is increased and more fluid then enters the interstitial compartment.
2. **Decreased blood colloid osmotic pressure.** Much of the osmotic pressure that pulls fluid into the capillaries is due to the presence of albumin and other plasma proteins made by the liver. The part of the blood osmotic pressure that is due to plasma proteins is often called *oncotic pressure* or blood colloid osmotic pressure. Any condition that decreases plasma proteins will decrease blood colloid osmotic pressure and cause edema. For example, if a clinical condition causes large amounts of albumin to leak into the urine, the liver will not be able to make albumin fast enough to replace it. As a result, the plasma protein level will fall, decreasing the blood osmotic pressure. Without this pulling force to return fluid to the capillaries, edema

**CLINICAL TIP**

You can tell if a child’s weight gain is due to normal growth or to the development of extracellular fluid volume excess by looking at the speed with which the increase develops. Sudden weight gain (e.g., 0.5 kg [1 lb] in 1 day) is due to the accumulation of fluid. Gain of 0.5 kg overnight is due to retention of about 500 mL of saline.

**CLINICAL TIP**

An infant’s urine output is important in monitoring both dehydration and edema. Weigh the diaper before and after use. A 1 g weight increase in the diaper equals approximately 1 mL of urine volume. Change the diaper frequently to minimize loss from evaporation.
CHAPTER 16

The cause of the edema that occurs in children who have nephrotic syndrome (see Chapter 25) is prolonged surgical procedures with significant blood loss. Intravenous fluids and blood may be infused during surgery to replace these losses, but plasma proteins are lost and not fully restored by infusion, causing edema in the postoperative period.

3. Increased interstitial fluid osmotic pressure. Ordinarily, only a few small proteins enter the interstitial fluid, and the interstitial fluid osmotic pressure is small. If the capillary becomes abnormally permeable to proteins, however, the influx of large amounts of proteins into the interstitial fluid causes a dramatic increase in interstitial fluid osmotic pressure. This increased pulling force keeps an abnormal amount of fluid in the interstitial compartment. This mechanism plays an important part in the edema caused by a bee sting or a sprained ankle. It occurs to a greater extent in burns, leading to swelling at the same time that there is a great loss of fluid volume through the burned skin (see Chapter 30).

4. Blocked lymphatic drainage. The lymph vessels normally drain small proteins and excess fluid from the interstitial compartment and return them to the blood vessels. If this process is blocked, fluid accumulates in the interstitial compartment. This may occur when a tumor blocks lymphatic drainage.
**BOX 16–3**  CLINICAL CONDITIONS THAT CAUSE EDEMA

<table>
<thead>
<tr>
<th>Edema Due to Increased Blood Hydrostatic Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Increased Capillary Blood Flow</strong></td>
</tr>
<tr>
<td>• Inflammation</td>
</tr>
<tr>
<td>• Local infection</td>
</tr>
<tr>
<td><strong>Venous Congestion</strong></td>
</tr>
<tr>
<td>• Extracellular fluid volume excess</td>
</tr>
<tr>
<td>• Right heart failure</td>
</tr>
<tr>
<td>• Venous thrombosis</td>
</tr>
<tr>
<td>• External pressure on vein</td>
</tr>
<tr>
<td>• Muscle paralysis</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Edema Due to Decreased Blood Osmotic Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Increased Albumin Excretion</strong></td>
</tr>
<tr>
<td>• Nephrotic syndrome (albumin leaks into urine)</td>
</tr>
<tr>
<td>• Protein-losing enteropathies (excess albumin in feces)</td>
</tr>
<tr>
<td><strong>Decreased Albumin Synthesis</strong></td>
</tr>
<tr>
<td>• Kwashiorkor (low-protein, high-carbohydrate starvation diet provides too few amino acids for liver to make albumin)</td>
</tr>
<tr>
<td>• Liver cirrhosis (diseased liver unable to make enough albumin)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Edema Due to Increased Interstitial Fluid Osmotic Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Increased Capillary Permeability</strong></td>
</tr>
<tr>
<td>• Inflammation</td>
</tr>
<tr>
<td>• Toxins</td>
</tr>
<tr>
<td>• Hypersensitivity reactions</td>
</tr>
<tr>
<td>• Burns</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Edema Due to Blocked Lymphatic Drainage</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Tumors</td>
</tr>
<tr>
<td>• Goiter</td>
</tr>
<tr>
<td>• Parasites that obstruct lymph nodes</td>
</tr>
<tr>
<td>• Surgery that removes lymph nodes</td>
</tr>
</tbody>
</table>

Edema causes swelling, which may be localized or generalized. The swelling of tissue may cause pain and restrict motion. Edema that is due to extracellular fluid volume excess or right-sided heart failure usually occurs in the dependent portion of the body. In a child who is walking, dependent edema is observed in the ankles; in a child who is supine in bed, it is seen in the sacral area. The skin over an edematous area often appears thin and shiny.

The main focus of clinical therapy for edema is to treat the underlying condition that caused the edema. Such conditions are discussed throughout this book. The edema from inflammation of an injury is initially treated with cold to reduce capillary blood flow and thus reduce blood hydrostatic pressure.

**NURSING MANAGEMENT**

A child or parent may make comments that alert the nurse to the development of edema. Shoes may become tight by the end of the day (dependent edema); the waistband of pants or a skirt may be “outgrown” suddenly (generalized edema or ascites, which is accumulation of fluid in the peritoneal cavity); the eyes may be puffy (periorbital edema); a ring may be too tight; fingers may “feel like sausages.” In many cases visual inspection is sufficient to recognize edema. Observe for the presence of **pitting edema**, a “pit” or concave indentation that remains after an edematous area is pressed downward by the examiner’s fingers. To detect changes in the amount of swelling, measure around the...
edematous part (Figure 16–14 ➤). If the edema is caused by extracellular fluid volume excess, daily measurements of weight and intake and output are a necessary part of the daily assessment. Nursing assessment should also focus on the integrity of the skin, presence of pain, restricted motion, and alterations in the child’s body image.

Elevation of an area of localized edema helps to reduce the swelling. The skin over an edematous area needs extra care because it is fragile (Figure 16–15 ➤). Carefully position an infant or child who is on bed rest and turn frequently to prevent pressure sores (see Chapter 30 ➤ for further information on pressure sores). Turning must be performed carefully to avoid skin abrasion by rubbing against the sheets. Pat the skin dry after cleansing rather than rubbing it. Trim the child’s fingernails smooth to prevent scratching. Teach parents skin care for the child at home. Teach older children to inspect their skin carefully to identify areas needing special care.

If restricted mobility is a problem, specific plans to help the child manage activities are needed. For example, if an edematous finger restricts the motion of a hand, food can be cut into bite-size portions before the meal is served, so that the child can still eat independently.

Discomfort from edema may require creative interventions by the nurse. If the child has a fluid restriction, access to fluids needs to be planned to provide satiety. Distraction with toys or activities appropriate to the child’s developmental level can be useful. Interventions to treat the underlying problem can also reduce the edema and its accompanying discomfort. Interventions for edema should be added to the nursing management of the underlying condition that causes the edema. Administration of the prescribed medical therapy and observation for the complications of therapy are nursing responsibilities.

Discuss with school-age children and adolescents feelings of embarrassment about the edematous appearance. They need to understand the reason for edema and be able to explain it to peers. Arrange for the child to meet other children with similar concerns.

Desired outcomes of care include maintenance of intact skin, normal respiratory sounds and effort, and normal weight patterns.

**ELECTROLYTE IMBALANCES**

All body fluids contain electrolytes, although the concentration of those electrolytes varies, depending on the type and location of the fluid. When a serum electrolyte value is reported from the laboratory, it provides information about the concentration of that electrolyte in the blood. It may not necessarily reflect the concentration of the electrolyte in other body compartments. Refer to Table 16–1 to see which electrolytes are of highest and lowest concentration in the blood and other fluid compartments.

Electrolytes are normally gained and lost in relatively equal amounts so the body remains in balance. However, when a child has an abnormal route of loss, such as vomiting, wound drainage, or nasogastric suction, electrolyte balance can be disturbed. Monitoring for signs of imbalance becomes important.

**Sodium Imbalances**

The serum sodium concentration reflects the osmolality of body fluids, that is, their degree of concentration or dilution. It refers to the number of moles of the substance per kilogram of water in the solution. Serum sodium concentration reflects the proportion of water and sodium in the extracellular compartment. When the osmolality of body fluids becomes abnormal, the cells shrink or swell. These cell size changes are due to osmosis, the movement of water across a semipermeable membrane into an area of higher particle concentration. Sodium levels are maintained at high extracellular and low intracellular levels by the sodium-potassium pump, which moves these electrolytes against their expected concentration gradients (Figure 16–16 ➤).
Sodium plays several important roles in the body and is an important cation (positively charged particle). It is important in blood pressure regulation and maintenance of fluid volume.

**Hypernatremia**

Hypernatremia is a condition of increased osmolality of the blood. The body fluids are too concentrated, containing excess sodium relative to water. A serum sodium level above 148 mmol/L in children (146 mmol/L in newborns) is diagnostic of hypernatremia.

Hypernatremia results from conditions that cause the body to lose relatively more water than sodium or to gain relatively more sodium than water (Table 16–4). Examples include children who do not have access to adequate water or are developmentally delayed and do not perceive thirst. Special circumstances in which a high solute intake may occur without adequate water include an infant formula that is too concentrated or one that is prepared with salt instead of sugar. A breast-fed baby not receiving adequate breast milk who has normal water loss may develop hypernatremic dehydration. This is a particular risk at 2 to 3 days of age, when babies generally have a diuresis, if the baby does not feed well or the mother does not yet produce an adequate amount of breast milk (Moritz, Manole, Bogen, & Ayus, 2005).

### Table 16–4 | CAUSES OF HYPERNATREMIA

<table>
<thead>
<tr>
<th>Loss of Relatively More Water than Sodium</th>
<th>Gain of Relatively More Sodium than Water</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes insipidus (not enough antidiuretic hormone)</td>
<td>Inability to communicate thirst</td>
</tr>
<tr>
<td>Diarrhea or vomiting without fluid replacement</td>
<td>Limited or no access to water</td>
</tr>
<tr>
<td>Excessive sweating without fluid replacement</td>
<td>High solute intake without adequate water (e.g., tube feedings)</td>
</tr>
<tr>
<td>High solute intake without adequate water (causes kidneys to excrete water)</td>
<td>Intravenous hypertonic saline</td>
</tr>
</tbody>
</table>

**Figure 16–16**

A. Water balance is maintained by the simple passage of molecules from greater to lesser concentration across cell membranes.

B. Sodium levels are maintained by an active transport system, the sodium-potassium pump, which moves these electrolytes across cell membranes in spite of their concentrations. At times, a pathophysiologic condition causes the pump to not function as quickly and efficiently as needed to maintain balance.
An infant or child who has hypernatremia is generally thirsty. The urine output is diminished unless the hypernatremia is caused by diabetes insipidus. A decreased level of consciousness manifested by confusion, lethargy, or coma results from shrinking of the brain cells. Seizures can occur when hypernatremia occurs rapidly or is severe. Symptoms in the neonate include decreased activity and alertness, loss of 10% or more of birth weight, and seizures. Severe hypernatremia can be fatal.

The major laboratory test that is diagnostic of sodium imbalance is serum sodium. Normal level for newborns is 131 to 146 mmol/L and for children 132 to 148 mmol/L. See Table 16–5 for a list of normal serum electrolyte values. Specific gravity of urine is concentrated in hypernatremia. Antidiuretic hormone (ADH) levels and 24-hour urinary output are helpful in diagnosing diabetes insipidus as the cause (see Chapter 30). Hypernatremia is treated by intravenous administration of hypotonic fluid, or fluid that is more dilute than normal body fluid. This therapy dilutes the body fluids back to normal concentration. If a child is dehydrated, isotonic fluids (those with the osmolality of body fluids) may be ordered first to replenish the volume, followed by hypotonic fluid to correct the osmolality. The underlying cause of the disorder is also treated.

### NURSING MANAGEMENT

Teaching can prevent many cases of hypernatremia. Be sure the breast-feeding mother has instruction and resources about lactation before discharge after delivery. If discharged soon after birth, be sure the infant has an appointment to have weight and alertness checked within the first few days, and instruct the parents about normal output of four to six wet diapers daily. By about 10 days, infants should have regained the birth weight. Assess the infant’s alertness and general neurological status.

When an infant is sick or developing slowly, parents sometimes want to feed the infant more concentrated formula to build the child’s strength. Parents and caregivers of bottle-fed babies should be taught never to give undiluted formula concentrate or evaporated milk due to the high sodium content.

Children with delayed development are at risk for hypernatremia since they may not be able to recognize thirst or obtain fluids when dehydrated. Teach parents about the child’s fluid requirements and ensure that nursing staff offers adequate fluids when the child is hospitalized.

Parents should be cautioned to keep salt out of reach, because eating handfuls of salt has caused hypernatremia. Teach parents to offer extra fluids during hot weather. See Families Want to Know: Preventing Heat-Related Illness on page 508. Teach oral rehydration therapy for use at home during mild vomiting and diarrhea (see p. 509).

<table>
<thead>
<tr>
<th>Table 16–5</th>
<th>NORMAL SERUM VALUES FOR ELECTROLYTES IN INFANTS AND YOUTH</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Newborn</strong></td>
<td><strong>Infant and Child</strong></td>
</tr>
<tr>
<td>Sodium</td>
<td>131–144 mmol/L</td>
</tr>
<tr>
<td></td>
<td>132–141 mmol/L</td>
</tr>
<tr>
<td>Potassium</td>
<td>Premature 4.5–7.2 mmol/L</td>
</tr>
<tr>
<td></td>
<td>Term 3.2–5.7 mmol/L</td>
</tr>
<tr>
<td></td>
<td>3.3–4.7 mmol/L</td>
</tr>
<tr>
<td>Calcium</td>
<td>Premature 3.5–4.5 mEq/L</td>
</tr>
<tr>
<td></td>
<td>(1.7–2.3 mmol/L)</td>
</tr>
<tr>
<td></td>
<td>Term 4–5 mEq/L (2–2.5 mmol/L)</td>
</tr>
<tr>
<td></td>
<td>4.4–5.3 mEq/L (2.2–2.7 mmol/L)</td>
</tr>
<tr>
<td>Magnesium</td>
<td>1.3–2.7 mg/dL (0.5–1.1 mmol/L)</td>
</tr>
<tr>
<td></td>
<td>1.6–2.7 mg/dL (0.7–1.1 mmol/L)</td>
</tr>
</tbody>
</table>

*Note: Laboratories may have slightly different levels of normal depending on assays performed. Always consult the normal values for your particular laboratory.
When a child is hospitalized for hypernatremia, monitor serum sodium level and measure intake and output and urine specific gravity. Specific gravity changes toward normal levels as therapy progresses. Frequently assess responsiveness to monitor the effect of hypernatremia on brain cells. As the concentration of body fluids returns to normal, the child will become more alert and responsive. Watch for rebound hyponatremia while monitoring the fluid replacement. Implement safety interventions such as raised bed rails for protection. Ensure adequate rest and introduce developmentally appropriate activities when the child is alert.

Water deprivation is a form of child neglect or abuse. In neglect, the parents simply do not provide adequate water for the child. A form of child abuse that sometimes includes water deprivation is Munchausen syndrome by proxy (see Chapter 6). A small child who is hospitalized with hypernatremia that does not have a detectable cause may be subject to water deprivation. Assess the child’s general condition, developmental tasks, the family dynamics, and the parent’s understanding of formula preparation and the child’s fluid intake needs.

Nurses can prevent hypernatremia in hospitalized infants and children by administering water between tube feedings, keeping water available, and offering it frequently. Offering frequent small amounts and using Popsicles and other creative interventions can increase children’s intake.

Desired outcomes of treatment for hypernatremia include balance of electrolytes and fluid in the intracellular and extracellular compartments, as well as alert level of consciousness.

**Hyponatremia**

In hyponatremia, the osmolality of the blood is decreased. The body fluids are too dilute, and contain excess water relative to sodium. Hyponatremia is the most common sodium imbalance in children (Greenbaum, 2004). A serum sodium level below 135 mmol/L in children (133 mmol/L in newborns) is diagnostic of hyponatremia.

**ETIOLOGY AND PATHOPHYSIOLOGY** Hyponatremia results from conditions that cause gain of relatively more water than sodium or loss of relatively more sodium than water (Table 16–6). Oral intake of water causes hyponatremia in unusual conditions such as forced fluid intake. More commonly, parents feed an infant only water or dilute formula to save money instead of regular-strength formula or breast milk. Excessive swallowing of swimming pool water by an infant can have the same effect. Infants are vulnerable to the type of hyponatremia caused by water intoxication, because they have a poorly developed thirst mechanism and may continue to drink, and then are unable to excrete excess water quickly due to immature kidney function (Chamley, Carson, Randall, & Sandwell, 2005). Exercise-associated hyponatremia can occur when persons in prolonged physical activity such as marathon running consume hypotonic fluids in the form of water or sports drinks above the levels lost in respiratory,

<table>
<thead>
<tr>
<th>Gain of Relatively More Water than Sodium</th>
<th>Loss of Relatively More Sodium than Water</th>
</tr>
</thead>
<tbody>
<tr>
<td>Excessive intravenous D5W (5% dextrose in water) rather than isotonic fluids for hospitalized children</td>
<td>Diarrhea or vomiting with replacement by tap water only instead of fluid containing sodium</td>
</tr>
<tr>
<td>Excessive tap water enemas</td>
<td>Excessive sweating such as in cystic fibrosis</td>
</tr>
<tr>
<td>Irrigation of body cavities with distilled water</td>
<td>Diuretics, especially thiazides</td>
</tr>
<tr>
<td>Excessive antidiuretic hormone</td>
<td>Forced excessive oral intake of tap water</td>
</tr>
<tr>
<td>Forced excessive intake of tap water</td>
<td>Excessive intake of water during exercise</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td></td>
</tr>
</tbody>
</table>
gastrointestinal, skin, and urinary routes (Exercise-Associated Hyponatremia Consensus Panel, 2005). Hospitalized children who are treated with hypotonic saline rather than isotonic solutions can also acquire hyponatremia. Young children are at particular risk because they commonly respond to surgery with increased levels of antidiuretic hormone (ADH) for 3 to 5 days postsurgery, causing decreased excretion of urine; use of hypotonic solutions during this period can cause hyponatremia. Additionally, they have a high brain-to-skull mass and are therefore at high risk of developing the neurologic complications of hyponatremia (Moritz & Ayus, 2003).

**CLINICAL MANIFESTATIONS** The child who has hyponatremia has a decreased level of consciousness, which results from edema of brain cells. Manifestations include anorexia, nausea, vomiting, confusion, headache, respiratory distress, muscle weakness, decreased deep tendon reflexes, agitation, lethargy, or confusion. The condition can progress to respiratory arrest, dilated pupils, decorticate posturing, and coma. If hyponatremia arises rapidly or is extreme, seizures may occur, and is a frequent cause of seizures in infants under 6 months of age. Severe hyponatremia can be fatal.

**COLLABORATIVE CARE**

Laboratory analysis of plasma or serum demonstrates a low sodium level.

Hyponatremia should be prevented in hospitalized children receiving intravenous solutions (particularly postoperatively) by administering isotonic rather than hypotonic solutions. In cases of improper formula preparation or fluid intake, hyponatremia is treated by feeding proper formula or restricting the intake of water. This therapy allows the kidneys to correct the imbalance by excreting excess water from the body. Intravenous **hypertonic fluid** (more concentrated than body fluid) may be administered for severe cases. Use of this concentrated saline is a way to rapidly increase body fluid concentration, but it must be monitored carefully because it can easily cause rebound hypernatremia. For exercise-associated hyponatremia, intravenous access is established at the first aid site, hypertonic saline is administered, and oxygen is delivered (Exercise-Associated Hyponatremia Consensus Panel, 2005). In cases of diabetes insipidus, treatment for the condition is needed (see Chapter 29).

**NURSING MANAGEMENT**

**Nursing Assessment and Diagnosis**

Monitor serum sodium level and measure intake and output. If an infant with hyponatremia has normal antidiuretic hormone (ADH) levels, and other causes have been ruled out, careful questioning about proper preparation of formula and feeding practices is needed. A toddler or school-age child may be subjected to forced fluid intake as a form of child abuse. Sensitive interviewing and a caring manner on the part of the nurse can help identify such problems in a family.

Because hyponatremia is characterized by a decreased level of consciousness, frequent assessment of responsiveness will be necessary to monitor the response to therapy. The child will become more alert and responsive as the concentration of body fluids returns to normal. Carefully monitor hospitalized children and those exercising for signs of hyponatremia.

The highest priority nursing diagnosis for hyponatremia addresses the risk for injury related to the child’s decreased level of consciousness and cerebral edema. The following nursing diagnoses might also apply:

- Self-Care Deficit related to weakness and fatigue
- Altered Health Maintenance related to parental information misinterpretation about infant formula
- Ineffective Breast-Feeding related to inadequate sucking by infant or inadequate milk production
**Planning and Implementation**

Nurses can prevent hyponatremia in hospitalized children by using normal saline instead of distilled water for irrigations and by avoiding tap water enemas. Verify intravenous types and amounts and question use of hypotonic fluids in a child with no intake of sodium. Teach parents to replace body fluids lost through diarrhea or vomiting with oral electrolyte solutions (see discussion of oral rehydration therapy earlier in this chapter). Teach the person with prolonged exercise to slowly increase exercise times, rehydrate according to thirst, and ensure intake of sodium-containing fluids. The child with diseases such as cystic fibrosis or who is taking thiazide diuretics needs intake above that recommended for usual maintenance needs.

**Evaluation**

Expected outcomes of nursing care for hyponatremia include the following:

- The child remains safe from injury.
- Balance of fluid and electrolytes is maintained.
- Proper intake of formula, breast milk, and other fluids is established.

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**Potassium Imbalances**

Potassium is an essential anion (negatively charged particle) that performs many necessary functions in the body. It is present in high levels in intracellular fluids and is active in enzyme performance in cells. It is needed for contractility of heart and skeletal muscle. Potassium intake in healthy children comes from potassium-rich foods such as fruits and vegetables. Potassium is absorbed easily from the intestine.

A potassium imbalance arises when the serum potassium concentration rises or falls outside the normal range. Potassium imbalances are caused by alterations in potassium intake, distribution, or excretion; or by loss of potassium through an abnormal route such as burns, emesis, or renal failure.

Most of the potassium ions in the body are found inside the cells. The sodium-potassium pump in cell membranes moves potassium ions into cells to maintain the high intracellular potassium concentration (see Figure 16–13). In addition, potassium ions can be shifted into or out of cells by various physiologic factors (Figure 16–17 ➤). Potassium is excreted from the body through urine, feces, and sweat. The hormone aldosterone increases potassium excretion in the urine.

**Hyperkalemia**

Hyperkalemia, an excess of potassium in the blood, is reflected by a level above 5.8 mmol/L in children or above 5.2 mmol/L in newborns.

**Etiology and Pathophysiology**

Hyperkalemia is caused by conditions that involve increased potassium intake, shift of potassium from cells into the extracellular fluid, and decreased potassium excretion. Renal insufficiency is a primary cause...
CHAPTER 16 of hyperkalemia (Burger, 2004a). Increased potassium intake is usually due to intravenous potassium overload. Excessive or too rapid intravenous administration of potassium-containing solutions can occur if the potassium requirement is overestimated or if the intravenous infusion runs in too quickly.

Blood transfusion is another source of potassium intake that may cause hyperkalemia. Potassium ions leak out of red blood cells that are stored in a blood bank. The longer the blood is stored, the more potassium leaks out of cells and accumulates in the fluid portion of the transfusion. Hyperkalemia from administration of stored blood arises when multiple units are transfused, as when infants receive exchange transfusions or children receive multiple blood transfusions after a serious injury or in surgery.

Shift of potassium from cells into the extracellular fluid occurs when there is massive cell death, as with a crush injury, in sickle-cell anemia (hemolytic crisis), or when chemotherapy for a malignancy is rapidly effective. In these situations, the dead cells release their high-potassium contents into the extracellular fluid. Potassium ions also shift out of cells in metabolic acidosis caused by diarrhea and in diabetes mellitus when insulin levels are low.

Decreased potassium excretion occurs with acute or chronic oliguria during renal failure, severe hypovolemia, and conditions that decrease the secretion of aldosterone by the adrenal cortex (lead poisoning, Addison’s disease, hypoaldosteronism). Several medications can cause hyperkalemia.

**CLINICAL MANIFESTATIONS** All clinical manifestations of hyperkalemia are related to muscle dysfunction because potassium plays a vital role in muscle activity. Hyperactivity of gastrointestinal smooth muscle causes intestinal colic, cramping, and diarrhea in some children. The skeletal muscles become weak, beginning typically with leg weakness and then ascending up the body. Weakness can progress to flaccid paralysis. The child is often lethargic. Dysfunction of cardiac muscle causes cardiac arrhythmias such as tachycardia and may result in heart failure and cardiac arrest. Abnormalities in the electrocardiogram include a prolonged QRS complex, a peak in T waves, and prolonged PR intervals. Renal signs include oliguria and anuria (Burger, 2004a).

**COLLABORATIVE CARE**

The major diagnostic tool is the serum laboratory test for potassium. In addition, observations of symptoms and abnormal electrocardiograph are indicative of hyperkalemia. Hyperkalemia is treated by management of the underlying condition that caused the imbalance. For mild cases, intake of potassium is restricted, and loop or thiazide diuretics may be administered. If the serum potassium concentration is very high or is causing dangerous cardiac arrhythmias, treatment to decrease the serum potassium level may be ordered. These treatments may remove potassium from the body or drive it from the extracellular fluid into the cells. Potassium is removed from the body by peritoneal dialysis or hemodialysis, and with a cation exchange resin (Kayexalate) or 70% sorbitol, both of which can be administered orally or rectally. Medical treatments that drive potassium ions into cells are intravenous sodium bicarbonate, intravenous insulin, glucose, and calcium gluconate.

NURSING MANAGEMENT

**Nursing Assessment and Diagnosis**

Monitor serum potassium levels. Ongoing assessment of muscle strength is important, because the muscle weakness may progress to flaccid paralysis. (This paralysis is reversible on correction of the potassium imbalance.) Diarrhea or colic can occur in infants and children. An older child may complain of intestinal cramping. Monitor the pulse rate carefully. Monitor urinary output, particularly in children with renal disease.
Nursing diagnoses for a child who has hyperkalemia depend on the severity of the clinical manifestations. The cause of the imbalance may also lead to useful diagnoses that guide teaching for the child and the parents regarding safety measures and accurate medication administration. The following nursing diagnoses may apply:

- Activity Intolerance related to decreased cardiac output secondary to cardiac arrhythmias
- Risk for Injury related to muscle weakness
- Self-Care Deficit: Hygiene and Dressing related to neuromuscular impairment
- Anxiety related to change in health status
- Ineffective Health Maintenance related to parental lack of exposure to potassium intake in chronic renal failure
- Ineffective Management of Therapeutic Regimen related to complexity of therapy

**Planning and Implementation**
Nursing care includes measures to prevent hyperkalemia from developing in hospitalized children. If hyperkalemia does develop, care shifts to administering intravenous solutions, monitoring cardiopulmonary status continuously, ensuring safety, promoting adequate nutrition, and preparing the child and family for discharge. For the child in the community, potassium levels are monitored when the child is taking a drug that can cause hyperkalemia, such as those used for cancer treatment.

**Prevent Hyperkalemia**
Any child who is receiving an intravenous infusion that contains potassium is at risk for hyperkalemia. Check that urine output is normal before administering intravenous potassium solutions. Observe the child closely and perform cardiorespiratory monitoring.

Be sure blood or packed red blood cells are fresh, especially for the child receiving multiple transfusions and for all neonates. Use a cardiac monitor during infusion of these products to watch for arrhythmias.

**Administer Intravenous Solutions**
Once a child is diagnosed as hyperkalemic, ensure that any infusions with added potassium are stopped. Several infusions may need to be managed, including glucose, bicarbonate, and calcium gluconate. Maintain the infusion at the ordered rate and monitor the child's condition frequently.

**Monitor Cardiopulmonary Status**
Upon diagnosis of hyperkalemia, an electrocardiogram is performed and a cardiac monitor is applied. Monitor for any changes in cardiac status and for cardiac arrhythmias. Report abnormal rate and character of pulse as well as shortness of breath.

**Ensure Safety**
Since the child is weak, side rails should be raised. Position the child carefully. Assist the child with activities requiring leg muscle strength, such as going to the bathroom, climbing into bed, or pushing up in bed. Encourage quiet activities appropriate for developmental level with frequent rest periods. Document and report any change in muscle weakness.

**Promote Adequate Nutritional Intake**
Adequate caloric intake is necessary to prevent tissue breakdown and the resultant potassium release from cells. Offer the child nourishing snacks if his or her appetite is decreased. Restrict potassium-rich foods.

**Discharge Planning and Home Care Teaching**
If the child has chronic renal failure or another condition that decreases aldosterone secretion, parents and the child need to be taught to restrict foods that are high in potassium (Families Want to Know: Potassium-Rich Foods). Most oral rehydration solutions, including Pedialyte, contain potassium and should not be used to provide fluid
for the child. Instruct the family not to use salt substitutes, which commonly contain potassium. Parents should check with the care provider and pharmacist before giving even over-the-counter products to the child, as some of these medications contain potassium. Management of renal failure at home with frequent visits for dialysis and other treatments can be challenging. Refer to Chapter 25 for further suggestions to help parents handle this condition.

**Evaluation**

Expected outcomes of nursing care for hyperkalemia include the following:

- The child returns to a state of fluid and electrolyte balance.
- Safety is maintained.
- The child receives adequate nutritional intake to provide essential potassium.
- Normal cardiac rate and rhythm is maintained.

**Hypokalemia**

Hypokalemia occurs when the serum potassium concentration is too low. Total body potassium may be decreased, normal, or even increased when the serum level is low, depending on the cause of the imbalance. Serum potassium levels below 3.5 mmol/L in children (3.7 mmol/L for newborns) are diagnostic of hypokalemia.

**Etiology and Pathophysiology**

Hypokalemia is caused by conditions that involve increased potassium excretion, decreased potassium intake, shift of potassium from the extracellular fluid into cells, and loss of potassium by an abnormal route.

- Increased potassium excretion through the gastrointestinal tract is the major cause of hypokalemia in children (Burger, 2004b). Loss of potassium occurs through vomiting and diarrhea (gastroenteritis). In the chapter opening vignette, Vernon had increased potassium excretion through diarrhea. Self-induced vomiting in bulimia is another example of this cause. Nasogastric suctioning (Figure 16–18) and intestinal decompression can cause potassium loss.

- Causes of increased urinary potassium excretion are osmotic diuresis (glucose present in urine), hypomagnesemia, hypercalcemia, increased aldosterone (hyperaldosteronism, congestive heart failure, nephrotic syndrome, cirrhosis), and increased cortisol (Cushing’s disease and syndrome). Eating large amounts of black licorice made from the root of *Glycyrrhiza glabra* increases renal retention of sodium and excretion of potassium (Burger, 2004b).

- Decreased potassium intake will lead to hypokalemia slowly, or more rapidly if combined with increased excretion or loss of potassium. Hospitalized children may be placed on NPO status and receive prolonged intravenous therapy without potassium.
Adolescents concerned about weight loss or those with anorexia nervosa may embark on diets low in potassium and may take medications that induce diuresis or diarrhea.

Shift of potassium from the extracellular fluid into cells occurs in alkalosis and hypothermia (unintentional or induced for surgery). Hyperalimentation often causes hypersecretion of insulin, which also shifts potassium into cells. Hypokalemia can also be caused by several medications.

**Clinical Manifestations**

Since the ratio of intracellular to extracellular potassium determines the responsiveness of muscle cells to neural stimuli, it is not surprising that the clinical manifestations of hypokalemia involve muscle dysfunction. Gastrointestinal smooth muscle activity is slowed, leading to diminished bowel tones, abdominal distention, constipation, or paralytic ileus. Skeletal muscles are weak and unresponsive to stimuli, deep tendon reflexes are diminished, and weakness may progress to flaccid paralysis. The respiratory muscles may be impaired. Cardiac arrhythmias can occur, particularly a prolonged QT interval, depressed ST segment, and flat or inverted T waves. Polyuria, polydipsia, and decreased urine specific gravity result from changes in the kidney caused by hypokalemia (Burger, 2004b; English, 2002).

**COLLABORATIVE CARE**

The major diagnostic tool is the serum laboratory test for potassium. In addition, observations of symptoms and an abnormal electrocardiograph are indicative of hypokalemia. Medical management of hypokalemia focuses on replacement of potassium while treating the cause of the imbalance. Potassium replacement may be given intravenously or orally.

**NURSING MANAGEMENT**

**Nursing Assessment and Diagnosis**

Monitor serum potassium levels. Observe for muscle weakness, which is frequently detected first in the legs. Parents may report that muscle weakness restricts the child’s activities and impairs interactions with peers. Skeletal muscle strength can be difficult to assess if the child is lethargic, as shown with Vernon at the beginning of the chapter.

Muscle weakness may affect the respiratory muscles. Assess the child frequently to determine the need for assisted ventilation. Cardiac monitoring is important for continued assessment of hypokalemia-associated arrhythmias.

Assess for diminished bowel sounds. Ask the parents if the child has recently been awakening to use the toilet at night or has begun bedwetting after previously being dry at night. These may be symptoms of polyuria associated with chronic hypokalemia.

The most important nursing diagnoses in the child with severe hypokalemia relate to cardiac arrhythmias and respiratory muscle weakness. The following nursing diagnoses may apply:

- Risk for Activity Intolerance related to decreased cardiac output secondary to cardiac dysrhythmias
- Ineffective Breathing Pattern related to respiratory musculoskeletal impairment
- Risk for Injury related to muscle weakness
- Self-Care Deficit: Hygiene and Dressing related to neuromuscular impairment
- Constipation related to decreased motility
- Anxiety related to change in health status
- Ineffective Health Maintenance related to management of potassium supplements or high-potassium diet

**NURSING ALERT**

Drugs that may cause hypokalemia:
- Beta-adrenergic agonists
- Insulin
- Potassium-wasting diuretics
- Parenteral penicillins
- Glucocorticoids
- Aminoglycoside antimicrobials
- Systemic antifungals
- Antineoplastics
- Laxatives, especially when abused
- Osmotic diuretics (mannitol)
• Ineffective Management of Therapeutic Regimen related to complexity of potassium therapy
• Imbalanced Nutrition: Less than Body Requirements related to lack of basic nutritional knowledge regarding safe weight-loss diet

Planning and Implementation
Nursing care of the child with hypokalemia focuses on ensuring adequate potassium intake, monitoring cardiopulmonary status, promoting normal bowel function, ensuring safety, providing dietary counseling, and preparing the child and family for discharge.

Ensure Adequate Potassium Intake
Since potassium is excreted from the body every day, daily potassium intake is necessary to prevent hypokalemia. A hypokalemic child who is able to eat should be given a high-potassium diet. Teach parents (and the child if old enough) which foods are high in potassium and how to incorporate them into the daily diet (see Families Want to Know on page 524).

Children who have no oral intake for a period of time should receive intravenous fluids that contain potassium. Calculate the dosage to ensure accuracy, and be sure that the infusion runs on schedule. Sometimes the child will complain of burning along the vein when potassium is infused. The infusion may need to be slowed temporarily to relieve pain and maintain the intravenous line. Remain vigilant to maintain patency of the vein in order to avoid infiltration, which can cause phlebitis and pain. A central line is a better choice than a peripheral line in order to decrease side effects of administration. Consult the hospital formulary for dilution and administration guidelines; it must be administered slowly to avoid arrhythmias and cardiac arrest. Ensure adequate fluid output for the child’s age to avoid hyperkalemia from potassium infusion.

Check serum potassium for high or low potassium levels. Analyze other electrolytes and acid-base balance. Monitor urine output. An oliguric child can develop hyperkalemia when receiving supplements.

Monitor Cardiopulmonary Status
Hypokalemia potentiates digitalis toxicity. A hypokalemic child who is receiving digitalis needs careful surveillance for digitalis toxicity, which is manifested as anorexia, nausea, vomiting, and bradycardia. Observe for these effects. Take the pulse rate and rhythm regularly. Monitor respirations and ease of breathing to watch for decreased respiratory muscle activity.

Promote Normal Bowel Function
Ensure adequate fluids and fiber in the diet. Monitor and record the number of stools and report inadequate stools.

Ensure Safety
Keep side rails up. Assist the child as needed to move into and out of bed. Reposition the child frequently to preserve the skin integrity of limbs that are not moved regularly. Perform passive range of motion if the child is not moving. Use supportive pillows to position the child properly.

Provide Dietary Counseling
The adolescent who is trying to lose weight and not consuming a nutritious diet needs dietary teaching. More intensive treatment will be needed for teens who are anorexic or bulimic (see Chapter 4 for interventions in these cases).

Discharge Planning and Home Care Teaching
Teach parents how to give potassium supplements, if prescribed. Liquid or powdered potassium supplements can be mixed with juice or sherbet to improve the bitter taste. The parent should call the mixture “medicine” so that the child does not learn to dislike all juices. Teach the parents signs of both hypokalemia and hyperkalemia and whom to call to report these symptoms. The signs must be reported promptly so medications can be adjusted.
Evaluation
Expected outcomes of nursing care during hypokalemia include the following:

- Normal rate and rhythm of heart and respiratory system is maintained.
- Regular bowel movements are established.
- The child is free from injury.
- The child and family have adequate knowledge regarding food sources of potassium.

Calcium Imbalances
A normal serum calcium concentration is important for many physiologic functions, including muscle and nerve function, secretion of hormones, bone formation and strength, and clotting of the blood. Calcium is the most abundant mineral in the body, and about 98% of it is located in the bones (Roberts, 2005). There are three forms of calcium in plasma—calcium bound to protein, calcium bound to small organic ions (e.g., citrate), and free ionized calcium (\(\text{Ca}^{++}\)), the only physiologically active form. A discussion of dietary calcium intake and its importance for bone formation can be found in Chapter 4.

Calcium imbalances are caused by alterations in calcium intake, absorption, distribution, or excretion. Calcium absorption requires vitamin D for maximum efficiency and is greatest in the duodenum. Calcium distribution involves calcium entry into and exit from bones and the distribution of different forms of calcium in the plasma. Excretion of calcium occurs in urine, feces, and sweat (Pathophysiology Illustrated, Figure 16–19 ➤).

Parathyroid hormone is the major regulator of the plasma calcium concentration. It increases this concentration by increasing calcium absorption, increasing calcium withdrawal from bones, and decreasing calcium excretion in the urine. The plasma calcium

PATHOPHYSIOLOGY ILLUSTRATED

Calcium Imbalance
Some causes of excess calcium in the blood (hypercalcemia)

- Vitamin D overdose
- Hyperparathyroidism
- Bone tumors and other cancers
- Thiazide diuretics
- Familial hypercalcemia

Some causes of decreased calcium in the blood (hypocalcemia)

- Insufficient dietary calcium and vitamin D intake
- Chronic diarrhea
- Laxative abuse
- Malabsorption
- Chronic renal insufficiency
- Hypoparathyroidism
- Alkalosis
- Large transfusion of citrated blood
- Rapid infusion of plasma expanders

Figure 16–19 ➤ A variety of conditions can lead to hypercalcemia and hypocalcemia.
concentration has an important influence on cell membrane permeability and influences the threshold potential of excitable cells. For this reason, calcium imbalances alter neuromuscular irritability.

**Hypercalcemia**

Hypercalcemia refers to a plasma excess of calcium (above 5.3 mEq/L [2.7 mmol/L] in children or 5 mEq/L [2.5 mmol/L] in newborns). Because so much calcium is stored in the bones, however, the serum levels of calcium may not reflect body stores.

**ETIOLOGY AND PATHOPHYSIOLOGY**

Hypercalcemia is caused by conditions that involve increased calcium intake or absorption, shift of calcium from bones into the extracellular fluid, and decreased calcium excretion. Hypercalcemia due to increased calcium intake or absorption may occur if an infant is fed large amounts of chicken liver (source of vitamin A), is given megadoses of vitamin D or vitamin A, or if a child or adolescent consumes large amounts of calcium-rich foods concurrently with antacids (milk-alkali syndrome). Infants with very low birth weight can develop hypercalcemia if they have inadequate phosphorus intake, as bone phosphorus and calcium will be resorbed. Hypercalcemia may also occur when children receiving total parenteral nutrition are given doses of calcium that are too high.

Most cases of hypercalcemia in children are due to a shift of calcium from bones into the extracellular fluid. The excessive amounts of parathyroid hormone produced in hyperparathyroidism cause calcium withdrawal from bones. Prolonged immobilization also causes withdrawal of calcium from bones. Often, the excess calcium ions are excreted in the urine. However, if calcium is withdrawn from bones faster than the kidneys can excrete it, hypercalcemia results. Hypercalcemia also occurs with many types of malignancies such as leukemias. The malignant cells produce substances that circulate in the blood to the bones and cause bone resorption. The calcium from the bones then enters the extracellular fluid, causing hypercalcemia. Bone tumors and chemotherapy destroy bone directly, leading to the release of calcium. Familial hypercalcemia and infantile hypercalcemia are rare congenital disorders.

Thiazide diuretics (e.g., thiazide and hydrochlorthiazide) decrease calcium excretion in the urine and may contribute to development of hypercalcemia. Other drugs that can cause hypercalcemia include lithium and theophylline (Carmichael & Alper, 2004).

**CLINICAL MANIFESTATIONS**

Hypercalcemia may have nonspecific symptoms, making diagnosis difficult. Many of the signs and symptoms of hypercalcemia are manifestations of decreased neuromuscular excitability. Constipation, anorexia, nausea, and vomiting can occur. Fatigue and skeletal muscle weakness predominate. Confusion, lethargy, and decreased attention span are common, and polyuria develops. Severe hypercalcemia may cause cardiac arrhythmias and arrest. Neonates with hypercalcemia have flaccid muscles and exhibit failure to thrive. Hypercalcemia increases sodium and potassium excretion by the kidneys and can lead to polyuria and polydipsia.

### COLLABORATIVE CARE

Serum calcium is tested although the blood levels may not reflect bone stores. Additional diagnostic laboratory analyses to assist in diagnosis of the cause include albumin, phosphate, magnesium, alkaline phosphate, electrolytes, blood urea nitrogen, creatinine, and parathyroid hormone.

Hypercalcemia is treated by increasing fluids and administering the diuretic furosemide (Lasix) to increase excretion of calcium in the urine. Treatment to decrease intestinal absorption of calcium involves effective use of glucocorticoids. Bone resorption can be decreased by administration of glucocorticoids and calcitonin. Phosphate is sometimes given to treat hypercalcemia, but it may cause dangerous precipitation of calcium phosphate salts in body tissues. Dialysis may be used, if necessary. Treatment of the underlying cause for the disorder is needed as well.
NURSING MANAGEMENT

Nursing Assessment and Diagnosis

Nursing assessment of a child with hypercalcemia includes monitoring serum calcium levels, level of consciousness, gastrointestinal function, urine volume, specific gravity, cardiac rhythm, and pH. With chronic hypercalcemia, assessment of activity tolerance and developmental level becomes important.

Many nursing diagnoses are appropriate for children who have hypercalcemia. Diagnoses that address cardiac and neuromuscular manifestation are especially important. The following nursing diagnoses may apply:

- Risk for Activity Intolerance related to decreased cardiac output secondary to cardiac arrhythmia
- Risk for Injury related to decreased level of response
- Risk for Injury related to neuromuscular impairment
- Risk for Injury related to possibility of spontaneous fractures
- Self-Care Deficit: Hygiene and Dressing related to neuromuscular impairment
- Anxiety related to change in health status
- Constipation related to decreased motility
- Risk for Imbalanced Nutrition: Less than Body Requirements related to anorexia and nausea
- Risk for Impaired Urinary Elimination related to renal calculi

Planning and Intervention

Carefully calculate calcium in total parenteral nutrition and other solutions, administer these solutions with caution, and use cardiac monitoring to prevent hypercalcemia in hospitalized children.

Interventions to increase fluid intake are important for children with hypercalcemia or those who are immobilized. A generous fluid intake, appropriate to the child’s age, is necessary to keep the urine dilute and to help reduce constipation (a common symptom of hypercalcemia). An acidic urine helps to keep calcium from forming stones. Because urinary tract infections may cause the urine to be alkaline, nursing interventions to prevent urinary tract infection are necessary. Thiazide diuretics, which decrease calcium excretion, should not be given to the hypercalcemic child. Provide a high-fiber diet to help reduce constipation.

Increasing mobility through assisted weight bearing helps to decrease the withdrawal of calcium from bones that is caused by immobility. If the hypercalcemia is caused by withdrawal of calcium from bones, the child is at risk for fractures with minor trauma and must be handled with special care. See Chapter 28 for further discussion of care following fractures and prolonged casting.

Teach parents to avoid giving calcium-rich foods and calcium antacids (e.g., Tums) to children with hypercalcemia. Vitamin D supplements should be avoided as they increase calcium absorption from the gastrointestinal tract.

Evaluation

Expected outcomes of nursing care include the following:

- The cardiac pump effectively maintains perfusion.
- The child is free from injury.
- Normal bowel excretion is maintained.
- Adequate nutritional status is maintained.

Hypocalcemia

Hypocalcemia is a serum deficit of calcium (below 4.4 mEq/L [2.2 mmol/L] in children or 4 mEq/L [2 mmol/L] in newborns). Remember that serum calcium levels may not reflect body stores of this mineral, as most of the body’s calcium is stored in bone.
Hypocalcemia

Hypocalcemia in infants is more frequently manifested as tremors, muscle twitches, and brief tonic-clonic seizures.

**NURSING ALERT**

Drugs that may cause hypocalcemia:
- Antacids (if overused)
- Laxatives (if overused)
- Oil-based bowel lubricants
- Anticonvulsants
- Phosphate-containing preparations
- Protein-type plasma expanders during rapid infusion
- Antineoplastics

**ETIOLOGY AND PATHOPHYSIOLOGY**

Hypocalcemia is caused by conditions that involve decreased calcium intake or absorption, shift of calcium to a physiologically unavailable form, increased calcium excretion, and loss of calcium by an abnormal route.

Decreased calcium intake or absorption causes hypocalcemia in children with chronic generalized malnutrition, or with a diet that is low in vitamin D and calcium. Female adolescents trying to lose weight or maintain a low weight often decrease foods that contain calcium and may develop chronic hypocalcemia. In these cases, premature bone loss and inadequate bone formation occur. (See Chapter 4 for further discussion of calcium intake during adolescence.) This deficit cannot be made up later in life, thus increasing the risk of osteoporosis.

Even with a normal calcium intake, hypocalcemia occurs if the mineral is not absorbed. If a child does not have enough vitamin D, calcium is not absorbed efficiently from the duodenum. Sunlight speeds formation of vitamin D in the skin. Children who are institutionalized without access to sunlight (e.g., severely developmentally delayed children), those with very dark skin, or children kept well covered when outside may become hypocalcemic because of the lack of vitamin D (see Chapter 4). Uremic syndrome is another cause of vitamin D deficiency. It interferes with the kidney's ability to activate vitamin D. High phosphate intake can cause hypocalcemia. Chronic diarrhea and steatorrhea (fatty stools) also reduce calcium absorption from the gastrointestinal tract.

The shift of calcium into a physiologically unavailable form occurs when calcium shifts into bone or free ionized calcium in plasma binds to proteins or small organic ions in the plasma. Too much calcium shifts into bones in various types of hypoparathyroidism, including DiGeorge syndrome (congenital absence of the parathyroid glands). Hypomagnesemia impairs parathyroid hormone function and may cause hypocalcemia. Some types of neonatal hypocalcemia are associated with delayed parathyroid hormone function or hypomagnesemia. Calcium shifts rapidly into bone when rickets is treated. A high plasma phosphate concentration causes plasma calcium to decrease. Alkalosis causes more calcium to bind to plasma proteins. The ionized hypocalcemia persists until the alkalosis resolves or the citrate is metabolized by the liver. Citrate in transfused blood products may bind with calcium so it is inactive. Children who receive liver transplants are hypocalcemic for several days because of impaired citrate metabolism.

Increased calcium excretion occurs in steatorrhea, when calcium secreted into the gastrointestinal fluid binds to the fecal fat in addition to the dietary calcium that is bound in the feces. A similar situation occurs in acute pancreatitis.

Loss of calcium by an abnormal route may contribute to hypocalcemia as calcium is lost from the body through burn or wound drainage or sequestered in acute pancreatitis. Many different medications can cause hypocalcemia.

**CLINICAL MANIFESTATIONS**

The signs and symptoms of hypocalcemia are manifestations of increased muscular excitability (tetany). In children they include twitching and cramping, tingling around the mouth or in the fingers, carpal spasm, and pedal spasm. Laryngospasm, seizures, and cardiac arrhythmias are the more severe manifestations of hypocalcemia and may be fatal. Hypocalcemia may cause congestive heart failure, especially in neonates.

Although these symptoms are diagnostic of acute calcium deficiency, a more common state in children and adolescents is chronic low intake of calcium. This may be manifested by spontaneous fractures in infants and in adolescents who exercise excessively.

**COLLABORATIVE CARE**

Laboratory measurement of calcium is the most useful diagnostic tool. Cardiac monitoring may be performed to observe for cardiac arrhythmias.

Hypocalcemia is treated by oral or intravenous administration of calcium. The original cause of the imbalance is also treated. If the hypocalcemia is due to hypomagnesemia, the magnesium must be replenished before the calcium replacement can be successful. When the cause is chronic low dietary intake, counseling is needed about high-calcium foods, and perhaps the necessity for vitamin D intake or supplements.
NURSING ALERT
Oral Calcium
Calcium tablets and powders are available for relief of acid indigestion and to increase calcium intake when it is deficient. Popular products contain calcium carbonate (i.e., Tums), calcium acetate, calcium citrate, tricalcium phosphate, calcium lactate, calcium gluconate, and calcium polycarbophil. Since so many forms exist, be sure that chewable tablets are chewed, sustained release tablets are swallowed whole, and powders are mixed and administered as recommended. The most common side effect is constipation; other side effects are hypercalcemia and renal calculi.

Intravenous Calcium
Intravenous calcium is administered to treat severe hypocalcemia such as in tetany due to parathyroid disease, in cardiac resuscitation, during exchange transfusions in newborns, and to relieve muscle cramps caused by insect bites. Intravenous calcium has several serious potential side effects, so nursing care centers on maintaining an intact intravenous line, continuous cardiorespiratory monitoring, and monitoring calcium and phosphate levels.

NURSING MANAGEMENT
Nursing Assessment and Diagnosis
Carefully assess growth in the young female who is trying to diet. When an adolescent female is very thin, be sure to ask about excessive sports and other activities, and about regularity of menstrual periods. If periods are irregular or not occurring, collect additional dietary information to help determine whether the girl is lacking in intake of calcium, calories, and other nutrients. These assessments are needed even if serum calcium values are normal. Look for signs of inadequate nutrition such as fat and muscle wasting, dry hair, and cold hands and feet.

In those who have acute hypocalcemia, assess for muscle cramps, stiffness, and clumsiness; grimacing caused by spasms of facial muscles and twitching of arm muscles; and laryngospasm. Increased neuromuscular excitability may be detected by testing for Trousseau’s sign or Chvostek’s sign. Many healthy newborns have a positive Chvostek’s sign; however, this assessment should be reserved for children over several months of age.

The effects of increased neuromuscular excitability in the child with hypocalcemia are the following nursing diagnoses:

- Risk for Injury related to potential for fractures
- Risk for Ineffective Breathing Pattern related to laryngospasm
- Risk for Activity Intolerance related to decreased cardiac output secondary to cardiac arrhythmias
- Disturbed Sensory Perception related to electrolyte imbalance
- Imbalanced Nutrition: Less than Body Requirement related to lack of basic nutritional knowledge of sources and recommended amounts of calcium intake

Planning and Implementation
To correct calcium deficiency in the hospitalized child, give oral or intravenous calcium as ordered. Monitor for complications of calcium supplementation. A 10% calcium gluconate solution should be readily available for emergency use in severe hypocalcemia. Calcium is never given intramuscularly because it causes tissue necrosis. See Medications Used to Treat Acute Hypocalcemia on page 532.

Take measures to ensure safety for the child who is hospitalized with hypocalcemia. Seizure precautions may be necessary. Explain the cause of muscle cramps to parents and older children.

Counsel the family about dairy products and nondairy foods rich in calcium (Families Want to Know: Calcium-Rich Foods). For the adolescent female whose weight and menstrual patterns show irregularities, total calories and calcium intake should be increased. Teaching may also be needed about proper calcium intake and its importance both to athletic performance and to prevention of osteoporosis. Encourage three glasses of nonfat milk per day. Teach ways to use milk in the diet. For example, sprinkle nonfat dry milk on cereal and other foods. If the child is lactose intolerant, emphasize nondairy sources of calcium and advise parents to purchase special milk treated with lactase. As this milk is more costly, inadequate family finances may be an impediment to its use. If a child has a health condition leading to chronic diarrhea, encourage increased intake of calcium-rich foods. Calcium supplements in the form of calcium carbonate tablets may be used.

Evaluation
Expected outcomes of nursing care for hypocalcemia include the following:

- Ingestion of recommended dietary allowances for calcium is maintained.
- The child displays calcium balance.
- The child is free from injury.

CLINICAL TIP
To test for Trousseau’s sign, apply a blood pressure cuff to the arm and leave inflated for 3 minutes. If a carpal spasm occurs, the Trousseau’s sign is positive. To test for Chvostek’s sign, tap the skin lightly just in front of the ear (over the facial nerve). If the corner of the mouth draws up because of muscle contraction, the Chvostek’s sign is positive. These findings may be indicative of hypocalcemia and/or hypomagnesemia.

To test for Claudius’s sign, apply a blood pressure cuff to the arm and leave inflated for 3 minutes. If a carpal spasm occurs, the Claudius’s sign is positive. To test for Chvostek’s sign, tap the skin lightly just in front of the ear (over the facial nerve). If the corner of the mouth draws up because of muscle contraction, the Chvostek’s sign is positive. These findings may be indicative of hypocalcemia and/or hypomagnesemia.

NURSING ALERT
Oral Calcium
Calcium tablets and powders are available for relief of acid indigestion and to increase calcium intake when it is deficient. Popular products contain calcium carbonate (i.e., Tums), calcium acetate, calcium citrate, tricalcium phosphate, calcium lactate, calcium gluconate, and calcium polycarbophil. Since so many forms exist, be sure that chewable tablets are chewed, sustained release tablets are swallowed whole, and powders are mixed and administered as recommended. The most common side effect is constipation; other side effects are hypercalcemia and renal calculi.

Intravenous Calcium
Intravenous calcium is administered to treat severe hypocalcemia such as in tetany due to parathyroid disease, in cardiac resuscitation, during exchange transfusions in newborns, and to relieve muscle cramps caused by insect bites. Intravenous calcium has several serious potential side effects, so nursing care centers on maintaining an intact intravenous line, continuous cardiorespiratory monitoring, and monitoring calcium and phosphate levels.
Magnesium is necessary for enzyme function in cells, acetylcholine release, glycolysis, stimulation of ATPases, and bone formation. Magnesium is a component of chlorophyll; thus, magnesium intake is aided by eating dark green leafy vegetables. Nuts and grains are also good sources of this mineral. Magnesium is absorbed primarily from the terminal ileum. It is distributed among the extracellular fluid (small amounts), the cells (larger amounts), and the bones (largest amounts). Magnesium excretion occurs in urine, feces, and sweat.

Magnesium imbalances are caused by alterations in magnesium intake, distribution, or excretion; by loss through an abnormal route; or by a combination of these factors. The plasma magnesium concentration influences the release of acetylcholine at neuromuscular junctions. Thus, magnesium imbalances are characterized by alterations in neuromuscular irritability.

**FAMILIES WANT TO KNOW**

**Calcium-Rich Foods**

When a child needs to increase sources of calcium, parents may be unfamiliar with the variety of foods that contain calcium. Although they are aware that dairy products have high calcium, there are many other foods that add a significant amount of calcium to the diet, and which may be more acceptable to families in certain cultures. Some possible sources of calcium include the following.

<table>
<thead>
<tr>
<th>Milk</th>
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<tr>
<td>Cheese</td>
<td>Chicken</td>
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<tr>
<td>Yogurt</td>
<td>Salmon (canned with bones)</td>
</tr>
<tr>
<td>Pudding</td>
<td>Grains (Cream of Wheat, farina, bran muffins)</td>
</tr>
<tr>
<td>Egg yolks</td>
<td>Sardines (canned)</td>
</tr>
<tr>
<td>Legumes</td>
<td>Tofu</td>
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<tr>
<td>Nuts</td>
<td>Fruit drinks with added calcium</td>
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Hypermagnesemia occurs when the plasma magnesium concentration is too high (above 2.4 mg/dL [0.99 mmol/L]). Keep in mind that the serum levels measured in the laboratory may not reflect body magnesium stores, because most of the magnesium in the body is located in the bones and inside the cells.

Hypermagnesemia is caused by conditions that involve increased magnesium intake and decreased magnesium excretion. Impaired renal function leading to decreased magnesium excretion is the most common cause of hypermagnesemia in children. In both oliguric renal failure and adrenal insufficiency, magnesium ions that cannot be excreted in the urine accumulate in the extracellular fluid.

Less frequently, increased magnesium intake may cause hypermagnesemia. Magnesium sulfate (MgSO₄) given to treat eclampsia in the mother before delivery causes hypermagnesemia in the newborn. Abnormally high amounts may also be taken in magnesium-containing enemas, laxatives, antacids, and intravenous fluids. Epsom salt is a readily available product and is a nearly pure magnesium sulfate preparation; its use as an enema has caused death in children. It has been used as a cathartic in the treatment of poisoning in the past but due to its potential for overdose, sorbitol is now preferred (Tofil, Benner, & Winkler, 2005). Aspiration of seawater, as in near-drowning, is an uncommon but potentially serious source of excessive magnesium intake. Children with Addison’s disease can have abnormally high magnesium levels.

Clinical manifestations of hypermagnesemia include decreased muscle irritability, hypotension, bradycardia, drowsiness, lethargy, and weak or absent deep tendon reflexes. In severe hypermagnesemia, flaccid muscle paralysis, fatal respiratory depression, cardiac arrhythmias, and cardiac arrest occur.

Hypermagnesemia is managed primarily by increasing the urinary excretion of magnesium. This is usually accomplished by increasing fluid intake (except in oliguric renal failure) and by the administration of diuretics. Dialysis may sometimes be necessary.

**NURSING MANAGEMENT**

Monitor serum magnesium levels. Take the child’s blood pressure (to watch for hypotension), heart rate and rhythm (to monitor for bradycardia and cardiac arrhythmias), respiratory rate and depth (to watch for respiratory depression), and deep tendon reflexes (to check muscle tone and paralysis or movement). Keep the side rails of the bed raised. Children with hypermagnesemia or oliguria should not be given magnesium-containing medications or sea salt.

Teach parents of children with chronic renal failure that these children should never be given milk of magnesia, antacids that contain magnesium, or other sources of magnesium; teach them to read labels and recognize ingredients. Caution all parents to avoid use of Epsom salts for children. When hypermagnesemia is treated with diuretics, monitor potassium levels to watch for hypokalemia.

Expected outcomes of nursing care include maintenance of electrolyte balance, normal neuromuscular tone, safety, and regular heart rate and rhythm.

**Hypomagnesemia**

Hypomagnesemia refers to a plasma magnesium concentration that is too low (below 1.5–1.7 mg/dL [0.62–0.70 mmol/L]). Remember that the serum levels of magnesium may not reflect body stores, as most of the magnesium in the body is found in cells and bones.

Hypomagnesemia is caused by conditions that involve decreased magnesium intake or absorption, shift of magnesium to a physiologically unavailable form, increased magnesium excretion, and loss of magnesium by an abnormal route. Hypocalcemia often accompanies and contributes to hypomagnesemia.

Neonates whose mothers are diabetic sometimes develop hypomagnesemia in the newborn period. Decreased magnesium intake or absorption can occur if a child who is not eating has prolonged intravenous therapy without magnesium. Chronic malnutrition...
is another cause of decreased magnesium intake. Magnesium absorption is decreased in chronic diarrhea, short bowel syndrome, malabsorption syndromes, and steatorrhea.

A shift of magnesium to a physiologically unavailable form may occur after transfusion of many units of citrated blood products, because magnesium bound to the citrate is not physiologically active. Such transfusions cause prolonged hypomagnesemia in liver transplant patients who have impaired citrate metabolism. Magnesium shifts rapidly into bones that have been deprived of adequate stores.

Increased magnesium excretion in the urine occurs with diuretic therapy, the diuretic phase of acute renal failure, diabetic ketoacidosis, and hyperaldosteronism. Chronic alcoholism, occasionally seen in adolescents, increases urinary magnesium excretion. Magnesium contained in gastrointestinal secretions is bound to fat and excreted in the stool.

Loss of magnesium by an abnormal route occurs with prolonged nasogastric suction and through sequestration of magnesium in acute pancreatitis. Several medications may cause hypomagnesemia, such as magnesium-wasting diuretics, some antineoplastic agents, systemic antifungals, aminoglycoside antibiotics, and laxatives without magnesium.

Hypomagnesemia is characterized by increased neuromuscular excitability (tetany). The clinical manifestations are hyperactive reflexes, skeletal muscle cramps, twitching, tremors, and cardiac arrhythmias. Seizures can occur with severe hypomagnesemia. Hypomagnesemia is associated with high mortality for children in the pediatric intensive care unit (Singhi, Singh, & Prasad, 2003).

Magnesium serum levels are measured, along with serum calcium and potassium, since these electrolyte disturbances often occur together. Hypomagnesemia is managed by administering magnesium and treating the underlying cause of the imbalance.

**NURSING MANAGEMENT**

In addition to monitoring serum magnesium levels, nursing assessment of hypomagnesemia includes monitoring deep tendon reflexes, testing for Trousseau's and Chvostek's signs (see page 531), monitoring cardiac function, and observing for muscle twitching. Children who are able to talk will report muscle cramping. Because magnesium levels are not routinely measured in many settings, request the test for any child who has risk factors and early manifestations of hypomagnesemia. When intramuscular or intravenous magnesium is ordered, administer carefully as directed and monitor vital signs. Electrocardiogram and renal studies may precede drug administration. Have resuscitative drugs and equipment readily available during drug administration.

Teach parents of a child with hypomagnesemia or continuing risk factors such as chronic diarrhea to include foods containing magnesium in the diet (see Families Want to Know: Magnesium-Rich Foods). Before administering magnesium supplements, verify that the child's urine output is adequate. Monitor deep tendon reflexes if intravenous magnesium is given, and observe for complications of magnesium supplementation.

Expected outcomes for nursing care include restoration and maintenance of electrolyte balance.

**FAMILIES WANT TO KNOW**

**Magnesium-Rich Foods**

Magnesium is a mineral with which most families are not familiar. When a child needs to increase magnesium intake, families can be encouraged to add two to three of the following foods to the child's daily diet:

- Whole-grain cereal
- Dark-green vegetables
- Soy
- Almonds
- Peanut butter
- Egg yolk
Clinical Assessment of Fluid and Electrolyte Imbalance

How can you assess children appropriately for fluid and electrolyte imbalance without thinking through the clinical manifestations of every possible disorder one after the other? First, perform a rapid risk factor assessment on each child to see which factors are present (Tables 16–7 and 16–8). Remember that most imbalances influence other factors so it is common to find more than one type of fluid and electrolyte problem. Examining several body systems such as cardiovascular, respiratory, and neurologic will be necessary to get a comprehensive picture of the child.

A risk factor assessment may be performed mentally during routine tasks. Look for factors that alter the intake, retention, and loss of isotonic fluid and water. This information is used to evaluate which fluid imbalance is most likely to occur in a particular child. Next, look for factors that alter electrolyte intake and absorption, distribution between plasma and other electrolyte pools, excretion, and abnormal routes of electrolyte loss. This information is used to evaluate which electrolyte imbalances are most likely to occur in the child. A review of pathophysiology is important to understand the role of the other electrolytes and substances, such as phosphorus, in the body. Apply growth and development to realize what types of problems might be most common in various age groups. For example, the newborn is more likely to be dehydrated due to lack of adequate intake, while the toddler more commonly has fluid loss from nausea and vomiting.

After evaluating possible imbalances for the child, perform a clinical assessment. Assessment of fluid imbalances is performed by assessing weight changes, vascular volume, interstitial volume, and cerebral function (Table 16–9). Assessment of electrolyte imbalances is performed by assessing serum electrolyte levels, skeletal muscle strength, neuromuscular excitability, gastrointestinal tract function, and cardiac rhythm (Table 16–10). Next, check for other manifestations that are specific to a particular high-risk imbalance (e.g., polyuria in hypokalemia). Evaluate any serum laboratory values available. This method of risk factor assessment followed by clinical assessment provides a rapid yet thorough approach to assessment for fluid and electrolyte imbalances.

### Table 16–7 | RISK FACTOR ASSESSMENT FOR FLUID IMBALANCES

<table>
<thead>
<tr>
<th>Isotonic Fluid (Extracellular Fluid Volume Imbalances)</th>
<th>Water</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Source of increased intake?</td>
<td>• Source of increased intake?</td>
</tr>
<tr>
<td>• Aldosterone secretion increased or decreased?</td>
<td>• Antidiuretic hormone secretion increased or decreased?</td>
</tr>
<tr>
<td>• Source of loss from the body?</td>
<td>• Source of unusual loss from the body?</td>
</tr>
</tbody>
</table>

### Table 16–8 | RISK FACTOR ASSESSMENT FOR ELECTROLYTE IMBALANCES

<table>
<thead>
<tr>
<th>Electrolyte Intake and Absorption</th>
<th>Electrolyte Shifts</th>
<th>Electrolyte Excretion</th>
<th>Electrolyte Loss by Abnormal Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Increased?</td>
<td>• From electrolyte pool to plasma?</td>
<td>• Increased?</td>
<td>• Vomiting?</td>
</tr>
<tr>
<td>• Decreased?</td>
<td>• From plasma to electrolyte pool?</td>
<td>• Decreased?</td>
<td>• Diarrhea?</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Nasogastric suction?</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Wound?</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Burn?</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Excessive sweating?</td>
</tr>
</tbody>
</table>
There are four acid-base imbalances. Two are the result of processes that cause too much acid in the body and are referred to as acidosis. The other two imbalances are the result of processes that cause too little acid in the body and are called alkalosis. An acid-base disorder caused by too much or too little carbonic acid is called a respiratory acid-base imbalance. A disorder caused by too much or too little metabolic acid is called a metabolic acid-base imbalance.

### Table 16–9 SUMMARY OF CLINICAL ASSESSMENT OF FLUID IMBALANCES

<table>
<thead>
<tr>
<th>Assessment Category</th>
<th>Specific Assessments</th>
<th>Changes with Fluid Imbalances</th>
</tr>
</thead>
</table>
| Rapid changes in weight | Daily weights | Weight gain—extracellular volume excess  
Weight loss—extracellular volume deficit; clinical dehydration |
| Vascular volume | Small-vein filling time  
Capillary refill time  
Character of pulse | Increased—extracellular volume deficit; clinical dehydration  
Increased—extracellular volume deficit; clinical dehydration  
Bounding—extracellular volume excess  
Thready—extracellular volume deficit; clinical dehydration |
| Postural blood pressure measurements | Postural drop—extracellular volume deficit; clinical dehydration |
| Lung sounds in dependent portions | Crakles—extracellular volume excess |
| Central venous pressure | Increased—extracellular volume excess |
| Tenseness of fontanel (infants) | Decreased—extracellular volume deficit; clinical dehydration |
| Neck vein filling (older children) | Full with upright—extracellular volume excess  
Flat when supine—extracellular volume deficit; clinical dehydration |
| Interstitial volume | Skin turgor  
Presence or absence of edema | Skin tents—extracellular volume deficit; clinical dehydration  
Edema—extracellular volume excess |
| Cerebral function | Level of consciousness | Decreased—clinical dehydration |

### Table 16–10 SUMMARY OF CLINICAL ASSESSMENT OF ELECTROLYTE IMBALANCES

<table>
<thead>
<tr>
<th>Assessment Category</th>
<th>Specific Assessments</th>
<th>Changes with Electrolyte Imbalances</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skeletal muscle function</td>
<td>Muscle strength</td>
<td>Weakness, flaccid paralysis—hyperkalemia; hypokalemia</td>
</tr>
</tbody>
</table>
| Neuromuscular excitability | Deep tendon reflexes  
Chvostek's sign (not infants)  
Trousseau's sign  
Paresthesias  
Muscle cramping or twitching | Depressed—hypercalcemia; hypermagnesemia  
Hyperactive—hypocalcemia; hypomagnesemia  
Positive—hypocalcemia; hypomagnesemia  
Positive—hypocalcemia; hypomagnesemia  
Digital or perioral—hypocalcemia  
Present—hypocalcemia; hypomagnesemia |
| Gastrointestinal tract function | Bowel sounds  
Elimination pattern | Decreased or absent—hypokalemia  
Constipation—hypokalemia; hypercalcemia  
Diarrhea—hyperkalemia |
| Cardiac rhythm | Arrhythmia  
Electrocardiogram | Irregular—hyperkalemia; hypokalemia; hypercalcemia; hypocalcemia; hypermagnesemia; hypomagnesemia  
Abnormal—hyperkalemia; hypokalemia; hypercalcemia; hypocalcemia; hypermagnesemia; hypomagnesemia |
| Cerebral function | Level of consciousness | Decreased—hyponatremia; hypernatremia  
Examples of Metabolic Acids  
Pyrubic acid  
Sulfuric acid  
Acetoacetic acid  
Lactic acid  
Hydrochloric acid  
Beta-hydroxybutyric acid |

**ACID-BASE IMBALANCES**

There are four acid-base imbalances. Two are the result of processes that cause too much acid in the body and are referred to as acidosis. The other two imbalances are the result of processes that cause too little acid in the body and are called alkalosis. An acid-base disorder caused by too much or too little carbonic acid is called a respiratory acid-base imbalance. A disorder caused by too much or too little metabolic acid is...
Acidosis: Relatively too much acid in the body
- Respiratory acidosis: Relatively too much carbonic acid
- Metabolic acidosis: Relatively too much metabolic acid
Alkalosis: Relatively too little acid in the body
- Respiratory alkalosis: Relatively too little carbonic acid
- Metabolic alkalosis: Relatively too little metabolic acid

**Respiratory Acidosis**

Respiratory acidosis is caused by the accumulation of carbon dioxide in the blood. Since carbon dioxide and water can be combined into carbonic acid, respiratory acidosis is sometimes called carbonic acid excess. The condition can be acute or chronic. It is controlled by the lungs.

**Etiology and Pathophysiology**

Any factor that interferes with the ability of the lungs to excrete carbon dioxide can cause respiratory acidosis. These factors may interfere with the gaseous exchange within the lungs, may impair the neuromuscular pump that moves air in and out of the lungs, or may depress the respiratory rate (Table 16–13; Figure 16–20 ➤).

As the Po2 begins to increase, the pH of the blood begins to decrease. Compensatory mechanisms begin to act in the form of nonbicarbonate buffers, additional

<table>
<thead>
<tr>
<th>Normal Blood pH and Gases</th>
<th>Infants</th>
<th>Children</th>
<th>Adolescents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial blood pH</td>
<td>7.18–7.50</td>
<td>7.27–7.49</td>
<td>7.35–7.41</td>
</tr>
<tr>
<td>Arterial blood Po2</td>
<td>60–70 mmHg (8.0–9.3 pKa)</td>
<td>80–108 mmHg (10.7–14.4 pKa)</td>
<td>80–100 mmHg (10.7–13.3 pKa)</td>
</tr>
<tr>
<td>Arterial blood Pco2</td>
<td>27–41 mmHg (3.6–5.5 pKa)</td>
<td>32–48 mmHg (4.3–6.4 pKa)</td>
<td>32–48 mmHg (4.3–6.4 pKa)</td>
</tr>
<tr>
<td>Arterial blood HCO3⁻ (bicarbonate)</td>
<td>19–24 mmol/L</td>
<td>18–25 mmol/L</td>
<td>20–29 mmol/L</td>
</tr>
</tbody>
</table>

**Acid-Base Imbalances**

Acidosis: Relatively too much acid in the body
- Respiratory acidosis: Relatively too much carbonic acid
- Metabolic acidosis: Relatively too much metabolic acid
Alkalosis: Relatively too little acid in the body
- Respiratory alkalosis: Relatively too little carbonic acid
- Metabolic alkalosis: Relatively too little metabolic acid
hydrogen ion excretion by the kidneys, and formation and decreased bicarbonate excretion by the kidneys. These compensatory mechanisms take several days to become active so the child manifests a changing clinical situation, depending on the underlying cause and the amount of compensation occurring (see Table 16–12).

**Clinical Manifestations**

Acidosis in the brain cells causes central nervous system depression, manifested by confusion, lethargy, headache, increased intracranial pressure, and even coma. Acute re-

---

**Table 16–13: CAUSES OF RESPIRATORY ACIDOSIS**

<table>
<thead>
<tr>
<th>Factors Affecting the Lungs</th>
<th>Factors Affecting the Neuromuscular Pump</th>
<th>Factors Affecting Central Control of Respiration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspiration</td>
<td>Flail chest</td>
<td>Sedative overdose</td>
</tr>
<tr>
<td>Spasm of the airways</td>
<td>Pneumothorax or hemothorax</td>
<td>General anesthesia</td>
</tr>
<tr>
<td>Laryngeal edema</td>
<td>Mechanical underventilation</td>
<td>Head injury</td>
</tr>
<tr>
<td>Epiglottitis</td>
<td>Hypokalemic muscle weakness</td>
<td>Brain tumor</td>
</tr>
<tr>
<td>Croup</td>
<td>High cervical spinal cord injury</td>
<td>Central sleep apnea</td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td>Botulism</td>
<td></td>
</tr>
<tr>
<td>Atelectasis</td>
<td>Tetanus</td>
<td></td>
</tr>
<tr>
<td>Severe pneumonia</td>
<td>Kyphoscoliosis</td>
<td></td>
</tr>
<tr>
<td>Cystic fibrosis</td>
<td>Poliomyelitis</td>
<td></td>
</tr>
<tr>
<td>Bronchopulmonary dysplasia</td>
<td>Muscular dystrophy</td>
<td></td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>Congenital diaphragmatic hernia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Guillain-Barré syndrome</td>
<td></td>
</tr>
</tbody>
</table>
Spiratory acidosis can lead to tachycardia and cardiac arrhythmias. The child’s arterial blood gases always show an increased Pco₂, the laboratory sign of increased carbonic acid. Serum pH can be decreased or normal.

### COLLABORATIVE CARE

Laboratory tests involve arterial blood gases, as described previously. Treatment of respiratory acidosis requires correction of the underlying cause. For example, treatment may include bronchodilators for bronchospasm, mechanical ventilation for neuromuscular defects, decreasing sedative use, or surgery for kyphoscoliosis.

### NURSING MANAGEMENT

#### Nursing Assessment and Diagnosis

Nursing assessment plays a pivotal role in decisions about interventions for respiratory acidosis, especially in chronic conditions such as cystic fibrosis and kyphoscoliosis. Assess respiratory rate, rhythm, and depth carefully. Take the apical pulse and be alert for tachycardia or arrhythmia. A cardiac monitor may be used. Obtain serial arterial blood gas measurements in acute conditions to evaluate changing status. Assess the level of consciousness and energy. Observe for chronic fatigue, headache, or decreased level of consciousness.

Several nursing diagnoses may apply to the child with respiratory acidosis. The most important of these addresses the child’s risk for injury. Other nursing diagnoses depend on the specific clinical manifestation and the particular cause of the acidosis. Examples include:

- Risk for Injury related to decreased level of consciousness
- Activity Intolerance related to decreased cardiac output secondary to cardiac dysrhythmias
- Ineffective Breathing Pattern (Hypoventilation) related to neuromuscular impairment
- Acute Pain (Headache) related to cerebral vasodilation
- Ineffective Family Management of Therapeutic Regimen related to complexity of bronchodilator therapy

#### Planning and Intervention

**Care in the Community**

Teach children at risk for respiratory acidosis and their parents preventive measures to use at home. For the child with a chronic condition such as cystic fibrosis, muscular dystrophy, or kyphoscoliosis, demonstrate deep breathing and encourage its use several times each day. Teach the family signs of infection—including fever, increased respiratory secretions, and discomfort with breathing—so the problems can be treated promptly to prevent further respiratory involvement. Position the child to facilitate chest expansion (Figure 16–21 ➤). Teach parents about proper administration of any necessary medications. For example, the child with cystic fibrosis may receive antibiotics to prevent respiratory infections. Teach parents and older children about home respirator use (Figure 16–22 ➤).

**Hospital-Based Care**

For the hospitalized child, the focus is on ensuring safety. Keep side rails raised, and turn and position the child frequently. Evaluate mental status and document and report any changes in alertness. When laboratory values of blood pH and Pco₂ are available, evaluate them promptly and report any changes or abnormalities. Administer medications as ordered. Carefully watch the doses of sedatives to avoid further respiratory depression. Provide suctioning and encourage deep breathing.
Evaluation
Expected outcomes of nursing care for the child with respiratory acidosis include the following:

- Safety is maintained for the child.
- Adequate rate and rhythm of respirations are manifested.
- Disorders that have contributed to the imbalance are corrected.

Respiratory Alkalosis
Respiratory alkalosis occurs when the blood contains too little carbon dioxide. It is sometimes called carbonic acid deficit.

Excess carbon dioxide loss is caused by hyperventilation, in which more air than normal is moved into and out of the lungs. Common causes of hyperventilation are listed in Box 16–6. Some of the most common causes in young children are hypoxia such as that from severe asthma, salicylate poisoning, and sepsis (Schwaderer & Schwartz, 2004).

In many cases, respiratory alkalosis only lasts for several hours. Renal compensation does not occur, as these compensatory mechanisms take several days to begin action. An example is the hyperventilation that occurs with acute anxiety. If the condition persists, however, the kidneys will begin to retain more acid and excrete more bicarbonate. Hydrogen ions will be released from body buffers to decrease plasma bicarbonate. While the imbalance continues, cellular function is thus protected by returning pH to normal levels.

Arterial blood gas measurements show a decreased Pco₂ in respiratory alkalosis. Blood pH is generally elevated. The lack of carbon dioxide causes neuromuscular irritability and paresthesias in the extremities and around the mouth. Muscle cramping and carpal or pedal spasms can occur. The child may be dizzy or confused.

Diagnosis is made by complete arterial blood gas measurements and thorough physical assessment. Clinical therapy focuses on correcting the condition that caused the hyperventilation so that the body’s compensatory mechanisms can return carbon dioxide levels to normal. Oxygen therapy may be helpful in cases of hypoxia, salicylates are removed from the body when poisoning is the cause (see Chapter 6 for further information on poisoning), drugs that have interfered with breathing are changed, sepsis is treated with effective medication, and anxiolytic medications may be used to treat anxiety.

NURSING MANAGEMENT
Assess the child’s level of consciousness and ask if the child feels light-headed or has tingling sensations or numbness in the fingers, toes, or around the mouth. Assess the rate and depth of respirations. Monitor the hospitalized child’s Po₂ with serial arterial blood gas measurements to evaluate changes in status. A careful assessment is needed regarding the cause of hyperventilation. Did an occurrence cause anxiety for the child? Is pain present (see Chapter 15)? Has the child received salicylates in any form? Is the child mechanically ventilated? Is there a central nervous system infection such as meningitis?

Nursing care for the child with respiratory alkalosis centers on teaching stress management techniques, maintaining pain control, promoting respiratory function, ensuring safety, maintaining fluid status, and providing health supervision and home care.

Teach Stress Management Techniques
When anxiety is the cause of respiratory alkalosis, instruct the child to breathe slowly, in rhythm with your own breathing. Teach stress control techniques such as relaxation and imagery for situations that cause anxiety (Table 16–14).
Maintain Pain Control
Use medications, imagery, distraction, positioning, massage, and other techniques to decrease pain and maintain pain management. Chapter 15 describes these and other measures to assist with pain control.

Promote Respiratory Function
Have the child cough, or suction as needed. Be certain that mechanical ventilation systems are working properly. Oxygen saturation is usually monitored continuously; observe and record results.

Ensure Safety
Provide a safe environment for the child who has a decreased level of consciousness. Be sure the child is supervised when sitting or standing up. Keep bed rails raised.

Regulate Fluid Status
Renal compensation to manage ongoing respiratory alkalosis requires adequate urinary output. Regulate fluid intake to ensure urine output unless fluids are restricted due to medical condition.

Care in the Community
Teach parents to keep aspirin and other salicylate products out of reach of children, preferably in a locked medicine box. Instruct parents to call the Poison Control Center immediately in case of poison ingestion.

Evaluation
Expected outcomes of nursing care for the child with respiratory alkalosis include the following:

- Normal respiratory rate and rhythm are manifested.
- Safety is maintained for the child.
- Fluid status is appropriately regulated.

Metabolic Acidosis
Metabolic acidosis is a condition in which there is an excess of any acid other than carbonic acid. For this reason, it is sometimes called noncarbonic acid excess.

Etiology and Pathophysiology
Metabolic acidosis is caused by an imbalance in production and excretion of acid or by excess loss of bicarbonate (Table 16–15). Excess accumulation occurs by one of two mechanisms. First, a child can eat or drink acids or substances that are converted to acid in the body. Examples include aspirin, boric acid, and antifreeze. Second, cells can make abnormally high amounts of acid that cannot be excreted. This is the case in ketoacidosis of untreated diabetes mellitus, untreated growth hormone deficiency, in children with bladder construction that uses part of the bowel, or the starvation that

### Table 16–14
**TECHNIQUES FOR REDUCING ANXIETY IN CHILDREN WITH PARETHESIAS**

<table>
<thead>
<tr>
<th>Infant</th>
<th>Toddler or Preschooler</th>
<th>Young School-Age Child</th>
<th>Older School-Age Child or Adolescent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calming touch</td>
<td>Stuffed toy to hug</td>
<td>Talking quietly about a happy event</td>
<td>Explaining the reason for the tingling and that it will go away</td>
</tr>
<tr>
<td>Quiet voice</td>
<td>Singing familiar quiet nursery songs</td>
<td>Telling a familiar story</td>
<td>Use of guided imagery</td>
</tr>
<tr>
<td>Swaddling</td>
<td>Acknowledging the child’s feelings</td>
<td>Reading a familiar book together</td>
<td>Familiar music on tape or radio</td>
</tr>
<tr>
<td>Holding quietly</td>
<td>Holding calmly</td>
<td>Explaining that the tingling will go away</td>
<td>Asking what the child does when anxious or “scared”</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Use of simple guided imagery</td>
<td>Talking about coping strategies</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Supportive listening</td>
<td></td>
</tr>
</tbody>
</table>

**NURSING ALERT**
The Po2 must be checked before any therapy for respiratory alkalosis is started, because it is dangerous to stop hyperventilation if oxygenation is poor. When Po2 is low, the child’s hyperventilation may be a protective mechanism to increase blood oxygenation. Other measures such as oxygen therapy or mechanical ventilation may need to start first, followed by treatment for the cause of respiratory alkalosis.
can occur in anorexia or bulimia. A disorder of excretion occurs in conditions such as oliguric renal failure (Figure 16–23 ➤).

Bicarbonate can be lost from the body through the urine or through excessive loss of intestinal fluid. Diarrhea, fistulas, and ileal drainage are all possible sources. Carbonic anhydrase inhibitors can cause loss of excess bicarbonate in the urine.

When the pH of the blood decreases below normal, the chemoreceptors in the brain and arteries are stimulated and respiratory compensation begins. The child's rate and depth of breathing increase and carbonic acid is removed from the body. The blood pH shifts to a more normal range even though the cause is not corrected. The underlying condition and the degree of compensation will alter the clinical laboratory values observed.

**Clinical Manifestations**

Laboratory values show decreased blood pH and decreased HCO₃⁻ and Pco₂. An attempt at respiratory compensation causes one of the most important signs of metabolic acidosis, increased rate and depth of respirations (hyperventilation) or *Kussmaul respirations*. Severe acidosis can cause decreased peripheral vascular resistance and resultant cardiac arrhythmias, hypotension, pulmonary edema, and tissue hypoxia. Confusion or drowsiness may result, as well as headache or abdominal pain.

**COLLABORATIVE CARE**

Laboratory tests include blood pH and arterial blood gases. Treatment of metabolic acidosis depends on identification and treatment of the underlying cause. For example, renal failure is treated with medications of dialysis, an intestinal fistula is repaired, and hyperalimentation formula is regulated to decrease acidosis. In severe metabolic acidosis, intravenous sodium bicarbonate may be used to increase the pH and to prevent cardiac arrhythmias. This treatment is difficult to manage, because renal excretion can cause excess retention of bicarbonate; therefore, intravenous sodium bicarbonate is used only in severe situations, such as prolonged cardiac arrest.

**NURSING MANAGEMENT**

**Nursing Assessment and Diagnosis**

When families bring a young child in for a health promotion visit, assess the risk of poisoning in the home and the family's knowledge of prevention techniques. For the child being treated for acidosis, assess the rate and depth of respirations. Evaluate the child's level of consciousness frequently. Be alert for signs or complaints of headache.
and abdominal pain. Serial arterial blood gas measurements will usually be obtained to evaluate changes in status.

The following nursing diagnoses can apply to the child with metabolic acidosis:

- Risk for Injury related to confusion/drowsiness or decreased responsiveness
- Risk for Decreased Cardiac Output related to cardiac dysrhythmias
- Ineffective Tissue Perfusion (Cerebral) related to tissue hypoxia
- Ineffective Family Management of Therapeutic Regimen related to complexity of management of diabetes mellitus

**Planning and Implementation**

Ensure safety, taking into account the child’s level of consciousness and alertness. Turn the child and change his or her position to prevent pressure on the skin. Limit the child’s activities to decrease cardiac workload.

Position the child to facilitate chest expansion. Provide oral care during rapid respirations because the mouth may become dry. Monitor intravenous solutions and laboratory values indicating acid-base balance. Report changes promptly.

Once the child is stabilized, provide teaching to compensate for knowledge deficits. This teaching for home prevention should take place at each health promotion visit for all children. Teach parents of young children to keep medications and acids locked in a secure place and out of reach to prevent poisoning (Figure 16–24 ➤). This includes medicines with aspirin as well as substances commonly kept in the garage for car maintenance. Teach about home management of diabetes and about early identification and treatment to avoid diabetic ketoacidosis. Expected outcomes of nursing care relate to prevention of acidosis and restoration of normal body balance during disease processes.

**Metabolic Alkalosis**

Metabolic alkalosis occurs when there are too few metabolic acids. It is sometimes called noncarbonic acid deficit.

A gain in bicarbonate or a loss of metabolic acid can cause metabolic alkalosis (Table 16–16). Bicarbonate is gained through excessive intake of bicarbonate antacids or baking soda or through metabolism of bicarbonate precursors such as the citrate contained in blood transfusions. Increased renal absorption of bicarbonate can occur in profound hypokalemia, primary hyperaldosteronism, or extreme deficit in extracellular fluid volume. Acid can be lost through severe vomiting, such as that seen in infants with pyloric stenosis and in continued removal of gastric contents through suction.

When the chemoreceptors in the brain and arteries detect the rising pH of metabolic alkalosis and respirations decrease, carbonic acid is retained in the body. This carbonic acid can neutralize the bicarbonate and return pH toward normal.

Blood pH, bicarbonate, and Pco2 are usually elevated in metabolic alkalosis. Hypokalemia often occurs simultaneously (refer to discussion of hypokalemia earlier in this chapter). Respiratory rate and depth usually decrease. Increased neuromuscular
irritability, cramping, paresthesia, tetany, seizures, and excitation can occur. Finally, this state can progress to weakness, confusion, lethargy, and coma.

Laboratory tests include blood pH and arterial blood gases. Clinical therapy is directed at treating the underlying cause of the condition. Increasing the extracellular fluid volume with intravenous normal saline is used to facilitate renal excretion of bicarbonate. Medications such as acetazolamide increase renal excretion of bicarbonate as well.

### NURSING MANAGEMENT

Assess the child’s level of consciousness frequently. Alertness may decrease after an initial period of excitement, so regular assessments are needed. Monitor neuromuscular irritability. Observe for nausea and vomiting. Assess the rate and depth of respirations carefully. Obtain serial arterial blood gas measurements as ordered.

Facilitate ease of respirations. Ensure safety by keeping bed rails elevated and by turning the child frequently. Position the child on the side to avoid aspiration of vomitus.

If antacids were the cause of the alkalosis, teach the child and parents about correct use of these medications.

### MIXED ACID-BASE IMBALANCES

It is possible for two acid-base imbalances to occur simultaneously. For example, a child with cystic fibrosis can develop respiratory acidosis from lung problems and concurrent metabolic alkalosis from vomiting during an illness. Treatment with diuretics may cause concurrent metabolic alkalosis resulting from extracellular volume depletion and hypokalemia in a child with congestive heart failure and chronic respiratory acidosis. In these cases, all underlying causes must be identified and treated. Care of children with mixed acid-base imbalances is often complicated, requiring hospitalization and careful management. Upon discharge, the nurse can teach parents about signs of imbalance that need to be reported and treated to prevent further complications. Evaluation of care is based on outcomes of adequate respiratory ventilation and metabolic balance.

### CRITICAL THINKING IN ACTION

Consider the scenario involving Vernon at the beginning of this chapter. He is an 18-month-old who has had vomiting and diarrhea for several days. Assessment of body weight loss, skin turgor, and level of activity suggest moderate dehydration. Vernon refuses attempts at feeding him orally, his pulse becomes rapid, and his blood pressure decreases. Voiding is decreased and capillary refill is slow. Vernon is admitted to the short-stay unit and an intravenous infusion is started.

**DISCUSSION**

1. Based on his age, what oral fluids might be best to offer to Vernon? What questions will you ask his mother about his normal fluid intake at home?

2. What additional assessment will you perform on Vernon to gather further information about his state of dehydration?

3. Since Vernon has had vomiting and diarrhea, he is probably deficient in an electrolyte present in high quantities in these body fluids. What electrolyte, in addition to sodium, is likely deficient?

4. A major nursing role is to plan care for Vernon while he is in the unit to rehydrate him. Calculate his replacement and maintenance fluid needs. Formulate a plan of care to include the amounts of oral rehydration therapy he should be offered over the next several hours.

Refer to the Companion Website for answers.
REFERENCES


