In this article, I focus on metabolic acidosis, which can occur in a variety of clinical contexts in the pediatric intensive care unit. I review the basic concepts of acid-base balance; examine the 2 types of metabolic acidosis, normal and elevated anion gap acidosis; and discuss the common causes of each type of metabolic acidosis. Case studies illustrate how 3 common but distinct clinical conditions can lead to metabolic acidosis and show that careful evaluation of the metabolic acidosis in the context of each clinical occurrence is essential for accurate diagnosis and timely treatment.

Basic Concepts of Acid-Base Balance

Optimal physiological functioning of metabolism, the cardiovascular system, and the neurological system depends on the ability of regulatory mechanisms to prevent and resolve fluctuations in pH, which is primarily determined by hydrogen ion concentration in plasma.\(^1\) Compared with the concentration of other ions, the concentration of hydrogen ions is relatively low in the extracellular space and is not measured directly. Concentration of hydrogen ions in the plasma is from 0.000035 to
0.000045 mEq/L. Because this number is cumbersome, the term pH, the negative logarithm of hydrogen ion concentration (Figure 1), is used to describe the level of hydrogen ions in the extracellular space.1,5

A high pH indicates a low hydrogen ion concentration, a low pH, a high concentration.2 Hydrogen ions exist in the form of fixed or volatile acids and are the product of normal metabolism and incomplete oxidation of fat and sugar. Hydrogen ions bound to other ions that neutralize the charge of the hydrogen ions are not free and therefore do not contribute to pH.5

First-Line Buffers: The Cellular Buffering System

Maintaining pH within a normal range is accomplished by either buffering or excreting acids. The cellular buffer system responds within seconds to fluctuations in acid concentration. In the extracellular space, the primary buffer is bicarbonate. Most acid in the extracellular space is in the form of carbonic acid. The goal ratio for bicarbonate to carbonic acid in the body is 20 to 1.2 When carbonic anhydrase is present, carbonic acid is easily formed by combining carbon dioxide and water or hydrogen and bicarbonate (Figure 2). Carbonic anhydrase also catalyzes the breakdown or dissociation of carbonic acid into carbon dioxide and water or hydrogen and bicarbonate.3 Carbonic anhydrase is found primarily in the walls of the lung alveoli and in the epithelial cells of the renal tubules.5

The formation or breakdown of carbonic acid is indicated by the direction of the arrows in the equation (Figure 2) and is determined by the acid concentration in the extracellular space. If the acid concentration is high, the pH is low. As a result, the carbonic acid is broken down to form hydrogen and bicarbonate or carbon dioxide and water. The breakdown or dissociation of carbonic acid ultimately facilitates the excretion of hydrogen ions. If the acid concentration is low, the pH is high. The body will always work to move the equation toward the middle. The hydrogen ions and bicarbonate will bond to form carbonic acid, and the carbon dioxide and water will bond to form carbonic acid, ultimately increasing the acid concentration and decreasing the pH.2

If acids enter the cells, phosphate buffers and hemoglobin neutralize the acids in the intracellular space. Hemoglobin and oxyhemoglobin are the primary intracellular buffers of carbonic acid. The oxyhemoglobin dissociation curve (Figure 3) helps explain the role of hemoglobin in the maintenance of an acid-base balance. Carbon dioxide enters the red blood cells in the capillaries and combines with water to form carbonic acid. As oxyhemoglobin releases oxygen to the cells, the hemoglobin becomes negatively charged. In respiratory acidosis, which is marked by increased carbon dioxide, red blood cells release oxygen to the cells more readily, causing the oxyhemoglobin dissociation curve to shift to the right. In respiratory alkalosis, which is marked by decreased carbon dioxide, red blood cells maintain a higher affinity for oxygen, as indicated by a shift of the oxyhemoglobin dissociation curve to the left. Other intracellular proteins include albumin and globulin, which work specifically during catabolism to neutralize the hydrogen released.5

Phosphate concentration is low in the extracellular space, but phosphates play an important role in intracellular buffering. Phosphates
are especially effective buffers in the renal tubules because the concentration of phosphate molecules is high and the pH within the tubules is significantly lower than the pH of the extracellular fluid. This difference in pH between the tubules and the extracellular fluid depends on the concentration of phosphates for intracellular buffering.

Second-Line Buffers: The Respiratory System

As described earlier, in a state of acid-base disturbance, carbonic acid is either formed or dissociated. The respiratory system responds to the changes in the ratio of bicarbonate to carbonic acid and begins to compensate within minutes. In acidosis, the cellular buffering system causes dissociation of carbonic acid to hydrogen dioxide and water and to hydrogen and bicarbonate. Chemoreceptors in the brain regulate the respiratory drive and respond to the increased concentration of circulating hydrogen ions by stimulating the respiratory system to increase respiratory rate and depth, in an effort to excrete carbon dioxide. In alkalosis, the chemoreceptors sense a low concentration of hydrogen ions and stimulate the respiratory system to slow respirations. Slow respirations cause retention of carbon dioxide, allowing the formation of carbonic acid to decrease the pH.

Third-Line Buffers: The Renal System

The renal system is the slowest, yet most effective buffering system. Kidneys respond to an alteration in acid-base balance within hours, and complete activation takes days. Renal compensation for acid-base imbalance involves the excretion or retention of hydrogen ions and the generation of bicarbonate. Bicarbonate ions are small and pass freely into the glomeruli. If bicarbonate ions are not reabsorbed into the plasma from the tubules, the body can be depleted of bicarbonate within several hours. Nearly 80% of the filtered bicarbonate is reabsorbed in the proximal tubules.

Bicarbonate reabsorption does not provide enough buffering activity to respond to the nonvolatile acids produced by metabolism. The kidneys generate bicarbonate in 2 ways. In one, the generation of bicarbonate is triggered by excessive hydrogen in the urine and involves activation of protein, ammonia, and phosphate buffer systems. In the other, glutamine is broken down in the proximal tubules of the kidneys. One molecule of glutamine produces bicarbonate ions and an ammonium ion. The bicarbonate ions move freely across the basolateral membrane of the proximal tubule and into the bloodstream.

Metabolic Acidosis

Metabolic acidosis, defined as a pH less than 7.35 without an elevated PaCO₂, is attributed to high hydrogen ion concentration or low bicarbonate concentration. The changes in ion concentration can be categorized as increased acid production; decreased acid excretion; and loss of base, commonly bicarbonate. The most common cause of metabolic acidosis in children is insufficient tissue perfusion, leading to an accumulation of acid. The body’s...
inability to compensate for an increase in hydrogen ions, by retaining or forming bicarbonate, leads to acidosis.

To help determine the cause of the metabolic acidosis, critical care nurses must measure the anion gap, the difference between cations (positively charged ions) and anions (negatively charged ions). The net charge of the cations and anions must be zero in the extracellular space to maintain electrochemical balance. In other words, the number of anions and cations within the plasma should be equal. Although other charged ions exist, sodium is the primary measured cation, and chloride and bicarbonate are the primary measured anions. Unmeasured anions include phosphates, sulfates, and proteins. Unmeasured cations include calcium, potassium, and magnesium.

Measurement of the anion gap requires simultaneous analysis of arterial blood gas and determination of electrolytes in venous blood. The arterial blood gas findings are used to determine if a metabolic acidosis is present. Once metabolic acidosis is confirmed, the results of the electrolyte assay provide the values needed to calculate the anion gap, including sodium, chloride, and calculated bicarbonate values (Figure 4).

A normal or expected anion gap is between 4 and 12. An expected gap exists because only the values of the measured cations and measured anions are used in the equation. Unmeasured cations and anions account for the gap and explain why the numbers of cations and anions are not equal in the equation.

Clinically, determining the presence or absence of an elevated anion gap assists clinicians in working through the differential diagnosis for metabolic acidosis. Elevated anion gap suggests an increased plasma level of unmeasured anions or a decreased level of unmeasured cations. In other words, the accumulation of acids is not adequately buffered by a base.

A nonelevated anion gap acidosis reflects the loss of bicarbonate, rather than an increase in acid production or a decrease in acid excretion. The gap is normal because chloride is the measured anion and its concentration increases as the concentration of bicarbonate decreases to compensate for imbalance. Table 1 lists common causes of metabolic acidosis associated with elevated and normal anion gaps.

**Case Studies**

The following case studies are presented to emphasize the clinical importance of metabolic acidosis and the value of anion gap measurement.

### Case Study 1

JT, a 5-year-old, previously healthy boy, was admitted to the pediatric intensive care unit after 2 weeks of polydipsia, polyuria, weight loss, fatigue, and irritability. His eyes were sunken, his mucous membranes were dry and cracked, and he had tenting of his skin. He was lethargic and irritable in response to stimulation. Heart rate was 155/min, and respirations were 62/min. Results of electrolyte and arterial blood gas analyses are listed in Table 2.

With the laboratory data provided, the anion gap can be calculated:

\[
anion\ gap = Na^+ - [Cl^- + HCO_3^-]
\]

\[131 - (96 + 10) = 25 \text{ mEq/L}
\]

JT’s history, clinical manifestations, and laboratory findings were consistent with new-onset diabetes and diabetic ketoacidosis. The laboratory data suggested metabolic acidosis (pH < 7.35, carbon dioxide < 45 mEq/L, and bicarbonate < 22 mEq/L) with an elevated anion gap (> 12 mEq/L). The cause of the acidosis was an increase in ketoacids in the bloodstream. Insulin deficiency in the blood stimulates the pancreas to release glucagon; the glycogen is converted to glucose in the liver, and the glucose is released into the

<table>
<thead>
<tr>
<th>Table 1 Common disorders of metabolic acidosis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Metabolic acidosis with elevated anion gap</strong></td>
</tr>
<tr>
<td><strong>MUDPILES</strong></td>
</tr>
<tr>
<td>Methanol ingestion</td>
</tr>
<tr>
<td>Uremia</td>
</tr>
<tr>
<td>Diabetic, alcoholic, or starvation ketoacidosis</td>
</tr>
<tr>
<td>Paraldehyde ingestion</td>
</tr>
<tr>
<td>Isoniazid, salicylate, or iron poisoning</td>
</tr>
<tr>
<td>Lactic acidosis</td>
</tr>
<tr>
<td>Ethylene glycol ingestion</td>
</tr>
</tbody>
</table>

---

Figure 4: Anion gap equation.
bloodstream. The blood glucose level increases; however, the cells cannot use glucose without insulin. Severe hyperglycemia leads to osmotic diuresis, which explains the polydipsia and polyuria experienced by JT and the signs of dehydration evident on clinical examination. Lipolysis that occurs as a result of insulin deficiency leads to oxidation of free fatty acids by the liver and subsequent production of ketoacids.

Calculating the anion gap helps clinicians decipher the cause of the acidosis. In this case, the accumulation of ketoacids in the bloodstream created an elevated anion gap metabolic acidosis. The metabolic acidosis, caused by an increase in ketoacids, was complicated by the acidosis that results from dehydration and poor tissue perfusion.

Case Study 2

PN, a 10-month-old, previously healthy girl, had had diarrhea for 4 days. Her mother reported that in the preceding 2 days, PN had taken a total of 180 mL (6 oz) of formula and had had only 2 wet diapers. The infant had had approximately 6 loose, watery stools per day for the preceding 3 days. On examination, PN was lethargic, pale, and cool. Her eyes were sunken, and she did not produce tears when she cried. Respirations were 65/min and breathing was unlabored. Laboratory tests included a basic metabolic panel, complete blood cell count, arterial blood gas analysis, and stool samples for culture. Electrolyte and arterial blood gas results are listed in Table 3.

The arterial blood gas analysis indicated a metabolic acidosis. The calculated anion gap was 8 mEq/L. PN’s clinical history and physical examination suggested that she was dehydrated from gastroenteritis. The laboratory data indicated a metabolic acidosis with a normal anion gap. The finding of a normal anion gap suggested that PN was deficient in bicarbonate. The infant most likely was losing bicarbonate through the gastrointestinal tract and excessive stool loss. The primary loss of bicarbonate created an alteration in the acid-base balance and led to metabolic acidosis. PN’s increased respiratory rate indicated an attempt to compensate for the acidosis by excreting carbon dioxide through the respiratory system. Additionally, chloride accumulated to compensate for the loss of bicarbonate. Because chloride is a measured anion, the hyperchloremia created a normal or expected anion gap value. Although loss of bicarbonate was the primary cause of the acidosis, PN’s state of dehydration and tissue hypoperfusion complicated the metabolic acidosis.

Case Study 3

CC, a 3-month-old girl, had surgery for repair of tetralogy of Fallot 1 day earlier and required a right-sided ventriculotomy. She was intubated, sedated, and receiving mechanical ventilation. She was cool and mottled peripherally, with weak central pulses. Heart rate was 178/min, blood pressure 65/40 mm Hg, respirations 24/min, oxygen saturation 95%, and central venous pressure 12 mm Hg. Mean urine output overnight was 0.4 mL/kg per hour.

Table 2 Case study 1 laboratory values

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Laboratory</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial blood gas</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.19</td>
<td>7.35-7.45</td>
</tr>
<tr>
<td>Paco₂, mm Hg</td>
<td>32</td>
<td>35-45</td>
</tr>
<tr>
<td>Pao₂, mm Hg</td>
<td>110</td>
<td>90-100</td>
</tr>
<tr>
<td>Bicarbonate, mEq/L</td>
<td>9</td>
<td>22-26</td>
</tr>
<tr>
<td>Electrolytes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sodium, mEq/L</td>
<td>131</td>
<td>135-145</td>
</tr>
<tr>
<td>Potassium, mEq/L</td>
<td>5.3</td>
<td>3.5-5.5</td>
</tr>
<tr>
<td>Chloride, mEq/L</td>
<td>96</td>
<td>95-108</td>
</tr>
<tr>
<td>Bicarbonate, mEq/L</td>
<td>10</td>
<td>20-29</td>
</tr>
<tr>
<td>Glucose, mg/dL</td>
<td>655</td>
<td>65-120</td>
</tr>
</tbody>
</table>

a To convert milligrams per deciliter to millimoles per liter, multiply by 0.0555.

Table 3 Case study 2 laboratory values

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Laboratory</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial blood gas</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.24</td>
<td>7.35-7.45</td>
</tr>
<tr>
<td>Paco₂, mm Hg</td>
<td>31</td>
<td>35-45</td>
</tr>
<tr>
<td>Pao₂, mm Hg</td>
<td>109</td>
<td>90-100</td>
</tr>
<tr>
<td>Bicarbonate, mEq/L</td>
<td>16</td>
<td>22-26</td>
</tr>
<tr>
<td>Electrolytes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sodium, mEq/L</td>
<td>140</td>
<td>135-145</td>
</tr>
<tr>
<td>Potassium, mEq/L</td>
<td>2.8</td>
<td>3.5-5.5</td>
</tr>
<tr>
<td>Chloride, mEq/L</td>
<td>114</td>
<td>95-108</td>
</tr>
<tr>
<td>Bicarbonate, mEq/L</td>
<td>18</td>
<td>20-29</td>
</tr>
<tr>
<td>Glucose, mg/dL</td>
<td>52</td>
<td>65-120</td>
</tr>
</tbody>
</table>

a To convert milligrams per deciliter to millimoles per liter, multiply by 0.0555.
Anaerobic metabolism ensues in the setting of tissue hypoxia, generating lactate, carbon dioxide, and hydrogen ions and ultimately creating an elevated anion gap acidosis.4

Treatment

Treatment of metabolic acidosis must be targeted at the underlying problem. Quick administration of sodium bicarbonate may be necessary to correct the pH and avoid the consequences of severe acidosis. However, investigating the cause of the acidosis and developing a treatment strategy that addresses the primary cause of the acid-base imbalance are critical.

In case study 1, JT had diabetic ketoacidosis. This type of acidosis will not resolve until the concentrations of glucose and other electrolytes are normal and the cycle of hyperglycemia and hypovolemia is stopped. With the administration of insulin, the blood glucose levels will slowly decrease and the signs and symptoms of hyperglycemia causing metabolic acidosis will subside.5

In case study 2, PN was losing bicarbonate in the stool as a result of gastroenteritis and was hypovolemic, both leading to metabolic acidosis. The underlying cause of the acidosis was loss of bicarbonate; therefore, the metabolic acidosis would resolve once the gastroenteritis was resolved and bicarbonate was not lost in the stool. Additionally, replacement of circulating volume improves oxygen delivery to the tissues and allows cells to return to aerobic metabolism. This change reduces the accumulation of acid caused by anaerobic metabolism.7 In addition, ensuring adequate replacement of ongoing losses is necessary to reverse metabolic acidosis.

Case study 3 required careful consideration of the factors influencing the low cardiac output. In order to correct the acidosis, support of cardiac output was needed.9 CC’s right ventricle was dysfunctional as a result of the surgery, and she might need an inotrope to improve contractility. Once the blood pressure improves after treatment with an inotrope, afterload reduction may be considered to reduce the workload of the ventricle, ultimately improving cardiac output and meeting the metabolic demands of the tissues.

Conclusion

Disruption of acid-base balance, specifically metabolic acidosis, can have serious consequences, including death. Metabolic acidosis is a common manifestation of illness; however, the causes of the acidosis vary widely. Calculating the anion gap is an important piece of the assessment to determine the cause of the acidosis. Detection of acid-base imbalance and assessment of the cause are vital skills of pediatric critical care nurses. CCN

Table 4 Case study 3 laboratory values

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Values</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Laboratory</td>
<td>Normal</td>
</tr>
<tr>
<td>Arterial blood gas</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.26</td>
<td>7.35-7.45</td>
</tr>
<tr>
<td>PacO₂, mm Hg</td>
<td>29</td>
<td>35-45</td>
</tr>
<tr>
<td>PacO₂, mm Hg</td>
<td>128</td>
<td>90-100</td>
</tr>
<tr>
<td>Bicarbonate, mEq/L</td>
<td>16</td>
<td>22-26</td>
</tr>
<tr>
<td>Electrolytes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sodium, mEq/L</td>
<td>140</td>
<td>135-145</td>
</tr>
<tr>
<td>Potassium, mEq/L</td>
<td>3.8</td>
<td>3.5-5.5</td>
</tr>
<tr>
<td>Chloride, mEq/L</td>
<td>105</td>
<td>95-108</td>
</tr>
<tr>
<td>Bicarbonate, mEq/L</td>
<td>14</td>
<td>20-29</td>
</tr>
<tr>
<td>Glucose, mg/dL</td>
<td>100</td>
<td>65-120</td>
</tr>
</tbody>
</table>

*To convert milligrams per deciliter to millimoles per liter, multiply by 0.0555.

Table 4 lists the electrolyte and arterial blood gas results. The arterial blood gas analysis indicated a metabolic acidosis, and the calculated anion gap was 21 mEq/L.

CC’s laboratory values revealed a metabolic acidosis with an elevated anion gap. Calculating an elevated anion gap helped determine the cause of the acidosis and ultimately guided the treatment strategy. CC’s history, clinical findings, and elevated anion gap acidosis suggested a low cardiac output state, causing increased production of lactic acid in the blood.

CC’s heart was recovering from open heart surgery and the effects of cardiopulmonary bypass. Presumably, the compliance and contractility of the right ventricle were compromised. CC was unable to generate cardiac output adequate to meet the metabolic demands of the tissues. Anaerobic metabolism ensues in the setting of tissue hypoxia, generating lactate, carbon dioxide, and hydrogen ions and ultimately creating an elevated anion gap acidosis.7 In addition, ensuring adequate replacement of ongoing losses is necessary to reverse metabolic acidosis.

Case study 3 required careful consideration of the factors influencing the low cardiac output. In order to correct the acidosis, support of cardiac output was needed.9 CC’s right ventricle was dysfunctional as a result of the surgery, and she might need an inotrope to improve contractility. Once the blood pressure improves after treatment with an inotrope, afterload reduction may be considered to reduce the workload of the ventricle, ultimately improving cardiac output and meeting the metabolic demands of the tissues.

Conclusion

Disruption of acid-base balance, specifically metabolic acidosis, can have serious consequences, including death. Metabolic acidosis is a common manifestation of illness; however, the causes of the acidosis vary widely. Calculating the anion gap is an important piece of the assessment to determine the cause of the acidosis. Detection of acid-base imbalance and assessment of the cause are vital skills of pediatric critical care nurses. CCN

dotmore

To learn more about pediatric care in the intensive care unit, read “Evaluating Central Venous Catheter Care in a Pediatric Intensive Care Unit” by Hatler et al in the American Journal of Critical Care, 2009;18:514-520. Available at www.ajcconline.org.

None reported.

Financial Disclosures

None reported.
References

1. Which of the following best describes the term pH?
   a. Positive algorithm of hydrogen ion concentration
   b. Negative logarithm of hydrogen ion concentration
   c. pH = \(10^{-pH}\)
   d. pH = \(HCO_3^- - Cl^- - CO_2\)

2. Which of the following bodily processes produces hydrogen ions?
   a. Incomplete oxidation of lipids and carbohydrates
   b. Cellular respiration
   c. Krebs cycle
   d. Renin-angiotensin system

3. If the body has too many hydrogen ions causing an acidosis, using the carbonic anhydrase equation, how would the body correct the acidosis?
   a. \(H_2O + CO_2 \rightarrow H_2CO_3\)
   b. \(H_2CO_3 \rightarrow H^+ + HCO_3^-\)
   c. \(H^+ + HCO_3^- \rightarrow H_2O + CO_2\)
   d. \(H_2CO_3 \rightarrow H_2O + CO_2\)

4. What is the anion gap for a patient with a sodium level of 138 mEq/L, a chloride level of 97 mEq/L, and a bicarbonate level of 22 mEq/L?
   a. 17
   b. 18
   c. 19
   d. 123

5. A patient who is receiving acetazolamide is at risk for which acid-base disturbance?
   a. Respiratory alkalosis
   b. Anion gap metabolic acidosis
   c. Normal anion gap metabolic acidosis
   d. Respiratory acidosis

6. When a patient has an anion gap metabolic acidosis, which of the following conditions should the clinician be looking for?
   a. Loss of bicarbonate
   b. Increased accumulation of acids
   c. Increased accumulation of buffering solutions
   d. Increased levels of unmeasured cations

7. Which of the following systems is identified as the most effective buffer system in the body?
   a. Cellular
   b. Bicarbonate
   c. Renal
   d. Respiratory

8. Diarrhea in pediatric patients may cause what type of acid-base abnormality?
   a. Anion gap metabolic acidosis
   b. Normal anion gap acidosis
   c. Respiratory acidosis
   d. Respiratory alkalosis

9. The conditions of diabetic ketoacidosis and lactic acidosis cause what type of acid-base abnormality?
   a. Anion gap metabolic acidosis
   b. Normal anion gap acidosis
   c. Respiratory acidosis
   d. Respiratory alkalosis

10. Critical management of metabolic acidosis must be targeted to what treatment plan?
    a. Quick administration of sodium bicarbonate
    b. Rapid volume replacement
    c. Administering inotropes for hypotension
    d. Identifying and treating the underlying cause

11. A shift of the oxyhemoglobin curve to the left causes what type of effect on oxygen’s attraction to hemoglobin?
    a. Higher affinity of oxygen to hemoglobin
    b. Higher affinity of oxygen to hemoglobin due to the increased presence of hydrogen ions
    c. Less affinity of oxygen to hemoglobin
    d. Less affinity of oxygen to hemoglobin due to an increased level of carbon dioxide

---

**Program evaluation**

- Objective 1 was met [ ]
- Objective 2 was met [ ]
- Content was relevant to my nursing practice [ ]
- My expectations were met [ ]
- This method of CE is effective for this content [ ]
- The level of difficulty of this test was: easy [ ] medium [ ] difficult [ ]
- It took me ___ hours/minutes to complete this program.

**Test answers:** Mark only one box for your answer to each question. You may photocopy this form.

1. a 2. b 3. a 4. a 5. a 6. a 7. a 8. a 9. a 10. a 11. a

**AMERICAN ASSOCIATION of CRITICAL-CARE NURSES**

For faster processing, take this CE test online at [www.ccnonline.org](http://www.ccnonline.org) (“CE Articles in this issue”) or mail this entire page to: AACN, 101 Columbia Aliso Viejo, CA 92656.

---

Downloaded from [http://ccn.aacnjournals.org](http://ccn.aacnjournals.org) by AACN on September 6, 2017