Assessing Tissue Oxygenation
Barbara E. Berry and Agnes Eugine Pinard

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Assessing Tissue Oxygenation

Barbara E. Berry, RN, PhD
Agnes Eugine Pinard, RN, BSN, MS/HSA

To provide the comprehensive nursing care that is necessary and is expected by informed consumers, nurses need an expanded knowledge base. This need is especially apparent in hospitals, where the acuity of patients is high. Accurate assessment, prompt recognition, and proper management of adverse changes are vital components in the nursing care of patients with compromised functioning. Although physical examination is important, the need for understanding the principles of gas diffusion and laboratory assessment of tissue oxygenation, including interpretation of hemoglobin concentration, oxygen saturation, cardiac output, and acid-base balance (Table 1), must not be overlooked.

DIFFUSION OF GASES

At sea level, atmospheric pressure is 760 mm Hg. The air we breathe is a mixture of gases containing 79% nitrogen, 21% oxygen, traces of carbon dioxide (approximately 0.04%), and other elements. The partial pressure of oxygen (P_{O_2}) can therefore be calculated by multiplying the percentage of oxygen by the atmospheric pressure:

\[
0.21 \times 760 \text{ mm Hg} = 159.6 \text{ mm Hg}.
\]

The partial pressure of carbon dioxide (P_{CO_2}) can be calculated in the same manner:

\[
0.0004 \times 760 \text{ mm Hg} = 0.3 \text{ mm Hg}.
\]

The concentrations of gases in alveolar air are not the same as the concentrations in atmospheric air, however, because alveolar air is only partially replaced by atmospheric air with each breath. As soon as atmospheric air enters the respiratory passages, it is exposed to dead space air and fluids that cover respiratory surfaces. Thus, oxygen reaches the alveoli at approximately 103 mm Hg (P_{A_{O_2}}). Likewise, the carbon dioxide level in the alveoli is approximately 40 mm Hg (P_{A_{CO_2}}).

According to the principles of diffusion, gas exposed to a liquid will dissolve in the liquid until equilibrium of the gaseous phase of the mixture is reached and will exert the same partial pressure in the gaseous phase of the liquid as it did in the gaseous mixture. Diffusion of gases in the lung illustrates this point. Alveolar
Table 1  Definitions of terms related to assessment of tissue oxygenation

**Acid-base ratio** – 1:20 ratio at an arterial blood pH of 7.4. The 1 represents acid or 1.2 mmol/L of carbonic acid (40 mm Hg PaCO₂); the 20 represents base or 24 mmol/L bicarbonate

**Acidemia** – pH lower than an acceptable level in arterial blood (ie, pH < 7.35)<sup>1,3</sup>

**Acidic substances** – substances that donate hydrogen ions in chemical reactions<sup>1,2,4</sup>

**Acidosis** – a pathophysiological state in which a marked base deficit is present (pH < 7.35)<sup>1,3</sup>

**Alkalemia** – pH greater than an acceptable level in arterial blood (ie, pH > 7.45)<sup>1,3</sup>

**Alkaline (or basic) substances** – substances that accept hydrogen ions in chemical reactions<sup>1,2,4</sup>

**Alkalosis** – a pathophysiological state in which a significant base excess is present (ie, pH > 7.45)<sup>1,3</sup>

**Anemia** – a condition that can result in tissue hypoxia and may be due to deficiency of hemoglobin in the blood, decreased number of red blood cells, inadequate flow of blood, or reduction in oxygen-carrying capacity of hemoglobin<sup>1</sup>

**Base excess** – a true nonrespiratory reflection of acid-base status reported in millimoles per liter of base greater than or less than the normal range; arrived at by multiplying the deviation from standard bicarbonate levels (24 mmol/L × 1.2)<sup>1,2,5</sup> (ie, 34 mmol/L – 24 mmol/L = 10 × 1.2 = +12, or 12 mmol/L – 24 mmol/L = -10 × 1.2 = -12)

**Bicarbonate (or HCO₃)** – an alkaline/basic substance in the blood<sup>1,5</sup>

**Buffer system** – a system in the body that prevents excessive changes in the pH of the body fluids<sup>1</sup>

**Cardiac output** – amount of blood pumped by the left ventricle into the aorta each minute (normal cardiac output in adults is 5 L/min)<sup>3</sup>

**Carbon dioxide combining power (carbon dioxide content)** – a laboratory value reported with serum electrolyte levels; a measure of the level of bicarbonate ions in the plasma, because bicarbonate is broken down by the addition of a strong acid, and the carbon dioxide emitted is a measure of the bicarbonate originally present (normal range is 22-26 mmol/L)<sup>2,4</sup>

**Hyperoxygenation** – a condition in which the partial pressure of arterial oxygen is greater than 100 mm Hg<sup>3</sup>

**Hypoxemia** – deficient oxygenation due to inadequate arterial blood tension or partial pressure of arterial oxygen<sup>1,5,4</sup> (ie, PaO₂ ranges from relatively normal, 70-100 mm Hg; to relatively safe, 45-70 mm Hg; to dangerous, < 45 mm Hg.)

**Hypoxia** – the condition of cellular oxygen deficiency<sup>7</sup>

**Oxyhemoglobin dissociation curve** – an S-shaped curve that demonstrates the relationship between PaO₂ and oxygen saturation of hemoglobin (ie, the progressive increased oxygen saturation or the percentage of hemoglobin that is bound with oxygen as PaO₂ increases and the desaturation that develops when PaO₂ decreases). Any PaO₂ less than 40 mm Hg is incompatible with life for more than a few minutes<sup>4,6</sup>

**P** – pressure<sup>5</sup>

**Po₂** – partial pressure of oxygen<sup>5</sup>

**Pco₂** – partial pressure of carbon dioxide<sup>5</sup>

**PAO₂** – partial pressure of alveolar oxygen<sup>1</sup>

**PAco₂** – partial pressure of alveolar carbon dioxide<sup>5</sup>

**Pao₂** – partial pressure of arterial oxygen<sup>5</sup>

**Paco₂** – partial pressure of arterial carbon dioxide<sup>5</sup>

**Pvo₂** – partial pressure of venous oxygen<sup>5</sup>

**Pvc0₂** – partial pressure of venous carbon dioxide<sup>5</sup>

**pH** – a logarithmic scale denoting hydrogen ion concentration (ie, pH ranges from 1 = acidic to 14 = alkaline/basic)<sup>1</sup>

**Polycythemia** – an increase in red blood cells most commonly associated with an increase in hemoglobin concentration in the blood<sup>1,3</sup>

**Respiratory insufficiency** – impairment of the normal ability to oxygenate arterial blood or to eliminate carbon dioxide, resulting in an inability to maintain normal arterial blood gas levels under conditions of increasing demand<sup>1</sup>

**Shunting** – passing of blood from the right side of the heart to the left side of the heart without gas exchange in the lungs<sup>1</sup>

**Tension** – partial pressure of a gas<sup>1</sup>
Oxygen and capillary blood are separated by a very thin alveolar membrane. Oxygen diffuses across this membrane until the partial pressure of the oxygen in capillary blood is the same as that in the alveoli (103 mm Hg). A small amount of blood from the right side of the heart that fails to pass through alveolar capillaries because of shunting mixes with the oxygenated blood going to the left side of the heart and slightly reduces the partial pressure of oxygen in the arterial blood (PaO₂) to approximately 95 mm Hg.

Because each gas is independent of the others in its ability to dissolve in a liquid, the principles of diffusion hold true for diffusion of carbon dioxide from the blood to the alveoli. That is, net diffusion occurs from areas of higher pressure to areas of lower pressure until equilibrium is reached. Blood from the right side of the heart contains carbon dioxide at a higher pressure (PvCO₂, ~46 mm Hg) than that of alveoli (~40 mm Hg), so some carbon dioxide diffuses out of capillary blood into the alveoli, from which it is expired into the atmosphere, and the blood that is returned to the left side of the heart and into systemic circulation has a partial pressure (PaCO₂) of approximately 40 mm Hg.

**OXYGENATION**

Pulmonary ventilation is defined as the inspiration and expiration of air (gases). Ventilatory failure is defined as having a PaO₂ of 50 mm Hg or less or a PaCO₂ of 50 mm Hg or greater. Although ventilation is the only method of eliminating carbon dioxide, factors other than ventilation influence oxygenation. Oxygen deficiency or tissue hypoxia has many causes (Table 2).

### OXYGEN TRANSPORT IN THE BLOOD

Hemoglobin transports almost all oxygen from the lungs to the tissues. Oxygen is transported in the blood in 2 forms: a negligible amount is dissolved in the plasma (~3% of total transported oxygen), and a considerably greater amount (97%) is bound to hemoglobin in red blood cells. The PaO₂ determines how much oxygen is transported in each form.

**Oxygen Transport in the Plasma**

Under normal conditions, 0.003 mL of oxygen can be dissolved in 100 mL of blood plasma for each 1 mm Hg of PaO₂. For example, 100 mL of blood plasma contains 0.3 mL of dissolved oxygen at a PaO₂ of 100 mm Hg:

\[
\text{If } \text{PaO}_2 \times 0.003 = \text{milliliters of dissolved oxygen in 100 mL of plasma,}
\]

\[
\text{then } 100 \times 0.003 = 0.3 \text{ mL of dissolved oxygen in 100 mL of plasma.}
\]

**Oxygen Transport in Red Blood Cells**

The amount of oxygen that can be delivered to the tissues also depends on the hemoglobin content of the blood. The normal range of hemoglobin in adults is 12 to 16 g per 100 mL (120-160 g/L) of blood, and each gram of hemoglobin can bind with approximately 1.34 mL of oxygen at full saturation. For example, approximately 20 mL of oxygen is transported in every 100 mL of blood when the hemoglobin content is 15 g, PaO₂ is 100 mm Hg, and the hemoglobin is 100% saturated with oxygen:

\[
\text{If hemoglobin content (in grams per milliliter of blood) } \times 1.34 \times \text{ oxygen saturation } = \text{milliliters of oxygen bound to hemoglobin per 100 mL of blood,}
\]

\[
\text{then } 15 \times 1.34 \times 1.00 = 20.1 \text{ mL of oxygen per 100 mL of blood.}
\]

However, oxygen transport can be reduced when either hemoglobin level or oxygen saturation is abnormal. For example, only 13.4 mL of oxygen is transported at a hemoglobin content of 10 g at the same PaO₂ and oxygen saturation (PaO₂ is 100 mm Hg, and the hemoglobin is 100%)

<table>
<thead>
<tr>
<th>Table 2 Some causes of oxygen deficiency or tissue hypoxia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breathing a hypoxic mixture of gases such as that at high altitude, where atmospheric pressure and oxygen tension are reduced, or near a fire, where atmospheric oxygen is consumed by combustion</td>
</tr>
<tr>
<td>Inability of the lung to oxygenate the blood</td>
</tr>
<tr>
<td>Interference with oxygen delivery to the tissues, as occurs in heart/pump failure</td>
</tr>
<tr>
<td>Inability of tissues to use oxygen, as occurs in cyanide poisoning</td>
</tr>
<tr>
<td>A decrease in oxygen content of blood, as occurs in anemia. (PaO₂ is normal, but the amount of hemoglobin is insufficient to carry an adequate amount of oxygen.)</td>
</tr>
</tbody>
</table>

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saturated with oxygen):

If hemoglobin content (grams per milliliter of blood) \(\times 1.34 \times\) oxygen saturation = milliliters of oxygen attached to hemoglobin per 100 mL of blood, then 10 \(\times 1.34 \times 1.00 = 13.4\) mL of oxygen per 100 mL of blood.

Total Oxygen Content in the Blood

Total oxygen content in the blood can be dramatically reduced when hemoglobin content, \(\text{PaO}_2\), or oxygen saturation is abnormal. For example, consider the situation when oxygen saturation is 70%, \(\text{PaO}_2\) is 40 mm Hg, and hemoglobin is 12 g/100 mL of blood:

\[
\text{If } 40 \times 0.003 = 0.12 \text{ mL of oxygen in plasma}, \text{ and } \\
12 \times 1.34 \times 0.70 = 11.26 \text{ mL of oxygen in red blood cells}, \text{ then total oxygen content } = \\
11.38 \text{ mL of oxygen in 100 mL of blood.}
\]

CARDIAC OUTPUT

When tissue oxygenation is assessed, another important consideration is cardiac output, which ultimately determines the amount of oxygen delivered. At a hemoglobin level of 15 g/mL, a \(\text{PaO}_2\) of 100 mm Hg, and an oxygen saturation of 100%, 20 mL of oxygen is transported in 100 mL of blood, which is equivalent to 200 mL of oxygen per liter of blood. Normal (adult) cardiac output is 5 L/min, so approximately 1 L of oxygen can be delivered to the body tissues per minute (5 L/min \(\times\) 200 mL of oxygen per liter of blood = 1000 mL or 1 L of oxygen delivered per minute). If the heart cannot pump enough blood (eg, ventricular failure, arrhythmias, reduced heart rate), the cardiac output and the amount of oxygen delivered to tissues are reduced. For example, if cardiac output is reduced by half, to 2.5 L/min, only 500 mL of oxygen will be delivered to the tissues per minute.

ASSESSMENT OF TISSUE OXYGENATION

Oxyhemoglobin Dissociation Curve

Exposing hemoglobin to oxygen tensions of 0 to 150 mm Hg results in an S-shaped curve known as the oxyhemoglobin dissociation curve (Figure 1). The equation that reproduces the oxyhemoglobin dissociation curve is calculated by most blood gas analyzers in clinical use. Normally, at a \(\text{PaO}_2\) of 27 mm Hg, hemoglobin is 50% saturated; at a \(\text{PaO}_2\) of 40 mm Hg, 75% saturated; at a \(\text{PaO}_2\) of 60 mm Hg, 90% saturated; at a \(\text{PaO}_2\) of 80 mm Hg, 95% saturated; and at a \(\text{PaO}_2\) of 97 mm Hg, 97% saturated1,3,7 (Figure 1).

Tissue oxygenation and pulmonary oxygen uptake depend critically on the relationship demonstrated by the oxyhemoglobin dissociation curve. Although hemoglobin has a certain affinity for oxygen that allows blood to oxygenate tissue appropriately, various factors can alter this affinity and change the position of the oxyhemoglobin dissociation curve1,2 (Figure 2).

For instance, a shift of the curve to the right results in a decreased affinity of hemoglobin for oxygen. Although this decreased affinity increases the release of oxygen and is beneficial for tissue oxygenation when \(\text{PaO}_2\) is in a safe or normal range, a deficiency in oxygenation of arterial blood limits the amount of oxygen available to the tissues despite easier release of oxygen to the tissues when the \(\text{PaO}_2\) is less than 38 mm Hg.1,6 On the other hand, a shift to the left increases the capacity of hemoglobin to carry oxygen, but decreases unloading of oxygen to the tissues.1

A shift of the curve to the right or left depends on the acidity of the blood, carbon dioxide tension, body temperature, and concentration of 2,3-diphosphoglycerate (an organic phosphate in red blood cells that increases in level when...
anemia or chronic hypoxemia occurs). An increase in any of these factors will cause a shift of the curve to the right, and a decrease in any of these factors will result in a shift of the curve to the left.4 A shift to the right due to increased body temperature reflects increased cell metabolism, and a greater need for oxygen and is therefore adaptive. A shift to the right may also be compensatory in conditions of anemia and chronic hypoxemia.1 However, decreased levels of the enzymes that produce 2,3-diphosphoglycerate, a circumstance that may occur in blood that has been stored for transfusion, will shift the curve to the left and threaten the release of oxygen to the tissues.3 On the other hand, a shift of the curve to the left is adaptive in fetal blood, because the hemoglobin in fetal blood has a greater affinity for oxygen than does adult hemoglobin and therefore requires a lower tissue Po2 to release comparable amounts of oxygen molecules from hemoglobin.6

**Oxygen Saturation**

Oximetry is a method of measuring oxygen saturation. Pulse oximetry is based on measurement of the proportion of light transmitted by oxygenated forms of hemoglobin; a sensor is placed over a finger, toe, earlobe, or the bridge of the nose, and a numerical output is produced.3 The measured saturation is accurate only if the probe adequately detects and measures pulsatile blood flow. Factors that may influence accuracy include nail polish, low perfusion states (which may be the result of hypovolemia or hypothermia), and administration of peripheral vasoconstriction agents, which make it difficult to identify a pulse signal.8

Pulse oximetry is generally accurate only for oxygen saturations greater than 80%; therefore, arterial blood gas analysis is recommended for oxygen saturations less than 80%.5 Moreover, most clinicians consider an oxygen saturation of less than 90% significant and will not rely exclusively on pulse oximetry for measurement in such situations.3

**Acid-Base Balance**

Body fluids must maintain a normal acid-base balance in order for normal cellular function to sustain health and life. Acid-base balance can be described by measuring the pH (or acidity) of a substance on a scale that ranges from 1 to 14 (Figure 3).14 Normally, blood has a pH of 7.35 to 7.45. A pH value outside this range indicates a serious acid-base imbalance. The body has numerous compensatory mechanisms to correct an abnormal pH; however, if these mechanisms fail (Figure 4), cellular functions are impaired, and death will eventually result.1,2

Respiratory acid-base imbalances (acidosis or alkalosis) are triggered by respiratory disorders that result from inspiratory and/or expiratory dysfunction. Metabolic acid-base imbalances (acidosis or alkalois) are trig-
Acidosis

Acidosis is the result of greater than normal amounts of acid or less than normal amounts of base (alkaline) in the blood (pH <7.35). Respiratory acidosis occurs with retention of excess carbonic acid (due to decreased expiration of carbon dioxide) and can be diagnosed on the basis of the increased PaCO₂ in arterial blood gas sampling (Table 3). Metabolic acidosis occurs when body fluids contain an excessive amount of metabolic acids or a deficit of bases and can be diagnosed on the basis of the decreased bicarbonate level or base excess in arterial blood samples (Table 3).

Alkalosis

Alkalosis is the result of less than normal amounts of acid or greater than normal amounts of base (alkaline) in the blood (pH >7.45). Respiratory alkalosis occurs when excess carbonic acid is excreted (via increased rate and depth of expiration of carbon dioxide) and can be diagnosed on the basis of the decreased PaCO₂ in arterial blood gas sampling (Table 3). Metabolic alkalosis occurs when body fluids contain an excessive amount of bases or a deficit of acids and can be diagnosed on the basis of the increased bicarbonate level or base excess in arterial blood gas sampling (Table 3).

Compensatory Mechanisms

The ratio of bicarbonate to carbon dioxide is 20:1, and this ratio is normally maintained through compensatory mechanisms or chemical buffers present in the extracellular fluid, body cells, blood cells, and plasma. Buffer systems maintain acid-base balance in 2 ways: by correcting or altering the component responsible for the imbalance and by compensating through alterations in the component that is not primarily responsible for the imbalance. These buffer systems can act within a fraction of a second to prevent excessive changes in pH.

Bicarbonate–Carbonic Acid Buffer System

The body’s major buffer system is the bicarbonate-carbonic acid compensatory mechanism. Normal blood pH is the result of a bicarbonate to carbon dioxide ratio of 20 to 1. The 20 in this ratio represents base or 24 mmol/L of bicarbonate. The 1 in this ratio represents acid or 1.2 mmol/L of

Table 3 Interpretation of arterial blood gas sampling*

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Low</td>
</tr>
<tr>
<td>pH</td>
<td>&lt; 7.35 Acidemia</td>
</tr>
<tr>
<td>PaCO₂, mm Hg</td>
<td>&lt; 35 Alkalemia</td>
</tr>
<tr>
<td>Bicarbonate, mmol/L</td>
<td>&lt; 22 Acidemia</td>
</tr>
<tr>
<td>Base excess</td>
<td>&lt; -2 Acidemia</td>
</tr>
<tr>
<td>PaCO₂, mm Hg</td>
<td>&lt; 70 Hypoxemia</td>
</tr>
</tbody>
</table>

*Statistical relationships are often used in medical laboratories to establish "normal ranges." Most laboratories consider 2 SDs from the mean as the normal range because that range includes 95% of the represented population. Therefore, normal ranges may vary slightly for different age groups and in different laboratories.
carbonic acid. The 1 in this ratio may also represent a PaCO₂ of 40 mm Hg, because carbon dioxide is a potential acid. That is, when carbon dioxide is dissolved in water, it becomes carbonic acid. Thus, when the level of carbon dioxide increases, the level of carbonic acid also increases, and when the level of carbon dioxide decreases, the level of carbonic acid also decreases. If the level of either bicarbonate or carbon dioxide is increased or decreased so that the 20:1 ratio is not maintained, acid-base imbalances occur.

A ratio of less than 20:1 (20/1) indicates acidosis. A ratio of more than 20:1 indicates alkalosis. The diagnosis of acidosis or alkalosis can be verified by using the 20:1 formula. For example, for a patient with a pH of 7.22, a bicarbonate level of 21 mmol/L, and a PaCO₂ of 55 mm Hg:

(Base) Given that the 20 in the 20:1 ratio represents 24 mmol/L bicarbonate, 21 mmol/L bicarbonate would be equivalent to a 17.5 mmol/L. First set up the simple proportion

\[
\frac{20}{24} = \frac{?}{21}
\]

and then cross-multiply to solve, \((20 \times 21)/24 = 420/24 = 17.5\).

(Acid) If the 1 in the 20:1 ratio represents a PaCO₂ of 40 mm Hg, then a PaCO₂ of 55 mm Hg would be equivalent to 1.37 mm Hg. Again, set up the simple proportion

\[
\frac{1}{40} = \frac{?}{55}
\]

and cross-multiply to solve, \((1 \times 55)/40 = 55/40 = 1.37\).

(Diagnosis) The ratio is less than 20:1 (17.5/1.37 = 12.8/1); therefore, the diagnosis is acidosis.

Respiratory Compensatory Mechanism

The respiratory center in the medulla is sensitive to concentrations of carbon dioxide and hydrogen ion in body fluids. Compensation by the respiratory center can occur rapidly (in minutes or seconds). When acidaemia (decreased blood pH) occurs, the respiratory center in the medulla is stimulated. This stimulation results in increases in the rate and depth of respirations, thereby reducing the carbon dioxide (carbonic acid) level and increasing the pH of the blood. When alkalemia (increased blood pH) occurs, the respiratory center in the brain is inhibited. This inhibition results in decreases in the rate and depth of respirations, retention of carbon dioxide (carbonic acid), and decrease in the
blood pH. The lungs compensate for both respiratory and metabolic imbalances. However, the lungs cannot compensate for acid-base disturbances when pulmonary dysfunction is severe. In these instances, the kidneys must provide compensation.²

Renal Compensatory Mechanism

Cells in the distal part of the renal tubules are sensitive to changes in the pH of the filtrate. When the pH is less than normal, hydrogen ions are excreted and bicarbonate is formed and retained. When the pH is greater than normal, hydrogen ions are conserved and base-forming ions are excreted. Renal compensation for imbalances is slow (hours to days). The kidneys cannot compensate for acid-base imbalances related to renal failure.²

Because total electrolyte levels must always be in electrochemical balance, electrolyte concentrations may change when acid-base imbalances occur. The primary electrolytes that are exchangeable and may be stimulated by acid-base imbalances to move in and out of intracellular and extracellular compartments are sodium, chloride, potassium, and bicarbonate (Table 4). Metabolic pH disturbances have a greater effect on electrolyte mechanisms than do

<table>
<thead>
<tr>
<th>Variable</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ionized calcium, mmol/L</td>
<td>1.15-1.27</td>
</tr>
<tr>
<td>Serum sodium, mmol/L</td>
<td>135-145</td>
</tr>
<tr>
<td>Serum potassium, mmol/L</td>
<td>3.5-5</td>
</tr>
<tr>
<td>Serum chloride, mmol/L</td>
<td>95-105</td>
</tr>
<tr>
<td>Carbon dioxide combining power, mmol/L</td>
<td>22-26</td>
</tr>
<tr>
<td>Hemoglobin, g/100 mL blood</td>
<td>12-16</td>
</tr>
<tr>
<td>Urinary pH</td>
<td>4.5-8 (normal, ~6)</td>
</tr>
</tbody>
</table>

*Statistical relationships are often used in medical laboratories to establish “normal ranges.” Most laboratories consider 2 SDs from the mean as the normal range because that range includes 95% of the represented population. Therefore, normal ranges may vary slightly for different age groups and in different laboratories.
respiratory pH disturbances.\textsuperscript{2,10}

Clinical states of acid-base imbalance may be described as acidosis or alkalosis. Acidosis is the result of greater than normal concentrations of acid or less than normal concentrations of base in blood. Respiratory acidosis is caused by any condition that interferes with excretion of carbon dioxide by the lungs, and metabolic acidosis is caused by abnormal losses of bicarbonate or by accumulation of excess metabolic acids\textsuperscript{2,5,10} (Tables 5 and 6).

### Table 5 Respiratory acidosis

<table>
<thead>
<tr>
<th>Clinical signs and symptoms</th>
<th>Arterial blood gas analysis</th>
<th>Other laboratory studies may reveal</th>
<th>Some compensatory mechanisms that may be occurring</th>
<th>Most appropriate management</th>
<th>Most appropriate nursing care</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decreased rate and depth of respirations</td>
<td>pH &lt; 7.35 (normal, 7.35-7.45)</td>
<td>Potassium level &gt; 5 mmol/L (normal, 3.5-5 mmol/L)</td>
<td>Respiratory—Hyperventilation will decrease carbonic acid level by blowing off excess carbon dioxide. However, the ability to control respirations may have been lost because of the condition that is interfering with gas exchange, and, in this situation, hyperventilation can be accomplished only by mechanical ventilation. Renal—Sodium is reabsorbed in exchange for hydrogen, which will contribute to the formation of ammonia and be excreted as urine; in addition, the reabsorbed sodium combines with bicarbonate to form sodium bicarbonate. Electrolytes—Chloride moves from blood to cells in exchange for bicarbonate; bicarbonate moves to blood and begins to neutralize excess carbonic acid; potassium moves from cells to blood in exchange for hydrogen ions.</td>
<td>Determining the cause of hypoventilation</td>
<td>Being aware of common causes, signs and symptoms, expected laboratory findings if acidosis is suspected</td>
</tr>
<tr>
<td>Decreased central nervous system activity (lethargy, lack of judgment, disorientation)</td>
<td>PaCO\textsubscript{2} &gt; 45 mm Hg (normal, 35-45 mm Hg)</td>
<td>Urinary pH &lt; 6 (normal, ~6)</td>
<td>Respiratory—Hyperventilation will decrease carbonic acid level by blowing off excess carbon dioxide. However, the ability to control respirations may have been lost because of the condition that is interfering with gas exchange, and, in this situation, hyperventilation can be accomplished only by mechanical ventilation. Renal—Sodium is reabsorbed in exchange for hydrogen, which will contribute to the formation of ammonia and be excreted as urine; in addition, the reabsorbed sodium combines with bicarbonate to form sodium bicarbonate. Electrolytes—Chloride moves from blood to cells in exchange for bicarbonate; bicarbonate moves to blood and begins to neutralize excess carbonic acid; potassium moves from cells to blood in exchange for hydrogen ions.</td>
<td>Maintaining adequate ventilation and hydration</td>
<td>Predicting which patients are at risk for respiratory acidosis, such as surgical patients who have chronic lung disease, are obese, or smoke</td>
</tr>
<tr>
<td>Headache or blurred vision, especially in the morning</td>
<td>Bicarbonate level normal (normal, 22-26 mmol/L)</td>
<td></td>
<td></td>
<td>Administering oxygen therapy for severe hypoxemia</td>
<td>Providing interventions that will help increase lung expansion (e.g., encouraging deep breathing, ambulation, use of incentive spirometer) for patients at risk for respiratory acidosis</td>
</tr>
<tr>
<td>Weakness, cardiac arrhythmia, seizures if hyperkalemia is present</td>
<td>Base excess normal (normal, -2 to +2)</td>
<td></td>
<td></td>
<td>Administering oxygen at low flow rates for patients with chronic lung disease in order to maintain the hypoxic drive (because the medulla of these patients responds to lack of oxygen rather than to low carbon dioxide levels)</td>
<td>Continuing preceding measures for patients with confirmed respiratory acidosis in addition to measures to improve respiratory function (e.g., encouraging fluid intake to help thin secretions and performing chest physiotherapy, postural drainage, and suctioning unless otherwise contraindicated by patient’s condition)</td>
</tr>
</tbody>
</table>

### Table 5 Respiratory acidosis (continued)

<table>
<thead>
<tr>
<th>Most appropriate management</th>
<th>Most appropriate nursing care</th>
</tr>
</thead>
<tbody>
<tr>
<td>Determining the cause of hypoventilation</td>
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</tr>
<tr>
<td>Improving respiratory function through use of bronchodilators and chest physiotherapy, antibiotics for respiratory infections, mechanical ventilation for a decompensating respiratory system</td>
<td>Beginning seizure precautions when potassium level is elevated</td>
</tr>
</tbody>
</table>

### CLINICAL STATES OF ACID-BASE IMBALANCE

Clinical states of acid-base imbalance may be described as acidosis or alkalosis. Acidosis is the result of greater than normal concentrations of acid or less than normal concentrations of base in blood. Respiratory acidosis is caused by any condition that interferes with excretion of carbon dioxide by the lungs, and metabolic acidosis is caused by abnormal losses of bicarbonate or by accumulation of excess metabolic acids\textsuperscript{2,5,10} (Tables 5 and 6).
Metabolic acidosis results from abnormal losses of bicarbonate as occurs with
- Severe diarrhea
- Prolonged vomiting of deep gastrointestinal contents
or by accumulation of metabolic acid, as occurs with
- Ketosis resulting when proteins and fats are burned for energy rather than carbohydrates, as occurs in
  - Diabetes mellitus
  - Increased metabolism (e.g., fever, anesthesia, infection)
  - Starvation
- Excessive ingestion of substances that increase concentrations of metabolic acids, such as
  - Salicylic acid
  - Ammonium chloride
  - Ferrous sulfate
  - Ethylene glycol (antifreeze)
  - Methyl alcohol (wood alcohol)
- Inadequate renal function (inability to excrete hydrogen or conserve bicarbonate)
- Anoxia (anaerobic metabolism resulting in lactic acidosis)

Clinical signs and symptoms
- Changes in sensorium (lethargy progressing to coma)
- Hyperventilation (indicating respiratory compensation)
- Weakness, cardiac arrhythmias if hyperkalemia present

Arterial blood gas analysis
- pH < 7.35 (normal, 7.35-7.45)
- Paco₂ normal (normal, 35-45 mm Hg)
- Bicarbonate level < 22 mmol/L (normal, 22-26 mmol/L)
- Base excess < -2 (normal, -2 to +2)

Other laboratory studies may reveal
- Carbon dioxide combining power < 22 mmol/L (normal, 22-26 mmol/L)
- Potassium level > 5 mmol/L (normal, 3.5-5 mmol/L)
- Urinary pH < 6 (normal, ~6)

Some compensatory mechanisms that may be occurring
- Respiratory—Hyperventilation begins involuntarily in an effort to decrease carbonic acid level and restore acid-base ratio.
- Renal—Sodium is reabsorbed in exchange for hydrogen, which is excreted in urine; in addition, sodium combines with bicarbonate to form sodium bicarbonate, which will help to restore acid-base ratio.
- Electrolytes—Potassium moves out of cells to blood in exchange for hydrogen, an attempt to neutralize excess acid.

Most appropriate management
- Determining the underlying mechanism
- Planning appropriate interventions to correct mechanism (e.g., administering insulin when ketoacidosis occurs)
- Assessing serum potassium level and treating as necessary
- Administering intravenous alkali, such as sodium bicarbonate (most frequently used, but care must be taken to avoid overalkalinization, which could result in arrhythmias and tetany—no further bicarbonate should be given once the pH reaches 7.2)
- Administration of salts of organic acids that are metabolized to bicarbonate, such as lactate, acetate, and citrate
- Administration of tromethamine

Most appropriate nursing care
- Being aware of common causes, signs and symptoms, expected laboratory findings if acidosis is suspected
- Predicting which patients who are at risk for metabolic acidosis, such as those who have diabetes mellitus, have advancing renal disease, are hypoxic, are malnourished, or are on low carbohydrate diets
- Teaching ways to avoid this imbalance (e.g., correct administration of medication, diet, testing urine and/or blood of diabetic patients) as part of discharge instructions, even though most of the treatment specifically for metabolic acidosis is medical

Alkalosis is the result of less than normal concentrations of acid or greater than normal concentrations of base in the blood. Respiratory alkalosis is caused by any condition that causes an increase in rate and depth of breathing, resulting in excessive elimination of carbon.
Metabolic alkalosis is caused by any condition that causes loss of metabolic acids from the body or retention of bicarbonate \(^{2,10}\) (Tables 7 and 8).

### ANALYSIS OF ARTERIAL BLOOD GASES

Analysis of arterial blood gas sampling (Figure 5) can be performed by following the 4 steps described in Table 9. Examples of

<table>
<thead>
<tr>
<th>Table 7 Respiratory alkalosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory alkalosis is brought on by any condition that causes an increase in the rate and depth of breathing that results in excessive elimination of carbon dioxide, as occurs with</td>
</tr>
<tr>
<td>Pain</td>
</tr>
<tr>
<td>Severe exercise</td>
</tr>
<tr>
<td>Hysteria and anxiety reactions</td>
</tr>
<tr>
<td>Hypoxia, anoxia</td>
</tr>
<tr>
<td>Intentional overbreathing</td>
</tr>
<tr>
<td>Damage to the respiratory center in medulla, as occurs in</td>
</tr>
<tr>
<td>Central nervous system disease (eg, meningitis, encephalitis)</td>
</tr>
<tr>
<td>Intracranial surgery</td>
</tr>
<tr>
<td>Overstimulation of the respiratory center, as occurs in</td>
</tr>
<tr>
<td>Fever</td>
</tr>
<tr>
<td>Drug overdose</td>
</tr>
<tr>
<td>Gram-negative bacteremia</td>
</tr>
</tbody>
</table>

#### Clinical signs and symptoms

- Deep, rapid respirations
- Light-headedness and alterations in consciousness, inability to concentrate, tetany, convulsions (a low \( \text{Paco}_2 \) causes cerebral vasoconstriction and thus cerebral ischemia)
- Cardiac arrhythmias if hypokalemia is present
- Numbness and tingling of extremities if hypocalcemia is present

#### Arterial blood gas analysis

- \( \text{pH} > 7.45 \) (normal, 7.35-7.45)
- \( \text{Paco}_2 < 35 \) mm Hg (normal, 35-45 mm Hg)
- Bicarbonate level normal (normal, 22-26 mmol/L)
- Base excess normal (normal, -2 to +2)

#### Other laboratory studies may reveal

- Potassium level < 3.5 mmol/L (normal, 3.5-5 mmol/L)
- Ionized calcium level < 1.15 mmol/L (normal, 1.15-1.27 mmol/L)
- Urinary \( \text{pH} > 7 \) or > 8 (normal, ~6)

#### Some compensatory mechanisms that may be occurring

- Respiratory—Decreasing rate and depth of respirations will increase retention of carbonic acid. However, the ability to control respirations may have been lost. In addition, attempts to slow rate and depth of respirations are often inappropriate in the presence of a physiological response to hypoxemia.
- Renal—Hydrogen is reabsorbed, and large amounts of potassium and sodium are excreted with excess bicarbonate.
- Electrolytes—Potassium moves from blood to cells in exchange for hydrogen ion; buffer systems attempt to neutralize excess alkaline; in the presence of alkalosis, increased amounts of calcium are bound to protein, producing hypocalcemia.

#### Most appropriate management

- Determining and eliminating the underlying cause of hyperventilation (ie, for hysteria, reassurance, sedation, and carbon dioxide inhalation)
- Providing oxygen and mechanical ventilation as needed
- Assessing serum levels of potassium and calcium and treating as necessary

#### Most appropriate nursing care

- Being aware of common causes, signs and symptoms, expected laboratory findings if alkalosis is suspected
- Assessing those patients with suspected respiratory alkalosis such as those with hysteria, hypoxemia, pain, drug overdose
- Eliminating the underlying cause (ie, providing oxygen to hypoxic patients, reassurance and emotional support to hysterical patients, and encouraging hyperventilating patients to hold their breath for short periods or breathe into a paper bag)
- Beginning safety and seizure precautions if signs or symptoms of hypocalcemia are present
arterial blood gas sampling with corresponding acid-base imbalances are given in Table 10. A practice test for interpreting results of arterial blood gas sampling is given in Table 11. Assessing trends of arterial blood gas findings often provides more information relating to progress of the condition than does a single isolated arterial blood gas analysis. If compensation occurs, intervention is often unnecessary. Remember, treatment is considered for an imbalance only

### Table 8 Metabolic alkalosis

Metabolic alkalosis is brought on by any condition that causes loss of metabolic acids from the body or retention of bicarbonate, such as:

- Loss of hydrochloric acid from the stomach, as occurs in Vomiting
- Gastrintestinal suctioning
- Loss of chloride causing reabsorption of bicarbonate, as occurs with Use of diuretics
- Excessive vomiting
- Retention of sodium causing reabsorption of bicarbonate, as occurs with Increased secretion of aldosterone (stress, trauma)
- Excessive ingestion of alkali, such as Bicarbonate of soda (baking soda)
- Milk of magnesia
- Intravenous administration of sodium bicarbonate for cardiopulmonary resuscitation

#### Clinical signs and symptoms
- Slow, shallow respirations (compensation by lungs)
- Dizziness, tingling of extremities, tetany, convulsions, hypertonicity of muscles
- Irritability, disorientation
- Cardiac arrhythmias if hypokalemia is present

#### Arterial blood gas analysis
- pH > 7.45 (normal, 7.35-7.45)
- PaCO₂ normal (normal, 35-45 mm Hg)
- Bicarbonate level > 26 (normal, 22-26 mmol/L)
- Base excess > +2 (normal, -2 to +2)

#### Other laboratory reports may reveal
- Carbon dioxide combining power > 26 mmol/L (normal, 22-26 mmol/L)
  - (ie, plasma bicarbonate)
- Potassium level < 3.5 mmol/L (normal, 3.5-5 mmol/L)
- Ionized calcium level < 1.15 mmol/L (normal, 1.15-1.27 mmol/L)
- Chloride level < 95 mmol/L (normal, 95-105 mmol/L)
- Urinary pH > 7 or > 8 (normal, ~6)

#### Some compensatory mechanisms that may be occurring
- Respiratory—Hypoventilation begins involuntarily in an effort to retain carbonic acid.
- Renal—Hydrogen is reabsorbed, and large amounts of potassium and sodium are excreted with excess bicarbonate.
- Electrolytes—Potassium moves from blood to cells in exchange for hydrogen; buffer systems attempt to neutralize excess alkaline; in the presence of alkalosis, increased amounts of calcium are bound to protein, producing hypocalcemia.

#### Most appropriate management
- Determining and treating the primary condition (ie, replacing fluids lost by vomiting or nasogastric suctioning)
- Assessing and correcting serum levels of potassium, calcium, chloride, and sodium as necessary

#### Most appropriate nursing care
- Being aware of common causes, signs and symptoms, expected laboratory findings if alkalosis is suspected
- Predicting which patients are at risk for metabolic alkalosis, such as patients who are self-medicating with antacids, patients undergoing gastrointestinal suctioning or therapy
- Taking safety and seizure precautions for patients who have signs and symptoms of hypocalcemia
- Providing discharge teaching (ie, overuse of antacids, misuse of baking soda in place of antacids, replacement of fluids lost through vomiting) that may prevent alkalosis from occurring in the home
Table 9 Steps in analysis of arterial blood gases

1. Examine the pH, PaCO₂, and bicarbonate level. Are they all within normal limits?
   If Yes → Normal arterial blood gas levels

2. Is the pH abnormal with abnormal level of the respiratory or acid (PaCO₂) parameter and/or the metabolic or base (bicarbonate) parameter (see Table 3)?
   If Yes → Acidosis and/or alkalosis

   a. Which parameter agrees with the pH?
      That is, if the pH indicates acidemia, does the PaCO₂ and/or bicarbonate level also indicate acidemia?
      Or, if the pH indicates alkalemia, does the PaCO₂ and/or the bicarbonate level also indicate alkalemia?
      If Yes → Acidosis

   b. Does the PaCO₂ agree with the pH?
      If Yes → Respiratory acidosis or alkalosis

   c. Does the bicarbonate level agree with the pH?
      If Yes → Metabolic acidosis or alkalosis

   d. Do both PaCO₂ and bicarbonate level agree with the pH?
      If Yes → Combination of respiratory and metabolic acidosis or respiratory and metabolic alkalosis

3. Is the pH abnormal with 1 parameter abnormal and in agreement with the pH, but the other parameter abnormal and not in agreement with the pH?
   If Yes → Partly compensated acid-base imbalance

   a. Is the PaCO₂ abnormal and in agreement with the pH, and the bicarbonate level abnormal, but not in agreement with the pH?
      If Yes → Partly compensated respiratory acidosis or alkalosis (predominantly the result of metabolic compensation)

   b. Is the bicarbonate level abnormal and in agreement with the pH, and PaCO₂ abnormal, but not in agreement with the pH?
      If Yes → Partly compensated metabolic acidosis or alkalosis (predominantly the result of respiratory compensation)

4. Is the pH normal, but the PaCO₂ and bicarbonate level are abnormal?
   a. Is the pH normal, but slightly alkaline (≥7.41)?
      If Yes → Compensated alkalemic imbalance

      Does the PaCO₂ (respiratory parameter) indicate alkalemia, but the bicarbonate level (respiratory parameter) indicates acidemia?
      If Yes → Compensated respiratory alkalosis

      Or, does the bicarbonate level (metabolic parameter) indicate alkalemia, but the PaCO₂ (respiratory parameter) indicates acidemia?
      If Yes → Compensated metabolic alkalosis

   b. Is the pH normal, but slightly acidic (≤7.39)?
      If Yes → Compensated acidotic imbalance

      Does the PaCO₂ (respiratory parameter) indicate acidemia, but the bicarbonate level (respiratory parameter) indicates alkalemia?
      If Yes → Compensated respiratory acidosis

      Or does the bicarbonate level (metabolic parameter) indicate acidemia, but the PaCO₂ (respiratory parameter) indicates alkalemia?
      If Yes → Compensated metabolic acidosis
Respiratory alkalosis

pH 7.52
PaCO₂ 30
HCO₃ 24 (or BE +2)

Metabolic alkalosis

pH 7.57
PaCO₂ 40
HCO₃ 36 (or BE +14)

Partly compensated respiratory alkalosis

pH 7.46
PaCO₂ 30
HCO₃ 20 (or BE -4)

Partly compensated metabolic alkalosis

pH 7.46
PaCO₂ 46
HCO₃ 31 (or BE +7)

Compensated respiratory alkalosis

pH 7.42
PaCO₂ 34
HCO₃ 21 (or BE -4)

Compensated metabolic alkalosis

pH 7.43
PaCO₂ 34
HCO₃ 30 (or BE +7)

When the pH is abnormal. Be alert for abnormal PaO₂, indicating hypoxia, and provide adequate supplemental oxygen. A case study is provided in Table 12 (page 40).

**SUMMARY**

Although physical examination remains an important part of the nursing assessment, assessment of tissue oxygenation provides additional vital information. Nurses who are caring for patients with compromised functioning must have a basic understanding of the physiological relationships between oxygen saturation, cardiac output, and acid-base balance and must be able to plan nursing care based on this knowledge.

**Acknowledgments**

The authors thank Debbie Newland, RN, BSN, associate nurse manager of the pediatric intensive care unit, and Barry Gelman, MD, associate professor of clinical pediatrics at the University of Miami School of Medicine and attending physician in the pediatric intensive care unit, Jackson Memorial Hospital, Miami, Fla, for their consultation and assistance with this article.

**References**

3. Shapiro BA. Peruzzi WT. Clinical
7. pH 7.51  
PaCO₂ 41  
HCO₃⁻ 32  

8. pH 7.47  
PaCO₂ 47  
HCO₃⁻ 30

9. pH 7.44  
PaCO₂ 46  
HCO₃⁻ 29

10. pH 7.68  
Paco₂ 30  
HCO₃⁻ 29

11. pH 7.51  
PaCO₂ 32  
HCO₃⁻ 25

12. pH 7.34  
PaCO₂ 49  
HCO₃⁻ 27

You may verify your answers by using the acid/base ratio:

\[
\frac{20}{1} \text{ represents base (alkaline) or 24 mmol/L bicarbonate} \\
\frac{1}{1} \text{ represents acid or 1.2 mmol/L of carbonic acid} \\
(40 \text{ mm Hg PaCO}_2)
\]

Therefore, a ratio < 20/1 = acidosis
a ratio > 20/1 = alkalosis

Answers

1. Respiratory acidosis
HCO₃⁻ 25 mmol/L
(24 mmol:20 = 25 mmol:?)
(20 x 25)/24 = 500/24 = 20.8

Paco₂ is 53 mm Hg
(40 mm Hg:1 = 53 mm Hg:?)
(1 x 53)/40 = 53/40 = 1.32

20.8
1.32 = 15.8
indicating acidosis

2. Respiratory acidosis
3. Compensated respiratory acidosis
4. Metabolic and respiratory acidosis
5. Normal
6. Normal

7. Metabolic alkalosis
HCO₃⁻ 32 mmol/L
(24 mmol:20 = 32 mmol:?)
(20 x 32)/24 = 640/24 = 26.67

Paco₂ is 41 mm Hg
(40 mm Hg:1 = 41 mm Hg:?)
(1 x 41)/40 = 41/40 = 1.02

26.67
1.02 = 26.15
indicating alkalosis

8. Partly compensated metabolic acidosis
9. Compensated metabolic alkalosis
10. Metabolic and respiratory alkalosis
11. Respiratory alkalosis
12. Partly compensated respiratory acidosis

Note: Table 12 follows on page 40.
Table 12  Case study: serial assessments, laboratory findings, results of arterial blood gas analysis, and interventions

A.G., an 86-year-old man, was admitted to the hospital at approximately 2 AM via the emergency department. On arrival, he was having difficulty breathing and was responsive only to painful stimuli. Vital signs at admission were body temperature, 97°F (~36.5°C); heart rate, 140/min; respirations, 37/min; and blood pressure, 177/110 mm Hg. Oxygen saturation measured by pulse oximetry was 76% on room air. Shortly after arrival, A.G. went into respiratory arrest, with subsequent bradycardia (heart rate, 35/min). Resuscitation was initiated with intubation, and oxygen was administered via a manual resuscitation bag. Further interventions were guided by serial physical assessments, results of laboratory tests, and arterial blood gas analyses.

<table>
<thead>
<tr>
<th>Findings</th>
<th>Conclusion/intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>March 24 at 2:16 AM</td>
<td>Partly compensated metabolic acidosis (possibly attributed to blowing off carbon dioxide via respirations with manual resuscitation bag)</td>
</tr>
<tr>
<td>( \text{PaO}_2, \text{ mm Hg} )</td>
<td>68</td>
</tr>
<tr>
<td>Oxygen saturation, %</td>
<td>91</td>
</tr>
<tr>
<td>( \text{pH} )</td>
<td>7.27</td>
</tr>
<tr>
<td>( \text{PaCO}_2, \text{ mm Hg} )</td>
<td>31</td>
</tr>
<tr>
<td>Base excess</td>
<td>-12</td>
</tr>
<tr>
<td>Bicarbonate, mmol/L</td>
<td>14</td>
</tr>
<tr>
<td>March 24 at 2:54 AM</td>
<td>Compensated metabolic acidosis</td>
</tr>
<tr>
<td>( \text{PaO}_2, \text{ mm Hg} )</td>
<td>103</td>
</tr>
<tr>
<td>Oxygen saturation, %</td>
<td>95</td>
</tr>
<tr>
<td>( \text{pH} )</td>
<td>7.35</td>
</tr>
<tr>
<td>( \text{PaCO}_2, \text{ mm Hg} )</td>
<td>34</td>
</tr>
<tr>
<td>Base excess</td>
<td>-8</td>
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<tr>
<td>Bicarbonate, mmol/L</td>
<td>18</td>
</tr>
<tr>
<td>March 24 at 6:30 AM</td>
<td>Normal acid-base balance</td>
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<tr>
<td>( \text{PaO}_2, \text{ mm Hg} )</td>
<td>309</td>
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<tr>
<td>Oxygen saturation, %</td>
<td>99</td>
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<tr>
<td>( \text{pH} )</td>
<td>7.38</td>
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<tr>
<td>( \text{PaCO}_2, \text{ mm Hg} )</td>
<td>40</td>
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<tr>
<td>Base excess</td>
<td>-2</td>
</tr>
<tr>
<td>Bicarbonate, mmol/L</td>
<td>23</td>
</tr>
<tr>
<td>White blood cell count, ( \times 10^9/\text{L} )</td>
<td>20.2</td>
</tr>
<tr>
<td>March 24 at 4:15 PM</td>
<td>Normal acid-base balance</td>
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<tr>
<td>( \text{PaO}_2, \text{ mm Hg} )</td>
<td>113</td>
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<tr>
<td>Oxygen saturation, %</td>
<td>98</td>
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<tr>
<td>( \text{pH} )</td>
<td>7.43</td>
</tr>
<tr>
<td>( \text{PaCO}_2, \text{ mm Hg} )</td>
<td>36</td>
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<tr>
<td>Base excess</td>
<td>0</td>
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<tr>
<td>Bicarbonate, mmol/L</td>
<td>24</td>
</tr>
<tr>
<td>March 25 at 5 AM</td>
<td>Normal acid-base balance</td>
</tr>
<tr>
<td>( \text{PaO}_2, \text{ mm Hg} )</td>
<td>97</td>
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<tr>
<td>Oxygen saturation, %</td>
<td>98</td>
</tr>
<tr>
<td>( \text{pH} )</td>
<td>7.45</td>
</tr>
<tr>
<td>( \text{PaCO}_2, \text{ mm Hg} )</td>
<td>35</td>
</tr>
<tr>
<td>Base excess</td>
<td>-1</td>
</tr>
<tr>
<td>( \text{HCO}_3, \text{ mmol/L} )</td>
<td>22</td>
</tr>
<tr>
<td>March 26 at 11:10 AM</td>
<td>Normal acid-base balance</td>
</tr>
<tr>
<td>( \text{PaO}_2, \text{ mm Hg} )</td>
<td>109</td>
</tr>
<tr>
<td>Oxygen saturation, %</td>
<td>98</td>
</tr>
<tr>
<td>( \text{pH} )</td>
<td>7.43</td>
</tr>
<tr>
<td>( \text{PaCO}_2, \text{ mm Hg} )</td>
<td>37</td>
</tr>
<tr>
<td>Base excess</td>
<td>0</td>
</tr>
<tr>
<td>Bicarbonate, mmol/L</td>
<td>24</td>
</tr>
</tbody>
</table>
**CE Test Instructions**

To receive CE credit for this test (ID C023), mark your answers on the form below, complete the enrollment information, and submit it with the $12 processing fee (payable in US funds) to the American Association of Critical-Care Nurses (AACN). Answer forms must be postmarked by June 1, 2004. Within 3 to 4 weeks of AACN receiving your test form, you will receive an AACN CE certificate.

This continuing education program is provided by AACN, which is accredited as a provider of continuing education in nursing by the American Nurses Credentialing Center’s Commission on Accreditation. AACN has been approved as a provider of continuing education by the State Boards of Nursing of Alabama (#ABNP0062), California (01036), Florida (#FBN2464), Iowa (#332), Louisiana (#ABN12), and Nevada. AACN programming meets the standards for most other states requiring mandatory continuing education credit for relicensure.

---

**CE Test Form**

**Assessing Tissue Oxygenation**

Objectives:
1. Describe the correlation of hemoglobin concentration to tissue oxygenation
2. Understand the role of laboratory assessment in evaluation of tissue oxygenation
3. Recognize the relationship of acid-base balance to tissue oxygenation

---

<table>
<thead>
<tr>
<th>Objective 1 was met</th>
<th>Agree</th>
<th>Neutral</th>
<th>Disagree</th>
</tr>
</thead>
<tbody>
<tr>
<td>Objective 2 was met</td>
<td></td>
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<tr>
<td>Objective 3 was met</td>
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<tr>
<td>The content was appropriate</td>
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<tr>
<td>My expectations were met</td>
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<tr>
<td>This method of CE is effective for this content</td>
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<td></td>
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</tbody>
</table>

Program evaluation

The level of difficulty of this test was:
- easy
- medium
- difficult

To complete this program, it took me ______ hours / minutes.

Mark your answers clearly in the appropriate box. There is only 1 correct answer. You may photocopy this form.

**Mail this entire page to** AACN, 101 Columbia, Aliso Viejo, CA 92656, (800) 899-2226

---
CE Test Questions
Assessing Tissue Oxygenation

1. Which one of the following statements is true regarding diffusion of gases in the lungs?
   a. PaO₂ is approximately 95 mm Hg.
   b. Partial pressure of oxygen at the point where it reaches the alveoli is approximately 130 mm Hg.
   c. The partial pressure of oxygen and carbon dioxide and their ability to diffuse into the capillary blood are dependent on one another.
   d. Partial pressure of carbon dioxide at the alveoli is approximately 30 mm Hg.

2. Which one of the following does not contribute to the decrease in the partial pressure of oxygen between inspired air and arterial blood?
   a. Dead space
   b. Water vapor in the airway
   c. Shunting of blood from the right side of the heart
   d. Partial pressure of carbon dioxide in arterial blood

3. Which one of the following best describes a patient in respiratory failure?
   a. 66-year-old man with congestive heart failure and a PaO₂ of 40 mm Hg
   b. 50-year-old woman with emphysema and a PaO₂ of 72 mm Hg
   c. 33-year-old asthmatic man with a PaO₂ of 55 mm Hg
   d. Both a and b

4. Which one of the following best describes pulmonary ventilation?
   a. Ability to maintain a PaO₂ greater than 50 mm Hg
   b. Inspiration and expiration of air (gases)
   c. Ability to maintain a PaO₂ greater than 30 mm Hg
   d. Dead space less than 30 mm Hg

5. Which one of the following statements is true regarding hemoglobin and its relationship to oxygen transport in the blood?
   a. Ninety-seven percent of oxygen transported in the blood is bound to hemoglobin.
   b. Each gram of hemoglobin can bind with approximately 1.34 mL of oxygen at full saturation.
   c. Oxygen transport can be reduced by either a hemoglobin level or oxygen saturation that is abnormal.
   d. All of the above

6. Which factor ultimately determines the amount of oxygen delivered at the tissue level?
   a. Serum potassium
   b. Cardiac output
   c. Core body temperature
   d. Peripheral vascular resistance

7. Which one of the following correlations is correct in accordance with the oxyhemoglobin dissociation curve?
   a. At a PaO₂ of 27 mm Hg, hemoglobin is 30% saturated.
   b. At a PaO₂ of 80 mm Hg, hemoglobin is 85% saturated.
   c. At a PaO₂ of 40 mm Hg, hemoglobin is 75% saturated.
   d. At a PaO₂ of 60 mm Hg, hemoglobin is 80% saturated.

8. Which one of the following does not affect the shift of the oxyhemoglobin dissociation curve to the right or left?
   a. Acidity of the blood
   b. Carbon dioxide tension in the blood
   c. Concentration of 2,3-diphosphoglycerate in red blood cells
   d. Serum sodium levels

9. Which one of the following factors influences the accuracy of oximetry, the measurement of oxygen saturation in the blood?
   a. Low perfusion states
   b. Hypothermia
   c. Oxygen saturations less than 80%
   d. All of the above

10. Which one of the following statements is true regarding the bicarbonate-carbonic acid buffer system?
    a. Normal blood pH is the result of a bicarbonate to carbon dioxide ratio of 20 to 1.
    b. Carbonic acid when dissolved in water becomes carbon dioxide.
    c. A ratio of less than 20:1 of bicarbonate to carbon dioxide indicates alkalosis.
    d. A ratio of more than 20:1 of bicarbonate to carbon dioxide indicates acidosis.

11. Which one of the following is not a likely cause of respiratory acidosis?
    a. Hypoventilation
    b. Damage to the respiratory center in the medulla
    c. Severe diarrhea
    d. Pulmonary damage or obstruction

12. Which one of the following is not a likely cause of metabolic alkalosis?
    a. Loss of hydrochloric acid secondary to vomiting
    b. Hyperventilation
    c. Excessive use of diuretics
    d. Intravenous administration of sodium bicarbonate
In the August 2002 issue, the article entitled “Met-hemoglobinemia: A Case Study” (2002;22:22-42), contained an error in Table 1. The methemoglobin level, reported as proportion of total hemoglobin, in Table 1 showed an incorrect reference range. The normal levels for methemoglobin are <0.01-0.02.

In the article entitled “Assessing Tissue Oxygenation” (June 2002;22:22-42), the reference to Figure 4 (p.27) was incorrectly placed in the middle of the sentence. The sentence that refers readers to Figure 4 should read: “The body has numerous compensatory mechanisms to correct an abnormal pH; however, if these mechanisms fail, cellular functions are impaired, and death will eventually result (Figure 4).” Moreover, this figure contained an error. The corrected figure is published below.

In Table 3 (p.28) of this article, the last parameter should be PaO₂, not PaCO₂.

In “Prone Positioning of Trauma Patients With Acute Respiratory Distress Syndrome and Open Abdominal Incisions” (June 2002;22:52-56), the company listed as the manufacturer of the CircOlectric bed was incorrect. This product is manufactured and distributed by Stryker Medical, Kalamazoo, Mich.

On page 53 of this article, nitrous oxide was incorrectly used instead of nitric oxide. The last sentence in the middle column and the third paragraph should read: “Other areas of research focus on the use of various techniques of ventilator support and the use of surfactant and inhaled nitric oxide in the treatment of ARDS.”

**Figure 4** Plasma pH. Normal plasma is slightly alkaline, with a pH of approximately 7.35 to 7.45. When the pH deviates outside this range in either direction, signs and symptoms of acid-base imbalance will occur. Without compensatory mechanisms or intervention, cellular dysfunction and death may occur.