Blood gas analysis is one of the major tools in assessing the respiratory status of the newborn. To adequately use this information, one must have a basic understanding of gas transportation and acid-base physiology. These topics are addressed in this chapter to provide a basis for applying these principles to the interpretation of neonatal blood gases. Common terminology is defined in Table 6-1.

**Transport of Oxygen and Carbon Dioxide**

**Oxygen**

Oxygen is used in aerobic reactions throughout the human body and is supplied to the tissues through the efforts of the respiratory and cardiovascular systems. The lungs are responsible for bringing an adequate supply of oxygen to the blood. Control of this process occurs mainly in response to the effect of carbon dioxide (CO₂) levels on receptors in the large arteries and the brain. At moderate to severe levels of hypoxemia, peripheral chemoreceptors take the dominant role in increasing ventilation, resulting in increased oxygen intake and lower than normal partial pressure of carbon dioxide in arterial blood (PaCO₂).¹

The cardiovascular system regulates the oxygen supply by altering cardiac output in response to the metabolic rate of peripheral tissues. Distribution of oxygen to specific tissues is determined by local metabolic activity. Oxygen transport is affected by:²

- partial pressure of oxygen in inspired air
- alveolar ventilation
- ventilation-to-perfusion matching
- arterial pH and temperature
- cardiac output
- blood volume
- hemoglobin
- hemoglobin’s affinity for oxygen

Oxygen transport to the tissues can be divided into a three-phase process, involving oxygen diffusion from the alveoli to the pulmonary capillaries (external respiration) (phase 1), gas transport in the bloodstream (phase 2), and diffusion of oxygen from the capillaries to the cells (internal respiration) (phase 3). The first two phases are discussed below.

Oxygen diffuses from the alveoli to the pulmonary capillaries. Oxygen enters the lung during inspiration and diffuses across the alveolar-capillary membrane, depending on the concentration gradient of oxygen in the alveolus and the capillary (Figure 6-1). Factors that interfere with oxygenation at this point include a decrease in minute ventilation, ventilation-perfusion mismatch, and alterations in the alveolar-capillary membrane.¹

Once in the blood, oxygen must be transported to the tissues. A small amount of oxygen (about 2–5 percent) is dissolved in the plasma; 95–98 percent is bound to hemoglobin. The total volume of oxygen carried in the blood is termed the arterial oxygen content and reflects both the oxygen combined with hemoglobin and the amount dissolved in the plasma.

The smaller, dissolved portion of oxygen is measured as the partial pressure of oxygen (PaO₂). Partial pressure refers to the force the gas exerts in the blood. Through simple diffusion, gases move from an area of higher pressure to an area of lower pressure. PaO₂ is the most
important factor in determining the amount of oxygen bound to hemoglobin. As PaO₂ increases, more oxygen diffuses into the red blood cells, where it combines with hemoglobin to form oxyhemoglobin.

Each hemoglobin molecule contains four atoms of iron and therefore can combine with four molecules of oxygen. When fully combined with oxygen, 1 g of hemoglobin carries 1.34 mL of oxygen.¹ The combination of oxygen and hemoglobin is expressed as oxygen saturation: a measure of the hemoglobin sites filled divided by the sites available (Figure 6-2).

Oxygen-hemoglobin saturation is plotted on an S-shaped curve known as the oxyhemoglobin dissociation curve (see Figure 4-10); this curve is based on adult hemoglobin at normal temperature and blood pH. Normal hemoglobin is 60 percent saturated at a PaO₂ of 30 mmHg and 90 percent saturated at a PaO₂ of 60 mmHg. At a PaO₂ of 90 mmHg, 95 percent of hemoglobin is saturated with oxygen.³

At the low PaO₂ values seen on the steep slope of the curve in Figure 4-10, a small increase in PaO₂ results in a large increase in oxygen saturation. Conversely, on the flat upper portion of the curve, a large increase in PaO₂ results in only a small increase in saturation. Hemoglobin cannot be more than 100 percent saturated, but PaO₂ can exceed 100 mmHg. At a PaO₂ of >100 mmHg, O₂ saturation cannot reflect PaO₂. For this reason, PaO₂ is a more sensitive indicator of high oxygen levels in the blood than is the measurement of saturation.⁴

Several factors change the affinity of hemoglobin for oxygen, shifting the curve to the left or to the right (see Figure 4-10). Alkalosis, hypocarbia, hypothermia, decreased amounts of 2,3-diphosphoglycerate (2,3-DPG), and the presence of fetal hemoglobin all shift the curve to the left.¹ An organic phosphate, 2,3-DPG is produced as a by-product of red cell metabolism. It binds with hemoglobin and decreases its oxygen affinity.

With a shift to the left, there is an increased affinity between oxygen and hemoglobin; therefore, hemoglobin more easily picks up oxygen and doesn’t release it until the PaO₂ level falls. This can impede oxygen release to the tissues, but enhances uptake of oxygen in the lungs.²

Acidosis, hypercapnia, hyperthermia, increased 2,3-DPG, and the presence of mature, or adult, hemoglobin move the curve to the right.¹ A shift to the right causes oxygen to bind less tightly to hemoglobin and to release from hemoglobin at higher levels of PaO₂, thereby enhancing oxygen unloading at the tissue level.²

Tip: An easy way to remember how shifts in the curve affect oxygen delivery is to think of it this way: left on the hemoglobin, right into the tissues.

**Carbon Dioxide**

Body cells produce CO₂ as a by-product of metabolism. Carbon dioxide diffuses from the cells down a concentration gradient, from areas of high partial pressure of CO₂ to areas of low partial pressure of CO₂. A small amount (8 percent) travels dissolved in the plasma; another small portion (2 percent) is transported in the plasma bound to proteins, forming carbamino compounds.¹ The remainder is transported within the red blood cells.

In red blood cells, about 10 percent of the CO₂ forms carbamino compounds by combining with amino acids contained in the globin portion of the hemoglobin. The remaining 80 percent is acted upon by carbonic anhydrase, which combines carbon dioxide and water to form carbonic acid (H₂CO₃) and then undergoes hydrolysis and forms bicarbonate (HCO₃⁻) and hydrogen ions.
The hydrogen ions are buffered by desaturated hemoglobin, and $\text{HCO}_3^-$ is transported out of the erythrocytes into the plasma (Figure 6-3). As oxygen is unloaded from hemoglobin along the tissue capillaries, more CO$_2$ can be transported because of the enhanced ability of deoxygenated hemoglobin to form carbamino compounds.

### Acid-Base Homeostasis

Normal function of the body’s cells depends on maintaining a biochemical balance within a narrow range of free H$^+$ concentration. Free H$^+$ is constantly released in the body as waste products from the metabolism of proteins and fats. The measurement of free H$^+$ present in the body in very low concentrations is expressed as pH, which is the negative logarithm of the H$^+$ concentration—that is, the more H$^+$ present in a solution, the lower the pH or the more acidic the solution. Conversely, the fewer H$^+$ present, the higher the pH or the more alkaline the solution. A pH of 7 is neutral, that is, neither alkaline nor acidic. A pH range of 7.35–7.45 is normal for cellular reactions in the human body.

Most of the acids formed by metabolism come from the interaction of carbon dioxide and water, which forms H$_2$CO$_3$, as illustrated in the following equation:

$$ \text{CO}_2 + \text{H}_2\text{O} \rightarrow \text{H}_2\text{CO}_3 \rightarrow \text{H}^+ + \text{HCO}_3^- $$

Carbonic anhydrase, an enzyme, accelerates this reaction. Carbonic acid is referred to as a volatile acid because it is transformed back into CO$_2$ in the lungs and exhaled, allowing the respiratory system to control the majority of acid-base regulation. Sulfurous, phosphoric, and other organic acids are nonvolatile acids that are eliminated in the renal tubules.

Changes in CO$_2$ affect pH by altering the amount of HCO$_3^-$ in the body. Changes in pH caused by changes in CO$_2$ tension are therefore termed respiratory. Hyperventilation causes a lower partial pressure of carbon dioxide (PCO$_2$), lower H$_2$CO$_3$ concentration, and increased pH. Hypoventilation has the opposite effect. Remember that concentrations of CO$_2$, H$_2$CO$_3$, and H$^+$ move in the opposite direction of pH:

$$ \uparrow \text{PCO}_2 \rightarrow \uparrow \text{H}_2\text{CO}_3 \rightarrow \uparrow \text{H}^+ \rightarrow \downarrow \text{pH} $$

$$ \downarrow \text{PCO}_2 \rightarrow \downarrow \text{H}_2\text{CO}_3 \rightarrow \downarrow \text{H}^+ \rightarrow \uparrow \text{pH} $$

Metabolic acids are formed in the body during the metabolism of protein, anaerobic metabolism resulting in the formation of lactic acid and keto acids, which are formed when glucose is unavailable as a fuel source. The kidneys provide the most important route by which metabolic acids can be excreted and buffered.
Hydrogen excretion takes place through the active exchange of sodium ions (Na⁺) for H⁺. The kidneys are also responsible for plasma levels of HCO₃⁻, the most important buffer of H⁺ (discussion follows). Therefore, pH changes that occur because of changes in bicarbonate concentrations are termed metabolic.

Tip: Remember the following equations:

\[ \begin{align*}
\text{up } HCO_3^- & \rightarrow \text{up } pH \\
\text{down } HCO_3^- & \rightarrow \text{down } pH
\end{align*} \]

**The Henderson-Hasselbalch Equation**

The concentration of H⁺ resulting from the dissociation of H₂CO₃ is determined by an interrelationship between bases, buffers, and blood acids. In blood gas analysis, the Henderson-Hasselbalch equation is used to calculate HCO₃⁻ if pH and PCO₂ are known. This equation describes the fixed relationship between H₂CO₃, HCO₃⁻, and H₂CO₃ concentration. When the equation is used in the clinical situation, H₂CO₃ is replaced by the amount of dissolved CO₂ in the blood, as shown in the following equation:

\[ pH = pK + \log \left( \frac{[HCO_3^-]}{s \times PCO_2} \right) \]

in which pK (a constant) = 6.1 and s (solubility of CO₂) = 0.0301. It is important to remember that this is a calculated bicarbonate value, not one that is measured.

**Buffer Systems**

Buffer systems are a combination of a weak acid and a strong base, which work by accepting or releasing hydrogen ions to maintain acid-base balance. The body has three primary buffers: plasma proteins, hemoglobin, and bicarbonate. Of these, HCO₃⁻ is the most important system and is regulated by the kidney. Bicarbonate ions are formed from the hydroxylation of CO₂ by water inside red blood cells, catalyzed by carbonic anhydrase. Once formed, HCO₃⁻ enters the plasma in exchange for chloride ions (Cl⁻) through an active transport mechanism known as the chloride shift, which is depicted in Figure 6-4. In blood gas analysis, bicarbonate and base deficit/excess are used in determining the nonrespiratory portion of the acid-base equation. Some centers provide both values in blood gas results; others report either HCO₃⁻ levels or base excess/deficit.

Bicarbonate is expressed in milliequivalents per liter (mEq/liter). The normal range is 22–26 mEq/liter. Base excess or base deficit is reported in mEq/liter, with a normal range of –4 to +4. Negative values indicate a deficiency of base or an excess of acid (metabolic acidosis); positive values indicate alkalosis. Clinically, the base excess or deficit is calculated from the Siggaard-Andersen nomogram (Appendix A).

The kidney has several mechanisms for controlling excretion of H⁺ and retention of HCO₃⁻. These are illustrated in Figure 6-5 and include the following:

- Resorption of filtered HCO₃⁻ (H⁺ is excreted in the renal tubular cells in exchange for Na⁺, which combines with HCO₃⁻ to form sodium bicarbonate, which enters the blood.)
- Excretion of acids (One example is phosphoric acid, which is formed from the combination of H⁺ and hydrogen phosphate.)
- Formation of ammonia (NH₃) (Elevated acid levels in the body result in the formation of NH₃, which combines with H⁺ to form ammonium, which is excreted in the urine.)
An acid-base ratio of 1:20—that is, 1 part carbonic acid to 20 parts bicarbonate—is needed to maintain a pH of 7.4. It is the ratio of PCO₂ to HCO₃⁻ that determines the pH; therefore, abnormalities can be compensated for by adding or subtracting on one side of the scale or the other. This is demonstrated in Figure 6-6.

If buffers cannot normalize the pH, compensatory mechanisms are activated. Healthy lungs are able to compensate for acid-base imbalances within minutes by altering the respiratory rate or volume to regulate CO₂ levels. The kidneys have a slower but more sustained response, either retaining or excreting HCO₃⁻ in response to changes in blood pH. The kidneys are also able to excrete additional H⁺ in combination with phosphate and ammonia. Renal compensatory responses are outlined in Table 6-2. In the neonate, compensatory mechanisms may be limited by respiratory disease and the inability of the immature neonatal kidney to conserve HCO₃⁻.

**Disorders of Acid-Base Balance**

Classification and interpretation of blood gas values are based on a set of normal values, such as the ones shown in Table 6-3. Because of immaturity and the presence of fetal hemoglobin, values for the term and preterm infant differ from those of the adult. In addition, the exact values accepted as normal vary from institution to institution and in the literature.

The terms applied to acid-base disorders can be a source of confusion. *Acidemia* and *alkalemia* refer to measurements of blood pH; *acidosis* and *alkalosis* refer to underlying pathologic processes.

As previously discussed, a blood pH < 7.35 is said to be acidic; a pH > 7.45 is alkaline. The PCO₂ and HCO₃⁻ levels, respectively, determine the respiratory and metabolic contributions to the acid-base equation. During a disturbance of acid-base balance, the body can attempt to return the pH to a normal level in one of two ways:

1. **Correction** occurs when the body alters the component responsible for the abnormality. If CO₂ levels are increased, for example, the body attempts to correct the problem by increasing the excretion of it. The neonate is often unable to correct an acid-base disturbance because of the limitations of immaturity (such as diminished response of chemoreceptors and decreased lung compliance).

2. **Compensation** occurs when the body alters the component not responsible for the abnormality. If the body is unable to correct the problem by altering the components, it will alter the compensatory mechanisms to maintain a normal pH.

**Table 6-3**

<table>
<thead>
<tr>
<th>Value</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.35–7.45</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>35–45 mmHg</td>
</tr>
<tr>
<td>PaO₂ term infant</td>
<td>50–70 mmHg</td>
</tr>
<tr>
<td>PaO₂ preterm infant</td>
<td>45–65 mmHg</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>22–26 mEq/liter</td>
</tr>
<tr>
<td>Base excess</td>
<td>−2 to +2 mEq/liter</td>
</tr>
<tr>
<td>O₂ saturation</td>
<td>92–94%</td>
</tr>
</tbody>
</table>

TABLE 6-4
Causes of Acid-Base Imbalances in Neonates

<table>
<thead>
<tr>
<th>Respiratory Acidosis</th>
<th>Metabolic Alkalosis</th>
<th>Respiratory Alkalosis</th>
<th>Metabolic Acidosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypoventilation</td>
<td>Gain of bases</td>
<td>Hyperventilation</td>
<td>Increased acid formation</td>
</tr>
<tr>
<td>Asphyxia</td>
<td>Bicarbonate admin.</td>
<td>Iatrogenic mechanical hyperventilation</td>
<td>Hyperpolarization</td>
</tr>
<tr>
<td>Apnea</td>
<td>Acetate admin.</td>
<td>Central nervous system</td>
<td>Hyperalimentation</td>
</tr>
<tr>
<td>Upper airway obstruction</td>
<td>Loss of acids</td>
<td>response to:</td>
<td>Loss of bases</td>
</tr>
<tr>
<td>Decreased lung tissue</td>
<td>Vomiting, gastric suctioning</td>
<td>Hyperp.</td>
<td>Diarrhea</td>
</tr>
<tr>
<td>Respiratory distress syndrome</td>
<td>Loss of acids</td>
<td></td>
<td>Renal tubular acidosis</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>H2CO3 reabsorption</td>
<td></td>
<td>Acetazolamide admin.</td>
</tr>
<tr>
<td>Pulmonary interstitial emphysema</td>
<td>Hypokalemia, hypochloremia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ventilation-to-perfusion mismatching</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meconium aspiration</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pneumonia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Transient tachypnea</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Persistent pulmonary hypertension</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>of the newborn</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac disease</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2. Compensation occurs when the body normalizes the pH by altering the blood gas component not responsible for the abnormality. If metabolic acidosis is present, for example, the lungs will excrete more CO2 to normalize the pH. If respiratory acidosis is present, the kidneys will excrete more H+ and conserve HCO3− in an attempt to compensate for the respiratory problem. Compensation is also limited in the neonate because of immaturity.

Respiratory Acidosis
Respiratory acidosis results from the formation of excess H2CO3 as a result of increased PCO2: (↑ PCO2 → H2CO3 → ↑ H+ → ↓ pH). Blood gas findings are a low pH, high PCO2, and normal bicarbonate levels.

Respiratory acidosis is caused by insufficient alveolar ventilation secondary to lung disease. Compensation occurs over three to four days as the kidneys increase the rates of H+ excretion and HCO3− reabsorption. Compensated respiratory acidosis is characterized by a low-normal pH (7.35–7.40), with increased CO2 and HCO3− levels as a result of the kidney retaining HCO3− to compensate for elevated CO2 levels.

Metabolic Alkalosis
Metabolic alkalosis results from an excess concentration of HCO3− in the extracellular fluid. Blood gas findings are high pH, high HCO3− level, and normal PCO2.

Metabolic alkalosis is caused by problems leading to increased loss of acids, such as severe vomiting, gastric suctioning, or increased retention or intake of bases, such as occurs with excessive administration of sodium bicarbonate. The lungs compensate by retaining CO2 through hypoventilation. The pH will be high normal (7.40–7.45), with high levels of CO2 and HCO3− ions.

Table 6-4 lists common causes of acid-base disturbances in the neonate.

Blood Gas Sampling
Blood gas analysis provides the basis for determining the adequacy of alveolar ventilation and perfusion. The accuracy of this test depends a great deal on the skill and knowledge of both the person drawing the sample and the person providing the analysis. It is therefore crucial that those performing and interpreting this
test understand appropriate techniques and potential sources of error.

Regardless of the type of sample obtained, attention should be given to the following factors:

1. **Infection control/universal precautions.** All types of blood gas sampling carry the risk of transmission of infection to the infant through the introduction of organisms into the bloodstream. In addition, the potential exposure of the clinician to the infant’s blood demands the use of appropriate precautions.

2. **Bleeding disorders.** The potential for bruising and excessive bleeding should be kept in mind, particularly if an arterial puncture is being considered.

3. **Steady state.** Ideally, blood gases should measure the infant’s condition in a state of equilibrium. After changing ventilator settings or disturbing the infant, a period of 20–30 minutes should be allowed for arterial blood to reach a steady state.1 The length of time needed to reach steady state varies from infant to infant.

**INTRAPARTUM TESTING**

**Fetal Scalp Sampling**

Scalp blood pH sampling in the fetus has been shown to be a useful tool for evaluating fetal well-being in the presence of suspect fetal heart tracings.8,9 Values are similar to umbilical cord gases obtained at delivery (Table 6-5). The accuracy of fetal scalp pH is diminished in the presence of scalp edema or caput succedaneum.10

A pH value of 7.25 or greater is classified as normal. Values of 7.20–7.25 are borderline and should be repeated in 30 minutes, and those below 7.20 are considered indicative of fetal acidosis.11 Despite its clinical value, scalp sampling is not widely practiced because it is technically difficult and invasive for both the mother and the fetus.10

Serum lactate has been used in research settings as a method of evaluating fetal well-being. The development of hand-held microvolume devices to measure blood lactate levels has made the use of lactate levels a promising alternative to fetal scalp pH testing. A randomized controlled trial comparing fetal scalp pH to fetal lactate levels found no difference in the predictive value of the two tests, but noted that measuring serum lactate provided quicker results and fewer sampling errors than did measuring scalp pH.12 A more recent randomized controlled trial again found there was no significant difference between lactate and pH analysis in predicting acidemia at birth.13

**Continuous Intrapartum Fetal Pulse Oximetry**

The intermittent nature of fetal scalp sampling combined with the technical difficulties in obtaining accurate specimens has prompted the development of techniques to more continuously monitor fetal well-being. As an adjunct to fetal heart rate monitoring, it is now possible to continuously monitor fetal oxygenation using pulse oximetry. Studies evaluating this technology have largely focused on whether fetal oximetry can clarify the condition of the fetus in the face of a nonreassuring fetal heart rate tracing, potentially reducing the rate of unnecessary cesarean section deliveries.14

**Cord Blood Gases**

Cord blood gases provide an accurate assessment of the fetus’s condition at the time of delivery, but do not predict long-term outcomes.15 Table 6-5 lists normal umbilical cord blood gas values.

Blood from the umbilical artery represents fetal status because this is blood returning from the fetus. Umbilical venous blood provides a measure of placental status. For example, in cases of cord compression, the placenta is functioning normally. Therefore, the venous gas is normal, and the arterial gas reflects a lower pH and increased PCO2. With decreased placental perfusion, the pH in the venous gas drops, as does the arterial pH.9

**ARTERIAL SAMPLING**

Arterial blood can be obtained from the neonate either from an indwelling line or through intermittent sampling of a peripheral artery. The choice of sample site depends on the clinical situation. An indwelling arterial catheter should be placed when it is anticipated that the neonate will require frequent arterial blood sampling. Many criteria are used to determine the need for an indwelling line: These include gestational age, disease process, and the percentage of oxygen required.

---

**TABLE 6-5 Normal Fetal Blood Gas Values**

<table>
<thead>
<tr>
<th>Value</th>
<th>Umbilical Artery</th>
<th>Umbilical Vein</th>
<th>Fetal Scalp</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>≥7.20</td>
<td>≥7.25</td>
<td>≥7.25</td>
</tr>
<tr>
<td>PCO2 (mmHg)</td>
<td>40–50</td>
<td>≤40</td>
<td>≤50</td>
</tr>
<tr>
<td>PO2 (mmHg)</td>
<td>18 ± 2</td>
<td>30 ± 2</td>
<td>≥20</td>
</tr>
<tr>
<td>Base excess (mEq/liter)</td>
<td>0 to −10</td>
<td>0 to −5</td>
<td>&lt;−6</td>
</tr>
</tbody>
</table>

by the neonate. Common sites for neonatal arterial sampling include the following:

1. **Umbilical artery.** The umbilical artery is usually readily accessible for line placement for three to four days and is often usable for up to two weeks after birth. Polyvinyl chloride (PVC) or silastic catheters are available in a variety of sizes, with 3.5 and 5.0 French being the most commonly used. A Cochrane Review of umbilical catheter materials suggests that catheters constructed of silastic material may be less thrombogenic than PVC catheters. The umbilical arterial catheter (UAC) should be positioned either between T6 and T10 or between L3 and L4. The distance for insertion of a high line can be estimated by measuring the distance between the infant’s umbilicus and shoulder tip and adding 2 cm. The following formula, based on the infant’s birth weight, can also be used:

\[ 3 \times \text{Weight (in kg)} + 9 \]

2. **Peripheral arterial lines** (radial, posterior tibial). These peripheral arteries can be accessed at any time beyond delivery with a small-gauge intravascular catheter. Risks for these lines include arteriospasms, tissue necrosis, thrombus formation, infection, and hemorrhage. UAC complications include hemorrhage, ischemic organ damage, infection, and thrombus formation. Other UAC complications include hemorrhage, ischemic organ damage, infection, and thrombus formation. UAC placement is unsuccessful in 10–15 percent of infants.

3. **Intermittent arterial samples.** Intermittent samples can be obtained from the radial, posterior tibial, or dorsalis pedis arteries. The femoral and brachial arteries are not recommended for sampling because of poor collateral circulation, close proximity of nerves, and the risk of complications such as nerve damage or circulatory impairment. See Figure 4-12 for a depiction of the radial site.

4. **Continuous blood gas monitoring.** Technological developments over the past 10–15 years have resulted in the development of devices that allow continuous monitoring of arterial blood gases, usually through an indwelling UAC. Studies evaluating these devices have generally been positive, demonstrating good correlation with intermittently drawn arterial gases. Of the values measured, pH, partial pressure of oxygen (PO2), and PCO2, PO2...
values were found to be the least accurate, but still within acceptable accuracy limits, in one study.27 See Chapter 4 for additional information on blood gas sampling techniques and considerations.

**Capillary Sampling**

Capillary blood can be “arterialized” by warming the skin to increase local blood flow. Samples can then be obtained from the outer aspects of the heel (see Figure 4-13) or from the side of a finger or toe. Transitional events during the first few hours of life and poor perfusion at any time diminish the accuracy of capillary gas measurements. Opinion is mixed as to the reliability of capillary blood gas values as estimators for arterial values. Some studies have found good correlation between arterial and capillary pH and PCO2.28–32 Others question the validity of capillary PCO2 values and suggest caution when basing treatment decisions on capillary blood gas values.33 Escalante-Kanashiro and Tantalean-Da-Fieno examined 75 paired arterial and capillary samples and found good correlation between pH (0.85), PCO2 (0.86), and oxygen (0.65). Neither tissue perfusion nor temperature significantly affected correlations, but the presence of hypotension did.34

Other research has also demonstrated a poor correlation between PO2 and PaO2.6,35 Given that finding, treatment decisions are not normally based on capillary PO2 alone.

**Errors in Blood Gas Measurement**

In examining blood gases, the clinician should be aware of potential sources of error that can affect the quality of the results:6,36,37

- **Temperature.** Most blood gas machines report results for 37°C (98.6°F). Hypothermia or hyperthermia can alter true arterial gas values.
- **Hemoglobin.** Calculated oxygen saturations are based on adult hemoglobin, not on fetal or mixed hemoglobins.
- **Dilution.** Heparin in a gas sample lowers the PCO2 and increases the base deficit without altering the pH.
- **Air bubbles.** Room air has a PCO2 close to 0 and a PO2 of 150 mmHg. Therefore, air bubbles in the sample decrease the PaCO2 and increase the PaO2 unless the PaO2 is >150 mmHg.

**Interpreting Blood Gases**

The blood gas report contains many pieces of information that must be examined and interpreted. Although oxygenation and acid-base status are interrelated, it is usually easier to consider these separately. The order in which to evaluate these parameters is a matter of personal preference, but it is important to use an organized, step-by-step approach to simplify the process and ensure that nothing is overlooked.

The following steps offer a systematic way of evaluating neonatal blood gases. Figure 6-7 illustrates the first five of these steps and is a useful way of visualizing the decision-making process.

**Step 1:** Assess the pH. A pH > 7.45 is alkalotic, and a pH < 7.35 is acidotic. When pH and either PCO2 or HCO3− are abnormal, the abnormal factor defines the origin of the imbalance. A normal pH should be further evaluated because compensation can normalize the pH while primary acid-base imbalances are present.

**Step 2:** Assess the respiratory component. A PCO2 > 45 mmHg lowers the pH. A PCO2 < 35 mmHg raises the pH.

**Step 3:** Assess the metabolic component. An HCO3− value < 22 mEq/liter lowers the pH. An HCO3− value > 26 mEq/liter raises the pH.

**Tip:** For primary abnormalities, remember the following:

- Abnormal pH and PCO2 = respiratory alkalosis (PaCO2 < 35 mmHg) or acidosis (PaCO2 > 45 mmHg)
- Abnormal pH and HCO3− = metabolic acidosis (HCO3− < 22 mEq/liter) or alkalosis (HCO3− > 26 mEq/liter)

Mixed problems: In some cases, abnormalities in both the metabolic and the respiratory systems may be present. This is more common in acidosis than in alkalosis. If both PCO2 and HCO3− are abnormal, consider the patient’s history to determine which problem came first or is more severe.
Step 4: Assess the compensation status. When the pH is abnormal, with one of the acid-base components (PCO₂ or HCO₃⁻) being abnormal and the other normal, the gas is said to be uncompensated.1 When both acid-base parameters are abnormal in opposite directions, the body is beginning to compensate for the primary abnormality. When the pH reaches the normal range, the gas is compensated.

When the pH is normal and respiratory and metabolic parameters are abnormal in opposite directions (e.g., one is acidic and one alkalic), it may be unclear which is the primary abnormality. Because the body does not normally compensate beyond the minimum acceptable pH, the pH usually leans in the direction of the primary problem. A pH of <7.4 in a compensated gas would result from a primary acidosis with an alkalic compensation, and a pH >7.4 would result from a primary alkalosis with an acidic compensation.1 Table 6-6 outlines the common findings in compensated gases.

Step 5: Complete the acid-base classification. Add the information from the blood gas analysis to the clinical assessment of the infant’s condition and knowledge of the pathophysiology of the infant’s disease process to determine a course of action. Remember, a blood gas result that is abnormal on paper may be quite acceptable given the infant’s gestational age or disease process. For example, a pH as low as 7.25 may be considered acceptable in a preterm infant. A pH of 7.45 may be desirable if a term infant has persistent pulmonary hypertension of the newborn (PPHN).

Step 6: Evaluate the oxygenation. Three pieces of information are routinely used to determine oxygenation: PaO₂, oxygen saturation, and the presence of cyanosis. The arterial blood gas value provides information about the pulmonary component of oxygenation, specifically the PaO₂.

- **PaO₂.** Normal values for PaO₂ in the term infant are 50–70 mmHg; in the preterm infant, they are 45–65 mmHg (fetal hemoglobin results in higher saturations at lower oxygen levels).6

Hypoxia (inadequate tissue oxygen supply) may result from a number of factors, including heart failure, anemia, abnormal hemoglobin affinity for oxygen, and a decreased PaO₂. Hypoxemia (low PaO₂) results from lung disease or cyanotic congenital heart disease.6 A PaO₂ value <45–50 mmHg is associated with vasoconstriction of pulmonary vasculature and vasodilation of the ductus arteriosus.38 Low PaO₂ levels are implicated in the etiology of PPHN. Hyperoxemia (PaO₂ >90–100 mmHg) should also be avoided, especially in the preterm infant, for whom high levels of oxygen in the blood are associated with retinal injury.38

Note: When interpreting neonatal PaO₂ values, it may be important to identify whether the sample is preductal or postductal in origin. (See Chapter 2 for a discussion of preductal and postductal gases.)

- **Oxygen saturation.** Although oxygen saturation levels may be reported as part of an arterial blood gas result, they are more commonly obtained from an oximeter. Saturations reported as part of the blood gas result may be measured or calculated from a nomogram. Calculated saturations predict saturation based on the pH and PaO₂ and have limited clinical value.1 Measured oxygen saturation is usually a good indicator of reduced arterial oxygen content. Because of the shape of the oxyhemoglobin dissociation curve, saturation is not a good indicator of hyperoxemia or pulmonary deterioration.1 PaO₂ readings of >100 mmHg occur on the flat upper portion of the curve; therefore, there is little change in oxygen saturation.

Concern regarding the incidence of retinopathy of prematurity (ROP) has prompted recommendations to maintain oxygen saturation at lower levels than those previously accepted. Tin and associates found that infants whose oxygen saturation (SpO₂) levels were maintained at between 70 and 90 percent were four times less likely to develop ROP requiring treatment than those given oxygen to maintain an SpO₂ of 88–98 percent.39 This is substantiated by the results of a survey of 142 U.S. NICUs that demonstrated that beyond the first two weeks of life, an SpO₂ of <93 percent was associated with a significant decrease in the incidence of Stage 3 or greater ROP and the need for retinal ablation.40 Chow and colleagues used research data to craft a practice guideline for their institution recommending that oxygen saturation levels be maintained between 85 and 93 percent. Implementation of this policy along with an education program for staff significantly decreased the rate of ROP observed in their NICU.41

- **Cyanosis.** Peripheral cyanosis is defined as a blue discoloration of the skin. It may be difficult to assess in dark-skinned infants. Central cyanosis is a blue discoloration of the mucous membranes and is a more reliable indicator of hypoxemia than peripheral cyanosis.
Cyanosis results from an increased amount of uncombined, or desaturated, hemoglobin. It is normally seen when the quantity of desaturated hemoglobin in the capillaries exceeds 5 g/100 mL. This corresponds to a PaO₂ of about 40 mmHg. Because cyanosis depends on the quantity of desaturated hemoglobin, an anemic infant may not look cyanotic despite having a low PO₂, and an infant with polycythemia may appear cyanotic despite adequate oxygenation because of an increase in total hemoglobin.

Step 7: Formulate a plan. By following steps 1–6, the nurse can interpret blood gas values. A plan should then be formulated to accomplish the following:

- **Correct acid-base imbalances.** Correction of acid-base imbalances is achieved, where possible, through treatment of the underlying cause. Treatment suggestions for the four primary acid-base imbalances follow.
  
  - **Respiratory acidosis.** Increase alveolar ventilation to remove CO₂ by applying nasal continuous positive airway pressure (CPAP) or mechanical ventilation. For infants already on mechanical ventilation, increase the tidal volume, rate, peak inspiratory pressure, or positive end-expiratory pressure (PEEP) to facilitate CO₂ removal (see Chapter 7). Sodium bicarbonate is not recommended for treating respiratory acidosis because it reacts with acids to form CO₂.
  
  - **Respiratory alkalosis.** For mechanically ventilated infants, reduce the tidal volume, rate, or pressure on the ventilator.
  
  - **Metabolic acidosis.** Where possible, treat the cause of the acidosis (e.g., correct hypovolemia, decrease the protein load in total parenteral nutrition). If the acidosis is severe, sodium bicarbonate can be administered at a dose of 2 mEq/kg or according to the following formula:

\[
\text{Base deficit} \times \text{Weight (in kg)} \times 0.3
\]

The amount of HCO₃⁻ calculated by this formula should theoretically correct half of the base deficit and should be administered slowly over 30–60 minutes. Fluid replacement may also be of benefit in treating metabolic acidosis by helping the infant metabolize lactic acid.°

**Note:** After fluid replacement, the infant may show a transient deterioration in acid-base status resulting from improved transport of acid from the peripheral to the central circulation.

- **Metabolic alkalosis.** Treat the cause by removing acetate from intravenous fluids, reducing diuretic doses, and replacing lost gastrointestinal secretions. Treat hyponatremia, hypokalemia, and hypochloremia.

- **Correct hypoxemia.** Hypoxemia secondary to ventilation-to-perfusion mismatching can be improved by administering supplemental oxygen. In addition, oxygenation can be improved by increasing the mean airway pressure in an infant on mechanical ventilation. Chapter 7 discusses mean airway pressure.

### Case Studies

The following case studies illustrate how the steps for interpreting blood gases might be applied for various infants.

**Case 1**

An infant born at 31 weeks gestation is two hours old with the following physical findings: respiratory rate 94 breaths per minute, heart rate 162 beats per minute, temperature 36.5°C (97.7°F), and grunting with moderate retractions.

Capillary blood gas results are as follows:

- pH 7.30
- PCO₂ 56 mmHg
- HCO₃⁻ 26 mEq/liter
- PO₂ 40 mmHg

The steps for analysis indicate the following:

1. The pH is low, indicating acidosis.
2. The PCO₂ is high, indicating a respiratory problem.
3. The metabolic component (HCO₃⁻) is normal.
4. No compensation is present (pH is not normal).
5. This is uncompensated respiratory acidosis.
6. Oxygenation is adequate.
7. Treatment should be aimed at improving alveolar ventilation. Depending on the infant’s clinical status and chest x-ray findings, treatment could consist of nasal CPAP or intermittent positive pressure ventilation.

**Case 2**

A 26-week-gestational-age infant is receiving total parenteral nutrition (TPN) with 3.5 g/kg of protein and 15 g/kg of glucose. The infant’s urine output is 7 mL/kg/hour, and the baby’s weight has dropped 30 g over the past 24 hours. Capillary refill is sluggish.

Capillary blood gas results are as follows:

- pH 7.24

<table>
<thead>
<tr>
<th>Base deficit ( \times ) Weight (in kg) ( \times 0.3 )</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium bicarbonate</td>
<td>2 mEq/kg</td>
</tr>
<tr>
<td>Sodium chloride</td>
<td>1 mEq/kg</td>
</tr>
<tr>
<td>Sodium acetate</td>
<td>1 mEq/kg</td>
</tr>
<tr>
<td>Sodium lactate</td>
<td>1 mEq/kg</td>
</tr>
</tbody>
</table>

Note: After fluid replacement, the infant may show a transient deterioration in acid-base status resulting from improved transport of acid from the peripheral to the central circulation.
Blood Gas Analysis

- PCO₂ 36 mmHg
- HCO₃⁻ 15 mEq/liter
- PO₂ 50 mmHg

The steps for analysis indicate the following:
1. The pH is low, indicating acidosis.
2. The respiratory component (PCO₂) is normal.
3. The HCO₃⁻ is low, indicating a metabolic problem.
4. There is no compensation (pH is not normal).
5. This is uncompensated metabolic acidosis.
6. Oxygenation is adequate.
7. Consider giving volume to compensate for hypovolemia and to help metabolize lactic acids, or reduce the amount of protein in the TPN feedings to lower the metabolic acid load.

CASE 3

A 28-week-gestational-age infant is on mechanical ventilation for respiratory distress syndrome. Settings are a rate of 40 breaths per minute, tidal volume of 5 mL/kg, PEEP +4, and fractional concentration of oxygen in inspired gas (FiO₂) 0.50.

Arterial blood gas results are as follows:
- pH 7.48
- PaCO₂ 27 mmHg
- HCO₃⁻ 22 mEq/liter
- PaO₂ 95 mmHg

The steps for analysis indicate the following:
1. The pH is high and shows an alkalemia.
2. The PCO₂ is low, indicating respiratory alkalosis.
3. The metabolic component (HCO₃⁻) is normal.
4. There is no compensation (pH is not normal).
5. This is uncompensated respiratory alkalosis.
6. The PO₂ is too high.
7. Reduce alveolar ventilation. Assess the infant’s chest expansion and spontaneous respirations to determine whether the tidal volume or the ventilator rate should be lowered. Reduce the FiO₂, ensuring that the oxygen saturation remains within the desired range.

CASE 4

A three-week-old infant underwent bowel surgery three days ago. On continuous gastric suction, the infant is receiving TPN with sodium and potassium acetate.

Capillary blood gas results are as follows:
- pH 7.51
- PCO₂ 43 mmHg
- HCO₃⁻ 34 mEq/liter
- PO₂ 52 mmHg

The steps for analysis indicate the following:
1. The pH is high, showing an alkalemia.
2. The respiratory component (PCO₂) is high normal.
3. The HCO₃⁻ is high, leading to metabolic alkalosis.
4. There is no compensation.
5. This is uncompensated metabolic alkalosis.
6. The PO₂ is adequate.
7. This infant’s kidneys have become efficient at conserving HCO₃⁻ and excreting H⁺, so treatment is not necessary. Keep in mind that further changes in the infant’s condition (atelectasis, pneumonia, or metabolic causes of acidosis) will likely exceed the infant’s ability to compensate and result in acidosis.

CASE 5

An infant born at 26 weeks gestational age is now three weeks old and receiving mechanical ventilation for chronic lung disease. The infant is on full nasogastric feedings.

Capillary blood gas results are as follows:
- pH 7.37
- PCO₂ 49 mmHg
- HCO₃⁻ 34 mEq/liter
- PO₂ 52 mmHg

The steps for analysis indicate the following:
1. The pH is normal.
2. The PCO₂ is high, suggesting respiratory acidosis.
3. The HCO₃⁻ is high, suggesting a metabolic alkalosis.
4. The pH is normal with abnormal CO₂ and HCO₃⁻; therefore, there is compensation. The pH is low normal; therefore, it is compensated acidosis. The high HCO₃⁻ does not fit with acidosis, but the high PCO₂ does.
5. This is compensated respiratory acidosis that fits with the clinical history of chronic lung disease.
6. Oxygen levels are satisfactory.
7. This infant’s kidneys have become efficient at conserving HCO₃⁻ and excreting H⁺, so treatment is not necessary. Keep in mind that further changes in the infant’s condition (atelectasis, pneumonia, or metabolic causes of acidosis) will likely exceed the infant’s ability to compensate and result in acidosis.

CASE 6

A term infant, with Apgar scores of 4 at one minute and 6 at five minutes and born through thick meconium, is pale, with retraction and grunting respirations. Temperature is 35.8°C (96.4°F).

Capillary blood gas results are as follows:
- pH 7.25
- PCO₂ 49 mmHg
- HCO₃⁻ 16 mEq/liter
- PO₂ 35 mmHg

The steps for analysis indicate the following:
1. The pH is low, indicating an acidosis.
2. The PCO₂ is high, suggesting a respiratory acidosis.
3. The HCO₃⁻ is low, suggesting a metabolic acidosis.
4. No compensation is present.
5. This is a mixed respiratory and metabolic acidosis that is uncompensated.
6. The PO₂ is low.
7. Warm the infant slowly. Improve the alveolar ventilation, and provide supplemental oxygen. Do not administer HCO₃⁻ unless ventilation is improved.

**Summary**

Interpretation of a blood gas requires a systematic approach based on an understanding of the physiology of gas transport and acid-base balance. Such an approach permits timely and appropriate interventions aimed at providing optimal care for the compromised infant.

**References**
