13 ARTERIAL BLOOD GASES AND ACID–BASE BALANCE

Jeffrey F. Barletta

OBJECTIVES

After completing this chapter, the reader should be able to

- Discuss the chemistry associated with acid–base balance
- Describe the components of an arterial blood gas analysis and their contribution to acid–base physiology
- Compare and contrast the physiologic approach to the Stewart approach for acid–base balance
- Describe the methods used by the human body to maintain acid–base balance
- Evaluate a patient's acid–base status and identify common causes given the clinical presentation, laboratory data, and arterial blood gas data
- List the four simple acid–base disorders, their accompanying laboratory test results, and possible causes
- Describe how the anion gap can be used to determine the primary cause of metabolic acidosis

Acid–base homeostasis is a fundamental component for the maintenance of normal metabolic function. Acid–base disorders, however, are extremely common in the intensive care unit and rapid, careful assessment is required to prevent unwanted morbidity and mortality. This chapter will provide a review of acid–base homeostasis, laboratory tests used to assess acid–base status, and a step-wise approach to classify acid–base disorders and their potential causes.

ACID–BASE CHEMISTRY

An acid is a substance that can donate a proton (e.g., $HCl \rightarrow H^+ + Cl^-$), while a base is a substance that can accept a proton (e.g., $H^+ + NH_3 \rightarrow NH_4^+$). Every acid has a corresponding base and every base has a corresponding acid. Some common acid–base pairs are carbonic acid/bicarbonate, ammonium/ammonia, monobasic/dibasic phosphate, and lactic acid/lactate.

The terms acid*emia* and alkal*emia* are used to describe an abnormal pH. Specifically, acidemia denotes a low pH, while alkalemia denotes a high pH. The terms *acidosis* and *alkalosis,* on the other hand, refer to the process by which either acid or alkali accumulate. It is, therefore, possible to have an acidosis, but not an acidemia. For this to occur (i.e., acidosis without acidemia), a corresponding alkalosis must also be present.

The acidity of a body fluid is determined by the concentration of hydrogen ion (H^+). Normal H^+ concentration is approximately 40 nanoequivalents/L. Because this is expressed in such small amounts (a nanoequivalent is one-millionth of a milliequivalent), acid–base status is measured in pH units using a logarithmic scale. Normal pH is 7.4 with a range of 7.35–7.45. The range of pH values considered compatible with life is 6.8–7.8, which corresponds to a hydrogen ion concentration of only 16 to 160 nEq/L.[1] In general, the body will tolerate acidemia much better than alkalemia. This is due to the fact that as pH decreases, a larger change in H^+ is required for a given change in pH.[2] In alkalemic states, small changes in H^+ can markedly affect pH. Furthermore, with alkalemia, the oxyhemoglobin dissociation curve will shift to the left and hemoglobin is less willing to release oxygen to the tissues (**Figure 13-1**).[3,4]

ARTERIAL BLOOD GASES

Assessment of acid–base status is determined using an arterial blood gas (ABG). Arterial blood reflects how well the blood is being oxygenated by the lungs, while venous blood reflects oxygen consumption by the tissues. It is important that arterial blood is used for these assessments as substantial differences may exist between the two particularly in the setting of critical illness.[5] Note that the "normal ranges" listed below are approximate, and there may be slight variability noted across different references or local laboratory standards.

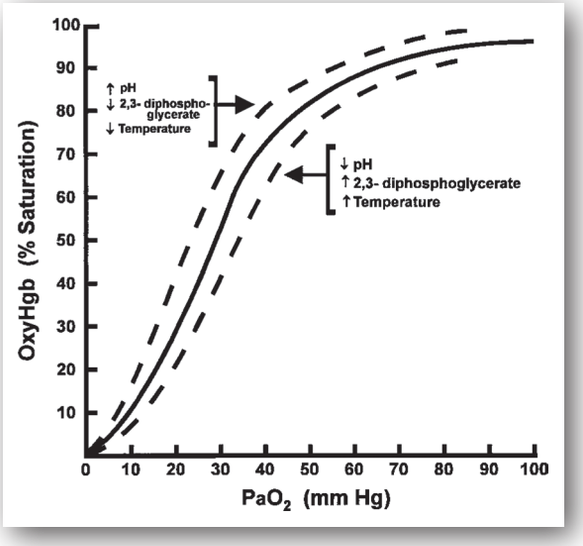

FIGURE 13-1. Oxygen–hemoglobin dissociation curve.

Arterial pH

Normal range: 7.35–7.45

The pH of arterial blood is the first value to consider when using an ABG to assess acid–base status. The pH is inversely related to hydrogen concentration. Generally speaking, pH values below 7.35 represent acidemia, and pH values above 7.45 represent alkalemia with 7.4 being the threshold for categorization during ABG assessment.

It is difficult to identify the pH value that will dictate the urgency whereby treatment must be initiated. Consequences of abnormal pH include arterial vasodilation, venous vasoconstriction, diminished myocardial contractility, impaired hepatic and renal perfusion, decreased oxygen-hemoglobin binding, and coma for acidemia, while cerebral vasoconstriction, reduced contractility, increased oxygen-hemoglobin binding, decreased oxygen delivery, and coma is encountered with alkalemia. These deleterious effects become more prominent when pH is <7.2 or >7.55.[6]

Arterial Partial Pressure of Carbon Dioxide

Normal range: 35–45 mm Hg (4.655–5.985 kPa)

Evaluation of *arterial partial pressure of carbon dioxide* ($PaCO_2$) provides information about the adequacy of lung function in excreting carbon dioxide. The amount of carbon dioxide dissolved in the blood is directly proportional to the concentration of carbonic acid ($PaCO_2 \times 0.03 = H_2CO_3^-$). Elevations in $PaCO_2$, therefore, will contribute to acidosis. Changes to ventilatory status that alter carbon dioxide concentrations will affect carbonic acid concentrations. Specifically, hypoventilation will lead to a higher $PaCO_2$, while hyperventilation will result in a lower $PaCO_2$. Regulation of ventilation is a major mechanism for respiratory compensation in the setting of primary metabolic disorders.

Arterial Partial Pressure of Oxygen

Normal range: 80–100 mm Hg (10.64–13.3 kPa)

Evaluation of the *arterial partial pressure of oxygen* (PaO_2) provides information about the level of oxygenation of arterial blood. The PaO_2 is important because it not only reflects the functional capabilities of the lungs, but also the rate at which oxygen can enter the tissues. Although there is no set cutoff for defining hypoxemia as it is typically relative to metabolic requirements, most would define clinically significant hypoxia at <60 mm Hg. Factors that influence PaO_2 are the amount of ventilation, the fraction of inspired oxygen (FiO_2), the functional capacities of the lung, and the oxyhemoglobin dissociation curve.

The oxyhemoglobin dissociation curve describes the relationship between PaO_2 and oxygen saturation (Figure 13-1). Oxygen saturation is the percentage of hemoglobin binding sites in the bloodstream occupied by oxygen. During states of acidemia, this curve will shift to the right whereby PaO_2 will be higher for a particular level of oxygen saturation. This is reflective of increased unloading of oxygen from hemoglobin. During alkalemia, on the other hand, this curve will shift to the left and higher PaO_2 values are required to maintain oxygen saturation. This will impair oxygen delivery to tissues. Other factors that can influence the oxyhemoglobin dissociation curve are temperature and the amount of 2,3-diphosphoglycerate in the red blood cells.

Although PaO_2 and oxygen saturation are both measurements of oxygenation, it is important to not confuse one with the other. For example, a PaO_2 of 80 mm Hg would typically not be considered abnormal as in most patients this is reflective of an oxygen saturation >93%. An oxygen saturation of 80%, on the other hand, would be considered critical and require immediate intervention. Furthermore, PaO_2 is expressed in mm Hg and values that exceed 100 can exist (especially when supplemental oxygen is administered). Oxygen saturation, however, cannot exceed 100%. Although PaO_2 assessment is crucial for determining pulmonary status, it does not directly impact acid–base balance.

Arterial Bicarbonate

Normal range: 22–26 mEq/L (22–26 mmol/L)

The concentration of *arterial bicarbonate* (HCO_3^-) reported from an ABG is not a direct measurement but calculated using the pH, $PaCO_2$ via the Henderson-Hasselbalch equation. It is important to compare this value (i.e., bicarbonate reading from the ABG) with the total CO_2 content (commonly referred to as *serum bicarbonate*) on an electrolyte panel. Under normal circumstances, the bicarbonate from the ABG is approximately 1.5–3 mEq/L less than the total CO_2 content from a plasma electrolyte panel (higher end of this range for venous samples).[7] Results should be interpreted with caution if this correlation does not exist.