Arterial Blood Gases:
Their Meaning and Interpretation*

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The measurement of arterial blood gases is essential in the management of respiratory failure and in the diagnostic assessment of the nature and severity of pulmonary disease. Adequate therapy for patients with acute respiratory insufficiency is often impossible without the information obtained from arterial blood gases. These studies must be readily available around the clock, including nights, weekends, and holidays and should not have to depend upon a technician called in from home. A ready knowledge of arterial blood gases in acute respiratory failure is just as important to good medical care as knowing the urine sugar and acetone in diabetic ketoacidosis or the blood count in an acute infection. In the practice of modern medicine, the lack of availability of arterial blood gases is unjustified and unacceptable. No hospital that maintains intensive care facilities or treats acutely ill patients can afford to be without blood gas equipment and properly trained personnel to provide accurate measurements.

The techniques for obtaining arterial blood samples are simple and can be done without difficulty by qualified laboratory technicians and nurses. A 10 cc glass syringe with a 20 or 22 gauge needle is flushed with aqueous heparin, and an arterial sample is obtained without air bubbles from a brachial, radial, or femoral artery. A glass syringe is preferable to one made of plastic because the glass plunger moves more freely and allows easy detection of arterial pulsation. After the puncture, the artery should be compressed by hand for no less than five minutes. If the blood gas analysis is not done immediately, the capped syringe should be placed in an iced container. Equipment for blood gas analysis is readily available and relatively easy to operate. The cost of basic equipment is approximately $3,000. An arterial blood sample of 3–5 cc is sufficient for a complete study. Micromethods are also available for “arterialized” capillary blood obtained by puncture of a hyperemic ear or fingertip. Only arterial blood is of consistent value in detecting the gas exchange in the lungs. Venous blood is unreliable because it has already passed through the systemic capillaries where oxygen and carbon dioxide exchange occurs. It is also influenced by changes in tissue metabolism and regional changes in circulation.

Oxygen and carbon dioxide are both physically dissolved in blood, and each exerts a tension or pressure (P₀₂ and P₉₄) which, in the capillaries of the lung, is in equilibrium with the gas pressures within the alveoli. The oxygen tension in the alveoli is determined by the partial pressure of the oxygen in the atmosphere and averages slightly above 100 mm Hg at sea level. The carbon dioxide in the alveolar gas averages 40 mm Hg. It comes almost entirely from the blood and reflects tissue metabolism (fig. 1).

The arterial oxygen tension is normally 5 to 10 mm Hg lower than the alveolar oxygen tension, due to shunting of the unoxygenated blood into the left atrium from the bronchial veins and the Thebesian
veins of the heart. Alterations in the distribution of ventilation and perfusion within the lungs also contribute to this alveolar-arterial oxygen gradient. As demonstrated in figure 2, during normal tidal breathing, about four times more ventilation goes to the bases of the lungs than to the apices. Perfusion is also distributed regionally with about 18 times more blood going to the bases than to the apices. This unequal matching of ventilation to perfusion accounts for lower tensions of oxygen and higher tensions of carbon dioxide in the alveoli at the bases of the lungs, and it is responsible for part of the alveolar-arterial oxygen difference.

The alveolar surface of the lungs is about 60 to 90 square meters with the pulmonary capillary volume approximately 70 cc. Gas exchange occurs within about 0.3 of a second and because of the enormous efficiency and reserve of this system, arterial blood gases remain within normal limits even with strenuous exercise when disease is not present. The physically dissolved oxygen in the arterial blood accounts for only a small fraction of the total oxygen that is available to the tissues. Sixty times more oxygen is carried by the red blood cells than is physically dissolved in the plasma. Venous blood returning to the lungs is about 75% saturated with oxygen. By the time it has traveled about one-third the distance through the pulmonary capillary bed, the hemoglobin has become fully oxygenated, and oxygen tensions in the alveoli and capillaries have reached equilibrium. Each gram of hemoglobin is capable of carrying 1.39 cc oxygen when fully saturated. In normal individuals with 15 g of hemoglobin the total oxygen content of arterial blood is about 20 cc per 100 ml of blood. Only 0.3 cc of oxygen is physically dissolved in each 100 ml of blood when the arterial $P_{O_2}$ is 100 mm Hg.

Hemoglobin saturation is calculated from the oxyhemoglobin dissociation curve (fig. 3). At arterial oxygen tensions ranging from 60 to 100 mm Hg, the dissociation curve is relatively flat with hemoglobin saturation changing only 8% (90% to 98%). Arterial oxygen tensions below 60 mm Hg are not normally encountered during life, except at very high altitudes and in disease. In systemic capillaries where oxygen tensions are as low as 30 to 40 mm Hg, the hemoglobin dissociation curve becomes quite steep, and oxygen is readily released from the red blood cells. Changes in pH, $P_{CO_2}$, and temperature all favor the release of oxygen from hemoglobin in the tissues and increase oxygen uptake by the red blood cells in the lungs.

Although the arterial $P_{O_2}$ is the most important blood gas indicator of oxygen transport, there are multiple factors that must be considered in the evaluation of tissue oxygen delivery. These include...
tissue oxygen consumption, cardiac output, and arterial oxygen content. An outline of factors relating to oxygen delivery is presented in Table 1.

In reporting arterial blood gases, most pulmonary laboratories make three direct measurements, which are summarized as follows:

1) \( \text{Pa}_O_2 \). This is the partial pressure of oxygen that is physically dissolved in the arterial blood; it is normally 5 to 10 mm Hg lower than the oxygen tension in the average alveolus. The normal arterial oxygen tension is 90 to 100 mm Hg at sea level.

2) \( \text{Pa}_{CO_2} \). This is the partial pressure of carbon dioxide that is dissolved in the blood and indicates the state of alveolar ventilation. The normal arterial carbon dioxide tension is 36 to 44 mm Hg.

3) \( \text{pH} \). This is an expression of the hydrogen ion concentration of the blood (negative logarithm) and relates directly to the ratio of the concentrations of bicarbonate and \( CO_2 \) in the blood. The normal range is 7.36 to 7.44.

In addition to these three direct measurements there are other determinations which are based on calculations and are reported from nomograms. These are summarized as follows:

1) Oxygen saturation of hemoglobin. This indicates the capacity of hemoglobin to carry oxygen and is measured from the oxyhemoglobin dissociation curve using the \( \text{Pa}_O_2 \) and \( \text{pH} \) determinations. The normal range for arterial blood is 95 to 98%.

2) Plasma bicarbonate (\( \text{HCO}_3^- \)). This is measured from a standard acid base nomogram using the \( \text{Pa}_{CO_2} \) and \( \text{pH} \) determinations. The normal range is 23 to 27 meq/liter (fig. 4).

3) Some laboratories report the base excess concentration. This is an expression of the
TABLE 1.
Oxygen Transport

A. Lungs
   O$_2$ loading of RBC's
B. Cardiovascular System
   1. Rate of blood flow.
   2. Distribution of flow between body tissues.
C. Erythrocyte
   1. Oxygen-carrying capacity of blood.
   2. Hemoglobin affinity for O$_2$ (O$_2$ release or loading at a given P$_{O_2}$).
D. Tissues
   Rate of O$_2$ consumption

base excess in meq/liter, as relates to a normal value of 0 for blood with pH 7.40 and P$_{CO_2}$ 40 mm Hg. Negative values indicate a base deficit or acid excess. The normal range is 0 plus or minus 2.3 meq/liter.

In the diagnostic evaluation of pulmonary disease, arterial blood gases provide a direct approach to ventilatory function since the primary purpose of the lungs is to oxygenate the blood and eliminate CO$_2$. Arterial blood gases are usually obtained at rest, following exercise, and after breathing 100% oxygen. This combination of studies provides considerable information in the evaluation of the nature and severity of respiratory disease. These data are much less influenced by patient cooperation and performance than many of the other tests performed in the pulmonary function laboratory.

In the management of patients with respiratory failure, it is frequently impossible to judge the level of alveolar ventilation or the adequacy of oxygenation by clinical evaluation alone. All of the signs and symptoms that suggest hypoxia or hypercapnia may be absent at times, and only by measurement of arterial blood gases can the physician assess the degree of respiratory impairment and render the appropriate therapy. Once active treatment has begun with oxygen or mechanical ventilation, it is important to evaluate the effectiveness of such therapy by measuring the arterial blood gases. Giving too much oxygen may seriously damage the lungs, while mechanical hyperventilation may cause severe alkalosis with possible coma and death. The delicate balance between too little and too much can best be reached and maintained by a knowledge of the state of the arterial blood gases.

BIBLIOGRAPHY
