Chapter 16: Blood Gases

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INTRODUCTION

Blood gases provide important clinical information for patients with respiratory disorders, compromised circulation, or abnormal metabolism. In recent years, a number of surrogates for blood gas analysis have entered the daily practice of emergency medicine. Leveraging the benefits of blood gases requires an understanding of the underlying physiology, appropriate use of arterial and venous blood gases, and knowledge of the advantages and limitations of noninvasive monitoring methods. This chapter will be limited to evaluation of oxygen and carbon dioxide levels; for information on carbon monoxide, please refer to chapter 222, "Carbon Monoxide."

RESPIRATORY PHYSIOLOGY

Several factors contribute to overall gas exchange in the lungs. Each breath (tidal volume) is composed of functional movement of air in and out of the alveolus and nonfunctional movement of air through bronchioles, bronchi, trachea, and nonperfused areas of lung (physiologic dead space). The physiologic dead space is approximately 30% of the tidal volume. The air remaining in the chest at the end of exhalation is referred to as the functional residual capacity. Dead space and the functional residual capacity do not contribute to gas exchange. The amount of air inhaled and exhaled in a minute is referred to as minute ventilation and is the product of the respiratory rate and tidal volume. Relatively small changes in the functional alveolar space demand large increases in the minute ventilation to maintain the same rate of gas exchange. Either raising the fraction of inspired oxygen (Fio_2) or increasing the surface area or functional residual capacity of the lung can increase total alveolar oxygen (O_2) content. Positive-pressure ventilation increases the functional residual capacity through recruitment of collapsed nonventilated alveolar space.

ESTIMATING OXYGEN DELIVERY TO THE ALVEOLAR SPACE

The F_{IO_2} is the fraction or percentage of oxygen in the space being measured. At sea level, room air is 21% oxygen (20% is often used for ease of calculation). As F_{IO_2} increases, the alveolar concentration of oxygen (P_{AO_2}) proportionately increases. Unless the patient is part of a closed system, like a ventilator circuit, the F_{IO_2} is only estimated. Each liter per minute of O_2 flow delivered via nasal cannula increases the F_{IO_2} by about 4%. Flow rates >4 L/min through a nasal cannula are poorly tolerated because of upper airway irritation. A simple O_2 mask provides an F_{IO_2} of 35% to 60% at flows of 10 to 15 L/min. A nonrebreather mask with a reservoir can deliver 95% O_2 at a flow rate of 10 to 12 L/min.

Comparing the measured arterial concentration of oxygen to the expected concentration of oxygen can be useful for determining the nature and severity of respiratory disorders. The approximate arterial oxygen concentration (P_{AO_2}) values that are expected in normal persons who are inhaling various concentrations of O_2 are listed in **Table 16–1**.

Table 16–1

Expected P_{AO_2} in Patients Inhaling Various Concentrations of Oxygen (mm Hg)

Fraction of inspired oxygen	0.21 (room air)	0.4	0.6	0.8	1.0
~P_{AO_2}*	105†	200	300	400	500

Abbreviation: P_{AO_2} = partial pressure of arterial oxygen.

*Assuming a patient with normal lung function at sea level and a partial pressure of carbon dioxide (P_{CO_2}) of 40 mm Hg.

†Calculation demonstrates slight overestimation using this technique.

The expected P_{AO_2} for a patient being given supplemental oxygen can be roughly estimated by multiplying the actual delivered percentage of O_2 by 5, the "**Five Times Rule**." Thus, a patient getting 60% O_2 would be expected to have a P_{AO_2} of about 60 × 5, or 300 mm Hg. For every 1000 ft (305 m) rise in altitude, barometric pressure drops approximately 25 mm Hg, and the atmospheric partial pressure of oxygen (P_{O_2}) drops about 5 mm Hg. The arterial oxygen estimate would therefore be reduced by the fraction of pressure at

elevation compared to sea level. The total alveolar oxygen pressure cannot be greater than atmospheric pressure unless the patient is receiving positive-pressure ventilation. Further discussion of O_2 and ventilation at altitude and depth is found in chapters 221, "High-Altitude Disorders" and 214, "Diving Disorders."

ALVEOLAR GAS EXCHANGE AND SYSTEMIC DELIVERY

Once oxygen or carbon dioxide reaches the alveolus, it moves across the interstitial space to either the red blood cell or alveolar space, respectively. The efficiency of diffusion depends on the distance across the alveolar-capillary membrane (interstitial space), the partial pressure of the gas in the alveolar space, and the solubility of the gas. Both oxygen and carbon dioxide are highly soluble, and carbon dioxide is 20 times more soluble than oxygen. As a result, increases in the distance across the interstitial space (as in pulmonary edema, for example) have greater effect on oxygen diffusion than carbon dioxide (CO_2) diffusion. Gas diffusion requires functioning alveolar space, and conditions that damage the alveolus prevent effective oxygen and carbon dioxide transport. Additionally, the alveolar space must be perfused by the pulmonary circulation. When portions of lung are perfused but not ventilated (as in pneumonia), or ventilated but not perfused (as in pulmonary embolism), there is a ventilation-perfusion mismatch. Either scenario may lead to hypoxemia, the first as deoxygenated blood passes through the nonfunctional lung and mixes with oxygenated blood in the left atrium, and the second when too little perfused lung is available for adequate oxygen loading.

The effectiveness of alveolar oxygenation can be estimated either by the alveolar-arterial gradient or the Pa_{O_2}/Fi_{O_2} ratio. The alveolar-arterial gradient is the difference between the partial pressure of oxygen in the alveolar space (estimated from Fi_{O_2} at atmospheric pressure) and the measured partial pressure of the gas in an arterial blood gas sample. The alveolar-arterial (A-a) gradient can be estimated by the following simplified formula:

$$P(A\text{-}a)o_2 = 147 - (Paco_2 \times 1.25) - Pao_2$$

A normal gradient for young adults is <15 mm Hg. The gradient increases with age at a rate of approximately 4 to 8 mm Hg per decade or can be estimated with the following formula: age/4 + 4. The Pa_{O_2}/Fi_{O_2} ratio correlates with the relative venous shunt across the pulmonary circulation and is calculated as the measured Pa_{O_2} divided by the Fi_{O_2} in decimal form. A healthy person on 40% oxygen would be expected to have a ratio of approximately 600, representing a normal physiologic shunt of approximately 5%. As the shunt increases, the ratio decreases. **Table 16–2** illustrates the fall in the Pa_{O_2}/Fi_{O_2} ratio with increasing physiologic shunt in hypothetical patients all receiving oxygen with an Fi_{O_2} of 40%.

Table 16–2

Interpretation of Pa_{O_2}/Fi_{O_2}

Pa_{O_2} (mm Hg)	Fi_{O_2} (mm Hg)	Ratio	QS/QT (%)	Impairment of Oxygenation
240	0.4	600	5	None
120	0.4	300	10	Minimal
100	0.4	250	15	Mild
80	0.4	200	20	Moderate
60	0.4	150	30	Severe*
40	0.4	100	40	Very severe*

Abbreviations: Fi_{O_2} = fraction of inspired oxygen; Pa_{O_2} = partial pressure of arterial oxygen; QS/QT = venous-arterial admixture (shunt).

*Ventilatory support and positive end-expiratory pressure to increase functional residual capacity and reduce the QS/QT to 15% should be considered.

The Pa_{O_2}/Fi_{O_2} ratio is the most frequently used parameter for evaluating the severity of lung failure and is included in the current definition for acute lung injury/acute respiratory distress syndrome.[1] The amount of oxygen transported to the body is determined to the greatest extent by the total blood hemoglobin content and cardiac output. Relatively little oxygen is dissolved in the plasma itself. The arterial O_2 content of blood (Ca_{O_2}) can be calculated with the following formula (hemoglobin [Hb] in grams/dL, Pa_{O_2} in mm Hg, and arterial oxygen saturation [Sa_{O_2}] expressed as a fraction of 1.0):

$$Ca_{O_2} = [Hb \times 1.39 \times Sa_{O_2}] + (Pa_{O_2} \times 0.0031)$$

As a result, systemic delivery of oxygen can only be practically modified by either increasing the hemoglobin content or oxygen saturation. Although increasing the hemoglobin by transfusion will increase O_2 content, various qualities of transfused blood limit its benefit to patients, and further discussion is beyond the scope of this chapter (see chapters 13, "Fluid and Blood Resuscitation in Traumatic Shock," and 238, "Transfusion Therapy").

Carbon dioxide is transported by the red blood cell as carbamino compounds bound to hemoglobin and other carbamino-containing proteins, as well as in the form of plasma bicarbonate. Most carbon dioxide (60% to 70%) combines with plasma water to form carbonic acid, which dissociates into bicarbonate and hydrogen ions. This reaction is catalyzed by carbonic anhydrase in the erythrocyte such that the reaction is almost instantaneous. The deoxygenated hemoglobin is a willing receptor for released hydrogen ions. As hemoglobin is oxygenated in the lung, hydrogen ions are released, driving the reaction back toward the production of CO_2.

ARTERIAL BLOOD GAS ANALYSIS

The amount of oxygen and carbon dioxide in the blood can be sampled and reported as the partial pressure of the gas. Blood gas analysis also typically includes a direct measurement of the serum pH and estimates of serum bicarbonate derived from the measured partial pressure of carbon dioxide (P_{CO_2}) and pH (see chapter 15, "Acid-Base Disorders"). Current tests often include other useful information such as direct measurement of lactic acid as lactate, total hemoglobin, and serum electrolytes.

An arterial blood sample is the reference standard for pH, oxygen, carbon dioxide, and lactate content providing a description of the oxygen and CO_2 content of the blood after leaving the pulmonary circulation and before any gas exchange in the peripheral tissues has occurred. In scenarios that require precise determination of these variables, an arterial sample is necessary.

Arterial blood gas samples are sometimes used for evaluation of serum hemoglobin and electrolytes. Blood gas analyzers typically have good concordance with the reference venous sample; however, there may be clinically significant variances when the result falls outside the normal range. Arterial electrolytes and hemoglobin results should be interpreted with caution when the results are significantly abnormal.[2,3,4,5,6]

MIXED VENOUS BLOOD GAS ANALYSIS

Mixed venous blood samples provide a source of information regarding the systemic uptake of oxygen and can be used to calculate cardiac output. The ideal sampling site for a systemic venous sample is at the pulmonary artery because blood from all body sites is

equally represented. Sampling blood from the pulmonary artery is limited to situations where the patient has a pulmonary artery catheter placed. More commonly, blood is sampled from the superior vena cava or right atrium after placement of a central venous catheter. Blood from the superior vena cava disproportionally represents cerebral and upper body blood flow but is generally useful for determining systemic oxygen uptake and lactic acid production.

Regular monitoring of the venous O_2 saturation at the superior vena cava ($S_{cv}O_2$) or right atrium ($S_{ra}O_2$) is currently recommended as part of the Surviving Sepsis Campaign.[7,8] Although measurement of the O_2 saturation from a superior vena cava sample is acceptable within the guidelines, this location may not accurately reflect the $S_{ra}o_2$, and neither the $S_{cv}o_2$ nor the $S_{ra}o_2$ may accurately reflect the mixed venous O_2 saturation at the pulmonary artery ($S_{v}o_2$) for critically ill patients.[9,10,11,12,13]

PERIPHERAL VENOUS GAS ANALYSIS

Peripheral venous samples are commonly used as the source for gas analysis due to their relative ease of collection and decreased patient discomfort. The substitution of venous blood for an arterial sample is appropriate in particular clinical scenarios as described below.

Peripheral venous blood gases are commonly used to monitor serum pH. When compared to arterial blood gases, the peripheral venous pH correlates very closely such that any differences are not usually clinically significant (+/– 0.05 pH units).[14] Venous Pa_{o_2} values do not correlate with arterial oxygen content and cannot be used for evaluation of oxygenation. Venous CO_2 values trend along with arterial CO_2, although they do vary somewhat (up to +/– 20 mm Hg). Normal venous CO_2 is predictive of normal Pa_{co_2}; however, the clinical outcomes of substituting venous CO_2 for evaluation of hypercarbia have not been described in the literature.[14,15,16,17,18,19]

It may be possible to derive a mathematical correction to venous blood gas values that better approximates arterial values. To date, no recognized validated approach has been identified.[20,21,22,23,24]

Peripheral venous estimations of serum lactate production are widely used for patient care; however, most studies describing the clinical utility of serum lactate reference an arterial or mixed venous sample. Peripheral venous lactate measurements correlate with arterial lactate; however, they are not equivalent, and a peripheral venous lactate level may be significantly higher than arterial lactate.[25,26,27]

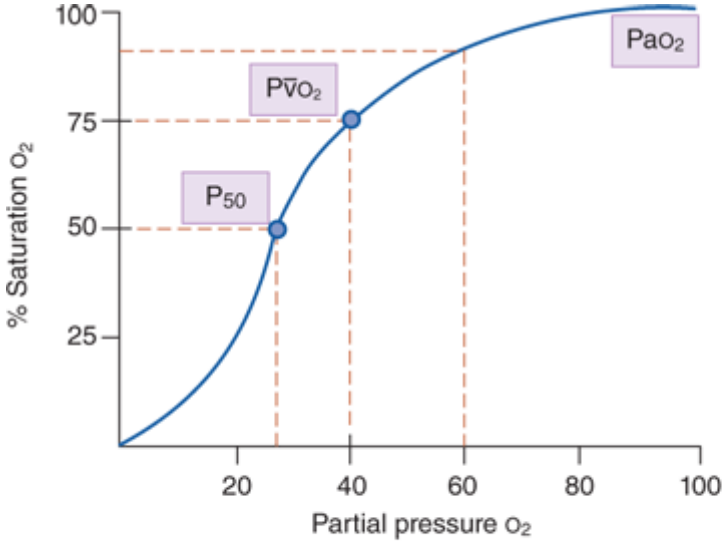
NONINVASIVE OXYGEN MONITORING

It is not possible to directly measure the arterial oxygen concentration by noninvasive means. In the clinical context, the arterial oxygen saturation is often as helpful for patient management and treatment decisions. For the last 30 years, the standard bedside tool for estimating arterial oxygen saturation has been the photometric pulse oximeter.

Pulse oximetry measures the relative absorption of oxygenated hemoglobin and deoxygenated hemoglobin and reports the percentage of oxygenated hemoglobin compared to the total. Pulse oximetry is fairly accurate, typically within 2% to 5% of the directly measured value performed at blood gas analysis. Importantly, the discrepancy between peripheral pulse oxygenation results and arterial gas saturation increases with hypoxia and poor circulation. Pulse oxygenation values greater than 92% are highly predictive of arterial saturations greater than 90%.[28] Pulse oxygenation values between 88% and 92% do not necessarily reflect normal arterial saturations and may overestimate arterial oxygen saturation. The pulse oximeter may overestimate saturation when arterial saturations fall below 80% to 90%, and marginally normal values should be interpreted with caution.[29,30] Carbon monoxide also causes hemoglobin to conform to the saturated state and will cause a false elevation in measured saturation (see chapter 222).

Although clinically useful, pulse oximetry does not allow for determination of the partial pressure of oxygen in the arterial blood, which, in combination with total hemoglobin content, is the primary determinant of peripheral oxygen delivery. A basic understanding of the oxygen saturation/dissociation curve is essential for practicing clinicians when using pulse oximetry to guide clinical therapy (**Figure 16–1**). Most notably, arterial oxygen content declines precipitously when the oxygen saturation falls below 92%.

FIGURE 16–1.
The oxyhemoglobin dissociation curve. This curve demonstrates the relationship of the partial pressure of oxygen (Po_2) in the plasma to the saturation of hemoglobin molecules with O_2. The P_{50} is the Po_2 at which hemoglobin is 50% saturated and correlates with Po_2 of 27 mm Hg normally. Normal mixed venous blood has an oxygen partial pressure ($Pmvo_2$) of 40 mm Hg and an oxyhemoglobin saturation of 75%. A partial pressure of arterial oxygen (Pao_2) of 60 mm Hg normally results in approximately 90% saturation of hemoglobin.



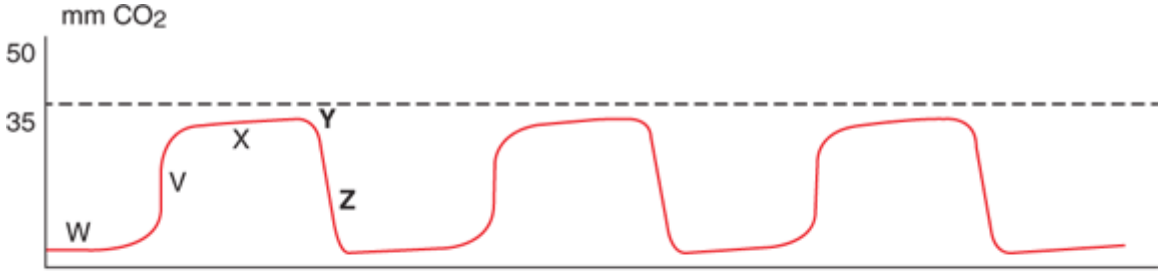
NONINVASIVE CARBON DIOXIDE MONITORING

The CO_2 content of arterial blood can be estimated by several noninvasive methods including direct transcutaneous measurement, cutaneous photometric measurement, or sampling of expired gases. Of these, only the later method is in common use in the ED.

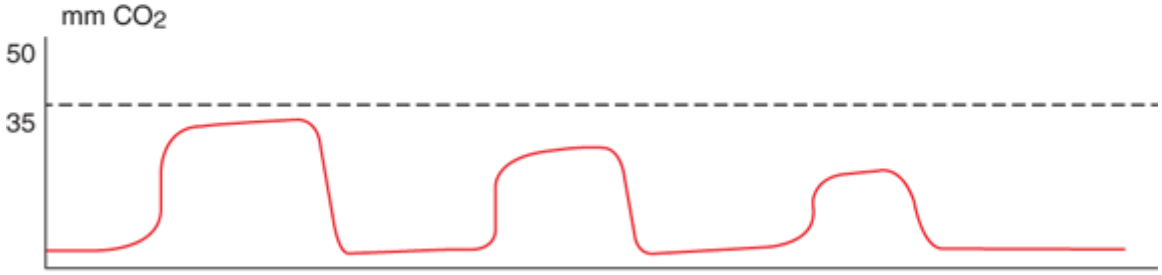
Expired gases can be directly sampled to determine their CO_2 content. The carbon dioxide content of expired gases at the end of the expiration phase of respiration (Et_{CO_2}) proportionally approximates the Pa_{CO_2} content in healthy individuals because CO_2 rapidly diffuses into the alveolar space along its pressure gradient, and arterial levels are typically only about 5 mm Hg higher than alveolar samples when pulmonary ventilation and perfusion are functioning normally. The CO_2 content of the entire volume of expired gas can be photometrically assessed by means of a **mainstream** detector, or a sample of the expired gases can be aspirated from airway via a **side stream** detector. Mainstream detectors are larger and must be inserted into the ventilator loop, either in a ventilator circuit or with the patient breathing through a mask or other tube. Side stream detectors are smaller and have the advantage of ease of use in nonintubated patients. Interpretation of the capnogram is not necessarily intuitive. A summary of typical capnogram waveforms is described in **Figure 16–2**.

FIGURE 16–2.

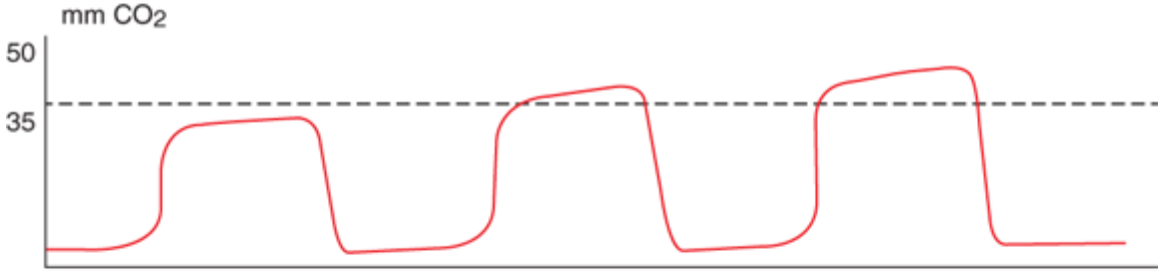
End-tidal capnogram. Capnogram A depicts a normal capnogram with inspiratory baseline (V), expiratory upstroke (W), expiratory plateau (X), end-tidal concentration (Y), and inspiratory downstroke (Z). Capnogram B represents apnea, which appears as serially decreasing end-tidal concentrations as little gas is expired. Capnogram C represents hypoventilation, which appears as an upward trend in the plateau and end-tidal concentration. Capnogram D represents rebreathing or air trapping, which appears as an increase in the baseline phase of the capnogram.



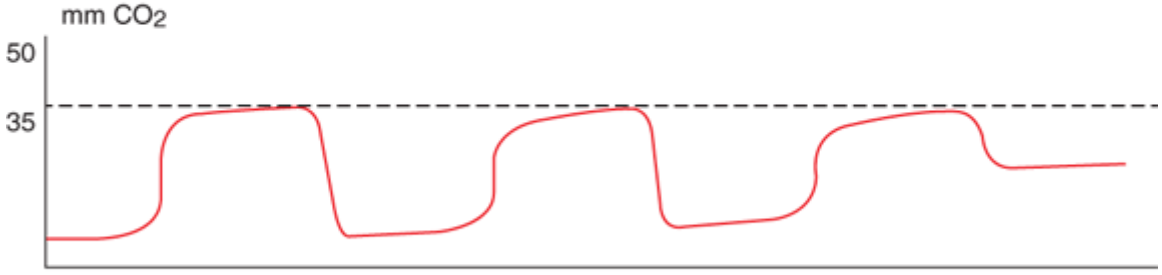
A



B



C



D

End-tidal CO_2 measurements ($Petco_2$) do not accurately represent $Paco_2$. Mainstream and side stream sampling may each include air that has not been involved in the gas exchange process, either from physiologic dead space or via environmental air that enters the sample. In either case the $Petco_2$ value will be lower than the arterial value. End-tidal CO_2 measurement is not as useful in obstructive pulmonary disease due to incomplete expiration of gases secondary to air trapping. In a study of ED patients with chronic obstructive pulmonary disease, end-tidal measurements did not significantly vary from presentation to admission and were not clinically different for patients discharged home as compared to those requiring admission.[31] Studies assessing the accuracy of $Etco_2$ measurements compared to arterial sampling are mixed. In one study of patients presenting to the ED with undifferentiated dyspnea, mainstream $Petco_2$ did correlate closely with $Paco_2$.[32] In a comparison of healthy individuals, neither mainstream nor side stream sampling accurately predicted the arterial Pco_2.[33] A convenience sample of ED patients with an indication for arterial blood gas did not have a close correlation of side stream $Etco_2$ with the $Paco_2$.[34] The precise role for $Etco_2$ measurements in the management of critically ill patients in the ED or intensive care unit is still to be determined.[35]

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