# 22

# Blood gas analysis

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## Abnormalities in oxygenation and ventilation

Hypoxemia is defined as a partial pressure of oxygen in the artery ($\mathbf{PaO_2}$) of less than 80 mm Hg. In general, there are five causes of hypoxemia: hypoventilation, decreased inspired oxygen content, ventilation/perfusion (V/Q) mismatch, intrapulmonary shunt, and diffusion impairment. Hypoventilation (increased partial pressure of carbon dioxide [$\mathbf{PaCO_2}$] in the blood) can be caused by primary central nervous system (CNS) suppression of ventilation, the administration of medications that suppress ventilation (opioids, anesthetic agents), neuromuscular disease affecting ventilation (tick paralysis, botulism), upper airway obstruction, pleural space disease, chest wall injury (flail chest, pain), and respiratory muscle fatigue. Decreased inspired oxygen content is associated with higher altitudes or from anesthetic machine errors. General etiologies are listed in Box 22.1.

V/Q mismatch and intrapulmonary shunting (V/Q mismatch taken to an extreme) are the most common causes of hypoxemia. **V/Q mismatch** occurs due to decreased ventilation to normally perfused lungs and results in a low V/Q ratio. This type of hypoxemia can be caused by narrowed airways as with asthma or chronic bronchial disease. The narrowing is either secondary to bronchospasm or increased mucus secretions, limiting oxygen entry into the alveolus. **Intrapulmonary shunting** is an extreme "low V/Q" state—a "zero V/Q state"—that occurs when collapsed alveoli or alveoli filled with nongas substance are perfused but (due to the collapse or nongas substance) are not ventilated at all (zero ventilation). Diseases like aspiration pneumonia and pulmonary edema commonly cause intrapulmonary shunting. Shunts can also occur due to abnormal blood flow, as with right-to-left shunting of blood due to cardiac abnormalities (extrapulmonary shunts: patent ductus arteriosus and septal defects), although these are less common than lung disease. **Diffusion impairment** results in incomplete arterialization of the pulmonary capillary blood due to thickening of the gas exchange layer in the alveoli. Under normal conditions the equilibration of oxygen occurs rapidly, but with a thickened gas exchange layer more time is needed, resulting in

**Box 22.1** Causes of hypoxemia and causes of hypoventilation

**Causes of hypoxemia**
- Hypoventilation
- Decreased inspired oxygen content
- Ventilation/ perfusion (V/Q) mismatch
- Intrapulmonary shunt
- Diffusion impairment

**Causes of hypoventilation**
- Central nervous system disease
- Medications (opioids, anesthetic agents)
- Neuromuscular disease
- Upper airway obstruction
- Pleural space disease (severe)
- Chest wall injury
- Respiratory muscle fatigue

*Advanced Monitoring and Procedures for Small Animal Emergency and Critical Care*, First Edition. Edited by Jamie M. Burkitt Creedon, Harold Davis.

hypoxemia. Diffusion impairment is believed to be an uncommon cause of hypoxemia in small animals.

When evaluating a patient's oxygenation and ventilation status, the gold standard is to obtain an arterial blood sample for direct analysis of the partial pressures of these gases in the blood: a **blood gas analysis**. Blood gases—the analyzers available, results, and interpretation—are the topics of this chapter.

## Blood gas analyzers

The types of analyzers available for use include benchtop and portable models. Dedicated blood gas benchtop analyzers are generally only used in academic facilities and large institutions due to their maintenance requirements and expense. Benchtop analyzers use a polarographic oxygen electrode and the Severinghaus carbon dioxide ($CO_2$) electrode to analyze the level of gas in the blood sample. The amount of $CO_2$ in the sample is evaluated based on comparison to a known amount of $CO_2$, which is supplied by conventional gas tanks. This severely limits the portability of the benchtop analyzers.

Portable (or "bedside") blood gas analyzers accurately measure oxygen and $CO_2$ using a small foil-wrapped cartridge containing a pH electrode, a polarographic oxygen ($O_2$) electrode, and a $CO_2$ electrode. When the cartridge is sealed, the $CO_2$ equilibrates with bicarbonate within the cartridge. Upon removal of the foil package, the gas surrounding the $CO_2$ is the known partial pressure of $CO_2$ in the ambient air, and the electrode calibrates to this pressure upon insertion into the machine.

There are many advantages to the use of portable blood gas analyzers. These machines are usually less expensive than the benchtop devices, can analyze full blood gases on a very small blood sample, and are portable. The availability of bedside analyzers has enhanced the care of critically ill veterinary patients, allowing for more frequent monitoring, diagnosis of ventilation and oxygenation problems, and the ability to guide therapy and prognosis of patients with significant respiratory disease.[1]

### *Sample analysis*

To be clinically useful, the arterial blood sample has to provide an accurate reflection of the patient's status. This requires appropriate blood sampling and handling. Please see Chapter 5, Arterial Puncture and Catheter Placement, and Chapter 47, Blood Sample Collection and Handling, for more information.

The basic method of determining the gas content in the blood involves obtaining an arterial blood sample and inserting it into the blood gas analyzer within a short period of time.

The blood gas analyzer measures the pH, the $PO_2$, and the $PCO_2$ of the sample, and the bicarbonate concentration and base deficit are calculated. The pH is measured using a pH-sensitive membrane. The pH is determined by measuring the voltage difference across this membrane.

The $PCO_2$ measurement is made by measuring the hydrogen ion difference between two solutions after allowing a chemical reaction that results in hydrogen ion production:

$$CO_2 + H_2O \rightarrow H_2CO_3 \rightarrow \mathbf{H^+} + HCO_3^-. \quad (22.1)$$

Resultant hydrogen ion production is determined using a pH-sensitive membrane similar to that used for the pH measurement. The hydrogen ion production is directly proportional to the $PCO_2$ of the blood sample.

The $PO_2$ is measured using a Clark electrode, which uses oxidation and reduction reactions involving the oxygen in the sample to quantify the partial pressure of oxygen present. The change in current as a result of the oxidation-reduction reactions is proportional to the $PO_2$ of the blood gas sample. Once pH, $PCO_2$, and $PO_2$ are measured, a microprocessor then calculates the bicarbonate, base deficit, and temperature-corrected gas values (see later).[2]

### *Temperature correction*

Blood gas analysis is performed at 37°C, which may or may not reflect patient body temperature and thus *in vivo* blood gas values. When the patient's temperature is entered into the blood gas analyzer, the machine can calculate the blood gas results accurate for the individual patient. However, changes in temperature alter gas solubility (Charles law) and alter hemoglobin's affinity for oxygen. Taken together with the fact that the metabolic, vascular, and respiratory effects of hyper- and hypothermia are not fully understood, temperature correction of blood gases remains controversial.[3]

## Blood gas sampling

The most commonly sampled arterial sites are the dorsal pedal (dorsal metatarsal) and femoral arteries. Less commonly sampled sites are the brachial, radial, auricular, and lingual arteries. Sampling is generally done using palpation and knowledge of anatomy. The dorsal pedal artery can be palpated over the dorsomedial aspect of the metatarsal; the femoral can be palpated on the medial aspect of the thigh. A single sample can be drawn using a needle and heparinized syringe, or a catheter can be placed for blood gas sampling and for direct blood pressure monitoring. The type of material used in

catheters can affect handling of the catheter with placement but also the likelihood of causing thrombophlebitis.[4] Monitoring for evidence of thrombosis is important, especially in the peripherally placed anatomic sites because this my compromise blood flow to distal tissues. Refer to Chapter 5, Arterial Puncture and Catheter Placement, for more information.

## Sample handling

To ensure accuracy of blood gas results, knowledge of appropriate sample handling is important. Exposure of the sample to room air and delayed analysis will alter the measurements. Air bubbles in the sample syringe should be expelled immediately. Due to gas diffusion, exposure to room air will cause a drop in the sample $PaCO_2$ ($PCO_2$ of room air is approximately zero) and change in the sample $PaO_2$ ($PO_2$ of room air is approximately 150 mm Hg at sea level). Analysis delay will also alter the accuracy of the blood gas results. Blood cells remain metabolically active for some time *ex vivo*, and cellular effects can alter the accuracy of the blood gas results when delayed samples are not chilled. If sample analysis must be delayed, the sample should be stored anaerobically in an ice water bath at 4°C; this allows up to a 6-hour delay in analysis without significantly affecting results.[5,6]

Blood gas analysis requires whole blood samples, and as such anticoagulants must be used to allow testing. Dilutional errors can affect sample results. Heparin is commonly used as an anticoagulant during blood sampling. Studies have shown that dilution of the sample with liquid heparin lowers the $PaCO_2$, affects the measure of oxygen, and also can affect electrolytes, lactate, and bicarbonate results. A <10% dilution with heparin minimizes effects on the analytes; this dilution can be achieved with the **evacuated syringe method** using a 3-cc syringe and 22-gauge needle. Liquid sodium heparin (1000 U/mL) 0.5 mL is drawn into the syringe, and then the plunger is drawn to the 3-cc mark to allow the heparin to coat the entire syringe. Then the contents of the syringe are removed by drawing back to the 3-cc mark and expelling all the air and any heparin forcibly. This air/heparin expel procedure is repeated three times, and the syringe is filled with at least 1 cc of blood, which represents a 3.9% dilution.[7] See Chapter 5, Arterial Puncture and Catheter Placement, for further details about this technique.

## Evaluating arterial blood gas results

Normal arterial blood gas values for dogs and cats are listed in Box 22.2.[8]

**Box 22.2** Normal arterial blood gas values for dogs and cats

**Dog[8] (at sea level)**
- $PaO_2$: 92 mm Hg (80–105)
- $PaCO_2$: 37 mm Hg (32–43)
- $SaO_2$: more than 95%

**Cat[8] (at sea level)**
- $PaO_2$: 105 mm Hg (95–115)
- $PaCO_2$: 31 mm Hg (26–36)
- $SaO_2$: more than 95%

### *Assessment of the partial pressure of carbon dioxide*

The partial pressure of carbon dioxide ($PCO_2$) is the amount of $CO_2$ dissolved in the blood and is determined by the balance between production in the tissues and elimination from the body.

$PCO_2$ is primarily a measure of ventilation because when $CO_2$ production increases, increases in respiratory rate and/or tidal volume (ie, minute ventilation) will normalize the $PCO_2$ in a healthy animal. Alterations in $PaCO_2$ are usually associated with neurologic or neuromuscular disease, medications, or pathology of the airway, pleural space, or chest wall.

Control of ventilation is primarily driven by the medulla. Increased $PCO_2$ diffuses into the cerebrospinal fluid and lowers the pH, which stimulates chemoreceptors and leads to increased ventilation. Hypoxemia can also trigger central chemoreceptors to increase ventilation when the $PaO_2$ is less than 60 mm Hg. The carotid bodies and aortic bodies are peripheral chemoreceptors that can stimulate ventilation secondary to decreased $PaO_2$, increased $PaCO_2$, or a drop in pH.[9]

Although this chapter focuses on arterial blood gas analysis, note that $PCO_2$ (unlike $PO_2$) is quite similar in the venous blood as in the arterial blood. Therefore venous blood can and is used to evaluate $PCO_2$, with normal values expected to be only a few millimeters of mercury higher than in arterial samples. The same is not true for evaluation of $PO_2$, which must be performed on arterial blood to provide information about the lung's ability to oxygenate the blood; venous $PO_2$ cannot provide such information.

#### Elevated $PCO_2$ (hypercapnia)

By definition respiratory acidosis or hypercapnia is an increase in the partial pressure of carbon dioxide ($PaCO_2$ >43mm Hg in the dog, >36 mm Hg in the cat). This most commonly occurs due to decreased alveolar ventilation,

which can occur for several reasons. CNS dysfunction, as with anesthetics, traumatic brain injury, neoplasia, infections, or cerebral edema, can affect CNS control of ventilation. Cervical diseases affecting the spinal cord, such as intervertebral disk disease, neoplasia, infection, or inflammation, can also lead to hypoventilation due to effects on the upper and lower (phrenic nerve) motor neurons responsible for diaphragmatic innervation. Peripherally mediated neuromuscular disease such as tetanus, botulism, and tick paralysis can also lead to a respiratory acidosis due to hypoventilation. Diseases of the pleural space, such as severe pleural effusion or pneumothorax, or diaphragmatic hernia can also result in hypoventilation. Obstructive diseases of the upper and lower airway alter airway resistance and lead to hypercapnia. Tracheal collapse, an upper airway mass, or a foreign body can lead to upper airway obstruction and hypercapnia. Lower airway diseases such as asthma or bronchitis can also cause hypercapnia. Respiratory acidosis will result when there is increased $CO_2$ production in the tissues without the appropriate compensation in ventilation, as in heat stroke and malignant hyperthermia; however, such situations are much less common than inadequate $CO_2$ elimination as the cause for hypercapnia.

Depending on the cause, interventions may vary significantly, so the primary goal is to determine the underlying cause and attempt to fix it (i.e., reverse respiratory depressant medications, decrease anesthetic depth, relieve pleural space disorders). A $PaCO_2$ greater than 60 mm Hg is a significant problem and warrants immediate correction of the underlying problem.

### Decreased $PCO_2$ (hypocapnia)

Respiratory alkalosis, or hypocapnia, is a decrease in the partial pressure of carbon dioxide ($PaCO_2$ <32 mm Hg in the dog, <26 mm Hg in the cat). This commonly occurs secondary to lung disease, which stimulates pulmonary afferents to drive ventilation, or poor oxygenation, which triggers both central and peripheral chemoreceptors and increases ventilation. Poor oxygenation can be caused by primary lung disease (leading to low $PaO_2$ or hypoxemia) or from disease states that affect the ability to transport or use oxygen. Examples of disease processes that lead to poor tissue oxygenation not associated with pulmonary disease include anemia, decreased cardiac output (shock, poor cardiac contractility), and mitochondrial dysfunction (as with sepsis or cyanide toxicity). Also animals with metabolic acidosis have a compensatory respiratory alkalosis (from hyperventilation → low $PaCO_2$) in an attempt to normalize the pH. See Chapter 50, Acid-Base Evaluation, for more information about acid-base status. A drop in the $PaCO_2$ below 25 mm Hg can result in compromised cerebral blood flow and should be addressed immediately.[10]

### *Assessment of the partial pressure of oxygen in arterial blood ($PaO_2$)*

Oxygen is carried in the blood in only two forms. One form is the portion of gas dissolved in the blood and measured as a partial pressure ($PO_2$). This represents only 2%–3% of the total arterial blood oxygen content.[11] The other form is the oxygen bound to hemoglobin in the red blood cells. In arterial blood gas analysis, the $PaO_2$ is measured to evaluate the lung's ability to oxygenate blood, often called simply "lung function." The diffusion of oxygen into the pulmonary capillaries occurs via diffusion down a concentration gradient. Alveolar partial pressure of oxygen ($PAO_2$) is higher than the partial pressure of oxygen in the pulmonary capillary as it leaves the pulmonary arteriole and approaches the alveolus. At the alveolar-pulmonary capillary junction, oxygen passively diffuses across the alveolar membrane into the pulmonary capillaries. The rate of diffusion is driven by Fick's law, which states that the diffusion of a gas across a membrane is directly proportional to the area of the tissue membrane and the pressure gradient, and inversely proportional to the thickness of the membrane.[12] Clinically, this means that diffusion will be altered if the surface area for gas exchange is decreased or if the respiratory membrane is thickened (which is uncommon in small animals). Decreased surface area for gas exchange occurs secondary to airway constriction (low V/Q) or lung unit collapse or consolidation (no V/Q: intrapulmonary shunting). $PaO_2$ is essentially a measure of how many perfused lung units are ventilated adequately to allow oxygen to diffuse into the pulmonary capillary as it heads to the left heart for systemic circulation. Hence $PaO_2$ is used as a primary measure of pulmonary function.

A $PaO_2$ less than 80 mm Hg is considered hypoxemia, and oxygen supplementation should be considered. A $PaO_2$ less than 60 mm Hg is considered severe hypoxemia, and immediate intervention is necessary.

## Pulmonary function assessment using arterial blood gas data

Once arterial blood gas values are attained, further analysis may be helpful to determine the severity of the pulmonary dysfunction and to rule out hypoventilation as the cause of hypoxemia. A summary of these analyses is available in Box 22.3.

**Box 22.3** Pulmonary function assessment using arterial blood gas data

**Alveolar-arterial oxygen gradient**

- A-a gradient $= PAO_2 - PaO_2$
  $= [FiO_2(P_b - P_{H20}) - PaCO_2/RQ] - PaO_2$
  - Normal ≤15 mm Hg at $FiO_2$ 0.21 (room air), normal ≤150 mm Hg at $FiO_2$ 1.0 (100% oxygen); values in excess of 15 mm Hg on room air indicate pulmonary dysfunction
  - Use: At any $P_b$, at any $PaCO_2$
  - Reliable only at room air or 100% oxygen

**The "120" rule**

- $PaCO_2 + PaO_2 \geq 120$: adequate lung function
- $PaCO_2 + PaO_2 < 120$: abnormal lung function
  - Use: Only at sea level on room air

**$PaO_2/FiO_2$**

- $PaO_2/FiO_2$ = "P:F ratio"
  - Normal ≥500 mm Hg; mild pulmonary dysfunction 300–500 mm Hg; moderate pulmonary dysfunction consistent with ALI 200–300 mm Hg; severe pulmonary dysfunction consistent with ARDS <200 mm Hg
  - Use: At any $FiO_2$
  - Expected values apply only to sea level; extrapolated, reliable values would be available at other $P_b$

**$FiO_2 \times 5$ rule**

- Normal $PaO_2 \geq (FiO_2, \%) \times 5$
  - Use: At any $FiO_2$
  - Expected value of $PaO_2 \geq 5 \times FiO_2$ applies only to sea level; extrapolated, reliable values would be available at other $P_b$

**Total arterial oxygen content**

- $CaO_2 = (SaO_2 \times Hgb \times 1.34) + (0.003 \times PaO_2)$
  - Normal in dogs: 16.9–18.0 mL/dL
  - Relevant at all $P_b$, $PCO_2$, and $FiO_2$

## Alveolar to arterial gradient (A-a gradient)

After gas exchange occurs, the oxygen content in the pulmonary capillary is normally the same as the oxygen content in the alveolus ($PaO_2$ ~105 mm Hg at sea level on room air) because diffusion is complete. Oxygenated pulmonary capillary blood then flows to the left heart for systemic circulation. However, a small amount of blood (from bronchial and Thebesian circulations) normally returns to the left heart deoxygenated; this small amount of deoxygenated blood mixes with the arterialized blood returning from the pulmonary capillaries, which drops the $PaO_2$ below the $PAO_2$. The normal difference between $PAO_2$ and $PaO_2$ (the "**A-a gradient**" or "A-a difference") should be less than 15 mm Hg and is generally considered "physiologic shunting." However, when an increased amount of blood enters the left atrium without being oxygenated (for instance, because it perfused lung units that were not well ventilated due to a low V/Q or no V/Q scenario), the excessive deoxygenated blood further dilutes the properly arterialized blood coming from functional lung units. This situation is commonly referred to as **venous admixture** or intrapulmonary **shunting.** To determine whether there is increased pulmonary shunting, the A-a gradient can be calculated; increased gradients are associated with underlying pathology and help direct diagnostics and intervention.

$$\text{A-a gradient} = PAO_2 - PaO_2 = [FiO_2(P_b - P_{H20}) - PaCO_2/RQ] - PaO_2 \quad (22.2)$$

Where the $FiO_2$ is the fraction of inspired oxygen (0.21 or 21% on room air), $P_b$ is the barometric pressure (~760 mm Hg at sea level), and $P_{H20}$ is the vapor pressure of water. $P_{H2O}$ does vary with temperature, but generally 47 mm Hg is used because this is the vapor pressure of water at 37°C (human body temperature). The $PaCO_2$ is measured from the arterial blood gas sample, and RQ is the respiratory quotient, which is approximately 0.8. This assessment also works when the patient is receiving 100% oxygen, in which case the expected gradient is less than 150 mm Hg. For fractional inspired oxygen levels between room air and pure oxygen, the expected gradient has not been established and must be extrapolated.[5]

## The "120" rule

Because the $PCO_2$ affects $PAO_2$ (see expanded Equation 22.2), when an animal is at sea level breathing room air, one can estimate what $PaO_2$ to expect when one knows the $PaCO_2$ by using **the "120" rule.** When an animal is breathing *room air at sea level*, the sum of $PaCO_2$ and $PaO_2$ is generally 120 to 160 mm Hg. When the sum of $PaCO_2$ and $PaO_2$ is less than 120, pulmonary dysfunction is present.[5] The major inherent limitation to this method is the requirement for room air, sea level conditions. The major advantage is its ease of use.

$$PaCO_2 + PaO_2 \geq 120\text{: adequate lung function}$$
$$PaCO_2 + PaO_2 < 120\text{: abnormal lung function} \quad (22.3)$$

### $PaO_2/FiO_2$ ("the P:F ratio")

Equation 22.3 can be used to evaluate pulmonary function at any $FiO_2$, which provides an advantage over the A-a gradient and the 120 rule. The value is acquired simply by dividing the $PaO_2$ by the $FiO_2$:

$$PaO_2/FiO_2 = \text{"P:F ratio"} \quad (22.4)$$

where the $FiO_2$ is expressed as a decimal. With normal pulmonary function, the **P:F ratio** should exceed 500 mm Hg. The P:F ratio can be used to approximate the severity of pulmonary dysfunction; values between 300 and 500 mm Hg are associated with mild dysfunction, between 300 and 200 mm Hg are considered to have moderate dysfunction, and less than 200 mm Hg are considered to have severe pulmonary dysfunction. This calculated ratio, along with several other criteria such as acute onset of respiratory distress, bilateral dorsocaudal pulmonary infiltrates, absence of fluid overload or congestive heart failure (pulmonary artery occlusion pressure <18 mm Hg; see Chapter 12), and an appropriate underlying disease process, is also used to identify patients with acute lung injury (ALI) and acute respiratory distress syndrome (ARDS).[13] Values less than 500 mm Hg indicate there is compromised pulmonary function and definitive intervention and diagnostics are indicated. The advantage of this method is that it is simple, quick, and can be used at any $FiO_2$. The main disadvantage is the method's disregard for the effect of ventilation ($PCO_2$), although this is only really an issue when making the calculation on room air.

### $FiO_2 \times 5$

Another way to approximate the expected $PaO_2$ is to multiply the $FiO_2$ (as a percentage) by 5. With normal pulmonary function the $PaO_2$ would be approximately 100 mm Hg, which is about 5 times the $FiO_2$. This can then be extrapolated out to estimate what the $PaO_2$ should be with normal pulmonary function. Values attained that are lower than this indicate dysfunction.[5] Advantages and disadvantages are identical to those described for the P:F ratio.

### Calculation of the total oxygen content

As stated earlier, $O_2$ is carried in the blood two ways: dissolved in plasma and attached to hemoglobin. Total blood $O_2$ content can be calculated easily using the blood gas analyzer results. For arterial blood,

$$CaO_2 = (SaO_2 \times Hgb \times 1.34) + (0.003 \times PaO_2) \quad (22.5)$$

where $CaO_2$ is the total arterial blood oxygen content, $SaO_2$ is the saturation of hemoglobin with oxygen expressed as a decimal (see later), and 1.34 and 0.003 are constants. Normal $O_2$ content in dogs is reported to be 16.9 – 18.0 mL/dL,[14] although an idealized canine value is closer to 20 mL/dL. Note that red blood cell mass has a far more profound effect on total blood oxygen content than does $PO_2$ within the survivable range.

## Venous samples

Although arterial samples are preferable for assessment of both oxygenation and ventilation, a venous sample can also help evaluate the respiratory system.

### Venous $PCO_2$ ($PvCO_2$)

There is an expected arterial-venous gradient for $PCO_2$. Venous blood contains $CO_2$ from the metabolically active tissue bed(s) upstream from where the sample was acquired, and as such, it generally has a $PCO_2$ approximately 5 mm Hg higher than arterial blood. $CO_2$ produced in the tissues is carried in several forms by the blood to the lungs for removal. The dissolved $PCO_2$ represents approximately 10% of the total $CO_2$.[5] Most of the $CO_2$ is buffered within the red blood cell and then transported as bicarbonate to the lungs where this process is reversed to facilitate removal of $CO_2$ by the lungs. The disadvantage of venous samples is that in certain disease states, the **arterial-to-venous $PCO_2$ gradient** may increase. Such disease states include anemia, where there is a decrease in the ability to buffer the $CO_2$. Venous stasis will also increase the gradient. As a general guide, a $PvCO_2$ less than 48 mm Hg indicates hypoventilation assuming no concurrent perfusion compromise.

### Venous $PO_2$ ($PvO_2$)

The $PvO_2$ can be used to determine the **oxygen extraction ratio (OER)**, a ratio that sheds some light on the adequacy of oxygen delivery in comparison with the patient's oxygen consumption. Traditionally, calculation of the OER requires a mixed venous sample collected from the distal port of a pulmonary arterial catheter; however, blood from a central venous line (a catheter with a distal port near the right atrium) is probably adequate for most purposes. See Chapter 15, Monitoring Tissue Perfusion: Clinicopathologic Aids and Advanced Techniques, for more information regarding the oxygen extraction ratio. Normal $PvO_2$ values range from 40 to 50 mm Hg. As a general rule, when $PvO_2$ is less than 30 mm Hg, this may be an indication of decreased oxygen delivery, and diagnostics to determine the cause should be considered if the patient's clinical condition supports this finding.

## Saturation of hemoglobin with oxygen

Blood gas analyzers often report the saturation of oxygen ($SO_2$). This value represents the amount of oxygen bound to the hemoglobin molecule and is reported as a percentage of hemoglobin saturation with oxygen. Blood in which all hemoglobin oxygen sites are bound with oxygen is 100% saturated; blood in which 75% of hemoglobin oxygen sites are bound with oxygen is 75% saturated. The arterial hemoglobin oxygen saturation ($SaO_2$) is calculated from the $pO_2$, pH, and bicarbonate values; it assumes normal conditions for the calculation.[5] The oxyhemoglobin equilibrium curve shows the relationship between the $PaO_2$ and the $SaO_2$ graphically; it is a sigmoid curve where initially there is rapid binding of oxygen and the hemoglobin molecule. Pulse oximetry is a noninvasive method of estimating the $PaO_2$ using the oxyhemoglobin dissociation curve. See Chapter 22, Pulse Oximetry and CO-oximetry, for more information about the oxyhemoglobin equilibrium curve and these monitoring tools.

## Summary

Blood gas analysis allows detailed assessment of a patient's respiratory function. Arterial blood gases provide the opportunity to monitor a patient's pulmonary function in response to time and therapy, particularly when simple tools like the A-a gradient and the P:F ratio are used. Arterial and venous blood gases can be used to inform the clinician and technician about the patient's ventilatory adequacy. Venous oxygen tension can be used as a marker of adequacy of tissue perfusion. Blood gas analysis is readily available and relatively inexpensive with the use of bedside monitoring equipment.

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