Oxygen Delivery

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Abstract: Early recognition of failure of oxygen delivery and knowledge of how medications can alter oxygen delivery allow clinicians to institute appropriate therapies in a timely manner and can result in improved patient outcomes. Oxygen delivery can be estimated and evaluated using a variety of methods, including arterial blood gas sampling, blood lactate quantification, echocardiography, and direct cardiac output measurement. Delivery can be enhanced by manipulating the components of the oxygen delivery formula. Cardiac output, hemoglobin concentration, oxygen saturation, and oxygen tension can all be improved through therapeutic or pharmacologic intervention.

Oxygen (O₂) has been recognized as necessary for the maintenance of life since Joseph Priestley described the death of mice deprived of oxygen in 1775. Since that time, the importance of oxygen for normal cellular function has been confirmed repeatedly. Oxygen is required in sufficient amounts to maintain an adequate ATP level in the electron transport system. ATP is needed for essential (e.g., membrane transport, cellular repair and maintenance) and facultative (e.g., biosynthesis, contractility, transport of various molecules) cellular functions.¹

Failure of oxygen delivery (DO₂) is encountered commonly in veterinary medicine in patients with shock, anesthesia-associated hypotension, primary respiratory disease, acetaminophen toxicosis (cats), or carbon monoxide poisoning. Failure of DO₂ results in cellular dysfunction and death. To restore normal cellular function and stabilize patients in shock, DO₂ must be restored and improved. To fully appreciate the importance of the body's DO₂ system, it is essential to understand that oxygen is poorly soluble in blood and that specialized physiologic mechanisms have developed to overcome this limitation.

This article provides an overview of oxygen transport and a definition of DO₂. Methods of evaluating DO₂ will be discussed, as well as therapeutic interventions to improve DO₂.

Oxygen Delivery

DO₂ (mL/kg/min) is equivalent to the product of cardiac output (CO, L/min) and arterial oxygen content (CaO₂, mL O₂/dL). CO is defined as the volume of blood pumped by the heart in 1 minute. Determinants of CO are heart rate and stroke volume (SV, mL/kg/beat). Cardiac index (CI, L/min/m²) is a commonly used indicator of heart function that correlates CO with patient size using body surface area.

The major determinants of CaO₂ are hemoglobin (Hb) concentration (g/dL), hemoglobin saturation, and dissolved oxygen present in arterial blood. Hemoglobin increases the oxygen-carrying capacity of the blood by a factor of approximately 10.² In healthy animals, approximately 97% of oxygen transported from the lungs to the tissues is carried in combination with hemoglobin.³

Oxygen Transport

Transport of oxygen can be divided into four phases⁴:

- Gas exchange in the pulmonary system
- Interaction of oxygen with hemoglobin
- DO₂ to target organs via the hematologic and cardiovascular systems
- Oxygen consumption (VO₂, mL/kg/min) at the tissue level

Under normal, resting physiologic conditions, tissue VO₂ is constant. VO₂ is affected by changes in metabolic rate caused by increased levels of physical activity or pathophysiologic stress (e.g., sepsis). VO₂ can be calculated using the following formula:

\[ VO₂ = CO \times (CaO₂ - CvO₂) \]

where CvO₂ is venous oxygen content (mL O₂/dL). The calculation can be made more practical by reducing the formula for oxygen content, resulting in the following equation:

\[ VO₂ = CO \times 1.34 \times O₂/g \times Hb \times (SaO₂ - SvO₂) \]

in which Hb is the hemoglobin concentration (g/dL), SaO₂ is the arterial oxygen saturation, and SvO₂ is the mixed venous oxygen saturation.
Oxygen Delivery

**TABLE 1  Blood Gas Formulas With Normal Values**

<table>
<thead>
<tr>
<th>Formula</th>
<th>Normal Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>$VO_2 = CO \times (CaO_2 - CvO_2)$</td>
<td>110–160 mL/kg/min</td>
</tr>
<tr>
<td>$VO_2 = CO \times 1.34 \times Hb (SaO_2 - SvO_2)$</td>
<td></td>
</tr>
<tr>
<td>OER = $VO_2/DO_2$</td>
<td>0.2–0.3</td>
</tr>
<tr>
<td>OER = $(SaO_2 - SvO_2)/SaO_2$</td>
<td></td>
</tr>
<tr>
<td>$CaO_2 = (1.34 \times Hb \times SaO_2) + (0.003 \times PaO_2)$</td>
<td>20 mL/dL</td>
</tr>
<tr>
<td>$CaO_2 = (1.34 \times Hb \times SaO_2)$</td>
<td></td>
</tr>
<tr>
<td>$DO_2 = (CaO_2) \times (CO)$</td>
<td>0.3 – 0.5 mL/kg/min</td>
</tr>
<tr>
<td>$DO_2 = (1.34 \times Hb \times SaO_2) \times (HR \times SV)$</td>
<td>(DO$_{crit}$)</td>
</tr>
<tr>
<td>$Cl = CO \times$ body surface area (m$^2$)</td>
<td>5 L/min/m$^2$</td>
</tr>
</tbody>
</table>

---

**FIGURE 1**

Normal oxyhemoglobin dissociation curve.

is arterial oxygen saturation (%), and SvO$_2$ is venous oxygen saturation (%). The constant, 1.34 mL O$_2$/g, is the Hüfner factor, a species-specific constant that describes the amount of oxygen in milliliters that can be carried per gram of hemoglobin. Recommendations for Hüfner factor values are 1.39 for cats$^5$ and 1.34 to 1.39 for dogs.$^6$–$^10$ However, using formulas to calculate physiologic variables has inherent flaws attributable to individual variability and differences in perfusion of different organ systems.$^11$ Some organs (e.g., liver, intestines) are more vulnerable to hypoxic insult than others.$^12$

Hemoglobin’s affinity for oxygen is typically described using the oxyhemoglobin dissociation curve (FIGURE 1). This curve describes the expected percentage of hemoglobin saturation at a given partial pressure of oxygen in arterial blood (PaO$_2$, mm Hg). Any alteration in hemoglobin’s affinity for oxygen can then be described as shifting the curve to the left or right. A shift in the curve to the right means that oxygen is bound more loosely to hemoglobin, resulting in facilitation of oxygen off-loading at the tissue level. A shift to the left indicates that the oxygen is more tightly bound to the hemoglobin molecule and is less likely to be released to tissue. Conditions typically associated with impaired DO$_2$, such as acidosis, hyperthermia, and increased CO$_2$, result in shifting of the oxyhemoglobin dissociation curve to the right, making it easier for oxygen to be released from the hemoglobin molecule. Conditions that shift the curve to the left include alkalosis, hypothermia, carboxyhemoglobinemia, and methemoglobinemia. The neonatal oxyhemoglobin dissociation curve is shifted to the left.

**Oxygen Consumption**

The oxygen extraction ratio (OER) is a calculation used to describe how efficiently the body is extracting delivered oxygen. The OER depends on DO$_2$ and VO$_2$ (TABLE 1). Normal OER has been determined to be approximately 0.25, meaning that 25% of the oxygen delivered to the tissues is extracted or consumed under normal physiologic conditions.$^{13}$

**Estimating Oxygen Delivery**

Lactate is a by-product of anaerobic metabolism and is often used to assess adequacy of DO$_2$ relative to VO$_2$. Impairment of DO$_2$ causes alterations in pyruvate metabolism, with subsequent accumulation of lactic acid.$^{13}$ Although lactate levels are useful for evaluating anesthetized patients or patients presenting in shock, elevated lactate levels can have many causes, including anaerobic metabolism from pathologic and nonpathologic conditions and failure of normal clearance.

The gold standard for determining CO is by thermodilution or indicator dilution using a Swan-Ganz catheter placed in the pulmonary artery. Although this technology provides a very accurate reading, it is not practical for clinical use in most veterinary practices. A newer technique, lithium dilution CO (LiDCO; LiDCO cardiac sensor system, LiDCO Ltd, Coppell, TX), uses indicator (lithium chloride) dilution in an animal monitored via arterial and venous sampling catheters.$^{14,15}$ A bolus of lithium chloride is injected into a peripheral vein, and the diluted concentration is measured using the peripheral arterial catheter.$^{15}$ LiDCO has been validated in a number of species, including humans, pigs, horses,$^{16}$ dogs,$^{15}$ and cats.$^{17}$ Using echocardiography, left ventricular SV can be estimated and used to calculate CO.$^{16}$

Determining CaO$_2$ is less technically demanding and can be performed in most fully equipped veterinary hos-
pitals. The first step is to determine hemoglobin concentration. This can be done by direct measurement or estimated by the formula \( Hb = 0.30 \times \text{hematocrit (HCT)} \). The second step in calculating \( \text{CaO}_2 \) involves determination of arterial hemoglobin saturation. This is done using arterial blood gas analysis to determine \( \text{SaO}_2 \) or pulse oximetry (SpO2) to assess hemoglobin oxygen saturation. Although blood gas analysis provides a value for \( \text{SaO}_2 \), this value is often derived by comparing measured \( \text{PaO}_2 \) to the normal oxyhemoglobin dissociation curve. Factors affecting this curve, such as changes in \( \text{pH} \), \( \text{CO}_2 \) level, temperature, and 2,3-diphosphoglycerate level, should be corrected before measurement if possible.

Pulse oximetry operates on the principle that red and infrared light are absorbed at different rates by oxygenated and reduced hemoglobin. The pulse oximeter measures the differential light absorption in pulsatile blood flow. This technology relies on the assumption that only two hemoglobin species (oxyhemoglobin and reduced hemoglobin) are present. Several factors can affect the accuracy of SpO2, including the presence of nonfunctional hemoglobin species (e.g., methemoglobin, carboxyhemoglobin), fluorescent light, motion, and low peripheral perfusion resulting in lack of pulsatile blood flow. Pulse oximetry errors usually result in an underestimation of actual \( \text{SaO}_2 \) and most often prompt institution of more aggressive therapy. When possible, \( \text{SaO}_2 \) should be used to calculate arterial oxygen content.

**Shock and Oxygen Delivery**

Under shock conditions, \( \text{DO}_2 \) fails to meet oxygen demand, leading to critical \( \text{DO}_2 \) (\( \text{DO}_{2\text{crit}} \)), which is defined as the level of \( \text{DO}_2 \) below which anaerobic metabolism begins and cellular function deteriorates. Due to the normally large oxygen reserves in hemoglobin, the consumption of oxygen is independent of \( \text{DO}_2 \) over a wide range. When the cellular requirements for oxygen begin to exceed the oxygen available, the body is in a state of shock. \( \text{DO}_{2\text{crit}} \) has been reported in the veterinary literature as ranging from 3.0 to 11.0 mL/kg/min. One possible explanation for the large variation in reported \( \text{DO}_{2\text{crit}} \) values in veterinary patients is that most of these studies are conducted on animals under sedation or anesthesia. It is reasonable to assume that the \( \text{DO}_{2\text{crit}} \) of a healthy animal under anesthesia may not translate directly to clinical patients.

The body has several specialized tissues for sensing decreased oxygen tension and maintaining \( \text{DO}_2 \): the glomus cells of the carotid bodies, neuroepithelial bodies in the airway mucosa, and smooth muscle cells of pulmonary resistance vessels and systemic arteries. Oxygen-sensing capabilities have also been described in neurons, alveolar epithelial cells, and T lymphocytes. The cells of the carotid bodies are activated when arterial oxygen pressure is less than 60 mm Hg. On activation, they cause a sensitization of breathlessness and an increase in respiratory rate and tidal volume. These specialized cells detect decreased \( \text{DO}_2 \) through hypoxic inhibition of potassium channels. The blocking of potassium channels ultimately results in neurosecretion of dopamine and alterations in blood vessel tone, blood flow, and respiratory pattern to optimize \( \text{DO}_2 \).

Under shock conditions, blood flow redistribution away from the kidneys, liver, splanchnic bed, and musculoskeletal system allows the whole body to maintain a state of supply independence for a longer period of time, meaning that aerobic metabolism is not limited by \( \text{DO}_2 \). Initially, tissues that are deprived of oxygen attempt to continue aerobic metabolism by increasing oxygen extraction. This increase in oxygen extraction is effective in most situations, but as shock continues, \( \text{DO}_2 \) falls below \( \text{DO}_{2\text{crit}} \) and the body’s oxygen reserves are quickly exhausted. Once \( \text{DO}_{2\text{crit}} \) is reached, serum lactate levels increase, indicating that anaerobic metabolism is taking place. At this point (i.e., the anaerobic threshold), \( \text{VO}_2 \) becomes supply-dependent (FIGURE 2) and \( \text{DO}_2 \) is the rate-limiting step to aerobic metabolism. If \( \text{DO}_{2\text{crit}} \) is not restored, shock progresses and, ultimately, complete circulatory collapse results.

**Drugs That Affect Oxygen Delivery**

Many drugs that are routinely administered in veterinary medicine may affect \( \text{DO}_2 \). These include nearly all drugs used for anesthesia and sedation. Therefore, it is extremely important to maximize \( \text{DO}_2 \) before induction of anesthesia or administration of sedation. Patients should be preoxygenated with a face mask, and blood pressure, hemoglobin concentration, and CO should be corrected before administration of any anesthetic drugs. When formulating an anesthetic plan for a patient in which \( \text{DO}_2 \) may be impaired, the clinician should select the medication least likely to result in an adverse effect. In general, opioids and benzodiazepines are considered safe because when used at recommended doses, they have few adverse cardiovascular effects, and the most significant adverse effect of these drugs is mild.
The use of propofol in veterinary medicine has increased. Dissociative drugs such as ketamine have sympathomi-

cation, and preload. Methods to improve DO$_2$ should focus on

- Failure of adequate oxygen delivery is a hallmark of shock.
- Many of the determinants of oxygen delivery can be manipulated by therapeutic intervention.

Improving Oxygen Delivery

CO can be manipulated by altering its primary determinants: chronotropy (heart rate), inotropy (force of contrac-

Key Points

- Under normal conditions, oxygen delivery far exceeds oxygen consumption.
- Failure of adequate oxygen delivery is a hallmark of shock.
- Many of the determinants of oxygen delivery can be manipulated by therapeutic intervention.

Anesthetic induction and maintenance drugs—including barbiturates, propofol, phenothiazines, opioids, α$_2$ agonists, and inhaled anesthetics—are capable of causing similar reductions in CO and can have deleterious effects on DO$_2$. The α$_2$ agonists are widely used in veterinary medicine for their ease of administration, predictable effect, and reversibility. However, these drugs have potent effects on the cardiovascular system and dramatically reduce CO and CI. Although CI has been shown to decrease by up to 50% with administration of medetomidine, it appears unlikely that DO$_{2\text{crit}}$ is reached. The use of supplemental oxygen is recommended when using medetomidine or medetomidine combinations to avoid potential hypoxia and decreased DO$_2$.

The use of propofol in veterinary medicine has increased in the past several years due to its desirable induction and recovery qualities. Although propofol is generally considered safer than barbiturates, its use can result in profound decreases in CO, myocardial contractility, systemic blood pressure, and right atrial pressure. Propofol can also induce apnea and, with repeated doses, may cause Heinz body formation that can impair DO$_2$ by causing hypoxemia and anemia.

Dissociative drugs such as ketamine have sympathomimetic properties that increase CO, contractility, blood pressure, and heart rate. These medications can provide some support to the cardiovascular system when used in critically ill animals, but they can also increase myocardial oxygen consumption.

Etomidate is an imidazole derivative that induces anesthesia in a manner similar to propofol. Single doses are capable of inducing brief hypnosis with very little adverse effect on CO. Unfortunately, the cost of stocking etomidate limits its use in general practice. All the volatile gas anesthetics currently used in veterinary medicine cause decreased CO and dose-dependent systemic hypotension.

Improving Oxygen Delivery

CO can be manipulated by altering its primary determinants: chronotropy (heart rate), inotropy (force of contradistinction), and preload. Methods to improve DO$_2$ should focus on the variables that are most easily manipulated, including CO, inspired oxygen content, and hemoglobin concentration. It is also important to reverse any anesthetic medications that can be reversed and to treat metabolic conditions that can shift the oxyhemoglobin dissociation curve.

Perhaps the most widely used and most available method of improving DO$_2$ is replenishment of circulating volume with intravenous (IV) fluids. Although crystalloid and colloidal solutions do not have oxygen-carrying properties, they improve CO and, subsequently, DO$_2$, based on the Frank-Starling law of the heart.$^{13}$ According to this law, the heart is obligated to pump the entire volume of blood that is returned from the peripheral and pulmonary circulation. All patients in which heart failure has been excluded as the cause of suspected or documented failure of DO$_2$ should receive IV fluids until circulating volume has been restored and ongoing losses have been controlled.

The anticholinergic medications atropine and glycopyrrolate are commonly used to increase heart rate. These medications exert their effect by abrogating vagal tone, allowing sympathetic stimulation to predominate. As the heart rate increases, so does myocardial oxygen consumption. If myocardial hypoxia is a factor in the disease process, as with cardiogenic forms of shock or underlying myocardial disease, caution should be used when administering anticholinergic drugs to avoid exacerbation of myocardial compromise.

Inotropy can be improved through the use of adrenergic agonists (TABLE 2). The adrenergic receptors (adrenoceptors) are a component of the sympathetic nervous system and can be classified according to type—α and β—and subtype (α$_1$; α$_2$; β$_1$; β$_2$). The α$_1$ receptors are found in both the central and peripheral nervous systems and are responsible for maintenance of vascular tone. The α$_2$ receptors are also found within the central and peripheral nervous systems and typically serve to produce inhibitory functions, although activation of α$_2$ receptors in the peripheral nervous system results in vasoconstriction. The β$_1$ receptors are primarily located in the heart, where activation increases chronotropy and inotropy. The β$_2$ receptors predominate in blood vessels, smooth muscle of the airways, and gastrointestinal tract. Activation of these receptors results in vasodilation and bronchodilation. Dopaminergic receptors are found in nearly every organ system, but the effects of these receptors on the central nervous and cardiovascular systems are most important for DO$_2$. Centrally, the dopaminergic receptors are responsible for cognition and locomotor activity. In the cardiovascular system, stimulation of only the dopaminergic receptors increases inotropy and vasodilation.

Common adrenergic agonists include epinephrine, nor-

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impar DO2 by limiting blood flow via peripheral vasoconstriction. Epinephrine and norepinephrine are α and β agonists that have positive inotropic and chronotrophic effects. Norepinephrine typically produces smaller changes in inotropy and CO than epinephrine. Phenylephrine is an α agonist that has little effect on cardiovascular variables such as CO, inotropy, and heart rate but causes a significant increase in systemic blood pressure. Dobutamine is primarily a β1 agonist that increases inotropy and chronotropy with little effect on the systemic vascular tree.32 Because of these properties, dobutamine should be considered the adrenergic agonist of choice in conditions resulting from systolic cardiac failure (e.g., cardiogenic shock). Another widely available adrenergic agonist is dopamine, which has both α and β effects. Dopaminergic effects predominate at doses of 1 to 3 μg/kg/min, and β adrenergic effects predominate at 4 to 10 μg/kg/min.33 At higher doses (10 to 15 μg/kg/min), the α effects begin to predominate, resulting in profound peripheral vasoconstriction and increased systemic vascular resistance. Dopamine is a less powerful positive inotrope than epinephrine or dobutamine, so it is often used in combination with other adrenergic agonists.35 When dopamine is combined with dobutamine, the result is a greater increase in inotropy with no significant impact on systemic vascular resistance.

Arterial oxygen content and, subsequently, DO2 can also be improved through increasing hemoglobin concentration through administration of red blood cells or hemoglobin-based oxygen carriers (HBOCs). The reported optimal hemoglobin concentration in healthy dogs and cats is approximately 12 g/dL (hematocrit 30%).2 When the hematocrit increases to 60%, blood viscosity is altered secondary to actual or relative polycythemia. Polycythemia may cause hyperviscosity and a resultant reduction in blood flow through the microvasculature, with possible induction of ischemic injury.34 The concept of a “transfusion trigger” (a hemoglobin concentration/hematocrit level below which administration of red blood cells is indicated) has been a topic of debate for many years. Although there is no consensus, human and veterinary literature suggests that the critical hemoglobin concentration may be as low as 3 to 5 g/dL (hematocrit 9% to 15%) before DO2 is adversely affected.2 DO2 can also be improved by eliminating nonfunctional hemoglobin species (e.g., treating methemoglobinemia in patients with acetaminophen toxicosis and carboxyhemooglobinemia in patients with smoke inhalation).

Although theoretically beneficial, in practical use, HBOCs may lead to profound vasoconstriction through free-radical scavenging of nitric oxide.35 HBOCs can also result in hemodilution due to their profound effect on colloid osmotic pressure. Polymerized hemoglobin tetramers behave as active osmoles and draw water from the interstitial space into the intravascular space, resulting in a dilutional decrease in hemoglobin.22 This hemodilution may negate the benefits of the increased oxygen-carrying capacity provided by the hemoglobin.

DO2 can also be improved through maximizing hemoglobin saturation and increasing the amount of oxygen that is carried in the blood in dissolved form. SaO2 can be improved through the elimination of nonfunctional hemoglobin species and the provision of supplemental oxygen. Raising the fraction of inspired oxygen (FiO2) helps to ensure that hemoglobin is maximally saturated on leaving the lungs, may increase the SaO2 from 88% to 95%, and can significantly improve DO2.

Dissolved plasma concentrations of oxygen can be raised minimally through increasing FiO2. This can be done by giving oxygen via nasal cannulation, oxygen cage, or mechanical ventilation. The amount of oxygen that is dissolved in the plasma depends on barometric pressure and the oxygen tension of inspired air. The oxygen tension in arterial blood is equal to the alveolar partial pressure of oxygen minus the normal physiologic gradient of 5 to 15 mm Hg. In healthy animals, the amount of oxygen that is dissolved in the blood is generally small. In the presence of anemia, nonfunctional hemoglobin species, or diffusion impairment in the lungs, increasing the FiO2 can result in an important improvement in this aspect of DO2. Supplemental oxygen should always be provided to a patient that is suspected of having DO2 impairment.

### Conclusion

The concept of DO2 may seem academic, but it is clinically relevant for conditions that occur frequently in general veterinary practice and daily in a busy emergency practice.

### TABLE 2 Adrenoceptor Selectivity and Activity of Common Adrenergic Agonists

<table>
<thead>
<tr>
<th>Agonist</th>
<th>α1</th>
<th>α2</th>
<th>β1</th>
<th>β2</th>
<th>Dopaminergic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isoproterenol</td>
<td>0</td>
<td>0</td>
<td>+++</td>
<td>+++</td>
<td>0</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>?</td>
<td>?</td>
<td>+++</td>
<td>++</td>
<td>0</td>
</tr>
<tr>
<td>Dopamine</td>
<td>++</td>
<td>+</td>
<td>++</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>+++</td>
<td>+++</td>
<td>++</td>
<td>+</td>
<td>0</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>+++</td>
<td>+++</td>
<td>+/++</td>
<td>/++</td>
<td>0</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>+/++</td>
<td>+</td>
<td>?</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

*α = relative activity.

Once DO₂ is understood, it can be broken down to its components, allowing interventions to be directed toward specific results.

References
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#### CE TEST

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<table>
<thead>
<tr>
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</thead>
</table>
| **1.** In the electron transport system, oxygen is responsible for maintaining an adequate level of | a. ADP.  
   b. ATP.  
   c. phosphocreatine.  
   d. pyrophosphate. |
| **2.** In veterinary medicine, DO₂ is not generally associated with | a. respiratory disease.  
   b. anesthesia-associated hypotension.  
   c. ethylene glycol toxicosis.  
   d. shock. |
| **3.** DO₂ is equal to the product of CaO₂ and | a. CO.  
   b. PaO₂.  
   c. CI.  
   d. CvO₂. |
| **4.** The amount of oxygen per milliliter that can be carried per gram of hemoglobin is described by | a. the OER.  
   b. HbO₂ × 1.2. |
| **5.** The oxyhemoglobin dissociation curve shifts to the left when | a. the oxygen-carrying capacity of hemoglobin increases.  
   b. the oxygen-carrying capacity of hemoglobin decreases.  
   c. oxygen is bound more tightly to hemoglobin.  
   d. oxygen is bound more loosely to hemoglobin. |
| **6.** A practical, widely available way of determining CO in veterinary practice is | a. Doppler echocardiography.  
   b. placing a Swan-Ganz catheter in the pulmonary artery.  
   c. using a peripheral arterial catheter to measure lithium chloride dilution.  
   d. indirect arteriography. |
| **7.** Sedatives and anesthetics that have a minimal impact on DO₂ include | a. barbiturates.  
   b. benzodiazepines.  
   c. phenothiazines.  
   d. α₂ agonists. |
| **8.** One of the most available methods of improving DO₂ is IV administration of | a. crystalloid and colloidal solutions.  
   b. glycopyrrolate.  
   c. magnesium sulfate.  
   d. atropine. |
| **9.** Adrenergic agonists are administered to improve cardiac | a. dromotropy.  
   b. preload.  
   c. inotropy.  
   d. chronotropy. |
| **10.** DO₂ can be improved by administering ______ to raise the hemoglobin concentration. | a. plasma  
   b. red blood cells  
   c. erythropoietin  
   d. iron supplements |

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