Mechanical ventilation is currently an uncommon supportive measure in veterinary medicine, and its use is largely restricted to academic institutions and specialty practices. As the discipline of veterinary critical care continues to grow, application of mechanical ventilation will likely become more widespread. Human intensive care physicians view mechanical ventilation as an essential aspect of critical patient care, with 1.5 million patients ventilated yearly in the United States. It is inevitable that mechanical ventilation will have a significant role in veterinary medicine.

Successful application of positive-pressure ventilation (PPV), the most prevalent form of mechanical ventilation, requires appropriate patient selection, an understanding of ventilator function, and most important, intensive nursing care. Many veterinary patients can benefit from PPV. It is hoped that the perception of the ventilator as a harbinger of death will change with time so that it can be viewed as a useful and lifesaving adjunct to critical care.

INDICATIONS
Mechanical ventilation is indicated in patients with severe hypoxemia despite oxygen therapy, severe hypercapnia, or excessive work of breathing that do not resolve with less invasive therapy. The prognosis varies with the underlying disease and the degree of pulmonary pathology. This article reviews the indications, goals, and prognosis of mechanical ventilation in small animals.
HYPOXEMIA

Recognition

Hypoxemia is defined as a partial pressure of oxygen in the arterial blood (PaO₂) of less than 80 mm Hg. A PaO₂ of less than 60 mm Hg is considered severe hypoxemia and warrants rapid intervention. Measuring PaO₂ requires an arterial blood sample and a blood gas analyzer.

π

Pulse oximetry measures the percent saturation of hemoglobin with oxygen (SpO₂) in pulsatile vessels. Although simple to use, pulse oximeters can be inaccurate, especially in moving, darkly pigmented, or poorly perfused animals; thus they should be interpreted with caution. Based on the oxyhemoglobin dissociation curve, an SpO₂ of 96% should approximate a PaO₂ of 80 mm Hg, whereas an SpO₂ of 91% should correspond to a PaO₂ of approximately 60 mm Hg. Pulse oximetry is not only prone to inaccuracy but also insensitive to changes in oxygenation at levels of PaO₂ greater than 100 mm Hg. For example, a patient receiving 100% inspired oxygen is expected to have a PaO₂ of approximately 500 mm Hg and a corresponding pulse oximeter reading of 99% to 100%. This patient’s ability to oxygenate could drop significantly so that the PaO₂ falls as low as 100 to 120 mm Hg without a corresponding change in the pulse oximeter reading. Therefore, pulse oximeter readings of 99% to 100% in animals receiving oxygen therapy can be interpreted only as adequate. There is no way to ascertain whether the degree of oxygenation is appropriate for the animal’s level of inspired oxygen. Accurate pulse oximeter readings of less than 99% in animals receiving supplemental oxygen can be considered abnormal.

Although the partial pressure of oxygen in venous blood (PvO₂) is not reflective of pulmonary function, when all other measurements are unavailable, it can be used as an indication that an animal is receiving adequate oxygen to the tissue. A PvO₂ of less than 30 mm Hg suggests that the total content of delivered oxygen is inadequate to meet the patient’s oxygen consumption. A low PvO₂ can be a consequence of reduced oxygen delivery and/or increased oxygen consumption and cannot be simply interpreted as an indicator of pulmonary dysfunction. When interpreting PvO₂ in patients with cardiovascular compromise, it is recommended that only central venous or mixed venous samples be evaluated. Peripheral venous samples can reflect abnormalities of the local tissue bed that are not globally representative of the patient.

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**Approximately 50% of dogs and cats ventilated for neuromuscular pathology are successfully weaned from the ventilator compared with 25% of those with pulmonary pathology.**
Cyanosis is blue discoloration of the mucous membranes indicating the presence of deoxygenated hemoglobin. Once cyanosis has been appreciated, the PaO₂ is 50 mm Hg or less and hypoxemia is severe; this is a late indicator of respiratory failure.\textsuperscript{12,13}

**Evaluation**

The PaO₂ must be interpreted with regard to the fraction of inspired oxygen (FIO\textsubscript{2}) at the time of the measurement. This evaluation allows a clinician to determine whether the animal’s ability to oxygenate is normal. Abnormal or lower than expected PaO₂ measurements can be quantified to reflect the degree of disease severity and allow comparisons between PaO₂ measurements in the same patient at different times and on varying levels of FIO\textsubscript{2}.

In patients with healthy lungs, the PaO₂ should be similar to the partial pressure of oxygen in the alveolus (P\textsubscript{A}O\textsubscript{2}). Calculation of the alveolar–arterial (A-a) gradient gives a measure of pulmonary dysfunction by evaluating the difference between these two partial pressures. The PaO₂ primarily depends on the inspired partial pressure of oxygen (P\textsubscript{I}O\textsubscript{2}) and the quantity of carbon dioxide (CO\textsubscript{2}) in the alveolus. PaO₂ is calculated by the alveolar air equation. The A-a gradient can then be determined by subtracting the PaO₂ from the P\textsubscript{A}O\textsubscript{2}.\textsuperscript{14} The A-a gradient (see the box on page 196) is one of the few calculations of oxygenation that accounts for the impact of changes in partial pressure of arterial carbon dioxide (P\textsubscript{A}CO\textsubscript{2}).\textsuperscript{14} A normal A-a gradient is less than 15 mm Hg when breathing room air (i.e., 21% oxygen).\textsuperscript{5} The normal A-a gradient increases as the FIO\textsubscript{2} increases (approximately 5 to 7 mm Hg per 10% increase in FIO\textsubscript{2}).\textsuperscript{15} If the A-a gradient is normal in a hypoxemic patient, the cause of hypoxemia is either hypercapnia or decreased inspired oxygen. An increased A-a gradient usually indicates the presence of pulmonary or cardiovascular pathology.\textsuperscript{14}

The PaO\textsubscript{2}:FIO\textsubscript{2} ratio (see the box on this page) is a less complicated method for quantifying the ability to oxygenate at different levels of FIO\textsubscript{2}. Dividing the value for PaO\textsubscript{2} by the decimal value of FIO\textsubscript{2} yields the ratio. A normal PaO\textsubscript{2}:FIO\textsubscript{2} ratio is 500. A PaO\textsubscript{2}:FIO\textsubscript{2} ratio of 300 to 500 is consistent with mild disease, 200 to 300 with moderate disease, and less than 200 with severe pathology.\textsuperscript{5,16} The PaO\textsubscript{2}:FIO\textsubscript{2} ratio is a quick method of evaluating oxygenation and is widely used in human medicine but is less accurate than the A-a gradient because it does not account for the influence of varying partial pressure of carbon dioxide (P\textsubscript{A}CO\textsubscript{2}) levels.

The five times rule is an approximate guideline for predicting the expected normal PaO₂ in a patient at sea level for a given FIO\textsubscript{2}. The FIO\textsubscript{2} measured as a percentage and multiplied by five is the approximate PaO₂ expected in an animal with normal lungs.\textsuperscript{16,17} Therefore, a patient on room air would be expected to have a PaO₂ of approximately 100 mm Hg, which should increase to approximately 500 mm Hg on 100% oxygen.

**Causes**

The three main causes of hypoxemia are a low FIO\textsubscript{2}, hypoventilation, and venous admixture\textsuperscript{5} (see the box on this page). A low inspired level of oxygen can occur with equipment malfunction or human error when establishing a patient on a breathing circuit. When hypoxemia is identified in an animal on a breathing circuit, it is essential to ensure that the oxygen supply is adequate before considering other causes of hypoxemia.

Hypoventilation by definition causes hypercapnia. In patients breathing room air, this elevated CO\textsubscript{2} level can cause significant dilution of the PaO₂ so that a patient can become hypoxemic. Patients with this hypoxemia readily respond to oxygen therapy.\textsuperscript{18} Therefore, hypercapnic animals should receive oxygen supplementation while the cause of elevated P\textsubscript{A}CO\textsubscript{2} is addressed. If hypercapnia is severe and the primary cause cannot be rapidly

<table>
<thead>
<tr>
<th><strong>The PaO\textsubscript{2}:FIO\textsubscript{2} Ratio</strong></th>
<th><strong>Causes of Hypoxemia\textsuperscript{5}</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>FIO\textsubscript{2} = 40% = 0.40 PaO\textsubscript{2} = 200 mm Hg PaO\textsubscript{2} : FIO\textsubscript{2} = 500</td>
<td>Low inspired oxygen\textsuperscript{a}</td>
</tr>
<tr>
<td>FIO\textsubscript{2} = 80% = 0.80 PaO\textsubscript{2} = 200 mm Hg PaO\textsubscript{2} : FIO\textsubscript{2} = 250</td>
<td>• High altitude</td>
</tr>
<tr>
<td>Assessment: Normal</td>
<td>• Equipment malfunction</td>
</tr>
</tbody>
</table>

\textsuperscript{a}Likely to be very oxygen responsive.
resolved, the animal may require PPV to restore a more acceptable P\textsubscript{CO\textsubscript{2}}.

Venous admixture, the most common cause of hypoxemia, refers to blood passing from the right to left side of the circulation without being fully oxygenated, thus diluting arterial blood with deoxygenated blood.\textsuperscript{5} Causes of venous admixture can be divided into right-to-left anatomic shunts, diffusion defects, and ventilation-perfusion (V/Q) mismatch. Clinically, V/Q mismatch is by far the most important cause of hypoxemia.\textsuperscript{5}

Pathologic anatomic shunts are associated with abnormal communication between the right and left sides of the circulation (e.g., right-to-left shunting ventricular septal defects, right-to-left shunting patent ductus arteriosus) and tend not to be oxygen responsive.\textsuperscript{16}

Diffusion defects are caused by thickening or pathology of the gas exchange surface of the alveoli. Although frequently discussed, diffusion defects are rare because the diffusion surface of the alveolus is highly protected by virtue of its anatomic structure.\textsuperscript{12,16} Although pulmonary parenchymal diseases such as pulmonary edema can increase the thickness of the pulmonary interstitium, there is minimal change to the barrier to gas exchange between the surface of the alveolar epithelial cell and the pulmonary capillary. The abnormality in gas exchange occurring in patients with pulmonary edema is largely due to alveolar flooding and collapse leading to V/Q mismatch.\textsuperscript{5}

Venous admixture occurring in patients with pulmonary parenchymal disease is due to discrepancies in the ventilation and perfusion of alveoli. These changes are best characterized by the V/Q ratio (Figure 1).\textsuperscript{19} The ideal alveolus is optimally ventilated and perfused, resulting in a V/Q of 1. Inadequately ventilated alveoli with low V/Q and collapsed alveoli, which have no V/Q, contribute to venous admixture and can result in hypoxemia or a lower than expected Pa\textsubscript{O\textsubscript{2}}.\textsuperscript{19}

Diseases that create partial filling of alveoli with fluid or partial obstruction of the terminal airways result in areas of low V/Q. These alveoli are still capable of gas exchange, and increasing the F\textsubscript{IO\textsubscript{2}} improves the arterial oxygen content of the capillaries perfusing these regions. In no-V/Q areas, the alveoli or airways are completely occluded (i.e., ventilation = 0) and are therefore not able to contribute to gas exchange even if the F\textsubscript{IO\textsubscript{2}} is increased. Oxygen therapy is therefore not effective in regions of no V/Q.\textsuperscript{17} Patients with a large proportion of no-V/Q regions usually have pulmonary parenchymal disease, fail to respond to oxygen therapy, and require ventilation. PPV may reopen some of these collapsed, no-V/Q alveoli and convert them into functional gas-exchange units—a process referred to as recruitment. This is one of the major benefits of PPV in patients with pulmonary parenchymal disease. In reality, most pulmonary diseases create a heterogenous mixture of low- and no-V/Q areas. As the relative proportion of no-V/Q regions increases, the effectiveness of oxygen therapy declines and the requirement for PPV increases.

The goals of mechanical ventilation include optimizing oxygenation and ventilation, reducing the work of breathing, and allowing patient stabilization while diagnostic and therapeutic plans are implemented with the ultimate goal of weaning the patient from the ventilator.
All pulmonary parenchymal diseases can create collapse or filling of alveoli, leading to V/Q mismatch. Examples include pneumonia, cardiogenic and noncardiogenic pulmonary edema, pulmonary contusions, infiltrative neoplasia, pulmonary hemorrhage, and compressive masses.¹²,¹⁶,²⁰

**Diagnostic Approach**

Initial management of hypoxemic patients should always include oxygen therapy. Evaluation of hypoxemic patients can follow a simple diagnostic algorithm (Figure 2).

For intubated patients on a breathing circuit, the first concerns are the adequacy of oxygen supply and correct placement of the endotracheal tube. Once the patient is receiving the appropriate FIO₂, the remaining causes of hypoxemia are hypoventilation or venous admixture.

Hypoventilation is identified by an elevated PCO₂ and results from an inadequate respiratory rate and/or tidal volume and causes an elevated PCO₂.²¹ Patients may have both hypoventilation and venous admixture; therefore, it is important to confirm the impact of an elevated PCO₂. Calculating the alveolar air equation in patients with hypoventilation alone reveals a normal A-a gradient, confirming that hypoventilation is the sole cause of hypoxemia. Animals with hypoventilation as their primary problem frequently have no evidence of pul-

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**Figure 2. Diagnostic approach to hypoxemic patients.**
Mechanical Ventilation: Indications, Goals, and Prognosis

The indications for ventilation include hypoxemia, hypercapnia, or excessive work of breathing that is not adequately responsive to other therapies.

Diopulmonary arrest. These animals should be promptly anesthetized and intubated to gain control over their ventilation. They can be manually ventilated on 100% oxygen with a nonrebreathing circuit or anesthetic machine until they can be safely established on a ventilator.

Patients that require a high FiO2 to maintain adequate oxygenation may also be candidates for mechanical ventilation because of the risk of oxygen toxicity. Exposure to 60% or more oxygen for longer than 24 hours is believed to lead to pulmonary damage. The full impact of these toxic effects may not be appreciated until the damage is irreversible. Mechanical ventilation can recruit collapsed alveoli and improve gas exchange so that patients may achieve acceptable blood gas values on a lower FiO2 with PPV compared with spontaneous breathing. Ventilatory assistance should therefore also be considered in patients that require an FiO2 of greater than 60% for longer than 24 hours.

**HYPERCAPNIA**

**Recognition**

The arterial CO2 level is an indicator of the adequacy of alveolar ventilation. A PaCO2 greater than 43 mm Hg in dogs and greater than 36 mm Hg in cats is considered elevated, whereas a PaCO2 greater than 60 mm Hg suggests severe hypoventilation and warrants therapy. An elevated Pco2 causes acidosis and can have serious metabolic and neurologic consequences. Elevated Pco2 leads to cerebral vasodilation and increased intracranial pressure. Patients with preexisting brain disease may not tolerate such fluctuations in intracranial pressure and often require their Pco2 to be maintained in a narrow range of 35 to 45 mm Hg to prevent deterioration of their neurologic status. Although a PaCO2 greater than 60 mm Hg despite therapy is an indication for PPV in most patients, a Pco2 of greater than 45 mm Hg may be an indication for PPV in animals with brain disease.

Although venous oxygen measurements do not reflect arterial oxygen values, venous PCO2 (PvCO2) values are usually 3 to 6 mm Hg higher than arterial values and therefore provide a good indication of ventilatory status. In animals with poor perfusion, a central venous sample is more accurate than a peripheral Pco2.

A capnograph, which allows measurement of end-tidal CO2 (ETCO2), can also be used to assess the adequacy of ventilation. These monitors measure the CO2 levels of expired gas, and the last portion of exhalation is assumed to have a composition similar to alveolar gas. ETCO2 is approximately 2 to 6 mm Hg lower than PaCO2 in dogs. ETCO2 is not an accurate reflection of PaCO2 in patients with poor perfusion or substantial alveolar dead space as seen in patients with pulmonary thromboembolism; therefore, it is important to compare the ETCO2 with at least one blood gas analysis when possible.

**Causes**

Hypoventilation leading to ineffective alveolar ventilation elevates Pco2. Effective alveolar ventilation is the portion of the tidal volume that reaches the alveoli and participates in gas exchange. A decrease in minute ventilation, which is the product of the tidal volume and the

Patients with a lower than expected ability to oxygenate and an abnormal A-a gradient have venous admixture. Most of these animals have pulmonary parenchymal disease and would be expected to have radiographic changes. A small number of patients with an abnormal A-a gradient have an anatomic shunt. These animals are identified usually by history and signalment and possibly by the presence of a cardiac murmur. They may have cardiac and/or pulmonary changes evident on thoracic radiographs.

The severity of pulmonary parenchymal disease can be estimated by calculating the A-a gradient, PaO2:Fio2 ratio, and response to oxygen therapy. Patients that do not achieve adequate oxygenation despite oxygen therapy are candidates for ventilation and should be recognized and treated aggressively before they are in danger of cardiopulmonary arrest. These animals should be promptly anesthetized and intubated to gain control over their ventilation. They can be manually ventilated on 100% oxygen with a nonrebreathing circuit or anesthetic machine until they can be safely established on a ventilator.

Patients that require a high Fio2 to maintain adequate oxygenation may also be candidates for mechanical ventilation because of the risk of oxygen toxicity. Exposure to 60% or more oxygen for longer than 24 hours is believed to lead to pulmonary damage. The full impact of these toxic effects may not be appreciated until the damage is irreversible. Mechanical ventilation can recruit collapsed alveoli and improve gas exchange so that patients may achieve acceptable blood gas values on a lower Fio2 with PPV compared with spontaneous breathing. Ventilatory assistance should therefore also be considered in patients that require an Fio2 of greater than 60% for longer than 24 hours.
respiratory rate, reduces the effective alveolar ventilation. **Dead space** refers to the portion of the tidal volume that does not participate in gas exchange.\(^{22}\) Diseases (e.g., pulmonary thromboembolism) that increase dead space can also lead to hypercapnia. Dead space can also be created by an increase in the length of the breathing circuit between the Y piece and the patient. This is especially important in very small patients in which an excessively long endotracheal tube or extensions such as an ETCO\(_2\) adapter can create significant dead space.\(^{22}\)

Hypoventilation is frequently due to neuromuscular disease and is a common indication for PPV. Impairment to the neuromuscular pathway controlling respiration can lead to hypoventilation, and patients may require PPV to maintain adequate minute ventilation and ensure an acceptable P\(_\text{CO}_2\). This includes lesions affecting the respiratory muscles themselves and/or the function of the central respiratory center, brain stem, cervical spinal cord, peripheral nerves, and/or neuromuscular junction.\(^{22,28-31}\)

Minute ventilation can also be reduced by large airway obstruction such as laryngeal paralysis or foreign body obstruction leading to hypercapnia. PPV is not indicated in patients with these conditions because specific therapy to relieve obstructions (e.g., tracheostomy) can resolve their hypercapnia.

Malfunction of one-way valves or scavenging systems and inadequate flow rates in a nonrebreathing circuit can be iatrogenic causes of hypercapnia. It is important to rule out these causes because they are reversible and affected patients do not require PPV.\(^{32}\)

Animals with pulmonary parenchymal disease frequently become hypoxemic while maintaining a normal or even reduced P\(_\text{CO}_2\). This is because CO\(_2\) is approximately 20 times more diffusible in water than in oxygen; therefore, less alveolar surface area is required to remove CO\(_2\) from blood than to add oxygen to it.\(^{18}\)

**Diagnostic Approach**

Evaluation of hypercapnic patients should follow a systematic approach. Hypoventilating animals breathing room air are likely to be hypoxemic as well as hypercapnic and should be given supplemental oxygen. As discussed previously, these patients should have a normal A-a gradient, and rapid resolution of their hypoxemia would be expected with oxygen supplementation.

If a hypercapnic animal is on a breathing circuit or anesthetic machine, the system should first be checked for a valve or scavenging system malfunction and inadequate flow rates. Excessive dead space between the Y piece and patient should be removed.

A primary cause of hypoventilation should be investigated and addressed when possible. Reversal of anesthetic or narcotic agents, decompression of cervical lesions, normalization of intracranial pressure, and intubation or medical management for airway obstructions may all increase effective ventilation in appropriate situations. A P\(_\text{CO}_2\) above 60 mm Hg (or \(>45\) mm Hg in patients with intracranial disease) despite specific treatment is sufficient indication for PPV.\(^{22}\)

**EXCESSIVE WORK OF BREATHING**

A patient that responds to oxygen therapy and has acceptable blood gas values may still require ventilation if its respiratory effort is not sustainable; work of breathing and muscle fatigue must be considered when evaluating a patient in respiratory distress.\(^{33}\) Human studies have demonstrated a four- to sixfold increase in inspiratory effort in acute respiratory failure.\(^{34}\) In fact, reducing the work of the respiratory muscles is the most common reason for mechanical ventilation in humans.\(^{34}\)

Likewise, animals with pulmonary pathology must expend more energy to maintain adequate gas exchange.\(^{35}\) This increase in work of breathing increases oxygen consumption and exacerbates hypoxemia.\(^{36}\) Dyspneic patients may also become hyperthermic, which further increases respiratory effort and oxygen consumption. Animals with excessive work of breathing can become exhausted and develop acute respiratory failure leading to cardiopulmonary arrest.\(^{35}\)

Making the decision to implement PPV in a patient based on clinical signs alone can be challenging. In some cases, the degree of respiratory distress may be so severe that it precludes any measure of oxygenation. Tests to measure or estimate work of breathing are not readily available, and clinicians are left with subjective evaluation as their only guide. A rule of thumb used by one human critical care specialist is that if a patient is distressed enough that mechanical ventilation is being considered, it is likely that mechanical ventilation is indicated.\(^{35}\) Patients demonstrating excessive work of breathing can acutely arrest. Early recognition of these patients and prompt intubation and mechanical ventilation can be lifesaving.

**GOALS OF VENTILATION**

The ultimate goal of mechanical ventilation is to wean the patient from the ventilator. The goals during ventilation are to optimize oxygenation and ventilation in a
patient while reducing work of breathing and minimizing complications. Because PPV is not benign, clinicians should ideally use the least-aggressive ventilator settings possible to maintain acceptable $\text{PaO}_2$ and $\text{PaCO}_2$ levels (i.e., $\text{PaO}_2$ of 80 to 120 mm Hg and $\text{PaCO}_2$ of approximately 40 mm Hg). From the time an animal is placed on a ventilator, efforts to reduce the level of support should be made. Many patients need long-term (>12 to 24 hours) ventilation; in these animals, the aim is to provide adequate medical and nursing care to minimize complications and maximize patient comfort.

**PROGNOSIS**

The prognosis in animals treated with mechanical ventilation has not been well established in the literature. The weaning and survival rates in veterinary patients are substantially lower than those in humans. This may be partly due to poor case selection, lack of experience, and the greater financial and time limits imposed on veterinarians.

One retrospective study of mechanical PPV in dogs and cats cited a 39% overall survival rate. Data from another veterinary study on long-term ventilation had an overall survival rate to discharge of 28%. The prognosis in patients requiring mechanical ventilation varies with the underlying disease process. Approximately 50% of veterinary patients that required ventilation because of neuromuscular pathology were successfully weaned compared with 25% of those requiring ventilation because of pulmonary pathology. Patients with a combination of pulmonary and extrapulmonary diseases had weaning rates between these two numbers.

In comparison, mortality rates in humans with nonpulmonary diseases or mild pulmonary changes are reportedly less than 5%. Severe pulmonary conditions such as acute respiratory distress syndrome are associated with a 40% to 50% mortality.

**CONCLUSION**

Mechanical ventilation is becoming a critical component of caring for animals with respiratory compromise. One of the most challenging aspects of mechanical ventilation is making the decision to initiate it. All clinicians will be faced with patients that require mechanical ventilation and need to be prepared to intubate and manually ventilate a patient to stabilize it. Mechanical ventilation can have a role in supporting patients with oxygenation and ventilation issues. The goal is to stabilize critically ill patients, maintain them until there is clinical improvement, and ultimately wean them from machine support. The prognosis for successful weaning depends on the underlying disease and the severity of concurrent issues.

**REFERENCES**


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1. Inhaled oxygen above ____% is associated with toxicity after 24 hours.
   a. 40  c. 70
   b. 60  d. 80
2. Which of the following is the most common cause of hypoxemia?
   a. diffusion defects
   b. hypoventilation
   c. venous admixture
   d. equipment failure

3. An $\text{SpO}_2$ of 91% should correspond to a $\text{PaO}_2$ of ___ mm Hg.
   a. 40
   b. 50
   c. 60
   d. 80

4. At sea level, the $\text{PaO}_2$ should be approximately ___ times the $\text{FiO}_2$.
   a. two
   b. three
   c. four
   d. five

5. The ________ is helpful in evaluating oxygenation because it accounts for the effect of $\text{CO}_2$.
   a. A-a gradient
   b. $\text{PaO}_2$:$\text{FiO}_2$ ratio
   c. V/Q ratio
   d. $\text{PvO}_2$

6. ________ is defined as the portion of the tidal volume not participating in gas exchange.
   a. Venous admixture
   b. Diffusion impairment
   c. V/Q mismatch
   d. Dead space

7. An animal that is hypoxemic from hypoventilation should have a normal
   a. $\text{Paco}_2$.
   b. A-a gradient.
   c. effective alveolar ventilation.
   d. $\text{PaO}_2$:$\text{FiO}_2$ ratio.

8. Cyanosis is not usually appreciated until the $\text{PaO}_2$ is ___ mm Hg or less.
   a. 50
   b. 60
   c. 70
   d. 80

9. The overall prognosis for survival of ventilated veterinary patients is approximately ___%.
   a. 5
   b. 15
   c. 35
   d. 50

10. The ETCO$_2$ is most likely to vary substantially from the $\text{Paco}_2$ when the
    a. respiratory rate is high.
    b. patient is in cardiovascular shock.
    c. mucous membranes are darkly pigmented.
    d. patient is on supplemental oxygen.