Review of Support of Ventilation in the Anesthetized Horse

John A. E. Hubbell, DVM, MS, Diplomate ACVA

Anesthetized horses hypoventilate. Combined with changes in body position to lateral or dorsal recumbency, this hypoventilation can lead to hypoxemia and insufficient delivery of oxygen to tissues. Healthy horses tolerate respiratory embarrassment for short periods, but most benefit from increased inspired oxygen concentrations and support of ventilation, either manually or through the use of a ventilator. Author's address: Department of Veterinary Clinical Sciences, College of Veterinary Medicine, Ohio State University, 601 Vernon L. Tharp Street, Columbus, Ohio 43210; e-mail: john.hubbell@cvm.osu.edu. © 2010 AAEP.

1. Introduction

The drugs used to induce and maintain anesthesia in the horse are respiratory depressants. The degree of respiratory depression imposed by anesthesia is dependent on the drugs and techniques used, the body position chosen (dorsal or lateral recumbency), and the duration of the anesthetic period. Frequently, respiratory depression in the horse is accompanied by lower arterial oxygen tensions (hypoxemia) than those seen in other species. A number of studies have been conducted to characterize this respiratory depression and hypoxemia, and some have proposed methods to improve ventilation and oxygenation. The purpose of this paper is to describe what is known about ventilation and oxygenation and to provide practical suggestions for employing ventilatory assistance in the anesthetized horse.

2. Overview of Ventilation and Oxygenation in the Horse

Hypoventilation

Respiratory depression can be assessed in a number of ways but fundamentally is determined by decreases in minute alveolar ventilation (the amount of fresh gas that reaches the alveoli in 1 min) and subsequent retention of carbon dioxide measured by increases in arterial carbon dioxide tension (PaCO₂) from its normal value of 40–45 mm Hg. Decreases in minute alveolar ventilation occur because of decreases in respiratory rate, decreases in tidal volume (the amount of gas in one breath), changes in the distribution of ventilation (distribution of a breath to alveoli that are not well-perfused or vice versa), or combinations of these changes. Decreases in respiratory rate are easy to detect, but changes in tidal volume are difficult to assess clinically and changes in the distribution of ventilation such as increases in dead-space ventilation (ventilation of areas where no gas exchange takes place) are limited to the laboratory. The relationship between PaCO₂ and minute ventilation is curvilinear, with increases in PaCO₂ to levels in excess of 80 mm Hg indicating that minute ventilation has decreased by 50%.
Correcting Hypoventilation

Horses anesthetized with IV techniques such as xylazine and ketamine or xylazine-diazepam-ketamine usually maintain PaCO₂ within normal limits (40–50 mm Hg) if anesthesia is limited to the induction doses (20–30 min), but hypoventilation (increases in PaCO₂) frequently occurs if anesthesia is extended with guaifenesin recipes.1,2 Hypoventilation occurs more rapidly when horses are induced with IV techniques and transitioned to inhalant anesthesia. Horses induced with xylazine-guaifenesin-ketamine or xylazine-diazepam-ketamine have increases in PaCO₂ to levels in excess of 50 mm Hg within 5 min of induction.3 Horses maintained under anesthesia with inhalant anesthetics that are allowed to spontaneously ventilate routinely have PaCO₂ in excess of 60 mm Hg, and levels may exceed 80 mm Hg.4 Hypoventilation can be corrected in almost all horses by increasing minute ventilation. Assisted ventilation (delivery of a tidal volume when the horse initiates a breath) does not normalize ventilation in anesthetized horses, but controlled ventilation (operator-determined respiratory rate and tidal volume) usually allows the anesthetist to establish the desired ventilatory values.

Hypoxemia

Hypoventilation in the anesthetized horse is frequently associated with hypoxemia (arterial oxygen tensions below 100 mm Hg), particularly when horses are breathing ambient oxygen tensions (20%). Arterial oxygen tensions (PaO₂) fall from standing levels (approximately 100 mm Hg) to 60–80 mm Hg within 5 min of the induction of lateral recumbency and fall even further if dorsal recumbency is employed.1,5 Hypoventilation contributes to the decreases in arterial oxygenation, but physiologic shunting of blood past unventilated alveoli and other ventilation/perfusion mismatches play larger roles.5–7 Decreases in arterial oxygen tension occur in all laterally and dorsally recumbent horses, and the decreases are exacerbated in horses with rounded or distended abdomens.8

Ideal levels of arterial oxygenation when increased oxygen concentrations are delivered (95–100% of the inspired air) are in excess of 400–450 mm Hg, but these levels are rarely attained. The appropriate target for PaO₂ is controversial, but most are concerned if levels fall below 50–60 mm Hg. The critical level for oxygen tension is determined by the oxyhemoglobin dissociation curve, because greater than 90% of the oxygen carried in the blood is bound to hemoglobin. Oxygen tensions in excess of 75 mm Hg are associated with greater than 95% saturation of hemoglobin. Even a PaO₂ of 50 mm Hg is associated with 90% saturation of hemoglobin. As oxygen tensions fall below 50 mm Hg, hemoglobin saturations decrease more linearly to the point where only 50% of hemoglobin is saturated at a PaO₂ in the range of 20–25 mm Hg. Other factors governing oxygen delivery to tissues include cardiac output, perfusion pressures, and physical factors related to positioning.

Assessing Ventilation and Oxygenation

The adequacy of ventilation and oxygenation is best assessed by obtaining a sample of arterial blood for blood-gas analysis. Portable self-calibrating point-of-care blood-gas analyzers are currently available that are easily used in both the hospital and ambulatory setting.18 Normal blood-gas values for anesthetized horses are presented in Table 1. Other methods of estimation of the adequacy of ventilation and oxygenation include measuring respiratory rate, watching for the degree of thoracic excursion during inspiration, visualizing the color of mucous membranes, employing monitoring equipment such as pulse oximetry, and measuring carbon dioxide tensions in the expired gasses. Pulse oximeters are used noninvasively to measure heart rate and estimate the percent saturation of hemoglobin. As pre-
The triggering mechanism is released when the chest wall has expanded appropriately, and the vault is fitted to the end of the tube and triggered. The cuff is inflated to seal the airway. The demand valve is triggered, and both nostrils are occluded. Because the horse is an obligate nasal breather, the thorax will expand, and a breath is delivered as above. If only a compressed gas source is available, a tube can be attached. The tube is advanced from the nostrils into the nasal cavity. Occlusion of the nostrils will cause the thorax to rise. When the thorax has risen appropriately, the nostrils should be released, and the horse will exhale. The duration of ventilation that can be accomplished through these methods is limited by the capacity of the compressed gas source.

Enhancing Oxygenation
Oxygenation can be enhanced if a compressed source of oxygen is available. As with emergency ventilation, oxygen can be supplied with an anesthetic machine or a demand valve, if available. Insufflation (the delivery of an oxygen flow to an open airway) is effective in normalizing PaO₂ if flow rates in excess of 15 l/min are used. The efficacy of insufflation is enhanced if the delivery tube is advanced into the pharynx or trachea. Most insufflation systems do not generate sufficient pressure to produce ventilation.

Enhancing Ventilation and Oxygenation During Anesthesia
Hypoventilation should be assumed when horses are anesthetized, particularly when inhalant anesthetics are used. Horses anesthetized with injectable agents for brief periods rarely require ventilatory support but would benefit from the delivery of enhanced oxygen tensions. Horses anesthetized through any method for extended periods frequently hypoventilate, and this hypoventilation can compromise oxygenation. If an extended procedure is contemplated, early ventilation with increased oxygen tensions provides the best chance for maximization of PaO₂. When inhalants are used, controlled ventilation provides consistent delivery of the anesthetic gas, helping establish and maintain a stable anesthetic level. It is the author's opinion that these factors must be balanced with the knowledge that the use of mechanical ventilation decreases cardiac output and frequently, arterial blood pressures and increases the depth of anesthesia more rapidly. Arterial blood pressures should be monitored closely, particularly in compromised patients.

Ventilation can be assisted in the anesthetized horse by compressing the rebreathing bag. Manual ventilation in the adult horse requires considerable expenditure of energy (both arms) to generate inspiratory pressures in the range of 20–30 cm H₂O. Respiratory rates of 6–10 breaths/min will usually produce PaCO₂ levels between 45 and 55 mm Hg in patients with normal pulmonary compliance. Inspiratory times (time for the delivery of the breath when there is a positive pressure applied to the airway) should be 1–3 sec in duration. Expiratory time (time when there is not a positive pressure applied to the airway) should always equal or preferably, exceed.

### Table 1. Expected Arterial Blood Gas Values in Awake and Anesthetized Horses

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<thead>
<tr>
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<th>Awake</th>
<th>Anesthetized</th>
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<tr>
<td><strong>pH</strong></td>
<td>7.4 ± 0.2</td>
<td>7.3–7.45</td>
</tr>
<tr>
<td>PaCO₂ (mm Hg)</td>
<td>40 ± 3</td>
<td>40–60</td>
</tr>
<tr>
<td>PaO₂ (mm Hg)</td>
<td>94 ± 3</td>
<td>100–500 (&gt;95% inspired oxygen)</td>
</tr>
<tr>
<td>Base Excess (mEq/l)</td>
<td>0 ± 2</td>
<td>0 ± 4</td>
</tr>
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Previously discussed, hemoglobin saturation in the horse remains in excess of 90%, even when PaO₂ values are in the range of 60 mm Hg. The accuracy of pulse-oximeter values in predicting actual hemoglobin saturation has been questioned, leading to the recommendation that any value below 95% saturation should be validated by other methods. Measurement of end-tidal carbon dioxide tensions potentially provides a method to continuously and noninvasively assess ventilation. End-tidal tensions should match alveolar tensions, because the gases exhaled at the end of expiration should be alveolar in origin. Recent studies have questioned the accuracy of this method of assessment of ventilation. No current noninvasive method effectively replaces arterial blood-gas analysis. On a practical basis, horses can be safely and effectively ventilated in the absence of arterial blood-gas analysis if appropriate guidelines are followed and attention is paid to ventilator-induced alterations in cardiac function.

3. **Practical Ventilatory Assistance in Anesthetized Horses**

Emergency Ventilation
The need for emergency ventilation associated with anesthesia in the horse has decreased dramatically with the increased use of ketamine as the primary anesthetic agent. Prior use of more respiratory-depressant drugs, such as thiopental or thiamylal, was associated with an increased incidence of apnea and severe respiratory depression. In a hospital setting, emergency ventilation can be performed using an anesthetic machine (if available) by either manually compressing the rebreathing bag or using a ventilator. Emergency ventilation can be accomplished in any location if there is a source of compressed air or oxygen and a means to deliver the gas under pressure to the horse’s airway. Emergency ventilation is most efficiently performed using a demand valve and an endotracheal tube. The endotracheal tube is placed into the trachea, and the cuff is inflated to seal the airway. The demand valve is fitted to the end of the tube and triggered. The triggering mechanism is released when the chest wall has expanded appropriately, and the horse is allowed to exhale. Alternatively, a smaller tube (12–14 mm inner diameter) can be inserted in one nostril and the demand valve attached. The
inspiratory time. The application of manual ventilation in the adult horse for periods in excess of 10–15 min is exhausting and precludes the anesthetist from completing other activities, such as monitoring the patient. Manual ventilation of the anesthetized foal requires less energy expenditure. The respiratory rates employed are in the range of 8–12 breaths/min using similar inspiratory pressures to those used in the adult. The indicated inspiratory pressures usually result in the delivery of normal or slightly reduced tidal volumes.

Ventilation is easily supported through the use of commercially available anesthetic ventilators. The ventilators function by compressing a re-breathing bag or bellows to deliver a mixture of oxygen and anesthetic gas. Most use compressed gases to compress the bellows, but the newest ventilator substitutes a linear actuator (similar to a piston) to deliver a tidal volume. Controlled ventilation rather than assisted ventilation should be used in the anesthetized horse, because horses do not consistently trigger the ventilator frequently enough to ensure normal PaCO₂ tensions. The primary determinants of appropriate controlled ventilation are tidal volume (12–15 ml/kg) and respiratory rate (6–8 breaths/min for adults and 8–10 breaths/min for foals). Inspiratory times are somewhat dependent on the capability of the ventilator, but they should never exceed 50% of any respiratory cycle (time from the beginning of one breath to the beginning of the next breath) and preferably, should be 20–30% of the cycle. Conventionally, this is noted as the ratio of inspiratory to expiratory time (I:E ratio). The lower the I:E ratio (1:4 being lower than 1:2 or 1:1), the less time that there is a positive pressure within the thorax. Excessively fast delivery of a breath (<1 sec) can change the distribution of ventilation because of differences in the compliance of lung segments and should be avoided. Generally, lower respiratory rates and higher tidal volumes should be used. Basic ventilatory guidelines are presented in Table 2.

**References and Footnotes**


### Table 2. Recommended Ventilatory Settings for Horses

<table>
<thead>
<tr>
<th></th>
<th>Adults</th>
<th>Foals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tidal Volume (ml/kg)</td>
<td>14–18</td>
<td>12–16</td>
</tr>
<tr>
<td>Respiratory Rate (breaths/min)</td>
<td>6–8</td>
<td>8–12</td>
</tr>
<tr>
<td>Inspiratory to Expiratory Ratio</td>
<td>1:2-1:4</td>
<td>1:4-1:5</td>
</tr>
<tr>
<td>Peak Inspiratory Pressure (cm H₂O)</td>
<td>25–40</td>
<td>15–25</td>
</tr>
</tbody>
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aTorpex, Boehringer Ingelheim Vetmedica, St. Joseph, MO 64506-2046.
bEquine Demand Valve, JD Medical Distributing Co, Inc., Phoenix, AZ 85029-4914.
cEquine endotracheal tube, Jorgensen Laboratories, Loveland, CO 80538-3683.
dTafonius, Hallowell EMC, Pittsfield, MA 01201-4714.