THE ABCS OF MECHANICAL VENTILATION
Amie Koenig, DVM, DACVIM, DACVECC
University of Georgia, Athens, GA

Key Points:
- Indications for ventilation include severe hypoventilation, hypoxemia and fatigue.
- Prognosis for ventilator patients varies with underlying reason for ventilation with hypoventilating patients faring better than those ventilated for lung disease.

Ventilation is the process of moving air in and out of alveoli which is important for the exchange of oxygen and carbon dioxide. The simplest form of artificial ventilation is provided by manually squeezing an Ambu or anesthesia bag. Long term mechanical ventilation had its start in the late 1920s in the form of the “iron lung,” a negative pressure ventilator that became instrumental in treating polio patients. Positive pressure ventilation was first developed during World War II as a result of military advances used to provide oxygen to fighter pilots. From these beginnings has risen modern ventilation, which today is used to provide positive pressure ventilation for anesthetic procedures as well as long term critical care patients.

Indications for Ventilation

Mechanical ventilation is indicated under three circumstances: 1) persistent hypoventilation, 2) severe hypoxemia that is unresponsive to oxygen therapy and 3) excessive work of breathing or fatigue.

Hypoventilation is defined as a PaCO₂ > 45 mmHg. Severe hypoventilation warranting mechanical ventilation is typically defined as PaCO₂ > 60 mmHg. Hypoventilation is most commonly seen in animals with neuromuscular disease, such as cervical disk extrusion, tetanus, or lower motor neuron disease, or in animals with certain toxicities (eg, ivermectin toxicosis) or drug overdoses. Severe pulmonary disease or thoracic cage damage (eg, flail chest) can also cause hypoventilation.

Hypoxemia is defined as PaO₂ < 60 mmHg. The first therapeutic measure for hypoxemia is to administer supplemental oxygen. Mechanical ventilation is indicated when PaO₂ remains less than 60 mmHg with the patient on oxygen or if the patient requires extremely high concentrations of oxygen (generally >60%) to maintain the PaO₂ in an acceptable range since high oxygen concentrations predispose to oxygen toxicity. Mechanical ventilation will improve oxygenation by recruiting alveoli for gas exchange.

Excess work of breathing or fatigue, is a subjective indication for ventilation. This assessment is sometimes difficult to make. In patients with severe lung disease, the animal must exert a large amount of energy just to obtain enough oxygen to supply the respiratory muscles. Oxygen consumption for breathing can increase from 2% of the full metabolic demand in a normal person to as high as 50% in a person with increased work of breathing (WOB). The amount of oxygen gained via that work is negated by the fact that large quantities of oxygen are consumed by the respiratory muscles. In addition to excess O₂ consumption, the extra WOB can lead to exhaustion and respiratory arrest. Signs of fatigue and impending respiratory arrest include lateral recumbency (cat), restlessness/thrashing in cage, short periods of apnea, fluid dripping from mouth or nose, or mentation changes (anxiety, moribund) to name a few.

If you are wondering whether the patient needs mechanical ventilation, it probably does!! It is better to obtain the airway and initiate mechanical ventilation before the animal arrests.
**Goals of ventilation**

The goals of mechanical ventilation are to 1) maintain adequate ventilation, typically a PaCO₂ of 35-60 mmHg, 2) maintain adequate oxygenation (PaO₂ = 80-120 mmHg) while minimizing the risk of oxygen toxicity (FiO₂ < 0.6), and 3) avoiding ventilation induced lung injury & other consequences of positive pressure ventilation.

**Providing Ventilation**

Initiating and maintaining mechanical ventilation requires a well planned and coordinated effort. The ventilator and all the necessary accoutrements should be set up prior to placing the patient on the ventilator. Except for the most critical patients, intravenous sedation or anesthesia is necessary for intubation and maintenance of mechanical ventilation. Intubation must be established quickly, especially in severely hypoxic patients, to prevent arrest. Neurologic patients can often be ventilated with a minimum of drugs if a tracheostomy tube is utilized which will reduce the sedation/anesthesia requirements which facilitates assessment of neurologic status and better maintenance of respiratory muscle strength, thus easier weaning from the ventilator.

There are several different modes of ventilation. Controlled ventilation is when the ventilator determines rate and tidal volume with no spontaneous breaths from the patient. In assisted ventilation, the patient can initiate breaths (determines rate) and ventilator aids in achieving the tidal volume. Synchronized Intermittent Mandatory Ventilation (SIMV) allows spontaneous ventilation with intermittent mandatory breaths being synchronized with a patient-initiated breath (ie, an assisted breath). SIMV was developed as a mode for weaning patients off the ventilator. It allows the patient to take more and more responsibility for breathing on its own as the number of synchronized mandatory breaths per minute are reduced. SIMV is also a good mode for patients that just a little assistance while building respiratory muscle strength, such as those with neuromuscular disease.

There are two ways that the ventilator can provide breaths: volume or pressure controlled. In Volume Controlled Ventilation (VCV), the ventilator delivers set tidal volume and inspiratory pressure is the dependent variable. That is, regardless of the pressure generated within the airways or alveoli, the ventilator will deliver the specified tidal volume. In Pressure Controlled Ventilation (PCV) the ventilator delivers gas until it senses the set inspiratory pressure; the tidal volume is the dependent variable. Given a set inspiratory pressure, a larger tidal volume will be generated in compliant lungs than in lungs that are less-compliant. It is important to watch for changes in the tidal volume over time, as if there is a kinked or partially obstructed ET tube, or if the patient’s lung compliance changes, the dependent tidal volume may be significantly altered. PCV is typically used for very small patients (usually less than 10 kg).

In hypoxic patients, application of positive end expiratory pressure (PEEP) is the ventilator’s greatest contribution to improved oxygenation. At the end of each breath, the ventilator continues to apply a small amount of positive pressure into the patient which recruits alveoli and holds them open for use in the gas exchange process. PEEP also prevents repetitive collapse and opening of the alveoli and therefore reduces ventilator induced lung injury (VILI).

General guidelines for ventilator settings for animals with normal lungs include a peak inspiratory pressure of 10-20 mmHg, PEEP of 0-2 mmHg, ventilatory rate of 10-20 bpm, tidal volume (V_T) of 6-10 mL/kg, minute ventilation (V_T x resps/min) of 150-250 mL/kg/min. For patients with abnormal lungs, these initial settings may be modified to include any of the following as needed to achieve the ventilatory goals: To improve ventilation (ie, you want to lower the PaCO₂), we try the following: 1) minimize dead space; 2) increase inspiratory pressure.
3) increase ventilatory rate, 4) decrease inspiratory time to ensure there is enough time for exhalation 5) increase tidal volume 6) consider permissive hypercapnia. To improve oxygenation (PaO₂ is too low) we may try the following: 1) many of the above changes, 2) increase PEEP, and 3) increase FiO₂, although preference is to maintain below 60% to prevent oxygen toxicity.

Lung Protective Strategy: Current recommendations for a “lung protective strategy” include low tidal volumes (6-8 mL/kg) coupled with increased PEEP. This allows recruitment of collapsed alveoli, prevents the cyclic opening and closing of alveoli between breaths, decreases the potential for volutrauma and barotrauma of the more normal alveoli, and reduces release of inflammatory mediators. Studies have shown reduced mortality in humans with ARDS ventilated with low tidal volumes and high PEEP compared to patients ventilated with higher tidal volumes. So ideally, tidal volume is not increased much above 10 mL/kg or so.

There are numerous potential complications which can result from mechanical ventilation. Hemodynamic compromise can occur because positive pressure within the thorax decreases venous return to heart, thereby decreasing preload and cardiac output. PPV can cause ventilator induced/associated lung injury due to cycling of alveoli open and closed, barotraumas (excessive pressure), and volutrauma (excessive volume). Oxygen toxicity and ARDS can also result. Pneumonia is a common sequel due to positional stasis and bacteria migrating down the ET tube into the lung. Chlorhexidine oral flushes, changing ET and ventilator tubes, rotating the patient, maintaining airway hydration, practicing good hand hygiene, and suctioning oropharyngeal secretions can reduce the incidence of pneumonia. Pneumothorax and pneumomediastinum can occur from alveolar rupture, typically due to over-distention of functional alveoli. Oral ulceration is also common from dessication of mucous membranes coupled with bacterial overgrowth and the trauma of the ET tube, gauze ET tube tie, pulse ox probe and mouth gag rubbing the tongue, lips and gingival

Prognosis

Mechanical ventilation is an expensive and labor-intensive undertaking. Patients require constant individual intensive care. Prognosis for successful weaning from mechanical ventilation varies with the patient’s underlying primary problem. Animals ventilated due to hypoventilation (neuromuscular disease) carry the best prognosis, with approximately 50% discharged home. In one study of cervical disk disease, 10/14 dogs (71%) were successfully weaned. In contrast, only about 15% of animals ventilated for hypoxemia are discharged from the hospital. Of 10 dogs ventilated due to pulmonary contusions, 3 went home, 1 was weaned but died acutely later in hospitalization, and 1 was euthanized despite marked pulmonary improvements. Five died or were euthanized while on the ventilator.