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An overview of positive pressure ventilation

Elena Vassilev, DVM and Maureen McMichael, DVM, DACVECC

Abstract

Objective: The purpose of this article is to give the reader a brief overview of the indications for positive pressure ventilation (PPV).
Data sources: Current human and veterinary literature.
Summary: There are numerous indications for PPV in veterinary medicine. These include both pulmonary parenchymal disease and diseases that affect ventilation. When choosing PPV, a clinician must have a comprehensive understanding of the different ventilation mode options available and the physiologic effects of ventilation on the patient.
Conclusions: PPV is becoming more widely used in veterinary medicine and is improving the survival of animals with hypoxemic and hypercapnic respiratory failure.

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Keywords: hypercapnia, hypoxemia, mechanical ventilation, PEEP (positive end-expiratory pressure), pressure control, respiratory failure, volume control

Introduction

Positive pressure ventilation (PPV) can be life-saving and has been used in human medicine for approximately 40 years.[1] Companion animals are reaping the benefits of human medical advancements, and in one recent study survival of mechanically ventilated veterinary patients was reported to be 39%.[2]

The objectives of mechanical ventilation are to improve pulmonary gas exchange by increasing alveolar ventilation, to increase arterial oxygenation, to increase lung volume, and to prevent atelectasis. Mechanical ventilation can increase the functional residual capacity (FRC), which is the residual volume of inspired gas in the lungs after a normal expiration. It can also decrease the work of breathing, reverse life-threatening hypoxemia, and treat respiratory acidosis.[3,4]

Since the inception of mechanical ventilation, there have been some dramatic changes in the design of ventilators and their use. The purpose of this article is to give the reader an overview of the basic principles, indications, and settings for mechanical ventilation in a critical-care setting.

Ventilation is defined as the movement of gas in and out of the pulmonary system. The ventilatory mechanism can be viewed as a to-and-fro, valveless pump consisting of the chest cage, ventilatory muscles, and those nervous system components that regulate and influence muscular performance.[5]

Mechanical ventilation may be indicated for any patient whose arterial partial pressure of oxygen (PaO_2) falls below 50–60 mmHg on supplemental oxygen or for those whose arterial partial pressure of carbon dioxide ($PaCO_2$) increases above 50–60 mmHg.[2,5,6] It is also indicated for any animal that is in danger of impending respiratory failure (i.e., using excess energy ventilating).[2,6]

Indications

Patients requiring ventilator care can be divided into those with difficulty oxygenating and those with difficulty ventilating (Table 1).[7]

In hypoxemic respiratory failure, the hypoxemia can be caused by insufficient gas exchange at the level of the alveoli and pulmonary vasculature, shunting, V/Q (ventilation/perfusion) mismatch, or decreased FiO_2.[7] The most common conditions causing hypoxemic

From the Pet Emergency and Specialty Center, 5232 Jackson Drive, #105, La Mesa, CA (Vassilev), and the Department of Small Animal Medicine and Surgery, College of Veterinary Medicine, Texas A&M University, College Station, TX (McMichael).

Address correspondence and reprint requests to:
Dr. Elena Vassilev, Pet Emergency and Specialty Center, 5232 Jackson Drive, #105, La Mesa, CA 91941. E-mail: elenavass@earthlink.net

Table 1: Two types of acute respiratory failure

Hypoxemic respiratory failure	Hypercapnic respiratory failure
Also called: type I ARF (acute respiratory failure), lung failure, oxygenation failure, respiratory insufficiency	*Also called:* Type II ARF, pump failure, ventilatory failure
Definition: the failure of lungs to provide adequate O_2 necessary to meet metabolic needs	*Definition:* the failure of the lungs to eliminate CO_2
Criteria: $PaO_2 < 60$ mmHg at an $FiO_2 > 0.5$ or $PaO_2 < 40$ mmHg on any FiO_2	*Criteria:* acute ↑ in $PaCO_2 > 50$ mmHg
Basic causes: V/Q (ventilation/perfusion) mismatch, alveolar hypoventilation, diffusion defect, anatomical shunts, inadequate FiO_2	*Basic causes:* pump failure (drive, muscles, work of breathing), ↑ CO_2 production, ↑ dead space

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ventilatory failure in companion animals are pulmonary contusion, acute respiratory distress syndrome (ARDS), near drowning, aspiration of gastric contents, pneumonia, heart disease, and inhalation of toxic gases.[8]

In hypercapneic respiratory failure, the patient has normal lung parenchyma but insufficient ventilatory function. The result is a respiratory acidosis due to inadequate removal of carbon dioxide (CO_2). The most common causes of ventilatory failure are injury or impairment at the level of the central nervous system (i.e., general anesthesia, opioids, pentobarbital), interference with the efferent neurons to the respiratory muscles (i.e., cervical neck injury or cervical disc disease), polyneuropathies (i.e., polyradiculoneuritis), and neuromuscular junction diseases (i.e., myasthenia gravis, tick paralysis, and botulism).[9]

Animals can also be presented with a combination of hypoxemic and hypercapneic respiratory failure. An example is the cervical disc disease dog that aspirates post-operatively.

In healthy humans, the ventilatory muscles consume 2–5% of the total oxygen intake. In states of compromised ventilation, the energy demands on the ventilatory muscles increase causing fatigue of the muscles of ventilation. During times of diminished arterial oxygen concentration, as much as 30–40% of the cardiac output (CO) can perfuse the diaphragm at the expense of the brain, kidneys, and other essential organs.[5]

Volume- and pressure-cycled ventilation

An inspiratory flow must be generated for a tidal volume to be delivered and mechanical ventilators facilitate this by creating either a positive or a negative pressure gradient. Negative pressure ventilators apply sub-atmospheric pressure outside of the chest to inflate the lungs. Negative pressure ventilation does not require an artificial airway as long as the patient's airway is patent. Negative pressure ventilators (i.e., iron lungs) are used mainly for research.[10] PPV refers to the application of any pressure higher than atmospheric pressure to the airway. Mechanical ventilation currently employed in a critical-care setting uses positive pressure to inflate the lungs. Inspiratory flow is generated by increasing the pressure at the airway entrance.[5]

The 4 physical factors involved in PPV are volume, pressure, flow, and time.[5] The physical factor that ends inspiration is traditionally used to classify the PPV mode.

- A volume-cycled ventilator cycles off when a preselected tidal volume is delivered.
- A pressure-cycled ventilator cycles off when a preset pressure level is reached.
- A flow-cycled ventilator cycles off when the inspiratory flow rate decreases to a set point.
- A time-cycled ventilator cycles off when a preset time has passed.[11]

Volume cycling delivers a preset tidal volume (V_t) with each breath. In this mode, the V_t is always the same but the airway pressure can vary. The major disadvantage of this mode is that it can cause alveolar over-distention and lung injury if the pressure required to deliver the V_t becomes too high.

Pressure cycling delivers a variable volume until a preset airway pressure is reached.[5] The major disadvantage of this mode is that the V_t may decrease as the lungs become less compliant. This will cause the $PaCO_2$ to increase. This method is useful in human pediatric patients with cuffless endotracheal tubes and is said to be more comfortable in awake humans.[4] Human and animal studies have shown no distinct cardiorespiratory advantage of pressure- *versus* volume-cycled ventilation.[12–14] Most new ventilators offer both volume- and pressure-cycled options.

Flow- and time-cycled options are used more commonly in human medicine than in veterinary medicine and will not be covered here.

High-frequency ventilation (HFV) delivers a small V_t (1–3 mL/kg) at a high frequency (100–300 breaths/min).[15] An advantage of this system is that it can be used in hypovolemic patients because the relatively low peak inspiratory pressure (PIP) does not decrease CO.[15]

It is an open system and can be used during pharyngoscopy and bronchoscopy. A disadvantage is that rapid, severe lung injury can occur if there is an outflow obstruction.[15]

Modes of mechanical ventilation

Intermittent mandatory ventilation (IMV) provides PPV at preset time intervals, independent of the patient's spontaneous breathing pattern. This mode allows the patient only 2 types of breaths: spontaneous or mechanically controlled.[16] Newer ventilators do not offer this option.[17]

Synchronized IMV (SIMV) synchronizes the patient's respiratory efforts with the ventilator-driven breaths. With SIMV, the patient can have 1 of 3 types of breaths: a spontaneous breath, a mechanically assisted breath, or a controlled mechanical breath. If the patient's respiratory rate is insufficient, a preset rate of breaths will be delivered.[16]

Figure 1a and b show SIMV with pressure or volume cycling, respectively.

The advantage of IMV/SIMV is that they both provide the security of a set number of mandatory breaths. They require the patient to perform respiratory work because of increased resistance of breathing circuits.[11] The disadvantage of IMV is that the mandatory breath can be delivered at the peak of a spontaneous breath causing hyperinflation and barotrauma.[15]

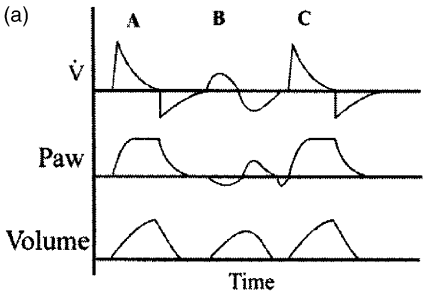
Assist-control (A/C) ventilation either delivers a preset ventilator driven breath or allows the patient to trigger a breath that is then assisted by the ventilator. This mode is ideally used for a patient who has normal respiratory drive, but the inability to generate a complete tidal volume. This mode of ventilation supports every patient breath.[16] One advantage is that each breath has full ventilatory support[15] (Figure 2).

A/C mode can cause excessive work of breathing if the inspiratory sensitivity setting is not sensitive enough, making it difficult for the patient to trigger a breath. Although this mode does not allow the patient to take a 'complete' spontaneous breath, it will support each inspiratory effort with a full V_t and may cause hypocapnia and respiratory alkalosis in a panting animal.

IMV, SIMV, and A/C can all be used with either volume- or pressure-cycled ventilation.

Pressure support ventilation (PSV) is the addition of a preset pressure plateau for the duration of a spontaneous breath, and is used to decrease the work of breathing.[4] Giving the spontaneously breathing patient a pressure boost with each breath relieves the respiratory muscles from work.[16] PSV is most often used during weaning to help overcome the resistance of the tubing. It prevents respiratory muscle atrophy and improves patient comfort. Its disadvantages are that PSV requires consistent spontaneous ventilation and can cause fatigue and tachypnea if the pressure is set too low[4] (Figure 3).

Positive end-expiratory pressure (PEEP) increases the end-expiratory pressure above atmospheric pressure, and is used to keep alveoli from collapsing at the end of expiration. PEEP is used in conjunction with other ventilator modes. The major indications for PEEP are in patients with decreased FRC and in patients with

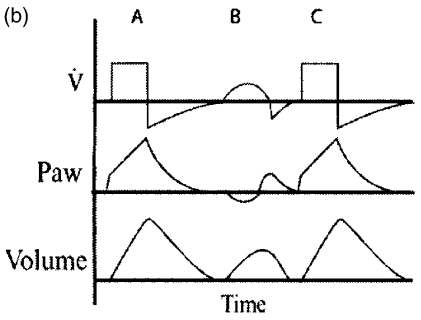


Breath A is controlled and time-triggered

Breath B is a spontaneous breath

Breath C is synchronized and assisted

Breaths A and C are full machine pressure breaths



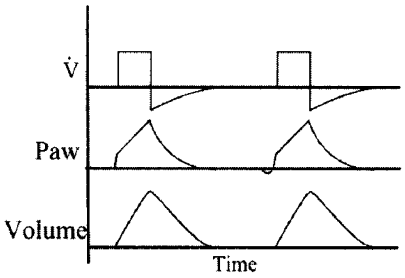
Breath A is controlled time-triggered

Breath B is a spontaneous breath

Breath C is synchronized and assisted

Breaths A and C are full machine volume breaths

Figure 1: Synchronized intermittent mandatory ventilation (SIMV) (a) pressure-cycled, (b) volume-cycled showing flow (V in L/min), airway pressure (Paw in cmH_2O), tidal volume (volume in mL) waveforms over time. (Reprinted by the permission of Dana Oakes Ventilator Management: A Bedside Reference Guide.)

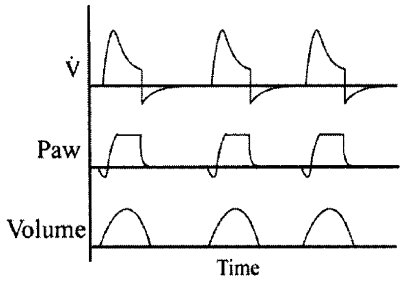


Breath A is controlled by machine-triggered

Breath B is assisted and patient-triggered

Each breath is a full machine pressure breath

Figure 2: Assist/control ventilation volume-cycled showing flow (V in L/min), airway pressure (Paw in cmH_2O), tidal volume (volume in mL) waveforms over time. (Reprinted by the permission of Dana Oakes Ventilator Management: A Bedside Reference Guide.)



All breaths are patient–triggered and flow–cycled

Figure 3: Pressure support ventilation (PSV) showing flow (V in L/min), airway pressure (Paw in cmH_2O), tidal volume (volume in mL) waveforms over time. (Reprinted by the permission of Dana Oakes Ventilator Management: A Bedside Reference Guide.)

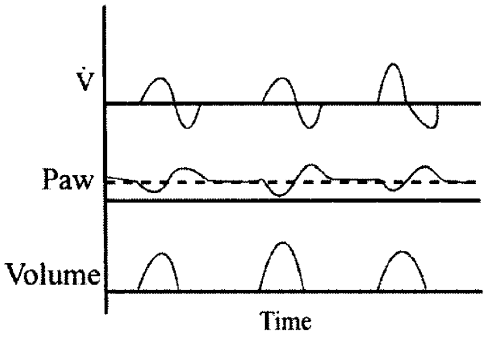
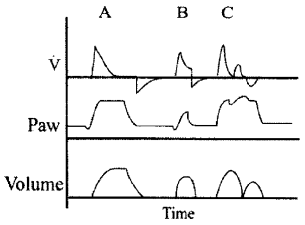


Figure 4: Continues positive airway pressure (CPAP) showing flow (V in L/min), airway pressure (Paw in cmH_2O), tidal volume (volume in mL) waveforms overtime. (Reprinted by the permission of Dana Oakes Ventilator Management: A Bedside Reference Guide.)



Breath A is a synchronized assisted patient-triggered breath

Breath B is a spontaneous pressure-supported breath at the low PEEP level

Breath C is a time-triggered controlled breath with a spontaneous breath at the high PEEP level

Figure 5: Bi-level showing flow (V in L/min), airway pressure (Paw in cmH_2O), tidal volume (volume in mL) waveforms overtime. (Reprinted by the permission of Dana Oakes Ventilator Management: A Bedside Reference Guide.)

intrapulmonary shunt (atelectasis). PEEP is applied to PPV breaths only.[5]

Continuous positive airway pressure (CPAP) is PEEP applied to a spontaneously breathing patient (Figure 4).

Bilevel positive airway pressure is a newer option offered on some ventilators, which applies independent airway pressure to both inspiration and expiration. The clinician sets both the high and low values of CPAP/PEEP. Spontaneous ventilation allows the patient to determine the rate and volume of each breath.[5] The patient triggers both the synchronized-assisted breaths and pressure-supported breaths. If no breaths occur over a period of time, then a mandatory breath is delivered (Figure 5).

The PIP is the maximal amount of pressure generated during inspiration and is measured at end-inspiration. High PIP can be used to recruit atelectic alveoli; however, excessive PIP is associated with barotrauma.[4,18] The degree of barotrauma varies directly with resistance, end-inspiratory flow, tidal volume, and respiratory system compliance.[18]

End-inspiratory pause or end-inspiratory plateau (EIP) refers to the period of time after the mechanical inspiration has ended and exhalation begins. End-inspiratory pressure represents the peak alveolar pressure. The difference between PIP and EIP is due to resistance of the system.[7] A plateau pressure of over 30 cmH_2O is associated with increased likelihood of barotrauma.[7]

Newer models of ventilators have graphics packages and graphics are used more to assist clinicians in finding optimal settings for each individual veterinary patient. Volume/time, pressure/time, flow/time, and pressure/volume loops are available. These can aid decisions regarding optimal PEEP and PIP. The useful

information gathered from these graphics includes the recognition of auto-PEEP and changes in resistance and compliance.

Ventilator settings

Ventilator settings are determined by the ventilator available and the underlying pulmonary pathophysiology of the animal to be ventilated. The clinician must decide on volume- or pressure-cycled ventilation, which mode of ventilation to use (SIMV, spontaneous, A/C), whether to add PEEP or pressure support, and then set the alarm parameters.

The decision to use a volume *versus* pressure cycle depends on the primary need to maintain V_t and $PaCO_2$ at the expense of the alveolar pressure or to limit the alveolar air pressure while allowing tidal volume and $PaCO_2$ to vary as system impedance varies.[19] Animal and human studies have demonstrated that there is no difference in gas exchange or pulmonary mechanics between the 2 cycles.[12–14,19]

The recommended tidal volume can range from 6 mL/kg in an ARDS patient to 15–20 mL/kg in an animal with normal lungs.[2,18] PEEP can range from 0 cmH_2O in an animal with normal lungs and can be increased up to 15 cmH_2O in an animal with severe lung disease.[2] PEEP can help maintain the FRC and prevent atelectasis and is often set initially at 3 cmH_2O.[7] In addition, the respiratory rate or frequency, the FiO_2, the inspiratory flow rate, and the inspiratory to expiratory (I:E) ratio must be set. A helpful formula to estimate the respiratory rate needed to achieve a certain $PaCO_2$ is:

$$\text{New RR} = (\text{RR} \times \text{PaCO}_2)/\text{Desired PaCO}_2.$$

Typical settings for an animal with hypercapneic respiratory failure (normal lungs) are an FiO_2 of 0.21–0.40, a V_t of 15 mL/kg, an RR of 12 bpm, SIMV mode, and a PEEP of 0 cmH_2O.[2] Depending on the initial $PaCO_2$, the RR and/or the V_t may be started higher to decrease the $PaCO_2$ more rapidly. In human beings an FiO_2 of 1.0 is the initial starting point at which mechanical ventilation is initiated in order to insure that hypoxemia does not complicate the initial acclimation to the ventilator.[7] An FiO_2 of 1.0 for longer than 48–72 hours caused death in animal models. The FiO_2 should not exceed 0.6 for prolonged periods.[7]

The typical initial settings for an animal with severe pulmonary parenchymal disease are an FiO_2 of 1.0, a V_t of 6–8 mL/kg, a RR of 15 bpm, SIMV mode, and a PEEP of 5 cmH_2O. The goal is to reduce FiO_2 to 0.6 as soon as possible. This is often done by adjusting PEEP upward first and then adjusting the V_t upward if oxygenation cannot be maintained on an FiO_2 of 0.6.[2]

The inspiratory time should be maintained between 1.5 and 2.5 seconds. The I:E ratio should be kept around 1:2. An I:E from 1:1.5 to 1:3 most closely resembles spontaneous breathing.[17] Prolonged inspiration causes a decreased CO, which can negatively affect perfusion. Whenever the respiratory rate or the V_t (or the minute volume on some ventilators) is increased, the I:E ratio will decrease. To adjust this back the flow rate can be adjusted upward. A simple formula for determining the appropriate flow rate for a given I:E ratio is as follows:[17]

For an I:E ratio of 1:3 in a 20 kg dog with an RR of 15 and a V_t of 200 mL

$$\begin{aligned}\text{Flow} &= \text{minute volume} \times \text{sum of I : E ratio}\\ &= V_t \times \text{RR} \times \text{sum of I : E ratio}\\ &= 200\ \text{mL} \times 15 \times (1+3)\\ &= 12\ \text{L/min}\end{aligned}$$

Physiologic and pathophysiologic effects of PPV

During normal breathing (negative pressure ventilation), pressures in the airways and pleura decrease during inspiration. PPV causes increased pressure during inspiration and increased intrathoracic pressure. Barotrauma is lung damage secondary to changes in intrathoracic pressure that cause extra-alveolar air.[7] The most common manifestations of barotrauma are pulmonary interstitial emphysema, pneumomediastinum, pneumothorax, and subcutaneous emphysema.[7]

Oxygen-induced lung injury is believed to be related to the generation of free radicals that can cause cellular injury. The extent of injury and damage appears to be related to the duration of exposure and the concentration of oxygen. Reducing the FiO_2 to ≤ 0.6 within 12 hours of starting ventilation minimizes the chance of oxygen-induced lung injury.[20]

Increased intrathoracic pressure compresses pulmonary blood vessels. This leads to decreased blood delivery to the left heart and a decrease in CO.[7] Higher mean airway pressures result in a further decrease of CO. An increase in the mean airway pressure has a more detrimental effect on venous return than increases in PIP. The mean airway pressure is a function of respiratory rate, PIP, inspiratory time, and PEEP.[7] Keeping these values as low as possible causes the smallest decrease in CO.

PPV results in maximum venous return during exhalation, while in spontaneous breathing venous return is greatest during inhalation.[7] Decreased CO due to PPV is most pronounced with high mean airway pressure, high lung compliance, and low circulating blood volume.[7]

PPV decreases urine output by increasing antidiuretic hormone and causes decreased production of atrial natriuretic factor favoring fluid retention.[7] An increased

incidence of gastric ulceration and a higher incidence of liver dysfunction in patients on PPV have been observed.[7] Decreased portal blood flow during PPV can result in decreased metabolism of certain drugs.[5]

Other physiologic alterations caused by PPV are decreased glomerular filtration rate (GFR) and intrarenal blood flow redistribution.[5] Decreased renal blood flow can result in decreased elimination of certain drugs and a thorough evaluation of drugs cleared by the kidney should be made in all ventilated patients.[5]

Because PPV may have adverse effects on other organs, it is essential to monitor perfusion parameters in addition to oxygenation (PaO_2, SpO_2) and ventilation ($PaCO_2$) parameters. Good indicators of perfusion are urine output, lactate levels, and peripheral temperature (core-web temperature gradient).[2]

Physiologic effects of PEEP

The PEEP increases FRC preventing collapse of alveoli. This minimizes the shear forces that result from opening and closing of alveoli. It also causes increases in the peak and mean airway pressures and increases in central venous and pulmonary arterial pressures. Decreases in aortic pressure and CO are also seen.[7] The cardiovascular effects of PEEP are similar to those of PPV and include a decrease in CO due to increased intrathoracic pressure.[7] The PEEP increases pulmonary vascular resistance and decreases venous return. This leads to an increase in the right ventricular afterload and even more importantly, decreases preload that results in a net increased right ventricular end diastolic volume.[7] At high PEEP values a diminished right ventricular ejection fraction occurs.[7] As the right ventricular ejection fraction decreases, the right ventricular end diastolic volume increases and shifts the intraventricular septum to the left, thus limiting the left ventricular distendability.[5,7] When PEEP is used in conjunction with PPV there can be a significant decrease in CO. Interestingly, a decrease in lung compliance (i.e., stiff lungs) as seen in many ARDS patients causes PEEP to have less of an effect on CO. The increase in airway pressure caused by PEEP is less readily transmitted to the rest of the thoracic cavity with decreased pulmonary compliance.[17]

Because PEEP causes increases in intracranial pressure by impeding venous return from the jugular veins, it should be used very cautiously in head trauma or in patients with space-occupying brain lesions.

Conclusions

In 2002, Drellich[2] reported a 55% weaning rate and a 50% survival to discharge rate among hypercapneic ventilatory failure veterinary patients. This is in contrast to a 20% weaning rate and an 11% discharge rate in hypoxemic ventilatory failure patients.[2] In a similar study, King et al. reported a 64% discharge rate in dogs with hypercapneic ventilatory failure.[21] King and Hendricks[22] reported a 39% survival rate in ventilator patients, with hypercapneic ventilatory failure patients having a better outcome than hypoxemic ventilatory failure patients.[2]

PPV can be life saving and should be instituted as early as possible in severely compromised patients.[23] A veterinary practice is much more likely to institute ventilator therapy if there is a ventilator on the premises, the staff are familiar with it, and are comfortable with the indications for PPV. Owning and using a critical-care ventilator has many advantages. PPV frees up personnel that would have had to breathe manually for the animal, and provides a higher level of care for critical-care patients than an anesthesia ventilator does. As ventilators become more sophisticated and user friendly, their use will most likely increase in veterinary medicine.

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