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Low tidal volume ventilation in healthy dogs

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Abstract

Objective – To determine if low tidal volume (V_T) ventilation is associated with the development of respiratory acidosis and changes in lung function in healthy dogs.
Design – Randomized prospective experimental cross-over study.
Setting – Pulmonary function laboratory at a university teaching hospital.
Animals – Five healthy Beagle dogs.
Interventions – Dogs were anesthetized and randomly mechanically ventilated with V_T of 6, 8, 10, 12, and 15 mL/kg while maintaining a constant minute volume.
Measurements and Main Results – Arterial blood gases and pulmonary mechanics were collected after 15 minutes of equilibration at each V_T. Repeated measures ANOVA was used to determine the effect of V_T with a P-value of <0.05 considered significant, and a Pearson product moment was used to determine correlation between V_T and pH and $PaCO_2$. V_T had a significant effect on $PaCO_2$ ($P < 0.001$) and on pH ($P < 0.001$) with lower V_T being associated with higher $PaCO_2$ and lower pH. There was a strong correlation between V_T and $PaCO_2$ ($r = –0.87$) and V_T and pH ($r = 0.83$). Increased airway pressures and pulmonary compliance were associated with increasing V_T.
Conclusions – There is a predictable decrease in the pH, decrease in airway pressure, decrease in compliance, and increase in the $PaCO_2$ associated with lower V_T. Low V_T ventilation is well tolerated in healthy dogs; the role of low V_T ventilation in dogs with acute lung injury/acute respiratory distress syndrome as well as the influence of positive end expiratory pressure requires further evaluation.

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Introduction

Intermittent positive pressure ventilation (IPPV) is critical to the support of pulmonary failure in dogs, particularly in animals with suspected acute lung injury or acute respiratory distress syndrome (ARDS). It is well recognized, however, that mechanical ventilation may have deleterious consequences, including the effects of volu-trauma and baro-trauma. These complications may occur even in the absence of pre-existing lung disease.[1] Resulting ventilator-induced lung injury may induce progressive respiratory failure, with increased morbidity, and even mortality. In human medicine, low tidal volume (V_T; ie, 6 mL/kg) ventilation has been demonstrated as the preferred technique in patients with

Abbreviations

ALI	acute lung injury
ARDS	acute respiratory distress syndrome
IPPV	intermittent positive pressure ventilation
MV	minute ventilation
PEEP	positive end expiratory pressure
$PeCO_2$	end-tidal CO_2
TLC	total lung capacity
V_A	alveolar ventilation
VILI	ventilator-induced lung injury
V_{DS}	dead space ventilation
V_T	tidal volume

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ARDS and is the first intervention associated with an improved survival rate.[2] Strategies to minimize lung damage associated with IPPV in dogs, including lower V_T, have not been investigated to date.

The normal V_T in dogs is reported as 10–15 mL/kg.[3] Predictable effects of lower V_T ventilation may thus include CO_2 retention with subsequent respiratory acidosis and hypoxemia. Hypoxemia is potentially surmounted with increasing inspired oxygen concentrations, but acidosis will persist and may be the limiting step in promoting low V_T ventilation. Additionally, a reduction in V_T will increase the patient's relative dead space ventilation (V_{DS}), as V_T is the sum of alveolar ventilation (V_A) and V_{DS}. V_{DS} may be calculated via the Bohr equation and consists of the anatomic dead space (eg, trachea and conducting airways) and alveolar dead space, which includes those alveoli that are ventilated but underperfused. Anatomic dead space is fixed, and is approximately 1/3 the normal V_T.[4] Therefore with decreasing V_T, even if minute ventilation (MV = Frequency × V_T) is held constant, there is an increasing proportion of each breath dedicated to V_{DS} and not available for gas exchange.

The goal of this study was to determine the effect of varying the V_T on gas exchange in healthy ventilated dogs. We hypothesized that decreasing V_T will be associated with increased $PaCO_2$ and the development of respiratory acidosis, even after controlling for MV. MV was controlled by adjusting the respiratory rate.

Materials and Methods

Five healthy laboratory beagle dogs of normal body condition with no abnormal history or physical examination findings were included in the study. Dogs were premedicated with hydromorphone[a] (0.1 mg/kg IM) and diazepamlink [b] (0.1 mg/kg IM) and general anesthesia was induced using propofol[c] (4–6 mg/kg IV). Orotracheal intubation was performed. Anesthesia was maintained with a propofol continuous rate infusion (CRI) (100–150 μg/kg/min). Routine anesthetic monitoring, included continuous ECG, pulse oximetry, and end-tidal CO_2 analysis was performed. Intravenous lactated Ringer's solution[d] was provided at a rate of 5 mL/kg/h. A 20-G over-the-needle arterial catheter was aseptically placed in the dorsal pedal artery for arterial blood gas analysis.

Dogs were ventilated in a randomized order at V_T of 6, 8, 10, 12 and 15 mL/kg using a specialized critical care ventilator.[e] The MV was held constant at 200 mL/kg/min. The inspired oxygen concentration was 25%, with zero end-expiratory pressure. The dogs were ventilated for 15 minutes at each V_T after which an arterial blood gas was collected from the arterial catheter. Lung mechanics, including static compliance, and airway pressures were recorded using commercial software contained within the ventilator. Static compliance was given by the formula (V_T/P_{plat} – PEEP; PEEP = positive end expiratory pressure). Oxygen saturation was measured via pulse oximetry and a modified Bohr equation ($V_{DS}/V_T = PaCO_2 – PeCO_2/PaCO_2$) was used to calculate the dead space fraction, using end-tidal CO_2 ($PeCO_2$) and arterial CO_2 ($PaCO_2$).

Following the completion of the study, dogs were recovered from anesthesia and returned to the laboratory animal facility. A single postprocedural dose of carprofen[f] (2.2 mg/kg SQ) was administered for any potential discomfort associated with endotracheal intubation and catheterization. The dogs were adopted out approximately 3 months after the conclusion of the study.

Repeated measures ANOVA were used to determine the effect of V_T on $PaCO_2$, airway pressures, static compliance, and pH with a P-value of <0.05 considered significant. All data were presented as mean ± SD. The correlation between V_T and pH and PCO_2 was calculated using the Pearson product moment, with a strong correlation defined as an $r > 0.8$. The incidence of mild (defined as <7.351) and severe acidosis (defined as <7.2), as well as hypoxemia (defined as PaO_2 < 80 mm Hg), were recorded using categorical data.[5]

Results

All dogs completed the study successfully. There were no unanticipated anesthetic or ventilatory complications.

As predicted, V_T had a significant effect on $PaCO_2$ ($P < 0.001$) and on pH ($P < 0.001$) (Table 1), with lower V_T being associated with higher $PaCO_2$ and lower pH. There was a strong correlation between V_T and $PaCO_2$ ($r = –0.87$) and V_T and pH ($r = 0.83$).

One dog, while being ventilated with a V_T of 6 mL/kg, developed mild hypoxemia (PaO_2 = 73 mm Hg, with 92% oxygen saturation). This individual dog has a $PaCO_2$ of 42 mm Hg with a V_T of 6 mL/kg. This hypoxemia resolved (PaO_2 = 87 mm Hg 96% saturation) during ventilation with a V_T of 8 mL/kg. Mild (>7.2) respiratory acidosis was observed in 12/25 arterial blood gases. All 5 dogs had a pH of <7.36 with a V_T = 6 mL/kg, 4 dogs with a V_T = 8 mL/kg, 2 dogs with a V_T = 10 mL/kg, and 1 dog with a pH of 12 mL/kg. No samples had a pH of <7.2.

As predicted, airway pressures significantly increased with higher V_T (Figure 1). Static compliance increased significantly from 21.3 ± 1.0 mL/cm H_2O at 6 mL/kg V_T to 24.5 ± 1.7 mL/cm H_2O at 15 mL/kg. Neither calculated dead space fraction nor pulse oximetry was significantly affected by changes in V_T.

Table 1: The effect of tidal volume on mean ± SD of PCO_2 and pH in healthy beagle dogs

	6 mL/kg	8 mL/kg	10 mL/kg	12 mL/kg	15 mL/kg
PCO_2 (mm Hg)	40.4 ± 2.2	38.6 ± 2.2	36 ± 2.7	32.8 ± 3.5	29.1 ± 1.7
pH	7.325 ± 0.02	7.336 ± 0.02	7.368 ± 0.03	7.39 ± 0.04	7.431 ± 0.03

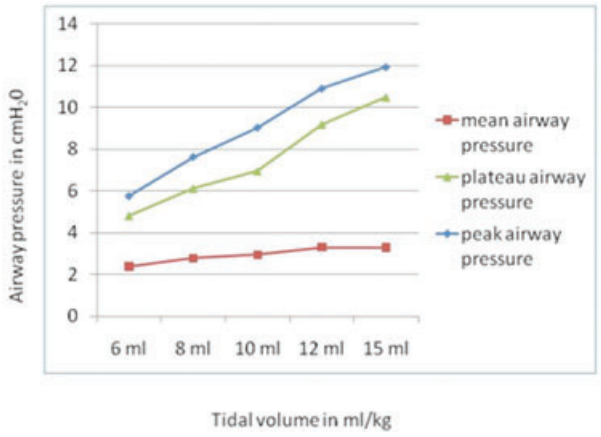

Figure 1: Peak, mean, and plateau airway pressures (cm H_2O) plotted against varying tidal volumes (mL/kg).

Discussion

The results of this study confirmed our hypothesis that decreasing V_T is associated with increased $PaCO_2$ and the development of mild respiratory acidosis, even after controlling for MV. These observations were likely related to greater proportion of fixed anatomic V_{DS}. Anatomic dead space is proposed to be 2.2 mL/kg; this assumes greater significance when the V_T is decreased from 15 mL/kg to 6 mL/kg.[4] However, a low V_T (6 mL/kg) was well tolerated in this population of healthy dogs, and there was no evidence of clinically significant acidosis due to CO_2 retention or hypoxemia.

As anticipated in this study, increasing V_T was associated with a significant rise in mean, peak, and plateau pressures. Peak pressure is defined as the highest pressure that is reached in the ventilatory circuit, while plateau pressure equivalent to the pressure in the alveoli.[6] Plateau pressure is usually slightly less than peak pressure, although is some cases, such as with very short inspiratory times, the peak pressure is much higher than the plateau pressure. Specific guidelines in human medicine from ARDSnet include trying to limit plateau pressure to less than 30 cm H_2O in patients with ARDS although the actual appropriate value as well as method of measurement is still debated.[1,7] Thirty centimeters of water pressure is considered in a healthy lung to represent total lung capacity and is typically equivalent to an inspiratory volume of approximately 80–90 mL/kg.[8] However, in people with severe ARDS, plateau pressures in excess of 30 cm H_2O may be associated with a V_T < 6 mL/kg due to the low lung compliance[1,2]

In the normal dogs in this study, compliance increased with increasing V_T. The phenomenon is mostly likely associated with recruitment of previously atelectatic lung lobes. Potentially, in dogs with ARDS, increasing V_T to 15 mL/kg may not be associated with recruitment, but rather propagation of lung injury, or the development of pneumothorax. ARDS is a syndrome that accompanies a variety of critical illnesses or injuries in people, and in dogs.[1,9,10] ARDS is identified by a variety of clinical parameters, including a pre-existing disease/injury, bilateral pulmonary infiltrates, decreased compliance, and the absence of congestive heart failure. ARDS is associated with a defect in the alveolar-capillary membrane, which permits flooding of the alveolar space with a protein-rich fluid. This results in hypoxemia due to intrapulmonary shunting and V–Q mismatch associate with derecruitment (ie, collapse) of alveoli and an increased work of breathing associated with a low compliance. In people with ARDS, low V_T ventilation has been enthusiastically embraced at medical centers as it results in improved clinical outcome. Hypercarbia is 1 predictable biochemical change associated with low V_T ventilation in people with ARDS, and is either ignored if possible (eg, permissive hypercapnea), treated by small increases in the V_T if the plateau pressure is low enough, or treated with sodium bicarbonate to increase the pH to a more physiological range.[11]

This study should be repeated in dogs with naturally developing ARDS, as the ability of those critically ill dogs to tolerate low V_T may be markedly different than these healthy beagles.

Of importance, this study did not evaluate the effects of PEEP. PEEP is applied by the ventilator, and prevents the end-expiratory pressure from decreasing below a specific set level. PEEP acts by limiting atelectasis, and by recruiting collapsed alveoli.[12] Moderate-to-high levels of PEEP are considered an essential aspect of ventilatory management of critically ill patients. As such, PEEP would not be anticipated to affect ventilatory values as much as oxygenation, as CO_2 is much more diffusible than oxygen. High PEEP in ventilated people may increase V_{DS} (eg, zone 1 ventilation) in people by limiting

perfusion to specific regions of the lung and this could affect CO_2.

Conclusion

This study demonstrates that despite predictable blood gas changes, low V_T ventilation (6 mL/kg) is well tolerated by healthy dogs, raising the possibility of using low V_T lung protective ventilation in dogs with ARDS in an effort to reduce ventilator-induced lung injury in these patients. Further work in dogs with ARDS is warranted to assess whether 6 mL/kg V_T provides adequate ventilation for these patients.

Footnotes

[a] Hydromrophone, Baxter Healthcare Corporation, Deerfield, IL.
[b] Diazepam, Hospira Inc, Lake Forrest, IL.
[c] Propofol, Propoflo, Abbott Animal Health, North Chicago, IL.
[d] Lactated Ringer's Solution, Baxter Healthcare Corporation.
[e] Puritan Bennett 840 Critical Care Ventilator, Puritan Bennett LLC, Boulder, CO.
[f] Carprofen, Pfizer Animal Health, Maddison, NJ.

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