A comparison of the cardiopulmonary effects of pressure controlled ventilation and volume controlled ventilation in healthy anesthetized dogs

Denise T. Fantoni, DVM, PhD; Keila K. Ida, DVM, MSc, PhD; Thomas F. T. Lopes, DVM, MSc; Denise A. Otsuki, DVM, PhD; José Otávio C. Auler Jr., DM, PhD and Aline M. Ambrósio, DVM, PhD

Abstract

Objective – To compare the effects of pressure controlled ventilation (PCV) with volume-controlled ventilation (VCV) on lung compliance, gas exchange, and hemodynamics in isoflurane-anesthetized dogs.

Design – Prospective randomized study.

Setting – Veterinary teaching hospital.

Animals – Forty client-owned bitches undergoing elective ovariohysterectomy.

Interventions – Dogs were randomly assigned to be ventilated with 100% oxygen using PCV (n = 20) or VCV (n = 20). The respiratory rate was 20/min and positive end-expiratory pressure (PEEP) was 5 cm H₂O, with a tidal volume of 10 mL/kg. Cardiac output (CO) was measured using thermodilution. Cardiopulmonary and blood gas data were obtained during spontaneous ventilation and after 30 (T30) and 60 minutes (T60) of controlled ventilation.

Measurements and Main Results – In dogs ventilated with PCV, at T30 and T60, PIP was lower (11.4 ± 1.9 and 11.1 ± 1.5 cm H₂O, respectively) and static compliance (CST) was higher (51 ± 7 and 56 ± 6 mL/cm H₂O, respectively) than in VCV group (PIP of 14.3 ± 1.3 and 15.5 ± 1.4 cm H₂O; CST of 34 ± 8 and 33 ± 9 mL/cm H₂O, P < 0.0001). Compared with spontaneous ventilation, both groups had decreased alveolar-arterial oxygen difference at T30 and T60 (PCV: 128 ± 32 mm Hg vs 108 ± 20 and 104 ± 16 mm Hg, respectively; VCV: 131 ± 38 mm Hg vs 109 ± 19 and 107 ± 14 mm Hg, respectively; P < 0.01), while CO was maintained at all time points.

Conclusions – Compared to spontaneous ventilation, both ventilatory modes effectively improved gas exchange without hemodynamic impairment. PCV resulted in higher lung CST and lower PIP compared to VCV.


Keywords: canine, mechanical ventilation, pulmonary function, respiratory mechanics

Abbreviations

CaO₂ arterial oxygen content
CI cardiac index
CO cardiac output
CST static compliance
CvO₂ mixed venous oxygen content
CVP central venous pressure
DO₂I oxygen delivery index
MPAP mean pulmonary artery pressure
MV mechanical ventilation
O₂ER oxygen extraction ratio
PAO₂ alveolar oxygen tension
P(A-a)O₂ alveolar-arterial oxygen gradient
PCV pressure controlled ventilation
PE’CO₂ end-tidal carbon dioxide tension
PEEP positive end expiratory pressure
PIP peak inspiratory pressure
PMEAN mean airway pressure
POAP pulmonary artery occlusion pressure

From the Departamento de Cirurgia, Faculdade de Medicina Veterinária e Zootecnia, (Fantoni, Lopes, Ambrósio) and Disciplina de Anestesiologia, Laboratório de Investigação Médica 8, Faculdade de Medicina, (Fantoni, Ida, Otsuki, Auler, Ambrósio) Universidade de São Paulo, São Paulo, SP, Brazil.

This study was funded by a grant from Fundação de Auxílio à Pesquisa do Estado de São Paulo – FAPESP 2001/11715-3.
The authors declare no conflict of interest.

Address correspondence and reprint requests to
Dr. Keila Ida, Laboratório de Investigação Médica LIM-08, Anestesiologia, Faculdade de Medicina, Universidade de São Paulo, Anestesiologia, Faculdade de Medicina, Universidade de São Paulo, Av. Doutor Arnaldo, 455, 2º andar - sala 2120, Cerqueira César, 01246-903, São Paulo, SP, Brazil.
Email: keilaida@usp.br

Submitted April 04, 2014; Accepted January 17, 2015.
Introduction

General anesthesia can result in hypoventilation and pulmonary atelectasis, leading to impaired gas exchange and hypoxemia. Mechanical ventilation (MV), also known as intermittent positive pressure ventilation, can prevent hypoventilation and hypoxemia, but its effects on respiratory mechanics and pulmonary function can vary according to the applied ventilation mode.

Volume-controlled ventilation (VCV) is a mode of MV that delivers a prescribed tidal volume ($V_T$) through a constant inspiratory flow, with peak airway pressure as a dependent variable (Figure 1). Although the VCV mode ensures the delivery of a constant $V_T$, it can result in barotrauma if the pressure required to deliver the $V_T$ is higher than the pulmonary impedance (eg, in some types of acute lung injury). Although peak inspiratory pressure (PIP) and inspiratory flow time ($T_{INSP}$) can be set to values that are less likely to produce barotrauma, VCV is not considered a safe ventilatory mode for patients with lung injury.

Pressure-controlled ventilation (PCV) is an MV mode that is characterized by a decelerating inspiratory flow pattern that rapidly achieves a preset pressure in the early inspiratory phase. The dependent variable is $V_T$, and the fixed pressure is achieved quickly and remains at this level until the $T_{INSP}$ set is achieved (Figure 1). In PCV mode, the rise time does not affect the $T_{INSP}$; however, it determines how quickly the ventilator achieves the set target pressure. The constant pressure throughout the inspiratory phase allows the alveoli to be kept opened during the entire $T_{INSP}$, which in theory maintains lung compliance and pulmonary gas exchange. In addition, the resulting $V_T$ varies with lung compliance and resistance, which can potentially reduce the incidence of barotrauma. However, PCV may deliver an insufficient $V_T$ in cases that the preset pressure is lower than the pressure necessary to overcome the impedance in the respiratory system.

The optimal ventilatory mode for managing lung compliance and supporting pulmonary function remains a subject of debate. In addition, the effects of different ventilatory modes on patient hemodynamics requires further investigation. The intrathoracic pressure produced by MV can decrease venous return, and thus preload, resulting in decreased cardiac output. Preservation of cardiovascular function must also be considered when planning a ventilatory strategy for an individual patient.

Because PCV has theoretical advantages over VCV on pulmonary mechanics, the present study aimed to compare the effects of PCV and VCV on static compliance ($CST$), alveolar-arterial oxygen gradient ($P(A-a)O_2$), and hemodynamics in isoflurane-anesthetized dogs. It was hypothesized that the PCV mode would result in better lung compliance and pulmonary gas exchange than the VCV mode, with no significant effects on hemodynamics.

Materials and Methods

Animals

Forty healthy, client-owned bitches undergoing elective ovariohysterectomy were prospectively randomly allocated to receive PCV ($n = 20$) or VCV ($n = 20$) during anesthesia. Randomization was determined via computer software, and group information was sealed within manila envelopes numbered from 1 to 40, which were opened consecutively as patients were enrolled.

Prior to inclusion in the study, each animal’s medical history was reviewed and a physical examination conducted. Animals were included in the study if they were between 3 and 6 years old, weighed between 10 and 20 kg, and had no known cardiorespiratory abnormalities detected by a complete physical examination, electrocardiogram evaluation, complete blood count, and serum biochemical analysis. This study was approved by the Bioethics Committee from Faculdade de Medicina Veterinária e Zootecnia de Universidade de São Paulo (protocol #21622011) and client consent was obtained before entry of any dog into the study.

Anesthesia

Food was withheld for 12 hours and water for 8 hours before anesthesia. Prior to anesthetic induction, all dogs received 4 mg/kg carprofen SC, which was continued for 3 days after surgery at a dosage of 2.2 mg/kg, PO, q 8 h. Anesthetic premedication consisted of 2 mg/kg meperidine and 0.05 mg/kg acepromazine, both administered IM.

Twenty minutes following premedication, a 20-Ga catheter was placed in the right cephalic vein, and propofol (5 mg/kg, IV) was administered through the catheter for induction of anesthesia. Following orotracheal intubation, animals were positioned in dorsal...
recumbency onto a heated mat, and the endotracheal tube was connected to a rebreathing circuit with a microprocessor-controlled anesthesia ventilator, incorporating a calibrated pneumotachograph of fixed-area type.\(^6\) Anesthesia was maintained with isoflurane vaporized in 100% oxygen, targeting an end-tidal isoflurane percentage between 1.4 and 1.6%. Airway gases were measured by a side-stream, nondispersive infrared gas analyzer.\(^7\) The gas analyzer was calibrated before each experiment and also measured the end-tidal carbon dioxide tension (PE\(_{\text{CO}_2}\)). During surgery, a rescue dose of fentanyl (4 \(\mu\)g/kg, IV) was administered if either heart rate or blood pressure increased 25% from baseline. The need for rescue analgesia, if any, was noted. Fluid therapy with lactated Ringer’s solution\(^8\) was administered at 5 mL/kg/h, IV, throughout the anesthetic period.

Once anesthetized, animals were allowed to spontaneously ventilate for 30 minutes during instrumentation. Subsequently, neuromuscular blockade was instituted by vecuronium\(^9\) infusion (0.3 mg/kg/h, IV) and a neuro-muscular activity monitor\(^10\) was used to ensure complete muscle paralysis and the absence of any inspiratory effort. The MV was then started using the VCV or PCV mode.

**Instrumentation and monitoring**

The metatarsal artery was catheterized with a 22-Ga catheter\(^11\) for continuous monitoring of mean arterial blood pressure (MAP) and for arterial blood-gas sampling. The pressure manometer for blood pressure measurement was placed at the level of the right atrium. The heart rate (HR) and rhythm were assessed by continuous ECG.\(^12\) A 7-Fr pulmonary artery catheter\(^13\) was aseptically and percutaneously introduced into the jugular vein using a modified Seldinger technique. The distal sensor of the catheter was advanced into the pulmonary artery, and placement was confirmed by observing characteristic waveforms. The pulmonary artery catheter was used for mixed-venous blood sampling,\(^14\) and for measurement of central venous pressure (CVP), cardiac output (CO), mean pulmonary artery pressure (MPAP), pulmonary artery occlusion pressure (PAOP), and core body temperature. Pressure transducers were connected to a multiparametric data collection system\(^15\) for continuous monitoring of the pressures and waveforms. Accuracy of the transducers was confirmed with a mercury column prior to each anesthetic procedure. The CO was determined in triplicate using the thermodilution technique by rapidly (10 seconds or less) administering 10 mL of room-temperature 5% glucose solution into the right atrium through the pulmonary artery catheter. The mean value of 3 measurements within \(\pm 10\%\) variation was recorded.

The cardiac index (CI) was calculated to normalize the data for body surface area in m\(^2\) by using a conversion factor appropriate for dogs (\(k \times \text{bodyweight}^{0.66}\), where \(k = 0.12\)).\(^16\) The systemic vascular resistance index (SVRI), pulmonary vascular resistance index (PVRI), stroke volume index (SVI), oxygen delivery index (DO\(_2\)I), oxygen consumption index (VO\(_2\)I), arterial oxygen content (Ca\(_{O_2}\)), mixed venous oxygen content (Cv\(_{O_2}\)), oxygen extraction ratio (O\(_2\)ER), alveolar oxygen tension (PA\(_{O_2}\)), P(A-a)O\(_2\), alveolar dead space-tidal volume ratio (V\(_{DlV}/V_{Talv}\)), and C\(_{ST}\) were calculated according to the following standard formulas listed in Table 1.\(^17\)

Tidal volume, minute volume (VE), PIP, \(P_{\text{PLAT}}\), mean airway pressure (\(P_{\text{MEAN}}\)) and C\(_{ST}\) were measured using a sensor for inspiratory and expiratory flow and volume measurements,\(^5\) located between the endotracheal tube and the Y-piece, and connected to dedicated software for data display.\(^9\) The \(P_{\text{PLAT}}\) and C\(_{ST}\) were measured during an end-inspiratory pause and PIP was measured at the end of inspiration before the inspiratory pause. The end-inspiratory pause technique was performed during a period of an inspiratory pause of 4 seconds in a completely relaxed patient (anesthetized and under neuromuscular blockade), which provided an environment with zero gas flow.
Study protocol

After instrumentation, baseline values for pulmonary and cardiovascular measurements were recorded during spontaneous ventilation. Subsequently, neuromuscular blockade was instituted and MV was initiated using PCV (PCV group, n = 20) or VCV mode (VCV group, n = 20) in a predetermined randomized fashion, with a respiratory rate of 20/min and positive end-expiratory pressure (PEEP) of 5 cm H2O. The VT was targeted to 10 mL/kg in both groups, which was achieved by adjusting FINSPI in the PCV group and inspiratory volume in the VCV group. In VCV-ventilated dogs, the PIP was limited to 20 cm H2O. Following baseline measurements, the dogs underwent ovariohysterectomy, performed by a single senior surgeon, using a routine midline celiotomy and using the same type of suture material for each surgery. Surgery was performed within a 30 minute time period, and timepoints T30 and T60 were defined as 30 and 60 minutes after the beginning of MV, respectively (Figure 2). Measurements were always taken with a closed abdomen.

Following the measurements at T60, animals were recovered from anesthesia. Following return of spontaneous breathing, the neuromuscular paralysis was reversed by neostigmine* (0.04 mg/kg IV) and atropine* (0.04 mg/kg IV), and extubation was performed when constant and repetitive swallowing reflexes were observed.

Statistical analysis

Normal distribution of data was verified using the Kolmogorov–Smirnov test. Body weights between groups were compared using one-way ANOVA. Within a group, variables were analyzed using ANOVA for repeated measures to investigate temporal differences. At each time point, all data were analyzed using one-way ANOVA to compare PCV and VCV groups. Tukey’s post hoc analyses were performed when significant time effects were detected within groups, and Student’s t test was used to investigate differences between groups. Data were analyzed using commercial software and values are presented as mean ± SD. A P value <0.05 was considered statistically significant.

Results

No significant differences in body weight were observed between groups (16.5 ± 2.2 kg in the VCV group and 16.1 ± 2.1 kg in the PCV group). Rescue analgesia or additional doses of vecuronium were not necessary in any study dog. Data summaries for blood-gas analyses and lung compliance are presented in Table 2 and for hemodynamics and oxygenation in Table 3.

Regardless of the ventilation mode, both groups had a significant increase in VE and VT at 30 and 60 minutes of MV compared to spontaneous ventilation (P < 0.001). At these corresponding time-points, these changes were associated with a significantly lower PIP (P < 0.01) and higher CST (P < 0.001) in the PCV group compared to the VCV group (Table 1). Despite differences in PIP and CST between groups, both ventilation modes resulted in a significant increase in pH and PaO2 (P < 0.001), and a significant decrease in PaCO2 (P < 0.0001), PE’CO2 (P < 0.0001), VDVT/VVTOT (P = 0.01), and P(A-a)O2 (P = 0.01) at T30 and T60 compared to spontaneous ventilation. These changes were not significantly different between groups (Table 1). DO2I, VO2I, O2ER, and hemodynamic

---

**Table 1:** Formulas used for calculation of physiological parameters used in the present study.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Formula</th>
</tr>
</thead>
<tbody>
<tr>
<td>SVRI (dyne sec/cm² m²)</td>
<td>(MAP - CVP)/CI × 80</td>
</tr>
<tr>
<td>PVRI (dyne sec/cm² m²)</td>
<td>(MAPAP - PAOP)/CI × 80</td>
</tr>
<tr>
<td>SVI (mL/beat/m²)</td>
<td>CI/HR</td>
</tr>
<tr>
<td>DO2I (mL/min/m²)</td>
<td>CI × CaO2 × 10</td>
</tr>
<tr>
<td>VO2I (mL/min/m²)</td>
<td>(CaO2 - CO2) × CI × 10</td>
</tr>
<tr>
<td>CaO2 (mL/dL)</td>
<td>(1.36 × Hb × SvO2) + 0.003 × PaO2</td>
</tr>
<tr>
<td>CO2 (mL/dL)</td>
<td>(1.36 × Hb × SvO2) + 0.003 × PaO2</td>
</tr>
<tr>
<td>O2ER (%)</td>
<td>[(CaO2 - CO2)/CaO2] × 100</td>
</tr>
<tr>
<td>PAO2 (mm Hg)</td>
<td>[(FiO2 × (Pb - PH2O))] - [(PaCO2 × (1/R))], where FiO2 is the fraction of inspired oxygen (1.0 = 100% O2), Pb is the barometric pressure (760 mm Hg), PH2O is the water vapor pressure in the respiratory system (47 mm Hg), and R is the respiratory quotient (0.8)</td>
</tr>
<tr>
<td>P(A-a)O2 (mm Hg)</td>
<td>PAO2 - PaO2</td>
</tr>
<tr>
<td>VT/VTalv (%)</td>
<td>[(PaCO2 - PE’CO2)/PaCO2] × 100</td>
</tr>
<tr>
<td>CST (mL/cm H2O)</td>
<td>VT/(PPLAT - PEEP)</td>
</tr>
</tbody>
</table>

---

*Veterinary Emergency and Critical Care Society 2016, doi: 10.1111/vec.12485*
variables did not change significantly over time within or between groups (Table 3).

**Discussion**

The key finding in this study was that PCV resulted in improved pulmonary compliance compared to VCV in healthy dogs undergoing isoflurane anesthesia. This difference was not associated with a significant improvement in gas exchange, which was adequate in both groups. In addition, both ventilatory modes effectively treated hypoventilation associated with isoflurane anesthesia, with no hemodynamic impairments.

Compliance is the ratio of change in volume to change in pressure. Because $V_T$ was maintained constant during MV in this study, the improved $C_{ST}$ during PCV indicated that less pressure was required to deliver the same $V_T$. The $C_{ST}$ reflects lung compliance without airway resistance because in this case, the volume change refers to $P_{PLAT}$, as shown in the equation $C_{ST} = V_T / (P_{PLAT} - PEEP)$. It is known that decelerating flow patterns improve the distribution of ventilation in a lung with heterogeneous mechanical properties (may occur in acute lung injury), which might explain the improved $C_{ST}$ in PCV-ventilated dogs of the present study. Although dogs were healthy, it is possible that during the SV portion of the study, when animals were receiving inhalant anesthesia in 100% oxygen, a change in compliance developed in some lung units, which could have led to maldistribution of $V_T$ and ventilation/perfusion ($V/Q$) mismatching. The presence of $V/Q$ mismatch that had developed during spontaneous ventilation was supported by the fact that gas exchange was improved in both groups when MV and PEEP was applied, regardless of the ventilation mode. Because a constant inspiratory pressure can be distributed more homogeneously by a decelerating inspiratory flow, PCV may have resulted in a more profound improvement in $C_{ST}$. However, dogs were ventilated for short periods of time, which might not have been long enough for the development of significant atelectasis that would account for differences in gas exchange between groups. Also, since the study population was healthy, the $PaO_2$ at baseline was already adequate, and the difference in $C_{ST}$ between groups could not have induced further improvements in oxygenation in the PCV group.

Differences in PIP were also noted between PCV and VCV modes. In the present study, they were attributed to the decelerating and constant flow associated with PCV and VCV, respectively. Usually, the decelerating flow in PCV mode produces an initial peak flow that quickly achieves the target pressure, which is maintained during the $T_{INSPI}$. In the VCV mode, however, the flow is fixed to deliver a total $V_T$, which may result in an inappropriately high PIP, regardless of the respiratory impedance. In this situation, ventilator-induced pulmonary injury can occur. In fact, this is the reason why PCV is usually chosen for VCV patients with lung injury. However, the animals from VCV group had no respiratory disease and the higher PIP was not associated with the development of any clinical pulmonary impairment. PIP could have varied depending on resistance of the different components of the respiratory system and the endotracheal tube, since it is measured during gas movement in the inspiratory phase (flow-dependent). Therefore, pulmonary pressure, as reflected by $P_{PLAT}$, was assessed without airway resistance by using the end-inspiratory pause technique. This condition allows the rearrangement of volume in the respiratory system, equalizing the different pressures from the alveoli to the

Table 2: Blood gas values and parameters of ventilation and respiratory mechanics in isoflurane-anesthetized dogs mechanically ventilated with VCV or PCV modes

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group</th>
<th>Baseline</th>
<th>30 minutes</th>
<th>60 minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>VCV</td>
<td>7.32 ± 0.04</td>
<td>7.38 ± 0.03*</td>
<td>7.39 ± 0.04*</td>
</tr>
<tr>
<td></td>
<td>PCV</td>
<td>7.32 ± 0.05</td>
<td>7.38 ± 0.02*</td>
<td>7.38 ± 0.03*</td>
</tr>
<tr>
<td>$PaCO_2$</td>
<td>VCV</td>
<td>54 ± 3</td>
<td>45 ± 4*</td>
<td>46 ± 4*</td>
</tr>
<tr>
<td></td>
<td>PCV</td>
<td>54 ± 4</td>
<td>46 ± 2*</td>
<td>47 ± 3*</td>
</tr>
<tr>
<td>$V_{DVL}/V_{TAL}$</td>
<td>VCV</td>
<td>15 ± 4</td>
<td>10 ± 2*</td>
<td>11 ± 3*</td>
</tr>
<tr>
<td></td>
<td>PCV</td>
<td>16 ± 3</td>
<td>11 ± 2*</td>
<td>11 ± 3*</td>
</tr>
<tr>
<td>$PaO_2$</td>
<td>VCV</td>
<td>453 ± 39</td>
<td>488 ± 19*</td>
<td>489 ± 14*</td>
</tr>
<tr>
<td></td>
<td>PCV</td>
<td>455 ± 31</td>
<td>488 ± 20*</td>
<td>490 ± 18*</td>
</tr>
<tr>
<td>$P(A-a)O_2$</td>
<td>VCV</td>
<td>131 ± 38</td>
<td>109 ± 19*</td>
<td>107 ± 14*</td>
</tr>
<tr>
<td></td>
<td>PCV</td>
<td>128 ± 32</td>
<td>108 ± 20*</td>
<td>104 ± 16*</td>
</tr>
<tr>
<td>$SaO_2$</td>
<td>VCV</td>
<td>100 ± 0</td>
<td>99 ± 0</td>
<td>100 ± 0</td>
</tr>
<tr>
<td></td>
<td>PCV</td>
<td>100 ± 0</td>
<td>99 ± 0</td>
<td>100 ± 0</td>
</tr>
<tr>
<td>$HCO_3^-$</td>
<td>VCV</td>
<td>21 ± 2</td>
<td>22 ± 2</td>
<td>21 ± 2</td>
</tr>
<tr>
<td></td>
<td>PCV</td>
<td>19 ± 4</td>
<td>21 ± 2</td>
<td>20 ± 2</td>
</tr>
<tr>
<td>VE</td>
<td>VCV</td>
<td>2.0 ± 0.5</td>
<td>3.3 ± 0.4*</td>
<td>3.4 ± 0.4*</td>
</tr>
<tr>
<td></td>
<td>PCV</td>
<td>2.1 ± 0.6</td>
<td>3.5 ± 0.7*</td>
<td>3.6 ± 0.7*</td>
</tr>
<tr>
<td>$V_T$</td>
<td>VCV</td>
<td>107 ± 20</td>
<td>167 ± 19*</td>
<td>170 ± 20*</td>
</tr>
<tr>
<td></td>
<td>PCV</td>
<td>99 ± 10</td>
<td>173 ± 33*</td>
<td>178 ± 33*</td>
</tr>
<tr>
<td>(mL)</td>
<td>VCV</td>
<td>14.3 ± 1.3</td>
<td>15.5 ± 1.4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PCV</td>
<td>11.4 ± 1.9</td>
<td>11.1 ± 1.5</td>
<td></td>
</tr>
<tr>
<td>$P_{PLAT}$</td>
<td>VCV</td>
<td>9.9 ± 0.3</td>
<td>10.2 ± 0.7</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PCV</td>
<td>8.4 ± 0.2</td>
<td>8.2 ± 0.2</td>
<td></td>
</tr>
<tr>
<td>$P_{MEAN}$</td>
<td>VCV</td>
<td>4.1 ± 0.8</td>
<td>3.7 ± 0.6</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PCV</td>
<td>3.5 ± 0.3</td>
<td>4.1 ± 0.7</td>
<td></td>
</tr>
<tr>
<td>$C_{ST}$</td>
<td>VCV</td>
<td>34 ± 8</td>
<td>33 ± 9</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PCV</td>
<td>51 ± 7*</td>
<td>56 ± 6*</td>
<td></td>
</tr>
</tbody>
</table>

Values are listed as mean ± SD. pH: arterial pH; $PaCO_2$: partial pressure of carbon dioxide in the arterial blood; $V_{DVL}/V_{TAL}$: instead: alveolar dead space-tidal volume ratio; $PaO_2$: partial pressure of oxygen in the arterial blood; $P(A-a)O_2$: alveolar-arterial oxygen difference; $SaO_2$: arterial oxygen saturation; $HCO_3^-$: plasma bicarbonate; VE: minute volume; $V_T$: tidal volume; PIP: peak inspiratory pressure; $P_{PLAT}$: plateau pressure; $P_{MEAN}$: mean airway pressure; $C_{ST}$: static compliance. †Within a group, values are significantly different from baseline ($P < 0.05$). ‡Within a time-point, values are significantly different from the VCV group ($P < 0.05$).
endotracheal tube. Therefore, in the absence of flow, the P_{PLAT} of the respiratory system measured at the tip of the endotracheal tube equaled the alveolar pressure.\(^6\)\(^9\) This technique revealed that the higher PIP in dogs ventilated with the VCV mode was not associated with a high P_{PLAT} or P_{MEAN}, which were not significantly different from PCV group.

Hemodynamic function was not impaired by either MV mode. A previous study also demonstrated that no significant hemodynamic alterations occurred in healthy, volume-loaded dogs during recruitment maneuvers and high PEEP, even when intrapulmonary pressures higher than those in the present study were applied.\(^2\) It is more likely that MV would impair CO in animals with a low C_{ST}, such as dogs with acute lung injury, regardless of the ventilatory mode.\(^2\)

Besides the differences in C_{ST} and PIP between groups, the values found for each measurement were not outside of the reference interval for dogs. Because the dogs of the present study were healthy and had normal pulmonary gas exchange, the advantages of the PCV mode over the VCV mode may not have been clinically relevant. However, since PCV mode was capable of improving compliance in dogs with normal lungs, these findings raise the possibility that PCV could have a greater impact and benefit in dogs with lung disease.

In conclusion, both PCV and VCV improved gas exchange in healthy dogs undergoing isoflurane anesthesia, without significant hemodynamic impairment. However, PCV resulted in higher C_{ST} and required a lower PIP than VCV to achieve the same V_{T}. Further studies are necessary to assess these ventilatory modes in dogs with respiratory impairments.

### Footnotes

\(^a\) www.randomization.com.
\(^b\) Carprofen, Agerer União Saúde Animal, Embu Guaçu, Brazil.
\(^c\) Dolosal, Cristália Produtos Químicos Farmacêuticos Ltda., Itapira, Brazil.
\(^d\) Acepran (0.2%), Veinil Ind. e Com. de Produtos Veterinários Ltda., Louveira, Brazil.
\(^e\) Safelet, Nipro Medical Corporation, Miami, FL.
\(^f\) Propovan, Cristália Produtos Químicos Farmacêuticos Ltda.
\(^g\) Linea A, Intermed, Cotia, Brazil.
\(^h\) Isoforine, Cristália Produtos Químicos Farmacêuticos Ltda.
\(^i\) PoetIQ, Criticare Systems Inc., Waukesha, WI.
\(^j\) Fentanest; Cristália Produtos Químicos Farmacêuticos Ltda.
References


