JOURNAL OF
Veterinary Emergency
AND Critical Care

Original Study

Journal of Veterinary Emergency and Critical Care **26**(4) 2016, pp 524–530
doi: 10.1111/vec.12485

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

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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 H_2O, 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 H_2O, respectively) and static compliance (C_{ST}) was higher (51 ± 7 and 56 ± 6 mL/cm H_2O, respectively) than in VCV group (PIP of 14.3 ± 1.3 and 15.5 ± 1.4 cm H_2O; C_{ST} of 34 ± 8 and 33 ± 9 mL/cm H_2O, $P < 0.0001$). 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 C_{ST} and lower PIP compared to VCV.

(J Vet Emerg Crit Care 2016; 26(4): 524–530) doi: 10.1111/vec.12485

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

Abbreviations

CaO_2	arterial oxygen content
CI	cardiac index
CO	cardiac output
C_{ST}	static compliance
CvO_2	mixed venous oxygen content
CVP	central venous pressure
DO_2I	oxygen delivery index
MPAP	mean pulmonary artery pressure
MV	mechanical ventilation
O_2ER	oxygen extraction ratio
PAO_2	alveolar oxygen tension
$P(A\text{-}a)O_2$	alveolar-arterial oxygen gradient
PCV	pressure controlled ventilation
$PE'CO_2$	end-tidal carbon dioxide tension
PEEP	positive end expiratory pressure
PIP	peak inspiratory pressure
P_{MEAN}	mean airway pressure
POAP	pulmonary artery occlusion pressure

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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.

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

P_{PLAT}	plateau airway pressure
PVRI	pulmonary vascular resistance index
SVI	stroke volume index
SVRI	systemic vascular resistance index
T_{INSP}	inspiratory time
V_T	tidal volume
VCV	volume controlled ventilation
V_{Dalv}/V_{Talv}	alveolar dead space-tidal volume ratio
VE	respiratory minute volume
VO_2I	oxygen consumption index

Introduction

General anesthesia can result in hypoventilation and pulmonary atelectasis, leading to impaired gas exchange and hypoxemia.[1] 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.[2,3]

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).[2] 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.[2,4–6]

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.[6–8]

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.[9] 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 (C_{ST}), alveolar-arterial oxygen gradient ($P(A\text{-}a)O_2$), 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,[a] 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 of 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[b] 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[c] and 0.05 mg/kg acepromazine,[d] both administered IM.

Twenty minutes following premedication, a 20-Ga catheter[e] was placed in the right cephalic vein, and propofol[f] (5 mg/kg, IV) was administered through the catheter for induction of anesthesia. Following orotracheal intubation, animals were positioned in dorsal

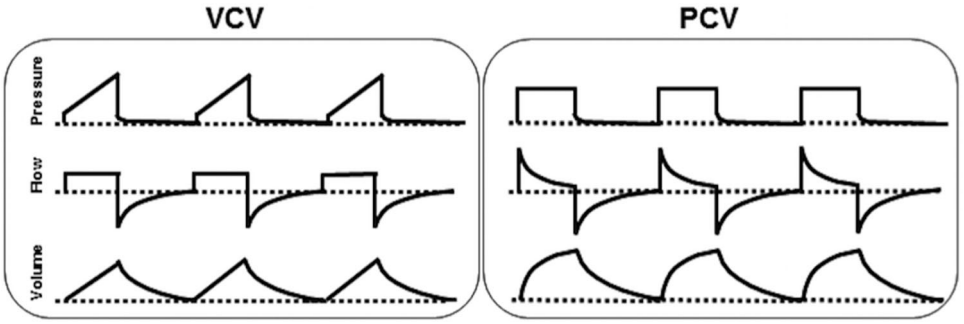

Figure 1: Pressure, flow, and volume waveforms of volume controlled ventilation (VCV) and pressure-controlled ventilation (PCV) modes. Note the square (constant) inspiratory flow and peak inspiratory pressure waveforms in VCV mode, and the decelerating inspiratory flow and square (constant) inspiratory pressure waveforms in PCV mode.

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.[g] Anesthesia was maintained with isoflurane[h] 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.[i] The gas analyzer was calibrated before each experiment and also measured the end-tidal carbon dioxide tension ($PE'CO_2$). During surgery, a rescue dose of fentanyl[j] (4 μg/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[k] 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[l] infusion (0.3 mg/kg/h, IV) and a neuro-muscular activity monitor[m] 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[e] 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.[n] A 7-Fr pulmonary artery catheter[o] was aseptically and percutaneously introduced into the right 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,[p] 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[m] 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 ± 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 $k = 0.12$).[10] The systemic vascular resistance index (SVRI), pulmonary vascular resistance index (PVRI), stroke volume index (SVI), oxygen delivery index (DO_2I), oxygen consumption index (VO_2I), arterial oxygen content (CaO_2), mixed venous oxygen content (CvO_2), oxygen extraction ratio (O_2ER), alveolar oxygen tension (PAO_2), $P(A\text{-}a)O_2$, alveolar dead space-tidal volume ratio (V_{Dalv}/V_{Talv}), and C_{ST} were calculated according to the following standard formulas listed in Table 1.[11]

Tidal volume, minute volume (VE), PIP, P_{PLAT}, mean airway pressure (P_{MEAN}) and C_{ST} were measured using a sensor for inspiratory and expiratory flow and volume measurements,[g] located between the endotracheal tube and the Y-piece, and connected to dedicated software for data display.[q] The P_{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.

Table 1: Formulas used for calculation of physiological parameters used in the present study[11]

SVRI (dynes seconds/cm^5/m^2) = [(MAP – CVP)/CI] × 80
PVRI (dynes seconds/cm^5/m^2) = [(MPAP – PAOP)/CI] × 80
SVI (mL/beat/m^2) = CI/HR
DO_2I (mL/min/m^2) = CI × CaO_2 × 10
VO_2I (mL/min/m^2) = (CaO_2 – CvO_2) × CI × 10
CaO_2 (mL/dL) = (1.36 × Hb × SaO_2) + 0.003 × PaO_2
CvO_2 (mL/dL) = (1.36 × Hb × SvO_2) + 0.003 × PaO_2
O_2ER (%) = [(CaO_2 – CvO_2)/CaO_2] × 100
PAO_2 (mmHg) = [FiO_2 × (Pb – PH_2O)] – [($PaCO_2$ × (1/R)], where FiO_2 is the fraction of inspired oxygen (1.0 = 100% O_2), Pb is the barometric pressure (760 mm Hg), PH_2O is the water vapor pressure in the respiratory system (47 mm Hg), and R is the respiratory quotient (0.8)
P(A-a)O_2 (mm Hg) = PAO_2 – PaO_2
V_{Dalv}/V_{Talv} (%) = [($PaCO_2$ – PE′CO_2)/$PaCO_2$] × 100
C_{ST} (mL/cm H_2O) = V_T/(P_{PLAT} – PEEP)

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 H_2O. The V_T was targeted to 10 mL/kg in both groups, which was achieved by adjusting P_{INSP} in the PCV group and inspiratory volume in the VCV group. In VCV-ventilated dogs, the PIP was limited to 20 cm H_2O. 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[r] (0.04 mg/kg IV) and atropine[s] (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[t] 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 V_T 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 C_{ST} ($P < 0.001$) in the PCV group compared to the VCV group (Table 1). Despite differences in PIP and C_{ST} between groups, both ventilation modes resulted in a significant increase in pH and PaO_2 ($P < 0.001$), and a significant decrease in $PaCO_2$ ($P < 0.0001$), PE′CO_2 ($P < 0.0001$), V_{Dalv}/V_{Talv} ($P = 0.01$), and P(A-a)O_2 ($P = 0.01$) at T30 and T60 compared to spontaneous ventilation. These changes were not significantly different between groups (Table 1). DO_2I, VO_2I, O_2ER, and hemodynamic

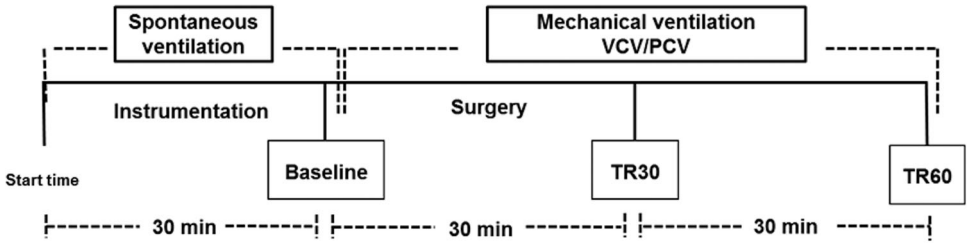

Figure 2: Time line of the study design.

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

			Mechanical ventilation	
Parameter	**Group**	**Baseline**	**30 minutes**	**60 minutes**
pH	VCV	7.32 ± 0.04	7.38 ± 0.03*	7.39 ± 0.04*
	PCV	7.32 ± 0.05	7.38 ± 0.02*	7.38 ± 0.03*
$PaCO_2$	VCV	54 ± 3	45 ± 4*	46 ± 4*
(mm Hg)	PCV	54 ± 4	46 ± 2*	47 ± 3*
V_{Dalv}/V_{Talv}	VCV	15 ± 4	10 ± 2*	11 ± 3*
(%)	PCV	16 ± 3	11 ± 2*	11 ± 3*
PaO_2	VCV	453 ± 39	488 ± 19*	489 ± 14*
(mm Hg)	PCV	455 ± 31	488 ± 20*	490 ± 18*
$P(A\text{-}a)O_2$	VCV	131 ± 38	109 ± 19*	107 ± 14*
(mm Hg)	PCV	128 ± 32	108 ± 20*	104 ± 16*
SaO_2	VCV	100 ± 0	99 ± 0	100 ± 0
(%)	PCV	100 ± 0	99 ± 0	100 ± 0
HCO_3^-	VCV	21 ± 2	22 ± 2	21 ± 2
(mmol/L)	PCV	19 ± 4	21 ± 2	20 ± 2
VE	VCV	2.0 ± 0.5	3.3 ± 0.4*	3.4 ± 0.4*
(L)	PCV	2.1 ± 0.6	3.5 ± 0.7*	3.6 ± 0.7*
V_T	VCV	107 ± 20	167 ± 19*	170 ± 20*
(mL)	PCV	99 ± 10	173 ± 33*	178 ± 33*
PIP	VCV	—	14.3 ± 1.3	15.5 ± 1.4
(cm H_2O)	PCV	—	11.4 ± 1.9†	11.1 ± 1.5†
P_{PLAT}	VCV	—	9.9 ± 0.3	10.2 ± 0.7
(cm H_2O)	PCV	—	8.4 ± 0.2	8.2 ± 0.2
P_{MEAN}	VCV	—	4.1 ± 0.8	3.7 ± 0.6
(cm H_2O)	PCV	—	3.5 ± 0.3	4.1 ± 0.7
C_{ST}	VCV	—	34 ± 8	33 ± 9
(mL/cm H_2O)	PCV	—	51 ± 7†	56 ± 6†

Values are listed as mean ± SD. pH: arterial pH; $PaCO_2$: partial pressure of carbon dioxide in the arterial blood; V_{Dalv}/V_{Talv}: instead: alveolar dead space-tidal volume ratio; PaO_2: partial pressure of oxygen in the arterial blood; $P(A\text{-}a)O_2$: 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 ($P < 0.05$).

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 (as may occur in acute lung injury),[4] 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,[12–16] 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.[9,16,17] Because a constant inspiratory pressure can be distributed more homogenously by a decelerating inspiratory flow, PCV may have resulted in a more profound improvement in C_{ST}.[2,5,7,8,16] 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_{INSP}. 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.[6,8,18] In fact, this is the reason why PCV is usually chosen over VCV for patients with lung injury.[2,4,5] 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 3: Hemodynamic and cardiopulmonary variables in isoflurane-anesthetized dogs mechanically ventilated with VCV or PCV modes

			Mechanical ventilation	
Parameter	**Group**	**Baseline**	**30 minutes**	**60 minutes**
HR	VCV	111 ± 22	118 ± 23	119 ± 21
(beats/min)	PCV	110 ± 19	118 ± 18	117 ± 14
MAP	VCV	78 ± 8	81 ± 8	79 ± 9
(mm Hg)	PCV	79 ± 9	84 ± 9	78 ± 9
CVP	VCV	8 ± 0.8	8 ± 0.5	7 ± 0.8
(mm Hg)	PCV	7 ± 0.8	8 ± 0.8	8 ± 0.5
MPAP	VCV	18 ± 2	18 ± 3	18 ± 2
(mm Hg)	PCV	17 ± 1	16 ± 2	17 ± 2
PAOP	VCV	7 ± 0.8	7 ± 0.9	7 ± 0.8
(mm Hg)	PCV	7 ± 0.7	6 ± 0.8	7 ± 0.5
CO	VCV	4.5 ± 0.5	4.5 ± 0.8	4.7 ± 0.8
(L/minute)	PCV	4.3 ± 0.8	4.3 ± 0.7	4.3 ± 0.6
CI	VCV	5.9 ± 0.8	5.9 ± 0.9	6.2 ± 0.8
(L/min/m^2)	PCV	5.5 ± 0.4	5.5 ± 0.6	5.5 ± 0.7
SVI	VCV	53.1 ± 2.4	50.0 ± 3.8	52.1 ± 3.6
(mL/beat/m^2)	PCV	50.0 ± 1.7	46.6 ± 2.6	47.0 ± 2.7
SVRI	VCV	1024 ± 67	1148 ± 171	1061 ± 75
(dynes seconds/cm^5/m^2)	PCV	1050 ± 118	1148 ± 178	1103 ± 50
PVRI	VCV	142 ± 19	140 ± 32	145 ± 41
(dynes seconds/cm^5/m^2)	PCV	149 ± 14	144 ± 32	149 ± 28
DO_2I	VCV	1225 ± 113	1370 ± 92	1347 ± 186
(mL/min/m^2)	PCV	1197 ± 96	1313 ± 158	1302 ± 152
VO_2I	VCV	153 ± 16	131 ± 11	133 ± 19
(mL/min/m^2)	PCV	143 ± 14	114 ± 16	100 ± 16
O_2ER	VCV	12 ± 1	10 ± 1	10 ± 1
(%)	PCV	12 ± 2	10 ± 1	8 ± 1

Values are listed as mean ± SD. HR: heart rate; MAP: mean arterial pressure; CVP: central venous pressure; MPAP: mean pulmonary arterial pressure; POAP: pulmonary arterial occlusion pressure; CO: cardiac output; CI: cardiac index; SVI: stroke volume index; SVRI: systemic vascular resistance index; PVRI: pulmonary vascular resistance index; DO_2I: systemic oxygen delivery index; VO_2I: systemic oxygen consumption index; O_2ER: oxygen extraction ratio.

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.[6,19] 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.[20] 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 Cst 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] Carproflan, Agener União Saúde Animal, Embu Guaçu, Brazil.
[c] Dolosal, Cristália Produtos Químicos Farmacêuticos Ltda., Itapira, Brazil.
[d] Acepran 0.2%, Vetnil Ind. e Com. de Produtos Veterinários Ltda., Louveira, Brazil.
[e] Safelet, Nipro Medical Corporation, Miami, FL.
[f] Propovan, Cristália Produtos Químicos Farmacêuticos Ltda.
[g] Línea 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.

[k] Lactated Ringer's solution, Cristália Produtos Químicos Farmacêuticos Ltda.
[l] Norcuron, Intervet Schering-Plough, Cotia, Brazil.
[m] TOF-Guard Biometer, Organon Teknika, São Paulo, Brazil.
[n] Viridia CMS, Hewlett-Packard, Andover, MA.
[o] Swan Ganz, Edward Lifesciences, Irvine, CA.
[p] ABL5, Radiometer, Copenhagen, Denmark.
[q] WinTracer 3.3 beta, Intermed.
[r] Normastig, União Química, São Paulo, Brazil.
[s] Hytropin, HypoFarma, Minas Gerais, Brazil.
[t] SigmaPlot, Systat Software, San Jose, CA.

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