Two levels of the inspired oxygen fraction in propofol-anesthetized dogs with high intracranial pressure: cardiopulmonary function

[*Dois níveis de fração inspirada de oxigênio em cães anestesiados com propofol e com pressão intracraniana elevada: função cardiopulmonar*]

P.C. *Ferro Lopes*[1], N. *Nunes*[2], E.A *Belmonte*[1], R. M. *Almeida*[1], L.G.G.G. *Dias*[1], G.B. *Pereira Neto*[1], A.L.G. *Souza*[1]

[1]Aluno de pós-graduação – Faculdade de Ciências Agrárias e Veterinárias – FCAV-Unesp– Jaboticabal, SP
[2]Faculdade de Ciências Agrárias e Veterinárias – FCAV-Unesp– Jaboticabal, SP

ABSTRACT

In the initial stage of traumatic brain injury, the use of 1.0 inspired oxygen fraction (FiO_2) is indicated. However, high FiO_2 has been correlated with atelectasis. Thus, the effects of $FiO_2 = 1.0$ and $FiO_2 = 0.6$ on the cardiopulmonary function in propofol-anesthetized dogs with high intracranial pressure (ICP) were evaluated. Eight dogs were anesthetized on two occasions, receiving, during controlled ventilation, an $FiO_2 = 1$ (G100) or an $FiO_2 = 0.6$ (G60). Propofol was used for induction ($10mg.kg^{-1}$) followed by a continuous rate infusion ($0.6mg.kg^{-1}.minute^{-1}$). An increase in the ICP was induced by temporary obliteration of the right jugular vein (OJ_v) 50 minutes after induction of anesthesia. The measurement was taken twenty minutes after OJ_v (T0) and then at 15-minute intervals (T15 to T60). Alveolar oxygen partial pressure in G60 was lower than in G100 during the whole procedure. Alveolar-arterial oxygen gradient in G100 was greater than in G60 at T0 and at T60. No differences were observed for arterial oxygen partial pressure/inspired oxygen fraction ratio, arterial-to-alveolar oxygen pressure ratio, respiratory index, venous admixture, oxygen delivery, oxygen consumption, oxygen extraction, heart rate, mean pulmonary arterial pressure, pulmonary arterial occlusion pressure, cardiac index, stroke index and systemic vascular resistance index. In G100, mean arterial pressure at T0 was higher than at T45. In dogs with high ICP, the cardiopulmonary function was not influenced by the different FiO_2 used.

Keywords: dog, total intravenous anesthesia, cerebral autoregulation, hemodynamic, venous admixture

RESUMO

No estágio inicial do trauma encefálico, o emprego de fração inspirada de oxigênio (FiO_2) de 1,0 é indicado. Todavia, altas FiO_2 têm sido correlacionadas com atelectasia. Assim, avaliaram-se os efeitos das $FiO_2 = 1.0$ e $FiO_2 = 0.6$ sobre a função cardiopulmonar em cães com pressão intracraniana (PIC) elevada e anestesiados com propofol. Oito animais foram anestesiados em duas ocasiões e receberam, durante a ventilação controlada, $FiO_2 = 1$(G100) ou $FiO_2 = 0,6$ (G60). Propofol foi usado para indução ($10mg.kg^{-1}$) e seguido por infusão contínua ($0,6mg.kg^{-1}minuto^{-1}$). O aumento da PIC foi induzido pela obliteração temporária da veia jugular (OJ_v). As mensurações foram realizadas 20 minutos após OJ_v (T0) e em intervalos de 15 minutos (de T15 a T60). A pressão parcial de oxigênio alveolar no G60 foi menor do que no G100 durante todo o procedimento. O gradiente alveolar-arterial no G100 foi maior do que no G60, em T0 e T60. Não foram observadas diferenças para: relação pressão parcial de oxigênio/fração inspirada de oxigênio, relação arterioalveolar, índice respiratório, mistura arteriovenosa, oferta de oxigênio, consumo de oxigênio, taxa de extração de oxigênio, frequência cardíaca, pressão da artéria pulmonar média, pressão de oclusão da artéria pulmonar, índice cardíaco, índice sistólico e índice de resistência vascular sistêmica. No G100, a pressão arterial média em T0 foi maior do que em T45. Em cães com alta PIC, a função cardiopulmonar não foi influenciada pelas diferentes FiO_2 empregadas.

Palavras-chave: cão, anestesia intravenosa total, autorregulação cerebral, hemodinâmica, mistura arteriovenosa

INTRODUCTION

Normal intracranial pressure (ICP) in dogs is 5 to 12mmHg, similar to that of humans for whom 20mmHg is an arbitrary upper limit beyond which treatment for ICP may be instituted. Causes of intracranial hypertension can be nonvascular (interstitial edema, intracellular swelling, masses or obstruction of cerebrospinal fluid) or vascular, which include cerebral vasodilatation caused by increased arterial partial pressure of carbon dioxide ($PaCO_2$), distention of cerebral vessels or venous outflow obstruction (Sturges and Lecouteur, 2009).

In the initial stage of traumatic brain injury, the use of 1.0 inspired oxygen fraction (FiO_2) is indicated (Falcão *et al.*, 2006). However, during anaesthesia, high FiO_2 has been correlated with atelectasis (Hedenstierna *et al.*, 1989), which can cause $PaCO_2$ increase (Haskins, 2007) that can interfere with cerebral blood flow (Falcão *et al.*, 2006) and promote the impairment of cerebral autoregulation (Häggendal and Johansson, 1965; McCulloch *et al.*, 2000).

In several investigations, atelectasis formation with subsequent pulmonary shunting was shown to be related to impairment of gas exchange (Brismar *et al.*, 1985). Thus, a low FiO_2 has been advocated to avoid pulmonary collapse (Magnusson and Spahn, 2003).

The hypothesis was that a $FiO_2 = 1.0$ has a detrimental effect on pulmonary gas exchange and can aggravate intracranial hypertension. Thus, this study was designed to establish the effects of two different levels of FiO_2 (1.0 and 0.6) on the cardiopulmonary function in propofol-anesthetized dogs with a high ICP.

MATERIALS AND METHODS

This study was approved by the regional Institutional Ethics and Animal Welfare Committee (Ref: 003933-07). After the study the animals were available for adoption.

Eight adult mongrel dogs weighing 10.1±2.0kg, 4 males and 4 females, were enrolled in the study. All animals were determined to be healthy based on clinical and laboratorial evaluation. Blood, urine, electrocardiogram and thoracic radiography exams were all done. They were provided with water and regular dog food and kept in individual cages at the Veterinary Teaching Hospital.

The dogs were anesthetized in 2 occasions, 21 d apart, and received oxygen at an FiO_2 of 1.0 (G100) or 0.6 (G60) in a randomized cross-over study.

Anesthesia was induced with 10mg.kg^{-1}of propofol (Fresofol 1%, Fresenius Kabi Brasil Ltda, Campinas, SP, Brazil) administered intravenously (IV). After endotracheal intubation, the dog received oxygen at the assigned FiO_2 and a flow of 30 or 50mL.kg^{-1}.minute^{-1} through an anesthetic circuit (Ohmeda - Excel 210SE, Madison, EUA – Proc. FAPESP 97/10668-4), that allowed partial rebreathing of gases, equipped with pressure/volumetric ventilator (Ohmeda). The oxygen concentration was checked using gas equipment (DX 2010LCD, Dixtal, Manaus, AM, Brazil - Proc. FAPESP 02/04625-0). Controlled ventilation began immediately, and the respiratory rate was adjusted to maintain end tidal carbon dioxide ($ETCO_2$) between 35-45 mmHg. $ETCO_2$ was monitored using a respiratory monitor (DX 8100, Dixtal, Manaus, AM, Brazil - Proc. FAPESP 03/11125-7). Adjustments were made in ventilation before the first measurement and, consequently, before intracranial hypertension induction and no more adjustments were made thereafter.

Anesthesia was maintained with a continuous rate infusion (CRI) (Infusion Pump 670T, Samtronic Ltda, São Paulo, SP, Brazil - Proc. FAPESP 03/11125-7) of propofol at a rate of 0.6mg.kg^{-1}.minute^{-1} IV. The propofol dose was obtained from previous studies. Additionally, lidocaine (Xylestesin 2% – Cristália Produtos Químicos Farmacêuticos Ltda – Itapira, SP) infiltration was performed on the right cerebral cortex and the right cervical region.

The dogs were positioned on left lateral recumbency. For assessment of the ICP, a fiber-optic catheter (Monitor de temperatura-pressão intracraniana, 110-4BT - Integra Neur Camino Labs, San Diego, EUA – Proc. FAPESP 00/01084-3) was surgically implanted on the surface of the right cerebral cortex using an access kit (Integra Neur Camino Labs – Proc. FAPESP 00/01084-3), according to the technique described by Bagley *et al.*, (1995). An intra-arterial catheter was then placed on the right dorsal pedal artery to assess the mean arterial pressure (MAP) and to obtain arterial blood, in order to evaluate blood gas variables (Roche Omi C blood gas analyzer, Roche Diagnostics GmbH, Mannheim, Germany). Before the beginning of each experiment the pressure transducer was checked against a mercury manometer. The zero reference point was at the level of the costochondral junction.

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From these data, the following parameters were calculated:

$PAO_2 = [FiO_2 \times (Pb - 47)] - (PaCO_2/RQ)$; where PAO_2 = alveolar partial pressure of oxygen, Pb = barometric pressure, $PaCO_2$ = arterial partial pressure of carbon dioxide and RQ = respiratory quotient, which was assumed equal to 0.8.

$AaDO_2 = PAO_2 - PaO_2$; where $AaDO_2$ = Alveolar-arterial oxygen gradient and PaO_2 = arterial partial pressure of oxygen.
$RI = AaDO_2/ PaO_2$; where RI = respiratory index.

Calculation of the PaO_2/FiO_2 ratio and the arterial-to-alveolar oxygen tension ratio (PaO_2/PAO_2) were also carried out.

Subsequently, a thermodilution catheter (Swan–Ganz mod 132 5 French; Edwards Lifesciences, Irvine, California, USA) was introduced surgically into the femoral vein and advanced into the lumen of the pulmonary artery in order to evaluate cardiac output (CO), which was measured directly by the thermodilution technique with a microprocessed device (Dixtal mod. DX 2010, CO module, Manaus, AM, Brazil – Proc. FAPESP 96/02877-0). The catheter's position was confirmed by observation of characteristic changes in the pressure tracings as the catheter tip was advanced from the right ventricle into the pulmonary artery. Central venous pressure (CVP), mean pulmonary arterial pressure (mPAP) and pulmonary arterial occlusion pressure (PAOP) were measured via the thermodilution catheter.

Seldinger's technique to introduce the thermodilution catheter in the jugular vein was not used, because the bilateral obliteration of the jugular vein could increase intracranial pressure excessively and promote irreversible damage.

The oxygen delivery ($DO_2 = CaO_2 \times CO \times 10$, where CaO_2 = oxygen content of arterial blood), the cardiac index (CI = CO/body surface area) and systemic vascular resistance index [SVRI = (MAP – CVP) x 80/CI] were calculated.

Heart rate (HR) was calculated from the simultaneous lead II electrocardiogram. The stroke index (SI) was calculated by (CO/HR)/body surface area.

Venous blood was obtained from the thermodilution catheter's main branch to evaluate blood gas variables. From these data, the following parameters were calculated:

$Qs/Qt = [(CcO_2 - CvO_2)/(CcO_2 - CaO_2)] \times 100$ where Qs/Qt = venous admixture, CcO_2 = oxygen content of end-capillary blood and CvO_2 = oxygen content of mixed venous blood.

$VO_2 = avDO_2 \times CO \times 10$ where VO_2 = oxygen consumption and $avDO_2$ = arteriovenous difference of oxygen content,
$TeO_2 = VO_2 /DO_2$ where TeO_2 = oxygen extraction.

Twenty minutes after implanting the fiber-optic catheter, an increase in ICP was induced for obliteration of the jugular vein. The right jugular vein was exposed surgically and subjected to a provisory ligature consisting of 2 cotton threads 3cm apart. The aim of this procedure was to obtain ICP values higher than 15mmHg. Lower values have been considered normal (Johnston *et al.*, 1991; Plöchl *et al.*, 1998). The ICP was measured and cerebral perfusion pressure (CPP) was calculated subtracting the ICP value from the MAP.

The first measurement of each variable was taken 20min after (T0) obliteration of the right jugular vein (OJ$_v$), and additional recordings were performed at 15-min intervals (T15, T30, T45, and T60).

Numerical data were subjected to one-way analysis of variance (ANOVA) to determine the difference between the different time points of the same group. Two-way ANOVA was used between groups. Bonferroni test was used for post-hoc multiple comparisons at a α level of 0.05. Analyses were performed using Prism 5 for Windows (GraphPad Software Inc, CA, USA).

In the recovery of anesthesia, the dog's wounds were covered with sterile bandages. Between anesthesia procedures animals were provided with water and regular dog food and kept in individual cages. Dogs were given carprofen (Rimadyl, 25mg; Pfizer, Guarulhos, São Paulo, Brazil), 4.4mg/kg orally once a day (SID) for 3 d, and benzathine penicillin G (Billi Farmacêutica, Santo Amaro, São Paulo, Brazil), 40.000U/kg intramuscularly once a day for 5d. One person was responsible for surgical wound care, medication and taking the dog for a walk twice a day.

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RESULTS

The baseline ICP was 15 (5)mmHg for G100 and 13 (4)mmHg for G60. By 20min after OJ_v (T0) the ICP had increased 46.67% in G100 and 38.46% in G60 (Figure 1). The ICP and CPP values did not differ significantly ($P>0.05$) between FiO_2 levels or between measurement times after OJ_v (Table 1).

Table 1. Cardiopulmonary measurements in dogs with high intracranial pressure anesthetized with propofol and mechanically ventilated with FiO_2 of 1.0 (G100) or 0.6 (G60)

Parameter	Group	Times				
		T0	T15	T30	T45	T60
PAO_2	G100	616 (4)**A**	613 (5)**A**	615 (4)**A**	612 (5)**A**	614 (9)**A**
(mm Hg)	G60	348 (6)**B**	346 (7)**B**	345 (5)**B**	347 (5)**B**	349 (5)**B**
$AaDO_2$	G100	95 (65)**A**	70 (39)	82 (45)	83 (21)	99 (53)**A**
(mm Hg)	G60	36 (14)**B**	41 (23)	47 (19)	45 (14)	47 (19)**B**
PaO_2/FiO_2	G100	521 (63)	543 (35)	534 (45)	529 (24)	516 (45)
(mm Hg)	G60	519 (27)	508 (32)	498 (36)	503 (25)	503 (34)
PaO_2/PAO_2	G100	0.85 (0.10)	0.88 (0.06)	0.87 (0.07)	0.86 (0.03)	0.84 (0.09)
	G60	0.89 (0.04)	0.88 (0.07)	0.86 (0.05)	0.87 (0.04)	0.87 (0.06)
RI	G100	0.20 (0.16)	0.13 (0.08)	0.16 (0.11)	0.16 (0.04)	0.20 (0.12)
	G60	0.12 (0.06)	0.14 (0.08)	0.16 (0.07)	0.15 (0.05)	0.16 (0.07)
Qs/Qt	G100	21 (8)	21 (7)	23 (14)	28 (18)	27 (12)
(%)	G60	17 (4)	22 (22)	16 (3)	20 (9)	17 (5)
DO_2	G100	294 (148)	274 (142)	284 (132)	277 (117)	285 (149)
(ml minute^{-1})	G60	258 (114)	243 (116)	238 (108)	226 (91)	238 (110)
VO_2	G100	69 (35)	57 (24)	61 (33)	52 (27)	55 (31)
(ml minute^{-1})	G60	61 (20)	69 (43)	66 (35)	56 (24)	67 (31)
TeO_2	G100	24 (6)	22 (8)	23 (9)	21 (12)	20 (7)
(%)	G60	25 (8)	28 (14)	29 (9)	27 (12)	29 (8)
HR	G100	135 (27)	137 (27)	130 (26)	130 (21)	136 (21)
(beat minute^{-1})	G60	144 (35)	139 (33)	137 (33)	137 (33)	139 (26)
MAP	G100	101 (8)**a**	95 (10)	95 (10)	92 (10)**b**	96 (9)
(mm Hg)	G60	105 (19)	103 (17)	104 (18)	101 (18)	101 (18)
mPAP	G100	8 (3)	10 (3)	9 (3)	9 (3)	10 (3)
(mm Hg)	G60	12 (4)	11 (5)	11 (5)	10 (5)	10 (5)
PAOP	G100	2 (3)	2 (3)	2 (3)	2 (3)	2 (3)
(mm Hg)	G60	5 (2)	3 (3)	3 (3)	3 (3)	3 (3)
CI	G100	4.2 (1.6)	3.8 (1.3)	4.0 (1.0)	3.9 (1.0)	3.9 (1.3)
(L minute^{-1} m^{-2})	G60	3.8 (1.0)	3.5 (0.9)	3.4 (0.9)	3.5 (0.6)	3.5 (0.9)
SI	G100	31 (11)	30 (10)	31 (9)	30 (5)	29 (8)
(L beat^{-1} m-^{2})	G60	27 (3)	26 (8)	26 (7)	26 (7)	26 (5)
SVRI	G100	2129 (802)	2208 (738)	1990 (526)	1966 (458)	2087 (569)
(dina seg cm^{-5}xm^{-2})	G60	2213 (430)	2460 (595)	2558 (774)	2349(485)	2361(581)
ICP	G100	22 (6)	21 (6)	22 (7)	22 (7)	23 (8)
(mm Hg)	G60	18 (5)	19 (6)	18 (4)	17 (5)	18 (6)
CPP	G100	79 (8)	74 (11)	72 (11)	70 (12)	74 (12)
(mm Hg)	G60	87 (21)	85 (19)	86 (20)	84 (20)	83 (21)

Data are expressed as mean (SD). Means with different upper case letters within each column differ significantly from one another and means with different lower case letters within each row differ significantly from one another. PAO_2, alveolar oxygen partial pressure; $AaDO_2$, Alveolar-arterial oxygen gradient; PaO_2/FiO_2, arterial oxygen partial pressure/inspired oxygen fraction ratio; PaO_2/PAO_2, arterial-to-alveolar oxygen tension ratio; RI, respiratory index; DO_2, oxygen delivery; VO_2, oxygen consumption; TeO_2, oxygen extraction; HR, heart rate; MAP, arterial mean pressure; mPAP, mean pulmonary arterial pressure; PAOP, pulmonary arterial occlusion pressure; CI, cardiac index; SI, stroke index; SVRI, systemic vascular resistance index; ICP, intracranial pressure; CPP, cerebral perfusion pressure.
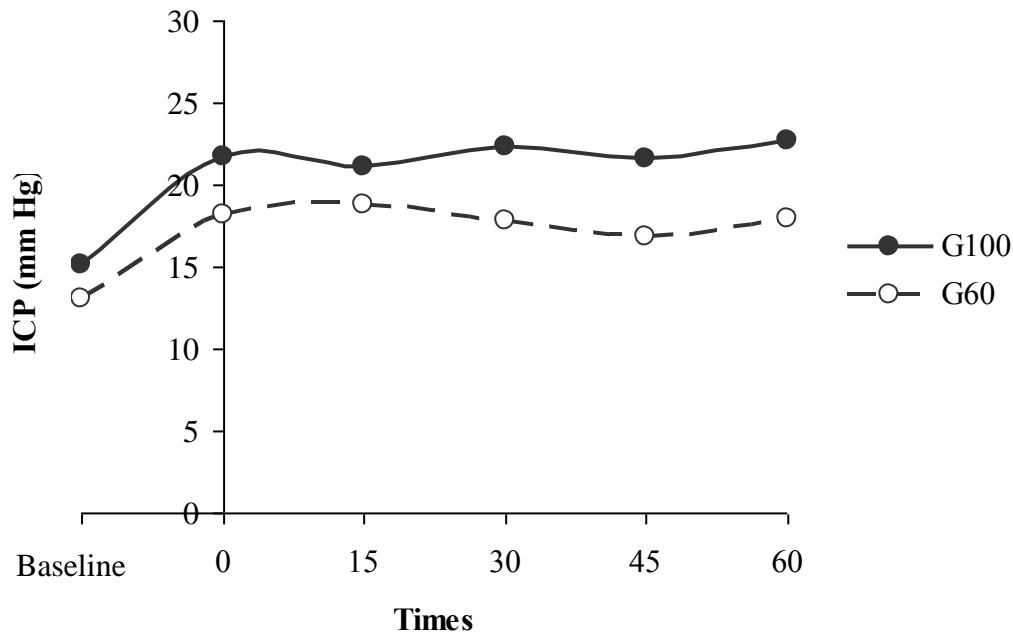
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Figure 1. Intracranial pressure (ICP) monitoring in dogs anesthetized with propofol, mechanically ventilated with FiO_2 of 1.0 (G100) or 0.6 (G60) and submitted to a temporary obliteration of the right jugular vein. Black filled line and circles represent the group that received $FiO_2 = 1.0$ (G100) and black spotted lines and open circles represent the group that received $FiO_2 = 0.6$ (G60).

Values of PAO_2 were significantly different (P<0.001) between groups throughout the entire procedure (Table 1), but they did not differ among measurements (P>0.05). With regard to $AaDO_2$, the values in G100 were higher than the means recorded in G60 at T0 (P<0.01) and at T60 (P<0.05).

PaO_2/FiO_2, PaO_2/PAO_2, RI, Qs/Qt, DO_2, VO_2, TeO_2, HR, mPAP, PAOP, CI, SI, SVRI (Table 1) and CVP did not differ significantly among measurements or between groups (P>0.05). In G100, MAP at T0 was higher than at T45 (P<0.05).

DISCUSSION

Propofol was used in this study because it is known to maintain or decrease the ICP while it maintains the CPP in patients with normovolemia and stable hemodynamics (McKeage and Perry, 2003), hence, it may commonly be selected for patients with suspicious ICP increase (Armitage-Chan *et al.*, 2007).

In patients with increased ICP, the mechanical positive pressure ventilation should be immediately available and instituted at induction, being recommended to maintain the normocapnia (Armitage-Chan *et al.*, 2007; Harvey *et al.*, 2007). Besides, for the initial stage of traumatic brain injury, 100% oxygen has been indicated (Falcão *et al.*, 2006). In this study, FiO_2 of 1.0 was chosen because the 100% oxygen has been used often during anesthesia procedure (Nunes *et al.*, 2011; Paula *et al.*, 2010). However, in dogs anesthetized with propofol ($0.7 mgkg^{-1}minute^{-1}$) and maintained in spontaneous breathing with different FiO_2 (1.0, 0.8, 0.6, 0.4 and 0.21), authors concluded that 60% of oxygen should be used (Nunes *et al.*, 2008).

Before occlusion of the jugular vein, ICP values were within the range of values considered normal (Johnston *et al.*, 1991; Plöchl *et al.*, 1998) (Figure 1), but after temporary obliteration of the right jugular vein, ICP increase was clinically significant. From T0, the administration of several FiO_2 did not cause

changes in ICP. The application of mechanical ventilation sought to minimize changes in $PaCO_2$, which can have some direct influence on ICP, CPP and cerebral autoregulation (Harvey *et al.*, 2007; McCulloch *et al.*, 2000).

The normal interval for CPP is between 50 and 150mmHg (Steiner and Andrews, 2006). Hence, in this study, CPP values were within this interval and held constant throughout the entire procedure (Table 1). This stability was attributed to MAP constancy, which is the most important factor in preserving CPP (Sponheim *et al.*, 2003).

PAO_2 is often used as an index of the oxygenating efficiency of the lung (Haskins, 2004). In this study, differences between groups (Table 1) can be explained by different FiO_2 used, because when an enriched O_2 mixture is inspired, the PAO_2 rises (Pasetto and Rinaldi, 2004).

Two factors may contribute to changes in $AaDO_2$: primary ventilation and perfusion mismatch (V/Q) and the decrease of the functional residual capacity (FRC) (Pasetto and Rinaldi, 2004). However, we believe that the difference observed between G100 and G60 (Table 1) was not resultant of FRC decrease, because all dogs were positioned in left lateral recumbency and the propofol rate was the same for the both groups, considering that this drug reduces the contractility of the canine diaphragm (Fujii *et al.*, 2004). Nevertheless, we suggest that V/Q was impaired in G100 when compared to G60.

PaO_2/PAO_2 ratio offers a sensible approach to presume the pulmonary *shunt* (José *et al.*, 2001). The normal value for PaO_2/PAO_2 is between 0.74 and 0.90 and, when this parameter is lower than 0.60, it means that the gas exchange is inefficacious (Kupe and Grant, 1998). Means of PaO_2/FiO_2 below 200mm Hg indicated severe Qs/Qt (Goldfarb *et al.*, 1975) and hypoxemia (Kupe and Grant, 1998). However, in this study, the values registered were greater than 500mm Hg, which is observed for a normal lung (Haskins, 2004).Thus, in this study, the PaO_2/FiO_2 and PaO_2/PAO_2 means were within the interval considered normal (Table 1) indicating lung oxygenating efficiency was similar in both groups.

RI is an index of oxygenation function of the lung and its increase reflects the presence of pulmonary *shunt* in a variety of conditions including atelectasis, pulmonary contusion and pulmonary troboembolism (Goldfarb *et al.*, 1975). During the whole procedure in this study, RI was stable (Table 1) and within the interval considered normal, from 0.1 to 0.37 (Goldfarb *et al.*, 1975) in all groups.

For Qs/Qt no significant difference between G100 and G60 was observed. In normal awake or anesthetized dogs, Qs/Qt may be up to 10% of the CO (Haskins, 2004). In both groups Qs/Qt means were higher than 10%, which are considered to be increased (Haskins, 2004).

Factors that can change DO_2 are: cardiac rhythm, blood pressure, body temperature, PaO_2, pH, CO and the quantity of haemoglobin (Mazzaferro, 2009), but, in this study, these parameters were stable, justifying the stability observed to DO_2. The use of different FiO_2 did not impair DO_2, VO_2 and TeO_2 (Table 1). Oxygen extraction normally ranges between 20% and 30% (Haskins, 2004). Thus, in this study, TeO_2 means were inside this interval. Besides, the stability of TeO_2 can be attributed to DO_2 constancy, because when DO_2 is decreased, the tissues continue to catch their normal amount of oxygen and, therefore, TeO_2 increases (Haskins, 2004).

Regarding HR, the values remained stable in all groups (Table 1), corroborating Nunes *et al.* (2008), who did not observe significant changes in HR and MAP in propofol-anesthetized dogs during spontaneous ventilation with $FiO_2= 1.0$ or $FiO_2= 0.6$. Besides, MAP was higher than 80mm Hg (Table 1), suggesting that perfusion was not impaired, because MAP > 65mm Hg is considered sufficient for perfusion maintenance of all vital organs and tissues (Miller, 1986).

The mPAP and PAOP are parameters used routinely in clinical emergency to estimate the left ventricle filling pressure (Mark, 1998) and left atrial pressure, respectively (Greene *et al.*, 1990; Mark, 1998). In normal awake dogs, the mPAP is 15 ± 4mmHg and PAOP is 5 ± 2mmHg. They are usually decreased by general anesthetics (Haskins, 2007) as it was observed in this study (Table 1). However, no differences between groups or among times were registered. Thus, as described by Nunes *et al.* (2008), the

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administration of different FiO_2 was not able to impair these parameters and consequently ventricular function and preload.

In normal awake dogs, the CI is 4.42 ± 1.24 L minute^{-1}m^{-2} and it is generally decreased by general anesthetics (Haskins, 2007). The values recorded in all groups (Table 1) were lower than the interval proposed by Haskins (2007), which can be justified by propofol-anesthesia and controlled ventilation. The stability of CI and HR can justify the SI constancy, which is the result of the relation between theses parameters (Shoemaker, 1995). Thus, the various FiO_2 used were not able to impair theses parameters.

The SVRI did not differ significantly between groups (Table 1). Considering that SVRI is obtained through mathematical calculation, the registered stability occurred due to the components of equation (MAP, CVP and CI) being stable and did not change with the use of different FiO_2. Thus, the influence of these parameters on the systemic vascular resistance index is clear.

CONCLUSION

In conclusion, in the dogs with high ICP and submitted to continuous infusion of propofol, the cardiopulmonary function was not influenced by the different FiO_2 used. Additionally, $FiO_2=1.0$ was not able to aggravate intracranial hypertension.

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REFERENCES

ARMITAGE-CHAN, E.A.; WETMORE, L.A.; CHAN, D.L. Anesthetic management of the head trauma patient. *J. Vet. Emerg. Crit. Care*, v.17, p.5-14, 2007.

BAGLEY, R.S.; KEEGAN, R.D.; GREENE, S.A. *et al*. Pathologic effects in brain after intracranial pressure monitoring in clinically normal dogs, using a fiberoptic monitoring system. *Am. J. Vet. Res.*, v.56, p.1475-1478, 1995.

BRISMAR, B.; HEDENSTIERNA, G.; LUNDQUIST, H. *et al*. Pulmonary densities during anaesthesia with muscular relaxation: a proposal of atelectasis. *Anesthesiology*, v.62, p.422-428, 1985.

FALCÃO, A.L.E.; FILHO DANTAS, V.P.; TERZI, R.G.G. Ventilação mecânica em pacientes com traumatismo cranioencefálico grave: controle pela pressão intracraniana e SjO$_2$. In: CARVALHO, C.R.R. (Ed). *Ventilação Mecânica Vol. II – Avançado*. São Paulo: Atheneu, 2006. p.181-194.

FUJII, Y.; UEMURA, A.; TOYOOKA, H. The recovery profile of reduced diaphragmatic contractility induced by propofol in dogs. *Anesth. Analg.*, v.99, p.99-113, 2004.

GREENE, S.A.; HARTSFIELD, S.M.; TYNER, C.L. Cardiovascular effects of butorphanol in halothane-anesthetized dogs. *Am. J. Vet. Res.*, v.51, p.1276-1279, 1990.

GOLDFARB, M.A.; CIUREY, T.F.; MCASLAN, T.C. *et al*. Tracking respiratory therapy in the trauma patient. *Am. J. Surg.*, v.129, p.255-258, 1975.

HÄGGENDAL, E.; JOHANSSON, B. Effects of arterial carbon dioxide tension and oxygen saturation on cerebral blood flow autoregulation in dogs. *Acta Physiol. Scand. Suppl.*, v.66, p.27-53, 1965.

HARVEY, R.C.; GREENE, S.A.; THOMAS W. Neurological disease. In: TRANQUILLI, W.J.; THURMON, J.C.; GRIMM, K.A. (Eds). *Lumb & Jones' Veterinary anesthesia and analgesia.* 4. ed. Oxford: Blackwell Publising, 2007. p.903-913.

HASKINS, S.C. Interpretation of blood gas measurements. In: KING, L.G. (Ed). *Textbook of respiratory disease in dogs and cats*. 1. ed. Philadelphia: Saunders, 2004. p.181-192.

HASKINS, S.C. Monitoring anesthetized patients. In: TRANQUILLI, W.J.; THURMON, J.C.; GRIMM, K.A. (Eds). *Lumb & Jones' Veterinary anesthesia and analgesia.* 4. ed. Oxford: Blackwell Publising, 2007. p.533-558.

HEDENSTIERNA, G.; LUNDQUIST, H.; LUNDH, B. *et al*. Pulmonary densities during anaesthesia. An experimental study on lung morphology and gas exchange. *Eur. Respir. J.*, v.2, p.528-535, 1989.

JOHNSTON, W.E.; VINTEN-JOHANSEN, J.; DEWITT, D.S. *et al.* Cerebral perfusion during canine hypothermic cardiopulmonary bypass effect of arterial carbon dioxide tension. *Ann. Thorac. Surg.*, v.52, p.479-489, 1991.

JOSÉ, A.; DIAS, E.C.; SANTOS, V.L.A. *et al.* Predictive value of blood gas analysis and oxygenation score in weaning of mechanical ventilation. *Rev. Bras. Ter. Intensiva*, v.13, p.50-57, 2001.

KUPE, T.J.; GRANT, B.J.B. Arterial blood gas monitoring: respiratory assessment. In: TOBIN, M.J. (Ed). *Principles and practice of intensive care monitoring*. New York: Mc Graw-Hill, 1998. p.197-215.

MAGNUSSON, L.; SPAHN, D.R. New concepts of atelectasis during general anaesthesia. *Br. J. Anaesth.*, v.91, p.61-72, 2003.

MARK, J.B. (Ed). *Atlas of cardiovascular monitoring.* New York: Churchill Livingstone, 1998. 362p.

MAZZAFERRO, E.M. Postoperative evaluation of the critically ill patient. In: SILVERSTEIN, D.C.; HOPPER, K. (Eds). *Small Animal Critical Care Medicine*. St. Louis: Saunders Elsevier, 2009. p.626-629.

McCULLOCH, T.J.; VISCO, E.; LAM, A.M. Graded hypercapnia and cerebral autoregulation during sevoflurane or propofol anesthesia. *Anesthesiology*, v.93, p.1205-1209, 2000.

MCKEAGE, K.; PERRY, C.M. Propofol: a review of this use in intensive care sedations of adults. *CNS Drugs*, v.17, p.235-272, 2003.

MILLER, E.D. Deliberate hypotension. In: MILLER, E.D. (Ed). *Anesthesia*. 2. Ed**.** New York: Churchill Livingstone, 1986. p.1949-1970.

NUNES, N.; LOPES, P.C.F.; SANTOS, P.S.P. *et al.* Hemodinâmica de diferentes frações inspiradas de oxigênio em cães submetidos à infusão contínua de propofol sob ventilação espontânea. *Cien. Rural*, v.38, p.729-735, 2008.

NUNES, N.; LEITE, A.V.; PAULA, D.P. *et al.* Intracranial variables in propofol or sevoflurane-anesthestized dogs subjected to subarachnoid administration of iohexol. *Arq. Bras. Med. Vet. Zootec.,* v.63, p.1315-1322, 2011.

PASETTO, A.; RINALDI, L. Total intravenous anesthesia and respiratory system. In: GULLO, A.; BERLOT, G. (Eds). *Perioperative and Critical Care Medicine: Educational Issues 2004*. New York: Spring, 2004. p.113-122.

PAULA, D.P.; NUNES, N.; NISHIMORI, C.T.D. *et al.* Efeitos da infusão contínua de propofol ou etomidato sobre variáveis intracranianas em cães. *Arq. Bras. Med. Vet. Zootec.,* v.62, p.302-308, 2010.

PLÖCHL, W.; COOK, D.J.; ORSZULAK, T.A.; DALY, R.C. Critical cerebral perfusion pressure during tepid heart surgery in dogs. *Ann. Thorac. Surg.*, v.66, p.118-124, 1998.

SHOEMAKER, W.C. Diagnosis and treatment of the shock syndromes. In: AYRES, S.M.; GRENVIK, A.; HOLBROOK, P.R.; SHOEMAKER, W.C. (Eds). *Textbook of Critical Care.* 3. ed. Philadelphia: W.B. Saunders, 1995. p.85-102.

SPONHEIM, S.; SKRAASTAD, O.; HELSETH, E. *et al.* Effects of 0.5 and 1.0 MAC isoflurane, sevoflurane and desflurane on intracranial and cerebral perfusion pressures in children. *Acta Anaesthesiol. Scand.*, v.47, p.932-938, 2003.

STEINER, L.A.; ANDREWS, J.D. Monitoring the injured brain: ICP and CBF. *Brit. J. Anaesth.*, v.97, p.26-38, 2006.

STURGES, B.K.; LeCOUTEUR, R.A. Intracranial hypertension. In: SILVERSTEIN, D.C.; HOPPER, K. (Eds). *Small Animal Critical Care Medicine*. St. Louis: Saunders Elsevier, 2009. p.423-429.