Effects on indicators of tissue perfusion in dogs anesthetized with isoflurane at two multiples of the minimum alveolar concentration

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OBJECTIVE
To investigate the effects of isoflurane anesthesia administered at 2 multiples of the minimum alveolar concentration (MAC) on tissue perfusion in dogs.

ANIMALS
8 healthy young adult Beagles.

PROCEDURES
A randomized crossover design was used. Dogs were anesthetized with isoflurane at 1.5 or 2.0 times the MAC for 2 hours, a 7-day washout period was provided, and dogs were reanesthetized with the alternate treatment. Various physiologic variables were monitored before anesthesia (baseline), at 20-minute intervals during anesthesia, and after anesthetic recovery. Various values were compared between MAC multiples by means of repeated-measures ANOVA, with the Tukey test used for multiple comparisons.

RESULTS
During anesthesia, mean arterial blood pressure, cardiac output, and mixed venous oxygen saturation were significantly greater when isoflurane was administered at 1.5 versus 2.0 times the MAC. Cardiac output gradually increased during anesthesia at 1.5 times but not at 2.0 times the MAC. Arterial blood lactate concentration did not differ between MAC multiples at any point; however, this concentration decreased with increasing anesthetic duration at both MAC multiples. Oxygen delivery differed between MAC multiples, and oxygen consumption differed from baseline during anesthesia at 2.0 times the MAC. Oxygen extraction was higher at 2.0 versus 1.5 times the MAC. Heart rate differed between MAC multiples only after anesthetic recovery.

CONCLUSIONS AND CLINICAL RELEVANCE
Isoflurane anesthesia impaired tissue perfusion in dogs, but these changes would not be clinically relevant with oxygen delivery at 100%. Peripheral tissue perfusion was maintained or improved with time. (Am J Vet Res 2016;77:24–31)

Isoflurane is the most commonly used inhalation anesthetic in veterinary medicine1–5; however, a main, deleterious effect of isoflurane is myocardial depression.6 Decreases in stroke volume and CO reportedly occur during isoflurane anesthesia and are ascribed to the direct effect of the anesthetic on myocardial fibers,1,6,7 which can compromise tissue perfusion. On the other hand, another known effect of isoflurane is vasodilation, which has been demonstrated in several studies of tissue perfusion, including those involving the coronary1–5 and cerebral arteries5,8–12 of humans, dogs, rats, and swine. Because of the myocardial depressant effect and concomitant vasodilator effect of isoflurane, reports conflict regarding the effects of this inhalation anesthetic on peripheral blood circulation. In many studies12,13 concerning the vasodilatory effect of isoflurane, blood flow was maintained or increased after occlusion of the middle cerebral artery12 or cardiopulmonary bypass13 when subjects were preconditioned with the agent; therefore, isoflurane was considered to protect against reperfusion injury or ischemia. To the authors’ knowledge, no studies have been conducted to investigate the effect of isoflurane on overall tissue perfusion.

Several variables are used to estimate the extent of tissue perfusion. Mixed venous oxygen saturation can be used to estimate the amount of oxygen returning from capillary circulation14 and is measured by use of blood samples collected from a pulmonary artery. Because SvO₂ measures the amount of oxygen returning from systemic circulation, this variable is consid-
ered a good indicator of overall perfusion. However, \( \text{SV}_o \) does not correlate well with \( \text{VO}_2 \) within single tissue beds and therefore is a poor indicator of specific tissue perfusion. Knowledge of arterial oxygen saturation and \( \text{SV}_o \) also allows calculation of \( \text{VO}_2 \) and \( \text{O}_2 \text{ER} \), which are important variables that can be calculated from blood gas measurements.

Another important indicator of tissue perfusion is arterial blood lactate concentration. Because blood lactate concentration changes noticeably after changes in \( \text{DO}_2 \), lactate values can be used as a prognostic indicator for critically ill patients. Concentrations > 2.5 mmol/L in anesthetized dogs can indicate impaired tissue perfusion. To the authors' knowledge, no studies have been reported regarding the effects of isoflurane alone on lactate concentration. In a study involving isoflurane-anesthetized dogs that received ropivacaine through an epidural catheter inserted at T1-2, blood lactate concentrations significantly decreased from preanesthetic values. The suggestion was that this decrease could have been a result of peripheral vasodilation caused by isoflurane, which would improve perfusion. However, isoflurane was not used alone in that study. Involving isoflurane-anesthetized dogs that received ropivacaine through an epidural catheter inserted at T1-2, blood lactate concentrations significantly decreased from preanesthetic values.

The purpose of the study reported here was to investigate the effects of isoflurane alone on overall and peripheral tissue perfusion in dogs by means of various perfusion indicators (arterial blood lactate concentration, \( \text{SV}_o \), \( \text{CPTG} \), \( \text{DO}_2 \), \( \text{VO}_2 \), and \( \text{O}_2 \text{ER} \)) and to compare indicator values between 2 anesthetic concentrations chosen to represent lighter and deeper anesthesia (1.5 and 2.0 times the MAC of isoflurane, respectively). We hypothesized that isoflurane anesthesia would maintain tissue perfusion even at the higher concentration, despite an expected decrease in arterial blood pressure. Maintenance of tissue perfusion during periods of hypotension would justify use of isoflurane for anesthesia for dogs in which hypotension might be expected.

**Materials and Methods**

**Animals**

Eight healthy young adult Beagles (mean ± SD age, 1.3 ± 0.1 years; mean body weight, 10.3 ± 2.4 kg) were included in the study. Dogs belonged to the experimental biotherium at the veterinary college. Dogs were excluded when they had any abnormalities in physiologic variables (heart rate, respiratory rate, rectal temperature, capillary refill time, and mucosal color), CBC results, and serum creatinine concentration and alanine transaminase activity or positive results of serologic testing for *Leishmania* infection (parasite endemic to the region). The study protocol was approved by the local Animal Usage Ethics Committee and was performed at the Experimental Laboratory of Anesthesiology, Faculty of Veterinary Medicine of Araçatuba, São Paulo State University.

**Anesthetic protocol**

The study was comprised of 2 phases: determination of the MAC of isoflurane for each dog and determination of the physiologic effects of isoflurane anesthesia at 1.5 and 2.0 times the MAC in all dogs. Prior to each anesthetic session, food was withheld from dogs for 12 hours and water was withheld for 2 hours.

To determine the MAC of isoflurane for each dog, isoflurane was administered via face mask at 5% in 100% oxygen at 3 mL/min. Once anesthesia was induced, dogs were endotracheally intubated, anesthesia was maintained with 2% isoflurane in oxygen at 2 L/min, and dogs were mechanically ventilated to achieve a peak airway pressure of 12 cm \( \text{H}_2\text{O} \) and positive end-expiratory pressure of 3 cm \( \text{H}_2\text{O} \). Respiratory rate varied from 15 to 25 breaths/min to maintain \( \text{Paco}_2 \) between 35 and 45 mm Hg. Saline (0.9% NaCl) solution was administered IV at a rate of 10 mL/kg/h through an infusion pump. Core temperature was monitored electronically and maintained at > 37.0°C by use of a warm-air blanket, which was positioned over the body without covering the limbs to avoid influencing peripheral temperatures. Room temperature was maintained at 25°C during all procedures.

After isoflurane had been administered to each dog at a stable rate for 15 minutes, as confirmed by a calibrated gas analyzer, two 22-gauge needles were inserted SC 5 cm below the elbow joint and 5 cm apart from each other, and an electrode was attached to each needle. Electrical stimulation was then administered, with each stimulus consisting of 2 short (10-millisecond) applications, followed by 2 prolonged (3-second) electrical currents at 50 V and 50 Hz, as described elsewhere. Isoflurane concentrations were decreased by 0.2% after each negative response (ie, no observable reaction) and increased 0.1% after the first positive response (flexion of the neck or gross purposeful limb movement) by each dog. Minimum alveolar concentration of isoflurane was calculated for each dog as the mean of the lowest concentration at which negative response was detected and the highest concentration at which a positive response was detected.

For the second phase of the study, dogs were assigned numbers through a random-number-generating system to first receive isoflurane at 1.5 or 2.0 times their individual MAC. Protocols for anesthetic induction, fluid administration, and mechanical ventilation were identical to those used for MAC determination.
22-gauge catheter was placed in the right metatarsal artery of each dog to perform invasive monitoring of arterial blood pressure and blood sample collection for blood lactate measurements. A Swan-Ganz catheter was placed in the left pulmonary artery through the left jugular vein for monitoring of hemodynamic variables and core temperature. Position of the Swan-Ganz catheter was confirmed by observation of pressure waveforms from the right atrium, right ventricle, and pulmonary artery. Additionally, the cuff at the distal end of the catheter was inflated to occlude the artery, confirming the correct catheter position. After complete instrumentation, the assigned MAC was maintained for at least 15 minutes before recordings were started.

Anesthesia was maintained for 2 hours, during which measurements were obtained. Afterward, the Swan-Ganz catheter was removed and dogs were extubated and allowed to recover fully from anesthesia. After a washout period of ≥7 days, dogs underwent the same process again, with the MAC of isoflurane applied at the alternate concentration to that initially received.

**Measurements of physiologic variables**

Measurements of physiologic variables were performed before anesthetic induction (baseline; did not include measurements obtained via pulmonary arterial catheterization); 20, 40, 60, 80, 100, and 120 minutes after dogs were completely instrumented, during anesthesia; and after dogs had completely recovered from anesthesia (with dogs in a standing position; did not include measurements obtained via pulmonary arterial catheterization).

Measurements were obtained of heart rate, MAP, CO (standard thermodilution method, with \( \frac{3}{4} \) mL of saline solution at 1° to 4°C), \( \text{ScVO}_2 \) (analyzed immediately by use of samples of mixed venous blood collected from the pulmonary artery through the Swan-Ganz catheter), CPTG, and arterial blood lactate concentration (analyzed immediately after blood sample collection). Hemodynamic values and core temperature were recorded directly from the monitor whereas peripheral temperature was assessed via infrared thermometer that was placed within an interdigital fold. Calculations were made to estimate \( \text{DO}_2 \) \((\text{arterial oxygen content } \times \text{CO } \times 10)/\text{body weight}\), \( \text{VCO}_2 \) \((\text{CaCO}_2 - \text{CVCO}_2 ) \times \text{CO } \times 10)/\text{body weight}\), and \( \text{O}_2 \text{ER} \) \((\text{CaCO}_2 - \text{CVCO}_2 )/\text{CaO}_2 \).

**Statistical analysis**

Data were first evaluated for normality of distribution by means of the Shapiro-Wilk test. Values were summarized as mean ± SD. Differences in values of physiologic variables between MAC multiples were assessed by performance of repeated-measures ANOVA. Multiple comparisons of values between MAC multiples at specific measurement points and within each MAC multiple between pairs of measurement points were performed with the Tukey test. Values of \( P < 0.05 \) were considered significant.

**Results**

**Animals**

The MAC of isoflurane was established for each of the 8 Beagles, with a mean ± SD value of 1.18 ± 0.14% and interindividual variation of 30%. All 8 dogs completed both sessions involving 2 hours of isoflurane anesthesia at 1.5 and 2.0 times their MAC (1.5 MAC and 2.0 MAC).

**Effect of MAC on physiologic variables**

Significant differences in physiologic variables between certain measurement points throughout the anesthetic sessions were detected for each MAC multiple (Table 1). After anesthetic induction, MAP decreased significantly at both MAC multiples, returning to baseline values at anesthetic recovery (Figure 1).

<p>| Table 1—Mean ± SD values of physiologic variables for 8 healthy young adult Beagles before (baseline), at various points during, and after (recovery) anesthesia at 1.5 or 2.0 times the MAC of isoflurane determined for each dog. |</p>
<table>
<thead>
<tr>
<th>Variable, by MAC multiple</th>
<th>Measurement point</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td></td>
</tr>
<tr>
<td>1.5</td>
<td>133 ± 18(b)</td>
</tr>
<tr>
<td>2.0</td>
<td>129 ± 11(b)</td>
</tr>
<tr>
<td>CPTG (°C)</td>
<td></td>
</tr>
<tr>
<td>1.5</td>
<td>3.2 ± 1.4</td>
</tr>
<tr>
<td>2.0</td>
<td>2.5 ± 0.9</td>
</tr>
<tr>
<td>DO(_2) (mL/kg/min)</td>
<td></td>
</tr>
<tr>
<td>1.5</td>
<td>36 ± 10(b)</td>
</tr>
<tr>
<td>2.0</td>
<td>27 ± 7(b)</td>
</tr>
<tr>
<td>V(_{CO}_2) (mL/kg/min)</td>
<td></td>
</tr>
<tr>
<td>1.5</td>
<td>5.6 ± 1.1(b)</td>
</tr>
<tr>
<td>2.0</td>
<td>4.2 ± 2.6(b)</td>
</tr>
<tr>
<td>O(_2)ER (%)</td>
<td></td>
</tr>
<tr>
<td>1.5</td>
<td>0.17 ± 0.04</td>
</tr>
<tr>
<td>2.0</td>
<td>0.22 ± 0.04</td>
</tr>
</tbody>
</table>

| a–cWithin a row, values with different superscript letters differ significantly (P < 0.05) as determined by the Tukey test. |
| _= Not measured. |

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Differences in MAP were also identified between MAC multiples from 60 minutes to 120 minutes of anesthesia, with the session involving 2.0 MAC resulting in a significantly lower MAP than the session involving 1.5 MAC. Cardiac output gradually increased with anesthetic duration at 1.5 MAC but not at 2.0 MAC. That variable also differed between MAC multiples from 60 to 100 minutes of anesthesia, with values significantly lower for 2.0 MAC than for 1.5 MAC. No differences over time were identified in $S\text{v}_2O_2$ for either MAC multiple, but values for 2.0 MAC were significantly lower than for 1.5 MAC from 60 to 120 minutes of anesthesia.

Arterial blood lactate concentrations did not differ significantly between MAC multiples; however, values slowly decreased with increasing anesthetic duration for both MAC multiples. No difference was identified in CPTG between MAC multiples or measurement points. Significant differences in $D\text{O}_2$ were identified between MAC multiples at 20 minutes of anesthesia and from 60 to 120 minutes of anesthesia. On the other hand, $V\text{O}_2$ only differed among measurement points for 2.0 MAC, with values increasing with increasing anesthetic duration. Oxygen extraction differed significantly between MAC multiples throughout anesthesia, with higher values achieved with 2.0 MAC.

At anesthetic recovery, heart rate was the only physiologic variable that differed between MAC multiples (Table 1), with values significantly higher for 1.5 MAC than for 2.0 MAC. Other than this difference, heart rates remained within reference limits (69 to 111 beats/min) throughout both anesthetic sessions.

**Discussion**

The mean MAC of isoflurane determined for the 8 healthy Beagles in the present study (1.18%) was close to that reported for 7 mixed-breed dogs (1.13%) in another study, but lower than that reported for 10 healthy mixed-breed dogs (1.35%) in a different study. Heart rates remained within reference limits throughout the experiment, and higher values were observed only when dogs were anesthetized at 1.5 times (and not 2.0 times) the MAC of isoflurane. Anesthesia without previous sedation can lead to more agitated recovery than with previous sedation because of catecholamine release, which in turn increases values
of cardiovascular variables. However, no long-term adverse effects of anesthesia were detected in any dog of the present study. Each dog recovered from anesthesia without complication.

Differences in MAP between MAC multiples in the present study supported the known hypotensive effect of isoflurane. This effect was dose dependent, given the greater decrease in MAP identified when dogs were anesthetized at 2.0 versus 1.5 times the MAC of isoflurane, as has been demonstrated in other studies. Values returned to preanesthetic MAPs for all dogs after anesthetic recovery.

Cardiac output was significantly lower when dogs were anesthetized at 2.0 times the MAC of isoflurane. At 1.5 times the MAC, CO increased with increasing anesthetic duration. Because isoflurane administration is known to decrease CO, the observed difference between MAC multiples was an expected result. However, the gradual increase identified for 1.5 times the MAC suggested that cardiac preload also increased. This phenomenon can be explained by the Frank-Starling mechanism, by which an increase in preload causes the heart to contract more intensively to pump the greater blood volume. The fluid administration rate used in the present study was fairly high (10 mL/kg/h) given that no hemorrhage existed that might have required fluid administration, and this high rate might have been caused by the increase in venous blood returning to the heart. Changes in blood volume could be better perceived when dogs were anesthetized at 1.5 versus 2.0 times the MAC of isoflurane because less anesthetic exposure would have less of an effect on the myocardium.

At 2.0 times versus 1.5 times the MAC, CO was significantly lower in conjunction with MAP values close to 50 mm Hg, corroborating findings in another study involving dogs. Also, anesthetic duration could have been a reason for the differences observed at 1.5 times the MAC, when there was an increase in CO over time. In a study of long-term isoflurane exposure in dogs, significant changes in cerebral blood flow were observed after 6 hours of anesthesia had elapsed.

Regarding tissue perfusion, arteriovenous oxygen content difference (AaO2) exceeded 70% in both groups, as would be expected when the inspired oxygen fraction (100% in the present study) is higher than that of atmospheric air. Values of AaO2 differed significantly between MAC multiples, suggesting that anesthesia at 2.0 times the MAC of isoflurane caused a greater reduction in tissue perfusion than did anesthesia at 1.5 times the MAC. However, this variable should be interpreted cautiously because values within reference limits can be observed even when tissue perfusion of some organs is impaired because of the mixture of venous blood from all tissues in the venous cava. Organs that receive a greater portion of CO than other organs may cause the mixed venous blood to appear analytically normal and mask the true saturation of poorly perfused tissues. In addition, when oxygen is not transferring into tissue, its concentration in venous blood will be similar to that in arterial blood. The importance of monitoring AaO2 relies on its relationship to CO because a low AaO2 indicates inadequate CO. Differences identified in the present study corroborate this information because CO also differed. Indeed, lower values of both CO and AaO2 were identified for the higher MAC of isoflurane evaluated.

Variables such as DlO2, VO2, and O2ER are also important for hemodynamic monitoring. The O2ER differed between MAC multiples at each measurement point during anesthesia and was higher for 2.0 times the MAC than for 1.5 times the MAC. An increase in O2ER occurs in tissues that receive inadequate blood flow. The higher O2ER identified for the higher MAC was, therefore, due to a lower CO and stroke volume. These results explain the findings in the AaO2 because an increase in the degree of O2ER would cause a decrease in the amount of oxygen in venous blood.

Similar to CO in the present study, DlO2 differed significantly between MAC multiples and increased with increasing anesthetic duration when dogs received 1.5 times the MAC of isoflurane. This similarity was due to the direct relationship between CO and DlO2. Oxygen delivery is commonly altered by anesthesia, but a decrease in metabolic rate and muscle activity will also cause a decrease in VO2, and as a result, the decrease in DlO2 is clinically irrelevant.

Although VO2 was significantly lower at 2.0 versus 1.5 times the MAC, VO2 did not differ between MAC multiples. Usually VO2 does not depend on DlO2, but when CO is low, VO2 becomes DlO2 dependent. Such a dependency could have occurred at 2.0 times the MAC because DlO2 was lower at that anesthetic concentration than at 1.5 times the MAC. Oxygen consumption did not differ between MAC multiples, probably because it was limited by the decrease in DlO2. Lower DlO2 together with the O2ER could have caused the observed difference in AaO2 between MAC multiples. However, DlO2 did not reach critical values (6 to 11 mL/kg/min) in the dogs of the present study; therefore, the differences were not clinically important.

When DlO2 fails to keep up with oxygen demand, anaerobic metabolism and serum lactate concentration increase. This situation makes lactate concentration an easy biomarker for quantification of tissue perfusion. Relationships exist between serum lactate concentration and prognosis for several types of illness. Lactate is also produced in nonpathological conditions and may increase during exercise without clinical relevance, therefore, high values must be interpreted cautiously.

Arterial blood lactate concentration was higher than the upper reference limit (2.0 mmol/L) throughout the present study and did not differ between MAC multiples. A decrease was observed at both MAC multiples with increasing anesthetic duration. High blood lactate concentrations (3.5 mmol/L) have also been identified in resting dogs. When inhalation anesthesia is used with no premedication, dogs can become agitated during anesthetic recovery, leading to an increase in muscle activity. That might be an explanation...
for the high values identified after anesthesia in the study dogs. In addition, muscle activity during physical restraint can cause an increase in blood lactate concentration to as high as 6.0 mmol/L, which may also explain the differences because the last sample was obtained with dogs restrained and after an agitated recovery. Before each aesthetic session, dogs were taken for a walk. Limitations of the present study were that the period between the walk and baseline measurements was too short to allow stabilization of lactate concentrations before measurements were obtained and that blood lactate concentration was measured with a portable analyzer, which can be less accurate, compared with results from wet chemistry analyzers.

The lack of difference between MAC multiples in arterial blood lactate concentrations suggested that hypotension and myocardial depression may not have influenced this variable. Rather, peripheral vasodilation may have improved perfusion of those tissue beds, decreasing lactate production in peripheral tissue over time. Findings indicated that the effects of isoflurane were influenced not only by concentration but also anesthetic duration, which is important when considering isoflurane anesthesia for prolonged procedures. In addition, although a significant difference was identified between MAC multiples, DO₂ values were within reference limits, probably because of the high inspired oxygen fraction and low metabolism. These factors might have contributed to the decrease identified in blood lactate concentrations during anesthesia.

In swine with experimentally induced hemorrhage, isoflurane anesthesia but not total IV anesthesia results in maintenance of blood lactate concentration within reference limits despite a decrease in MAP. Results of the present study involving healthy dogs suggested the opposite. High arterial blood lactate concentrations, together with the differences between MAC multiples in SvO₂, indicated that isoflurane anesthesia impaired overall tissue perfusion. However, lactate values did not differ between MAC multiples but DO₂ did; consequently, DO₂ and CO could be considered to have been impaired by the higher isoflurane concentration (2.0 times the MAC), whereas lactate values were more related to VO₂ in tissues, which was similar between MAC multiples.

Peripheral vasodilation can be confirmed by the CPTG, which represents the delivery of arterial blood to peripheral tissues. Provided that the difference between core and peripheral body temperatures remains between 3°C and 7°C, peripheral perfusion can be considered normal. In the present study, CPTG was < 7°C during both anesthetic sessions and was even < 3°C for many measurements. Although rectal temperature has been used for this variable, core temperature is considered more reliable. In a human study conducted with a portable analyzer, which can be less accurate, compared with results from wet chemistry analyzers.

The CPTG is an important variable for relating hemodynamic changes to the extent of peripheral perfusion. However, strategies were used in the present study to ensure reliability of this variable. Room temperature was maintained at 25°C for all procedures, and a warm-air blanket was used to maintain the core temperature of each dog at > 37°C. The highest core temperature identified was 37.5°C, and no differences in this variable were identified between MAC multiples or measurement points. The CPTG results suggested that despite the difference in DO₂ between MAC multiples, that difference was not clinically relevant to the peripheral tissues because blood flow was most likely maintained. However, CPTG is merely an estimation of blood flow and it is impossible to accurately determine the degree of perfusion by CPTG alone. Furthermore, invasively measured variables can be more precise in quantifying actual delivery of arterial blood to tissues.

A few alternative methods for monitoring tissue perfusion were not used in the present study. Splanchnic perfusion can be assessed through gastric tonometry and microcirculation can be assessed through orthogonal polarization spectrometry or near-infrared spectroscopy. These techniques were unavailable and should be considered in the design of future studies conducted to evaluate the influence of isoflurane on tissue perfusion.

In the study reported here, the distress and physical restraint of dogs associated with placement of the Swan-Ganz catheter before anesthesia for measurement of baseline physiologic values were concerns. For that reason, pulmonary arterial catheterization was instead performed after anesthesia was induced, enabling measurement or calculation of CO, SvO₂, CPTG, DO₂, VO₂, and O₂ER during anesthesia. However, because of this choice, no baseline data were available to compare with hemodynamic data obtained during isoflurane anesthesia. Other studies are suggested that would allow comparison of hemodynamic values in awake and isoflurane-anesthetized dogs.

Despite the limitations of the present study, findings suggested that isoflurane anesthesia impaired tissue perfusion in dogs, as indicated by its effects on CO, MAP, SvO₂, and arterial blood lactate concentrations. However, these changes were clinically irrelevant because most SvO₂ values were within reference limits and arterial blood lactate concentration gradually decreased with increasing anesthetic duration. Because of low metabolism and high inspired oxygen fraction, the degree of tissue perfusion may have improved with time, as suggested by the CPTG, VO₂, and arterial blood lactate concentration. Additional studies are suggested to evaluate the effects of prolonged isoflurane exposure on tissue perfusion in dogs.

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Footnotes

a. Anesthetic machine Nikkei K, Takaoka, São Paulo, Brazil.

b. Mechanical ventilator Nikkei K, Takaoka, São Paulo, Brazil.

c. Rotating pump ST 550T2, Santronic, São Paulo, Brazil.

d. Warm air blower unit TC3000, Gaymar Industries Inc, Orchard

Park, NY.

e. Quick CaTM calibration gas, Datec-Engstrom Division Instru-

mentarium Corp, Helsinki, Finland.

f. Stimulator Grass-S48 Astronmed Inc. Los Angeles, Calif.

g. Nipro 22G, Nipro Medical Corp, Sorocaba, Brazil.

h. Pediatric Swan-Ganz catheter 152FS (75 cm), Edwards Life-

sciences, São Paulo, Brazil.

i. Accutrend, Roche, São Paulo, Brazil.

j. DX 2020 CO module, Dixtal Biomedica Indústria e Comércio

Ltda, Manaus, Brazil.

k. Infrared Thermometer Fluke 62, Fluke, São Paulo, Brazil.


