Intracranial pressure and cardiopulmonary variables during isoflurane or sevoflurane anesthesia at various minimum alveolar concentration multiples in normocapnic dogs

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Objective—To compare effects of isoflurane and sevoflurane on intracranial pressure and cardiovascular variables at 1.0, 1.5, and 2.0 times the minimum alveolar concentration (MAC) in mechanically ventilated normocapnic dogs.

Animals—6 healthy male Beagles.

Procedures—The individual MAC was determined for each agent with an electrical stimulus. After a minimum of 1 week, anesthetic induction by use of a mask with one of the inhalation anesthetics selected randomly was followed by mechanical ventilation and instrumentation for measurement of intracranial pressure and cardiovascular variables. Heart rate; systolic, mean, and diastolic arterial blood pressures; central venous pressure; mean pulmonary arterial pressure; pulmonary artery occlusion pressure; cardiac output; intracranial pressure (ICP); core body temperature; end-tidal inhalation anesthetic and carbon dioxide concentration; and arterial blood gas values were measured after attaining equilibrium at 1.0, 1.5, and 2.0 MAC of each inhalation anesthetic. Cardiac index, systemic vascular resistance, pulmonary vascular resistance, and cerebral perfusion pressure (CPP) were calculated.

Results—Mean ICP did not differ within and between anesthetics at any MAC. Compared with equipotent concentrations of isoflurane, the CPP and mean values for systolic, mean, and diastolic arterial blood pressures were increased at 2.0 MAC for sevoflurane, whereas mean values for mean and diastolic arterial blood pressures and systemic vascular resistance were increased at 1.5 MAC for sevoflurane.

Conclusions and Clinical Relevance—Although ICP was similar in healthy normocapnic dogs, CPP was better maintained during 2.0 MAC for sevoflurane, compared with isoflurane. (Am J Vet Res 2013;74:369–374)
extracranial vessels, respectively. The ICP will increase when the volume of one (or more) of these compartments increases beyond its compensatory capacity.

Cerebral blood flow is determined by the CPP and the cerebral vascular resistance. These variables are maintained by multiple mechanisms including cerebral autoregulation of blood flow, CMR, PA\textsubscript{CO\textsubscript{2}}, PA\textsubscript{O\textsubscript{2}}, and the autonomic nervous system. Cerebral blood flow and its effect on CBV have great potential to influence ICP and the changes in ICP tend to parallel changes in CBF or CBV. Although CBV is the smallest of the compartments, its importance in neuroanesthesia lies in the fact that it can be altered rapidly.

Inhalation anesthetics suppress the CMR in a dose-dependent fashion,\textsuperscript{1-3} causing a parallel decrease in CBF. These drugs also elicit intrinsic cerebral vasodilatory effects, resulting in a decrease in cerebral vascular resistance that tends to increase CBF.\textsuperscript{4} Therefore, the net effect of inhalation anesthetics on CBF is a balance between these 2 responses. The CBF:CMR ratio is altered (increased) by inhalation anesthetics. This alteration is dose dependent, and under steady-state conditions, there is a positive correlation between MAC multiples and the CBF:CMR ratio.\textsuperscript{5} However, this relationship between CBF and MAC may differ depending upon species and other circumstances.\textsuperscript{2,6,7} Also, compared with isoflurane, sevoflurane is less vasoactive and better maintains autoregulation of CBF in humans.\textsuperscript{8-12}

We hypothesized that administration of either isoflurane or sevoflurane would cause a dose-dependent increase in ICP in dogs but that sevoflurane would cause less change in ICP, compared with isoflurane, at equipotent dosages. The specific objective of the study reported here was to compare effects of isoflurane and sevoflurane on CBF and cardiopulmonary variables at 1.0, 1.5, and 2.0 MACs in healthy normocapnic dogs during controlled ventilation.

**Materials and Methods**

**Animals**—Six sexually intact male Beagles with a mean ± SD body weight of 11.8 ± 0.6 kg that were between 11 and 13 months of age and determined to be healthy (on the basis of findings on physical examination, CBC, and serum biochemical profile) were included in the study. The dogs were current on their vaccination and deworming and were housed in an Association for Assessment and Accreditation of Laboratory Animal Care International–accredited facility located in the College of Veterinary Medicine at Washington State University. They were fed commercial dog food and had free access to water. The study was approved by the Washington State University Institutional Animal Care and Use Committee.

**Experimental protocol**—The study was conducted in 2 parts separated by at least 1 week: determination of individual MACs for isoflurane and sevoflurane and measurement of ICP and cardiopulmonary variables at each of 1.0, 1.5, or 2.0 MAC multiples for both isoflurane and sevoflurane. The order of study of each anesthetic and each MAC multiple within anesthetics was randomized.

**Determination of MAC**—On the day of the MAC determination, food was withheld from the dogs for 12 hours but water was available until 1 hour before the procedure. Each dog was randomly assigned to undergo anesthetic induction with either isoflurane\textsuperscript{a} or sevoflurane\textsuperscript{a} in oxygen via a mask to allow placement of a cuffed endotracheal tube, and then positioned in left lateral recumbency. All dogs were mechanically ventilated via IPPV to maintain the end-tidal CO\textsubscript{2} pressure between 35 and 40 mm Hg and were monitored via side-stream capnography.\textsuperscript{a} An IV catheter\textsuperscript{a} was placed in the right cephalic vein, and electrolyte solution\textsuperscript{a} was administered at a rate of 10 mL/kg/h during the procedure. Core body (esophageal) temperature was maintained between 37.3\textsuperscript{°} and 38.5\textsuperscript{°}C with an active air warming system\textsuperscript{a} and a heated table.\textsuperscript{a} Pulse oximetry, oscillometric arterial blood pressure, esophageal temperature, heart rate, and lead II ECG were continuously monitored with a multiparameter anesthetic monitoring system.\textsuperscript{a} An elbow connector with a sampling port was placed between the endotracheal tube and Y-piece of the circle anesthetic circuit for measurement of end-tidal airway gas concentrations of CO\textsubscript{2} and inhalation anesthetic agent with a calibrated anesthetic gas analyzer.\textsuperscript{a} The gas analyzer used Raman scattering to detect individual agents and was able to detect a mixture of inhalation anesthetics. The sampling port of the elbow connector allowed passage of an 8F plastic tube, the free end of which rested in the endotracheal tube proximal to the level of the thoracic inlet. The airway gas samples were drawn through this plastic tubing at a rate of 210 mL/min. At each end-tidal anesthetic agent concentration, 20 minutes were allowed for equilibration during isoflurane and 10 minutes were allowed for sevoflurane. For each inhalation anesthetic, MAC was determined with a supramaximal noxious electrical stimulus (50 V, 50 Hz, and 10 milliseconds) applied via 29-gauge needle electrodes placed 2 cm apart in the buccal mucosa for the lesser of 60 seconds or until a positive response was obtained with a Grass stimulator.\textsuperscript{13,14} A gross, purposeful movement in response to the applied electrical stimulus was interpreted as substantial movement of the limbs or head but did not include chewing, coughing, muscle twitching, blinking, nystagmus, increase in respiratory rate, or swallowing.\textsuperscript{13,15} When the dog had a positive response, the end-tidal anesthetic agent concentration was increased by 0.10% and the noxious stimulus was applied again after re-equilibration. When a negative response was obtained, the end-tidal anesthetic agent concentration was decreased by a 0.10% increment and the noxious stimulus was reapplied after the equilibration period. The procedure for obtaining both the lowest anesthetic concentration with a negative response and the highest concentration with a positive response was performed in triplicate. The MAC was calculated as the mean of the lowest end-tidal anesthetic agent concentration at which the dog had a negative response and the highest end-tidal anesthetic agent concentration at which the dog had a positive response to the noxious stimulus.\textsuperscript{14} After the last measurement with the first inhalation anesthetic, its administration was discontinued. At the first sign of light anesthesia (eg, coughing or movement), the
other inhalation anesthetic was introduced and allowed to equilibrate for 40 minutes at an end-tidal anesthetic agent concentration equal to 1.2 times estimated MAC and the MAC determination process was repeated.

Measurement of ICP and cardiopulmonary variables—In the second part of the study, the dogs were prepared and instrumented during anesthesia with an inhalation anesthetic chosen randomly as for the MAC determinations. During instrumentation, end-tidal anesthetic agent concentration was kept at 1.3 times the determined end-tidal CO2 concentration guided the adjustment of IPPV to maintain the Paco2 between 35 and 45 mm Hg. An indwelling catheter was placed in the dorsal pedal artery for collection of arterial blood samples and direct monitoring of arterial blood pressure with a mercury-calibrated transducer attached to a pressure module and monitor. For determination of CO2, a flow-directed Swan-Ganz thermodilution catheter was inserted percutaneously via a catheter introducer into the right jugular vein and advanced into the pulmonary artery. Correct catheter placement was determined at the time of placement and before measuring CO2 by observing the characteristic pressure waveform displayed on the pressure monitor. The thermodilution catheter was connected to a CO module and monitor; iced 5% dextrose solution maintained at 0°C to 4°C was used as the injectate, and 5 mL of injectate was rapidly injected as a bolus through the proximal port of the thermodilution catheter during the expiratory phase of the respiratory cycle. The CO was determined as the mean of 3 measurements at each time point. Mean pulmonary artery pressure was determined with the distal port of the thermodilution catheter and a calibrated pressure transducer connected to the physiologic recorder. Core body temperature was recorded with a thermistor placed within the pulmonary artery via the Swan-Ganz catheter.

Intracranial pressure was measured with a calibrated, fiberoptic transducer connected to a digital ICP monitor via a described aseptic surgical technique. Briefly, a burr hole was drilled at a point on the right side of the skull approximately midway between the dorsal midline and the dorsal aspect of zygomatic arch and midway between the lateral canthus of the eye and the caudal aspect of the temporalis muscle. A bolt accompanying the fiberoptic transducer was screwed into the hole until it was secure and then the dura was incised. The fiberoptic pressure transducer was advanced through the bolt into the brain parenchyma.

Intracranial pressure, heart rate, SAP, MAP, MPAP, PAOP, CVP, core body temperature (ie, pulmonary arterial temperature), CO, end-tidal anesthetic agent concentration, and end-tidal CO2 concentration were measured after equilibration at 1.0, 1.5, and 2.0 MACs with each of the inhalation anesthetics. Cardiac index was calculated as CO/body weight (kg). Systemic vascular resistance was calculated as ([MAP – CVP]/CO) × 80. Pulmonary vascular resistance was calculated as ((MAP – PAOP)/CO) × 80. Cerebral perfusion pressure was calculated as MAP – ICP. Arterial blood was sampled for analysis of arterial pH, PaO2, and PaCO2 immediately prior to measurement of ICP and cardiovascular variables at each MAC.

After the last measurement with the first inhalation anesthetic, its administration was discontinued. At the first sign of light anesthesia (eg, coughing or movement), the other inhalation anesthetic was introduced and allowed to equilibrate for 40 minutes at 1 of the 3 MACs randomly chosen. The ICP and cardiopulmonary measurements were repeated for each of the MACs with the second agent. At the end of the experiment, the dogs were euthanized with an overdose of pentobarbital sodium administered IV.

Statistical analysis—The data were analyzed via 2-way repeated-measures ANOVA with post hoc

Table 1—Mean ± SD values for cardiopulmonary variables in 6 dogs during anesthesia with isoflurane or sevoflurane at 1.0, 1.5, and 2.0 MAC.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Isoflurane 1.0 MAC</th>
<th>Isoflurane 1.5 MAC</th>
<th>Isoflurane 2.0 MAC</th>
<th>Sevoflurane 1.0 MAC</th>
<th>Sevoflurane 1.5 MAC</th>
<th>Sevoflurane 2.0 MAC</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICP (mm Hg)</td>
<td>18.7 ± 4.3</td>
<td>20.2 ± 4.9</td>
<td>17.8 ± 7.1</td>
<td>16.7 ± 5</td>
<td>19.3 ± 7.2</td>
<td>18.8 ± 5.9</td>
</tr>
<tr>
<td>CPP (mm Hg)</td>
<td>75.5 ± 14.9†</td>
<td>94 ± 17.2†</td>
<td>86.7 ± 20.2†</td>
<td>83.7 ± 17.9†</td>
<td>71 ± 24.3</td>
<td>58.3 ± 20.3†</td>
</tr>
<tr>
<td>ETCO2 (mm Hg)</td>
<td>30.8 ± 9.4</td>
<td>34.3 ± 9.9</td>
<td>33.8 ± 1</td>
<td>31.6 ± 5.4</td>
<td>34.8 ± 1.9</td>
<td>34.2 ± 1.9</td>
</tr>
<tr>
<td>SAP (mm Hg)</td>
<td>140.2 ± 29.6†</td>
<td>111 ± 25.7†</td>
<td>78.8 ± 26.5±</td>
<td>141.5 ± 27.6†</td>
<td>122.6 ± 18.1†</td>
<td>110 ± 21.9±</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>94.2 ± 14.2†</td>
<td>74 ± 14.1±</td>
<td>54.7 ± 12.7±</td>
<td>99.5 ± 14.9†</td>
<td>86.7 ± 10.9±</td>
<td>76 ± 13.8±</td>
</tr>
<tr>
<td>DAP (mm Hg)</td>
<td>76.7 ± 10.6†</td>
<td>60.2 ± 11.6±</td>
<td>44.5 ± 8.0±</td>
<td>82.7 ± 10.8†</td>
<td>72.3 ± 8.7±</td>
<td>62.7 ± 11.4±</td>
</tr>
<tr>
<td>CO (L/min)</td>
<td>2.6 ± 0.5†</td>
<td>2.3 ± 0.5†</td>
<td>1.4 ± 0.6§</td>
<td>2.6 ± 0.4†</td>
<td>2.2 ± 0.4</td>
<td>1.8 ± 0.2†</td>
</tr>
<tr>
<td>Cardiac index (L/kg/min)</td>
<td>0.22 ± 0.05†</td>
<td>0.2 ± 0.04†</td>
<td>0.12 ± 0.05†</td>
<td>0.23 ± 0.04‡</td>
<td>0.19 ± 0.03</td>
<td>0.15 ± 0.01†</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>125 ± 24</td>
<td>124 ± 10</td>
<td>115 ± 13</td>
<td>128 ± 20</td>
<td>120 ± 20</td>
<td>123 ± 12</td>
</tr>
<tr>
<td>SVR (dynes · cm–5)</td>
<td>2,602.8 ± 494.3</td>
<td>2,152.7 ± 572.5</td>
<td>2,756.6 ± 671.8</td>
<td>2,733.7 ± 521.7</td>
<td>2,855.1 ± 643.4*</td>
<td>2,930.3 ± 399</td>
</tr>
<tr>
<td>PVR (dynes · cm–5)</td>
<td>245.8 ± 111.8</td>
<td>215.4 ± 62.9</td>
<td>272 ± 51.8</td>
<td>190.1 ± 101.5</td>
<td>235.1 ± 88.7</td>
<td>261.6 ± 107.1</td>
</tr>
<tr>
<td>CVP (cm H2O)</td>
<td>6.8 ± 2.4</td>
<td>8.2 ± 3</td>
<td>7.4 ± 3.9</td>
<td>6.6 ± 2.1</td>
<td>6.8 ± 2.5</td>
<td>7.4 ± 1.9</td>
</tr>
<tr>
<td>PAOP (mm Hg)</td>
<td>10.4 ± 2.7</td>
<td>12.2 ± 4.8</td>
<td>12.2 ± 3.3</td>
<td>12 ± 4.2</td>
<td>11.8 ± 3.4</td>
<td>12 ± 3</td>
</tr>
<tr>
<td>MPAP (mm Hg)</td>
<td>18.2 ± 2.3</td>
<td>18.6 ± 3.4</td>
<td>16.8 ± 2.8</td>
<td>17.8 ± 2.9</td>
<td>18.2 ± 2.8</td>
<td>17.6 ± 2.2</td>
</tr>
<tr>
<td>pH</td>
<td>7.39 ± 0.02</td>
<td>7.37 ± 0.01</td>
<td>7.36 ± 0.02</td>
<td>7.41 ± 0.04</td>
<td>7.38 ± 0.02</td>
<td>7.38 ± 0.02</td>
</tr>
<tr>
<td>Paco2 (mm Hg)</td>
<td>38.8 ± 2.2</td>
<td>39.2 ± 2.8</td>
<td>39.6 ± 3.3</td>
<td>34.9 ± 5.6</td>
<td>41.5 ± 2.0</td>
<td>39.1 ± 3.4</td>
</tr>
<tr>
<td>PaO2 (mm Hg)</td>
<td>527 ± 32</td>
<td>513 ± 41</td>
<td>501 ± 39</td>
<td>501 ± 44</td>
<td>542 ± 34</td>
<td>517 ± 33</td>
</tr>
<tr>
<td>HCO3 (mmol/L)</td>
<td>23.8 ± 1.3</td>
<td>22.7 ± 1.3</td>
<td>22.4 ± 2.0</td>
<td>23.3 ± 1.9</td>
<td>24.4 ± 0.9</td>
<td>23.3 ± 1.7</td>
</tr>
</tbody>
</table>

*Significantly (P < 0.05) different between anesthetics at same MAC. †Significantly (P < 0.05) different within anesthetic for 1.0 versus 1.5 MAC. §Significantly (P < 0.05) different within anesthetic for 1.0 versus 2.0 MAC. ¶Significantly (P < 0.05) different within anesthetic for 1.5 versus 2.0 MAC. ETCO2 = End-tidal concentration of CO2. PVR = Pulmonary vascular resistance.
comparison of means by the Tukey-Kramer test performed with commercially available statistical analysis software. The level of significance was set at P < 0.05.

Results

Data are presented as mean ± SD (Table 1). In the first part of the study, the MAC for sevoflurane was 2.04 ± 0.14% and for isoflurane was 1.52 ± 0.18%. Cardio-pulmonary data from dogs during the MAC determinations were not reported. In the second part of the study, mean values for ICP at the MACs within and between anesthetics were not significantly different. The mean value for CPP was significantly lower during isoflurane anesthesia, compared with sevoflurane anesthesia at 2.0 MAC. In the isoflurane group, the mean value for CPP at 1.0 MAC was significantly different from that at 1.5 and 2.0 MAC. In the sevoflurane group, CPP was significantly different only between the 1.0 and 2.0 MACs. Mean values for SAP, MAP, and DAP were significantly higher at 2.0 MAC for sevoflurane, compared with isoflurane. The MAP and DAP were significantly higher at 1.5 MAC for sevoflurane, compared with isoflurane. In the isoflurane group, both SAP and DAP were significantly different among all 3 MACs. In the sevoflurane group, SAP and DAP were significantly different between 1.0 and 1.5 MAC and between 1.0 and 2.0 MAC (Table 1). Values for MAP were significantly different between 1.0 and 2.0 MAC for both isoflurane and sevoflurane. In the isoflurane group, MAP was also significantly different between 1.5 and 2.0 MAC. The SVR was significantly higher at 1.5 MAC with sevoflurane, compared with the equipotent concentration of isoflurane. Mean values for CO and cardiac index were not different between anesthetics at the same MACs. In the isoflurane group, CO and cardiac index were significantly different between 1.0 and 2.0 MAC and 1.5 and 2.0 MAC, whereas a significant difference was noted only between 1.0 and 2.0 MAC in the sevoflurane group. Mean values for MPAP were significantly different only between the 1.5 and 2.0 MAC for isoflurane. There were no significant differences detected either within or between inhalation anesthetic groups for end-tidal CO₂ concentration, heart rate, pulmonary arterial blood temperature, pH, Paco₂, PaO₂, HCO₃⁻ concentration, PAOP, pulmonary vascular resistance, or CVP.

Discussion

In this study, MACs were determined for isoflurane and sevoflurane in 6 young Beagles to allow subsequent measurement of ICP at various MAC multiples. Considering that the observers were not unaware of the treatment groups, it is possible that some bias in the determination of the MACs could have occurred. Minimum alveolar concentration of anesthetic at 1 atm that induces immobility in 50% of animals exposed to a noxious stimulus was used to define MAC. The mean MACs determined for the dogs in the present study were similar to values reported for isoflurane and sevoflurane in dogs. Electrical current was used as the supramaximal noxious stimulus for the MAC determinations and yields results similar to those obtained with mechanical stimulation. Equilibration times were based on previous MAC studies in dogs.

A fiberoptic transducer system was used to measure ICP in the present study, and its use has been described in dogs. The values reported here were similar to the values reported (mean ± SD, 15 ± 5.25 mm Hg) in a recent study but somewhat higher than reported values of ICP (8 to 14 mm Hg) in clinically normal dogs of other breeds. The relative position of the body and head may affect the brain-to-heart hydrostatic gradient leading to variability in measured ICP. Thus, the differences in reported values could be due to positioning, concurrent drug administration, or perioperative instrumentation (eg, presence of a Swan-Ganz catheter).

In each part of the present study, isoflurane and sevoflurane were studied on the same day. This allowed the humane measurement of ICP for both anesthetics in the same dog without subsequent recovery from anesthesia. Although an effort was made to ensure adequate washout of the first inhalation anesthetic before measurements were begun with the second inhalation anesthetic, an effect of the initial anesthetic on the measured variables during the second anesthetic cannot be ruled out. The order of study of inhalation anesthetics was randomized to minimize such effects.

In the present study, a dose-dependent change in ICP was not observed during anesthesia with either isoflurane or sevoflurane. These findings agree with previous studies conducted by Takahashi et al in dogs, which detected no significant change in ICP with sevoflurane at 0.5, 1.0, and 1.5 MAC. Although CBF was not measured in the present study, other investigators have found that increasing MACs of both isoflurane and sevoflurane were not associated with increased CBF in dogs and rabbits. Other studies further found that neither isoflurane nor sevoflurane affects CSF dynamics in a way that would increase CSF volume or pressure. It is possible that in these healthy normocapnic dogs with (presumably) normal intracranial compliance, alterations in CBF due to either isoflurane or sevoflurane were inadequate to alter CBV to an extent that would lead to significant changes in ICP.

Cerebral blood flow autoregulation prevents alterations in CBF due to changes in CPP. The CPP may be decreased by either arterial hypotension or increased ICP. The CPP and MAP were higher at equipotent concentrations of sevoflurane, compared with isoflurane, but were significantly higher only at 2.0 MAC. Also, the SVR was higher at all the MACs with sevoflurane, compared with isoflurane, but significance was achieved only at 1.5 MAC. Other reports describing anesthetic effects in horses, children, and cats, indicate that sevoflurane may cause less systemic vasodilation and maintain better vascular tone, compared with isoflurane.

The CBF is maintained constant between CPP of 50 and 150 mm Hg. Cerebral blood flow autoregulation is maintained by isoflurane at 1.0 MAC but is impaired at 2.0 MAC. In isoflurane-anesthetized dogs at 1.0 MAC, CBF was decreased when CPP decreased to < 43 mm Hg. In the present study, 5 dogs at 2.0 MAC for isoflurane and 2 dogs at 1.5 MAC for isoflurane had CPP < 43 mm Hg. These findings lead to speculation that brain hypoperfusion could be more likely to occur in dogs at moderate to deep concentrations of isoflurane. Only 2 dogs had their CPP decreased to < 50 mm Hg.
(38 and 45 mm Hg) in the sevoflurane group at 2.0 MAC. Studies have found no change in CBF velocity in response to phenylephrine-induced increase in MAP during anesthesia with 1.2 to 1.5 MAC for sevoflurane. Thus, there is some evidence that, compared with isoflurane, sevoflurane may cause less impairment of autoregulation. The present study supports the expectation that dogs anesthetized with sevoflurane may maintain better CPP, compared with isoflurane, particularly during deeper (ie, 2.0 MAC) anesthesia. The ultimate effect on CBF will be determined by the dose of the inhalation anesthetic, the hemodynamic status, and the intracranial compliance of the animal.

A dose-dependent decrease was observed in arterial blood pressure (SAP, MAP, and DAP) and CO during both isoflurane and sevoflurane anesthesia, as reported in previous studies. However, CO was similar during equipotent dosages of isoflurane and sevoflurane. Further, in the present study, SVR did not decrease in a dose-dependent fashion and no change in heart rate was observed within or between either anesthetic. Thus, the dose-related response in blood pressure may be due to changes in stroke volume. The dose-related decreases in blood pressure associated with inhalation anesthetics are mostly related to decreased stroke volume in animals, in contrast to humans, in which blood pressure decreases primarily due to a decrease in SVR.

We used IPPV to maintain normocapnia to minimize the influence of altered PaCO₂ on CBV and ICP. It is possible that IPPV itself may influence ICP due to changes in venous outflow from the brain, but this potential effect was present for all dogs and therefore did not likely contribute to any of the observed differences between anesthetics.

During the second part of the experiment, dogs were anesthetized for a mean ± SD of 5.2 ± 0.2 hours, until the last measurement was made. Previous investigations in goats and dogs but not in humans have found the effect of inhalation anesthetic drugs on CBV to be time dependent. Contribution of any such effect in the present study was minimal because the inhalation agents and different MACs within each inhalation agent were randomized.

In conclusion, ICP did not change during 1.0, 1.5, or 2.0 MAC anesthesia with either isoflurane or sevoflurane in the Beagles of the present study. Although ICP was similar during isoflurane or sevoflurane anesthesia, the CPP was better maintained during 2.0 MAC for sevoflurane. The present study was conducted in young dogs with apparently normal brains and intracranial compliance; therefore, these results should be only carefully extrapolated to animals with altered brain physiology.

References

17. Schwartz AE, Maneksha FR, Kanchuger MS, et al. Flumazenil...


