Hemodynamic effects of sevoflurane in cats

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Objective—To determine hemodynamic effects of 3 concentrations of sevoflurane in cats.

Animals—6 cats.

Procedure—Cats were anesthetized with sevoflurane in oxygen. After instruments were inserted, end-tidal sevoflurane concentration was set at 1.25, 1.5, or 1.75 times the individual minimum alveolar concentration (MAC), which was determined in another study. Twenty-five minutes were allowed after each change of concentration. Heart rate; systemic and pulmonary arterial pressures; central venous pressure; pulmonary artery occlusion pressure; cardiac output; body temperature; arterial and mixed-venous pH, PCO$_2$, PO$_2$, oxygen saturation, and hemoglobin concentrations; PCV; and total protein and lactate concentrations were measured for each sevoflurane concentration before and during noxious stimulation. Arterial and mixed-venous bicarbonate concentrations, cardiac index, stroke index, rate-pressure product, systemic and pulmonary vascular resistance indices, left and right ventricular stroke work indices, Pa$_{O_2}$, mixed-venous partial pressure of oxygen (PvO$_2$), oxygen delivery, oxygen consumption, oxygen-extraction ratio, alveolar-to-arterial oxygen difference, and venous admixture were calculated. Spontaneous and mechanical ventilations were studied during separate experiments.

Results—Mode of ventilation did not significantly influence any of the variables examined. Therefore, data from both ventilation modes were pooled for analysis. Mean arterial pressure, cardiac index, stroke index, rate-pressure product, left ventricular stroke work index, arterial and mixed-venous pH, Pa$_{O_2}$, and oxygen delivery decreased, whereas Pa$_{CO_2}$, PvO$_2$, and mixed-venous partial pressure of CO$_2$ increased significantly with increasing doses of sevoflurane. Noxious stimulation caused a significant increase in most cardiovascular variables.

Conclusions and Clinical Relevance—Sevoflurane induces dose-dependent cardiovascular depression in cats that is mainly attributable to myocardial depression. (Am J Vet Res 2004;65:20–25)

Sevoflurane was introduced in North America and Europe during the past 5 years.1 Its potential benefits include a low blood-gas partition coefficient and good tolerance in children when administered by face-mask. These characteristics suggest that its use may be advantageous whenever fast induction and rapid recovery are desired.

Cats seem to be particularly sensitive to the cardiovascular depressant effects of potent inhalation anesthetics.2 This may contribute to the higher morbidity and mortality rates reported in cats anesthetized with these agents, compared with morbidity and mortality rates in anesthetized dogs.3 Sevoflurane has been proposed for clinical use in veterinary patients, including cats. However, to our knowledge, the cardiovascular effects of sevoflurane in cats have only been partially reported.4,5 The study reported here was conducted to determine the hemodynamic effects of 3 concentrations of sevoflurane in cats. We hypothesized that sevoflurane would depress the cardiovascular system in a dose-dependent manner and the depression would be greater during mechanical ventilation than during spontaneous respiration. Moreover, to mimic clinical conditions, hemodynamic measurements were repeated during application of a noxious stimulus.

Materials and Methods

Animals—Six healthy adult domestic shorthair cats that weighed (mean ± SEM) 3.47 ± 0.44 kg were used in the study. The minimum alveolar concentration (MAC) of sevoflurane had been determined in each cat by use of the tail-clamp method in a study reported elsewhere. Food was withheld from cats for 12 hours before experiments were initiated. The study was approved by an institutional animal care and use committee.

Induction of anesthesia and insertion of instruments—Anesthesia was induced with sevoflurane in oxygen by use of an induction box. Anesthesia induction was completed by use of a facemask; each cat was then intubated with a cuffed endotracheal tube, and anesthesia was maintained by administration of sevoflurane in oxygen via a Bain circuit with an oxygen flow rate of 500 mL/kg/min.

A catheter was passed through the lumen of the endotracheal tube to a point where the distal tip of the catheter was level with the end of the endotracheal tube. This catheter was used to sample end-tidal gases. A 22-gauge, 2.5-cm catheter was inserted in a cephalic vein, and lactated Ringer's solution was administered at the rate of 3 mL/kg/h. A 5-F, 7.5-cm introducer was placed in a jugular vein. A 4-F, 75-cm thermodilution catheter was inserted by use of fluoroscopic observation through the introducer and positioned with its distal port and thermistor in the pulmonary artery; this catheter was used for measurement of cardiac output, mean pulmonary arterial pressure (MPAP), pulmonary artery occlusion pressure (PAOP), central venous pressure (CVP), and core body temperature and collection of mixed-venous blood samples (ie, samples from the pulmonary artery). A 24-gauge, 9-cm catheter was inserted in a femoral artery by use of the Seldinger technique; this catheter was used for measurement of arterial pressure and collection of arterial blood samples.

Each cat was positioned in right lateral recumbency. An ECG (lead II), heart rate (HR), systolic arterial pressure

Received November 7, 2002.
Accepted July 14, 2003.
From the Department of Surgical and Radiological Sciences, School of Veterinary Medicine, University of California, Davis, CA 95616. Supported by the Center for Companion Animal Health, School of Veterinary Medicine, University of California, Davis. Presented in part at the 27th Annual Meeting of the American College of Veterinary Anesthesiologists, Orlando, Fla, October 2002. The authors thank Jennifer Bolich for technical assistance. Address correspondence to Dr. Pypendop.
(SAP), diastolic arterial pressure (DAP), mean arterial pressure (MAP), MPAP, and CVP were continuously monitored and recorded by use of a physiograph and acquisition software. All pressure transducers were calibrated against a mercury manometer before each experiment, and a value of zero was established at the level of the sternum. Inspired and expired O₂, CO₂, and sevoflurane concentrations were continuously monitored by use of a spectrometer calibrated with 60 mL of 3 calibration gases of known concentration (1%, 2%, and 5% sevoflurane). The calibration was repeated every 80 minutes, corresponding to the internal-calibration interval of the spectrometer. Expired gases were collected manually (20 mL collected during 7 to 10 respiratory cycles) and analyzed in triplicate by use of the spectrometer, and the mean value for the 3 measurements was calculated. Arterial and mixed-venous pH, PaCO₂, mixed-venous partial pressure of CO₂ (PvCO₂), PaO₂, and mixed-venous partial pressure of O₂ (PvO₂) were measured and bicarbonate concentration calculated by use of a blood gas analyzer that adjusted measurements on the basis of body temperature. Arterial and mixed-venous blood hemoglobin concentration and oxygen saturation were measured by use of a hematocrit. Lactate concentration, PCV (by use of a microcentrifugation technique), and total protein (TP) concentration (by use of a refractometer) were measured in arterial blood samples. Cardiac output was determined, in triplicate, by use of a thermodilution technique, and a cardiac output computer. Three milliliters of iced 5% dextrose was injected through the proximal port of the thermodilution catheter for each determination. The mean value of the 3 measurements was then calculated. Core body temperature was maintained between 38° and 39°C throughout the study by use of warm-water circulated blankets and forced-air blankets, as needed.

Cardiac index (CI), stroke index (SI), rate-pressure product (RPP), systemic vascular resistance index (SVRI), pulmonary vascular resistance index (PVRI), left ventricular stroke work index (LVSWI), right ventricular stroke work index (RVSWI), arterial oxygen concentration (CaO₂), mixed-venous oxygen concentration (CvO₂), oxygen delivery (DO₂), oxygen consumption (VO₂), oxygen-extraction ratio (O₂ extraction), alveolar-to-arterial difference in partial pressure of oxygen (PAO₂ – PaO₂), and venous admixture (Qₐ/Qₗ) were calculated by use of standard equations. Barometric pressure at the time of the study, which was used to calculate alveolar Po₂, was obtained from the climate station at the University of California, Davis.

Spontaneous respiration and mechanical ventilation were studied during separate experiments. For mechanical ventilation, a pressure-cycled ventilator was used. Intermittent positive-pressure ventilation was started after instruments were inserted and was provided for the remainder of that experimental period.

Experimental protocol—Ninety minutes after induction of anesthesia and subsequent insertion of instruments, the sevoflurane concentration was randomly set at 1.25, 1.5, or 1.75 times the individual MAC that had been determined for each cat. Concentrations were administered in accordance with a Latin-square design. Twenty-five minutes was allowed after each change of concentration for conditions to equilibrate. Samples of end-tidal gas were then manually collected for measurement of end-tidal sevoflurane concentration. Heart rate, SAP, DAP, MAP, CVP, MPAP, and PAOP were recorded. Samples (1 mL) of arterial and mixed-venous blood were collected and immediately placed on ice until analyzed; these samples were analyzed within 20 minutes after collection. Cardiac output was measured. After each set of measurements, a supramaximal noxious stimulus was applied to the tail for 5 minutes. The stimulus involved a 20-cm Martin forceps closed to the second ratchet. The proximal 10 cm of the tail was used for application of the stimulus, and the actual location of the forceps differed at each stimulation. With the stimulus ongoing, the measurements were repeated.

At the end of the experiment, the thermodilution catheter and introducer were removed, and a compressive bandage was applied over the jugular vein for 15 minutes. The catheter in the femoral artery was removed, and the femoral artery was sutured and the skin closed. Cefazolin was administered (22 mg/kg, IV), and the cats were allowed to recover.

Statistical analysis—Data were analyzed for the effects of MAC, ventilation mode, and noxious stimulation by use of a repeated-measures ANOVA. Data from both modes of ventilation were then pooled, and the analysis was repeated. Post hoc comparisons among MAC (stratified on the basis of stimulus) or between stimulus modes (stratified on the basis of MAC) were performed by use of least-square means. We did not use adjustments for multiple comparisons. Statistical significance was set at values of P < 0.05. Data were reported as mean ± SEM.

![Figure 1](image1.png) Figure 1—Effect of sevoflurane concentration on mean arterial pressure before (diamond) and during (square) noxious stimulation in 6 cats. Values reported are mean ± SEM for each minimum alveolar concentration (MAC) multiple of sevoflurane tested and represent pooled data from the spontaneous respiration and mechanical ventilation experiments. Within a stimulation category, value differs significantly (P < 0.05) from value for 1.25 MAC. *Within a MAC category, value differs significantly (P < 0.05) from value before stimulation.

![Figure 2](image2.png) Figure 2—Effect of sevoflurane concentration on cardiac index before (diamond) and during (square) noxious stimulation in 6 cats. Values reported are mean ± SEM for each MAC multiple of sevoflurane tested and represent pooled data from the spontaneous respiration and mechanical ventilation experiments. See Figure 1 for key.
Table 1—Effects of various concentrations of sevoflurane before and during noxious stimulation on selected variables in 6 cats

<table>
<thead>
<tr>
<th>Variable</th>
<th>Before</th>
<th>During</th>
<th>Before</th>
<th>During</th>
<th>Before</th>
<th>During</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (BPM)</td>
<td>190 ± 4</td>
<td>193 ± 5</td>
<td>187 ± 4</td>
<td>184 ± 4</td>
<td>188 ± 6</td>
<td>184 ± 6</td>
</tr>
<tr>
<td>SAP (mm Hg)</td>
<td>79 ± 3</td>
<td>89 ± 4a</td>
<td>70 ± 3a</td>
<td>70 ± 3</td>
<td>74 ± 3</td>
<td>79 ± 4</td>
</tr>
<tr>
<td>DAP (mm Hg)</td>
<td>51 ± 2</td>
<td>61 ± 4a</td>
<td>45 ± 2†</td>
<td>47 ± 2†</td>
<td>47 ± 3</td>
<td>47 ± 4†</td>
</tr>
<tr>
<td>CVP (mm Hg)</td>
<td>10 ± 1</td>
<td>11 ± 1</td>
<td>11 ± 1</td>
<td>11 ± 1</td>
<td>11 ± 1</td>
<td>11 ± 1</td>
</tr>
<tr>
<td>MPAP (mm Hg)</td>
<td>17 ± 1</td>
<td>18 ± 1</td>
<td>17 ± 1</td>
<td>18 ± 1</td>
<td>17 ± 1</td>
<td>18 ± 1</td>
</tr>
<tr>
<td>PAOP (mm Hg)</td>
<td>11 ± 1</td>
<td>12 ± 1</td>
<td>11 ± 1</td>
<td>12 ± 1</td>
<td>12 ± 1</td>
<td>12 ± 1</td>
</tr>
<tr>
<td>SI (l[m]baths/m³)</td>
<td>5.7 ± 0.5</td>
<td>6.5 ± 0.7</td>
<td>4.8 ± 0.4†</td>
<td>5.3 ± 0.4**</td>
<td>4.6 ± 0.3†</td>
<td>5.2 ± 0.4**</td>
</tr>
<tr>
<td>SVRI ([dyne x s]/cm²/cm²)</td>
<td>3,941 ± 205</td>
<td>3,951 ± 246</td>
<td>4,049 ± 249</td>
<td>3,804 ± 227</td>
<td>4,171 ± 184</td>
<td>3,922 ± 258</td>
</tr>
<tr>
<td>PVR ([dyne x s]/cm²/cm²)</td>
<td>464 ± 59</td>
<td>464 ± 78</td>
<td>496 ± 61</td>
<td>502 ± 81</td>
<td>469 ± 79</td>
<td>487 ± 67</td>
</tr>
<tr>
<td>RPP (beats x mm Hg)</td>
<td>14,991 ± 698</td>
<td>17,102 ± 994*</td>
<td>12,958 ± 637</td>
<td>13,382 ± 691†</td>
<td>13,856 ± 809</td>
<td>14,438 ± 780t</td>
</tr>
<tr>
<td>LVSWI (g x m²/m²)</td>
<td>4.0 ± 0.5</td>
<td>5.3 ± 0.8*</td>
<td>3.0 ± 0.4</td>
<td>3.3 ± 0.4t</td>
<td>2.7 ± 0.3</td>
<td>3.4 ± 0.4</td>
</tr>
<tr>
<td>RSVWI (g x m²/m³)</td>
<td>0.6 ± 0.1</td>
<td>0.6 ± 0.1</td>
<td>0.4 ± 0.1</td>
<td>0.5 ± 0.1</td>
<td>0.4 ± 0.1</td>
<td>0.4 ± 0.1</td>
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<tr>
<td>PCV (%)</td>
<td>25 ± 1</td>
<td>25 ± 1</td>
<td>26 ± 1</td>
<td>26 ± 1</td>
<td>26 ± 1</td>
<td>26 ± 3</td>
</tr>
<tr>
<td>TP (g/dL)</td>
<td>4.3 ± 0.1</td>
<td>4.0 ± 0.1</td>
<td>4.2 ± 0.1</td>
<td>4.0 ± 0.1</td>
<td>4.2 ± 0.1</td>
<td>3.7 ± 0.3</td>
</tr>
</tbody>
</table>

Values reported are mean ± SEM and represent pooled data from the spontaneous respiration and mechanical ventilation experiments.

*Within a minimum alveolar concentration (MAC) category, value differs significantly (P < 0.05) from value before stimulation. 
†Within a row, value differs significantly (P < 0.05) from corresponding value for 1.25 MAC.

HR = Heart rate. BPM = Beats per minute. SAP = Systolic arterial pressure. DAP = Diastolic arterial pressure. CVP = Central venous pressure. MPAP = Mean pulmonary arterial pressure. PAOP = Pulmonary artery occlusion pressure. SI = Stroke index. SVRI = Systemic vascular resistance index. PVR = Pulmonary vascular resistance index. RPP = Rate-pressure product. LVSWI = Left ventricular stroke work index. RSVWI = Right ventricular stroke work index. TP = Total protein.

Results

Sevoflurane concentrations—The MAC of sevoflurane determined in the same cats in another study4 and used in this study was 3.41% ± 0.27% (range for each cat, 2.64% to 4.47%). The ratio for the measured sevoflurane concentration-to-target sevoflurane concentration was 0.99 ± 0.02 for all cats at all measurement times.

Effects of mode of ventilation—Mode of ventilation did not significantly influence any of the variables examined at any sevoflurane concentration administered. Therefore, data from both modes of ventilation were pooled for subsequent analysis, and results reported here were for pooled data from the spontaneous respiration and mechanical ventilation experiments.

Effects of sevoflurane before noxious stimulation—We did not detect significant differences in HR, CVP, MPAP, PAOP, SVRI, PVR, RSVWI, PCV, TP concentration, PCO₂, arterial and mixed-venous bicarbonate concentrations, SaO₂, SvO₂, arterial and mixed-venous hemoglobin concentrations, lactate concentration, CaO₂, CVO₂, VO₂ extraction, PAO₂ – PaO₂, and
Q₁/Q₇ among the 3 doses of sevoflurane tested in the study.

The MAP, CI, SI, LVSWI, and DO₂ were significantly higher at 1.25 MAC, compared with values at 1.5 and 1.75 MAC of sevoflurane, but we did not detect significant differences for these variables between 1.5 and 1.75 MAC (Fig 1 and 2; Table 1 and 2). The PaO₂ was significantly lower at 1.75 than at 1.25 MAC, but we did not detect differences in PaO₂ between 1.5 and 1.75 MAC. The PVO₂ was significantly higher at 1.75 MAC than at 1.25 and 1.5 MAC, whereas we did not detect differences in PVO₂ between 1.25 and 1.5 MAC. Arterial and mixed-venous pH decreased significantly; whereas PaCO₂ increased significantly, with the administration of increasing doses of sevoflurane; values for these variables differed significantly among the 3 sevoflurane concentrations. Increasing MAC of sevoflurane significantly decreased RPP; however, when pairwise comparisons were made for the sevoflurane concentrations, we did not detect a significant effect on RPP.

Effects of noxious stimulation—The MAP, SAP, DAP, CI, SI, RPP, LVSWI, and DO₂ were significantly higher during noxious stimulation than before stimulation for some concentrations of sevoflurane (Fig 1 and 2; Table 1 and 2). Moreover, a significant interaction between sevoflurane MAC and noxious stimulation was detected for DAP.

Discussion

The study reported here revealed the hemodynamic effects of sevoflurane administered to cats at 1.25, 1.5, and 1.75 MAC. Although the effects of sevoflurane on variables such as HR, blood pressure, arterial blood gas tensions, and pH have been studied in cats, we are not aware of a more complete characterization of the hemodynamic changes induced by sevoflurane administration to cats.

The individual MAC values were determined in another study and used in this study to improve accuracy. The measured sevoflurane concentrations were extremely close to the target concentrations, as illustrated by the ratio for measured sevoflurane concentration to target sevoflurane concentration.

Sevoflurane has a low blood:gas partition coefficient of 0.68; therefore, partial pressures in the blood and brain equilibrate rapidly with alveolar partial pressure. Twenty-five minutes was allowed after each change of concentration for equilibration, which should have been sufficient for equilibration with the vessel-rich group, including the brain.

In the study reported here, we chose to compare 1.25, 1.5, and 1.75 MAC of sevoflurane. Given the fact that cats appear to be extremely sensitive to the cardiovascular-depressant effects of volatile anesthetics, we elected not to study the effects of 2.0 MAC of sevoflurane because of the potential severity of the hemodynamic depression at that concentration that has been observed in other species by other investigators at our institution. Moreover, 2.0 MAC is of little clinical relevance, representing an extremely deep plane of anesthesia. In 1 study, 2.0 MAC of sevoflurane did not result in life-threatening hypotension; however, the MAC value (2.38%) in that study was lower than the lowest individual MAC value (2.64%) in our cats. The 2.0 MAC of that study corresponds to approximately 1.5 times the mean MAC in our study.

Two modes of ventilation, spontaneous respiration and mechanical ventilation, were used in the study reported here. We expected that cardiovascular depression induced by sevoflurane would be greater during mechanical ventilation because of the effects of increased intrathoracic pressure on hemodynamics and the effects of decreased PaCO₂. Moreover, we hypothesized that the concentration of sevoflurane administered would influence the magnitude of the effect for mechanical ventilation. In the mechanical ventilation experiment, we intended to maintain end-tidal CO₂ between 35 and 40 mm Hg. However, in the spontaneous respiration experiment, end-tidal CO₂ was < 40 mm Hg at all sevoflurane concentrations, whereas in the mechanical ventilation experiment, all cats breathed spontaneously between the ventilator cycles, resulting in CO₂ tensions lower than targeted. This explains the lack of difference on PaCO₂ between the spontaneous respiration and mechanical ventilation experiments and, possibly, the lack of effect of ventilation mode on any cardiovascular variable in our study.

In an attempt to induce similar effects among cats despite the lack of control on end-tidal CO₂, peak inspiratory pressure and respiratory rate were maintained constant throughout the study. In this study, mechanical ventilation did not result in increased cardiovascular depression in sevoflurane-anesthetized cats.

The study was designed to characterize the dose-dependent manner of the cardiovascular depression induced by sevoflurane in cats. Although it would have been preferable to compare the effects in sevoflurane-anesthetized cats with values for awake control cats, the technical difficulty of maintaining cardiovascular instrumentation and performing cardiovascular measurements in awake cats prevented this. However, it has been reported in other species that sevoflurane produced cardiovascular depression when compared to the awake state.

Cardiovascular effects of sevoflurane have been reported in various species. In most studies, the effects have been described as being similar to those induced by isoflurane, with the exception of a lower HR. According to those studies, sevoflurane induces a dose-dependent decrease in arterial pressure, systemic vascular resistance, and cardiac output. However, in newborn pigs, cardiovascular depression induced by sevoflurane is significantly less than that induced by isoflurane at equipotent concentrations. Similar observations have been reported in cats when a rebreathing system was used but not when a nonrebreathing system was used. In cats, sevoflurane decreased MAP, arterial pH, PaO₂, and arterial bicarbonate concentration and increased PaCO₂ in a dose-dependent manner. In that study, most effects were significant when results for 1.0 MAC were compared with results for 2.0 MAC, but not when results for 1.0 MAC were compared with those for 1.5 MAC; further-
more, the effects on MAP were similar during spontaneous respiration and controlled ventilation.

In the study reported here, sevoflurane induced dose-dependent cardiovascular depression characterized by a decrease in MAP, CI, SI, RPP, LVSWI, and DO₂. Although values for these variables were significantly higher at 1.25 MAC, compared with values at 1.5 MAC, little difference was found between values at 1.5 and 1.75 MAC. Analysis of these results indicates that a ceiling for the cardiovascular-depressant effects of sevoflurane in cats may be reached at approximately 1.5 MAC; however, further studies at additional MAC multiples would be needed to substantiate this.

Mean arterial pressure decreased with the administration of sevoflurane at 1.5 and 1.75 MAC, compared with MAP at 1.25 MAC. This decrease in MAP is similar to that reported in various species15,18,20,21 and for most inhalant anesthetics.22 However, in the study reported here, the decrease in MAP was attributable to a decrease in CI because SVRI did not change within the range of sevoflurane concentrations used. This is in contrast to the effects of sevoflurane in other species,11,12,15,21 in which the dose-dependent decrease in MAP is related to a combination of negative inotropic activity and vasodilation.

The decrease in CI was related to a decrease in SI because HR did not change significantly in the cats during the study. This is similar to the effects of sevoflurane in other species in which HR remains stable11,13,21 or even increases.21 In 1 study3 in cats anesthetized with sevoflurane, HR reportedly increased in a dose-dependent manner. In our study, SI most likely decreased because of a dose-dependent negative inotropic effect, based on the fact that sevoflurane can cause myocardial depression.13,22 Indeed, indices of preload and afterload such as CVP, PAOP, or SVRI remained unchanged throughout our study.

Rate-pressure product has been used as an indicator of myocardial oxygen consumption.15-20 Because of the dose-dependent hypotension induced by sevoflurane, RPP decreased as the anesthetic concentration increased. This decrease in myocardial oxygen consumption may have been related to decreased myocardial workload, as illustrated by the decrease in LVSWI, which is related to both hypotension and decreased SI. A dose-dependent decrease in myocardial oxygen consumption during sevoflurane-induced anesthesia has been reported in other studies.12,22 The effect on DO₂ was related to the cardiovascular-depressant effects of sevoflurane (ie, decreased CI) because CaO₂ did not change.

Sevoflurane induced dose-dependent respiratory depression, indicated by the increase in PaCO₂. Similar effects have been reported15-18,21,27-29 in various species, including cats; however, based on the comparison of results for several studies,11,21 the magnitude of these effects seems to be less in cats than in other species. The increase in PaCO₂ resulted in a mild decrease in blood pH. Although PaO₂ decreased significantly (mainly because of changes in ventilation), this effect was not considered clinically important, and PaO₂ remained well above 500 mm Hg. The increase in PVO₂ may reflect the right shift of the hemoglobin dissociation curve related to the increase in PaCO₂.

Noxious stimulation during inhalant anesthesia generally stimulates the cardiovascular system, and this response may or may not be attenuated by increasing the anesthetic concentration. In the study reported here, noxious stimulation resulted in significant increases in SAP, DAP, MAP, CI, SI, and LVSWI. A significant interaction between MAC and noxious stimulation was observed only for DAP, suggesting that increasing the dose of sevoflurane did not blunt the response to noxious stimulation for the other variables. Although to our knowledge the effects of noxious stimulation during sevoflurane-induced anesthesia in cats have not been reported, these effects are similar to those reported for isoflurane-induced anesthesia in cats.30 The fact that increasing the concentration of sevoflurane failed to attenuate the cardiovascular response to noxious stimulation is not surprising because when administered alone in humans, > 2.0 MAC of sevoflurane was needed to block this response.31

Similar to other inhalants, sevoflurane induces dose-dependent cardiovascular depression in cats; however, a ceiling effect may be reached at approximately 1.5 MAC, although additional studies at additional MAC multiples would be needed to confirm this effect. The apparent lack of effect of sevoflurane on systemic vascular resistance may limit the potential of this agent to cause hypotension in cats, despite substantial myocardial depression.

References

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