Effects of increasing infusion rates of dopamine, dobutamine, epinephrine, and phenylephrine in healthy anesthetized cats

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Objective—To determine the cardiopulmonary effects of increasing doses of dopamine, dobutamine, epinephrine, and phenylephrine and measure plasma concentrations of norepinephrine, epinephrine, and dopamine in cats anesthetized with isoflurane.

Animals—6 healthy adult cats.

Procedures—Each cat was anesthetized with isoflurane (1.5% minimum alveolar concentration) on 4 occasions. Cardiopulmonary measurements were obtained after a 30-minute stabilization period; 20 minutes after the start of each infusion dose; and 30, 60, and 90 minutes after the infusion was discontinued. Cats received 5 progressively increasing infusions of epinephrine or phenylephrine (0.125, 0.25, 0.5, 1, and 2 μg/kg/min) or dobutamine or dopamine (2.5, 5, 10, 15, and 20 μg/kg/min). The order of treatment was randomly allocated.

Results—All 4 treatments increased oxygen delivery. Heart rate (HR) increased during administration of all drugs except phenylephrine, and mean arterial pressure increased during administration of all drugs except dobutamine. A progressive metabolic acidosis was detected, but whole-blood lactate concentration only increased during administration of epinephrine and dobutamine. Systemic vascular resistance index increased during administration of phenylephrine, decreased during administration of dobutamine, and remained unchanged during administration of dopamine and epinephrine. A positive inotropic effect was detected with all treatments.

Conclusions and Clinical Relevance—During anesthesia in cats, administration of dopamine, dobutamine, and epinephrine may be useful for increasing cardiac output, with dopamine having the most useful effects. Administration of phenylephrine increased cardiac and systemic vascular resistance indexes with minimal effect on HR and may be useful for increasing mean arterial pressure without increasing HR. (Am J Vet Res 2006;67:1491–1499)

Anesthesia in cats continues to be a challenge for all practitioners. Results of 3 studies1,2 examining morbidity and death rate associated with anesthesia in small animal practices indicate that cats have higher death rates than dogs. These studies have used the

Received November 9, 2005.
Accepted December 27, 2005.
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Supported by the Center for Companion Animal Health, School of Veterinary Medicine, University of California.
The authors thank Drs. Tony Yaksh and Mike Rathbun for assistance with the statistical analysis.
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ABBREVIATIONS
- ASA American Society of Anesthesiologists
- MAC Minimum alveolar concentration
- MAP Mean arterial pressure
- CVP Central venous pressure
- SVRI Systemic vascular resistance index
- PVRI Pulmonary vascular resistance index
- CaO2 Arterial oxygen content
- CvO2 Venous oxygen content
- SVI Stroke volume index
- CI Cardiac index

ASAs patient classification scheme in which a patient classified as ASA 1 or 2 is basically considered healthy and ASA 3, 4, and 5 represent increasing degrees of physiologic dysfunction; a patient classified as ASA 5 has an extremely poor prognosis. In 1 study,1 the death rate in healthy cats (classified as ASA 1 and 2) undergoing anesthesia was 0.18%, compared with 0.11% in dogs, whereas in a second study,2 death rates were similar (0.11% to 0.1% for healthy cats and dogs, respectively). However, in the first study, the death rate for sick cats (classified as ASA 3, 4, and 5) was 3.33% (3.12% for dogs), whereas in the second study, the odds ratio for anesthetic complications was 5.3 in a similar group of cats (ie, complications in sick cats were 5.3 times more likely than in clinically normal cats), compared with an odds ratio of 2.5 for dogs.2 Another study3 reports a death rate of 0.112% for healthy cats and 1.4% for cats classified as ASA 3 to 5. These death rates do not compare favorably with anesthetic-associated deaths in humans in which the rate is 0.01% for patients classified as ASA 1 to 5.3

Part of the reason for increased death rate in cats is that they develop considerable cardiovascular depression with inhalant anesthesia. The depressant effect of halothane in cats was detected at a surgical plane of anesthesia (1.3 to 2.0 × MAC) with a decrease in MAP from 89 to 49 mm Hg, a decrease in heart rate from 208 to 142 beats/min, and a decrease in cardiac output from 0.82 to 0.32 L/min when comparing values in awake cats with values in anesthetized cats, respectively.4 Isoflurane induced similar changes with a decrease in MAP from 95 to 60 mm Hg at 1.3% inspired isoflurane (<1 × MAC) and to 40 mm Hg at 2.5% inspired isoflurane (1.5 × MAC), respectively.5 Aortic blood flow decreased to 53% and 33% of the value in awake cats at these concentrations. Our most recent experiments in cats during anesthesia have examined the effect of opioids at reducing the amount of inhalant anesthetic needed for anesthesia, and we have found that with a
number of various drugs, there is a ceiling effect at approximately a 30% reduction of MAC. In 1 study, the addition of alfentanil to reduce the required amount of isoflurane resulted in a 2-fold increase in MAP and cardiac output. However, although the reduction of inhalant anesthetic is helpful in improving cardiovascular function, it is often necessary, in a clinical setting, to use catecholamines to increase arterial blood pressure to within acceptable limits.

The purpose of the study reported here was to determine the cardiopulmonary effects of increasing doses of dopamine, dobutamine, epinephrine, and phenylephrine and measure plasma concentrations of norepinephrine, epinephrine, and dopamine in cats anesthetized with isoflurane. We hypothesized that dopamine, dobutamine, and epinephrine would increase cardiac output in a dose-dependent manner but that phenylephrine would increase blood pressure with no change in cardiac output.

**Materials and Methods**

The study was approved by the Animal Care and Use Committee at the University of California, Davis. Six adult cats weighing 5.4 ± 0.8 kg (mean ± SD) were used in the study. Each cat was determined to be healthy on the basis of physical examination findings and findings detected during a 2-week observation period and results of CBC and serum biochemical analyses.

Cats were gang housed and fed ad libitum. Food was withheld for 12 hours before each experiment. The cardiovascular response to 5 incremental doses of dopamine, dobutamine, epinephrine, or phenylephrine was tested in each cat. Dopamine and dobutamine were studied at infusion rates of 2.5, 5, 10, 15, and 20 μg/kg/min, and epinephrine and phenylephrine were studied at infusion rates of 0.125, 0.25, 0.5, 1, and 2 μg/kg/min. Cats were anesthetized on 4 occasions. The order of treatment was allocated randomly. On the day of each experiment, the cat was placed in an induction chamber and anesthesia was induced with isoflurane in oxygen. After the cat lost its righting reflex, it was removed from the chamber and a face mask was used to deliver isoflurane in oxygen until endotracheal intubation could be performed. Anesthesia was maintained with isoflurane in oxygen by use of a Bain nonrebreathing circuit with a flow rate of 200 mL/kg/min. The cat was placed on a heating blanket, and core body temperature was maintained between 37° and 39°C. A catheter was placed in a cephalic vein, and lactated Ringer’s solution was administered IV at a rate of 3 mL/kg/h. A 5-F Swan Ganz catheter was advanced into the pulmonary artery via a jugular cut down during fluoroscopic guidance and was used to measure core body temperature, CVP, pulmonary arterial occlusion pressure, and cardiac output by use of thermodilution. A catheter was placed in the femoral or brachial artery by use of a surgical cut down. This was used for measurement of arterial blood pressure (by use of a calibrated pressure transducer) and for collection of arterial blood for measurements of pH, blood gas values, and plasma catecholamine concentrations. Electrocardiography (lead II by use of foot pad electrodes) was performed in each cat. After the cat had been instrumented, the end-tidal concentration of isoflurane was adjusted to 1.5 × MAC on the basis of previously published values (MAC = 1.28%). This value was used to approximate a surgical depth of anesthesia. The anesthetic circuit was connected to a ventilator and the end-tidal carbon dioxide tension was monitored continuously from the same catheter used for sampling the inhalant. Ventilation was adjusted to prevent the end-tidal carbon dioxide tension from becoming > 40 mm Hg; however, cats were able to override the ventilator, so spontaneous ventilation was possible and occurred in some cats. End-tidal samples were collected by hand sampling from a catheter placed in the endotracheal tube, with the end of the sampling catheter 1 cm from the end of the endotracheal tube, and isoflurane concentrations were measured by use of an infrared analyzer calibrated with known concentrations of isoflurane. After a 30-minute stabilization period, the following baseline measurements were obtained: arterial and mixed venous blood gas values and pH; PCV; total plasma protein, whole-blood lactate, and hemoglobin concentrations; MAP; core body temperature; CVP; pulmonary arterial occlusion pressure (PAOP); mean pulmonary arterial pressure (MPAP); and cardiac output. Hemoglobin concentration was measured by use of the cyanmethemoglobin method and a spectrophotometer. These values were used to calculate the following parameters:

\[
\text{SVRI} = \left(\frac{\text{MAP} - \text{CVP}}{\text{CI}}\right) \times 80 \text{ dynes} \cdot \text{sec} / \text{cm}^2 \cdot \text{m}^2,
\]

\[
\text{PVRI} = \left(\frac{\text{MPAP} - \text{PAOP}}{\text{CI}}\right) \times 80 \text{ dynes} \cdot \text{sec} / \text{cm}^2 \cdot \text{m}^2,
\]

\[
\text{CaO}_2 = \left(\frac{1.32 \times [\text{hemoglobin concentration} \times 10]}{[\text{arterial oxygen saturation of hemoglobin} / 100]}\right) + \left(\frac{0.0031}{[\text{Paco}_2]}\right) \text{ mL/L},
\]

\[
\text{CVO}_2 = \left(\frac{1.32 \times [\text{hemoglobin concentration}] \times [\text{mixed venous oxygen saturation of hemoglobin} / 100]}{[\text{arterial oxygen consumption of hemoglobin} / 100]}\right) + \left(\frac{0.0031}{[\text{Pvo}_2]}\right) \text{ mL/L},
\]

\[
\text{oxygen delivery} = \text{CaO}_2 \times \text{CI} / 1,000 \text{ mL/min/m}^2,
\]

\[
\text{oxygen consumption} = \text{CI} \times ([\text{CaO}_2 / 1,000] - \text{CVO}_2 / 1,000) \text{ mL/min/m}^2,
\]

\[
\text{oxygen utilization ratio} = \text{oxygen consumption/oxygen delivery},
\]

\[
\text{CI} = \frac{\text{Cardiac output} (\text{mL/min}) / (\text{1.000}^{0.21}) \times 10 / 10^3}{\text{mL/min/m}^2},
\]

\[
\text{SVI} = \left(\frac{\text{CI} / 1,000}{\text{heart rate}}\right) \text{heart rate mL/m}^2.
\]

The lowest infusion rate of the test drug was then started, this infusion was maintained for 20 minutes, and measurements were repeated. This process was repeated for the next 4 increasing doses. After the highest dose of drug was administered, the infusion was stopped and measurements were repeated 30, 60, and 90 minutes later. Catheters were removed, and vessels were closed with 6-0 or 7-0 polyglactin suture. At least 2 weeks were allowed between each experiment.

At each sampling point, arterial blood was collected and placed in tubes containing EDTA, which had been kept on ice. Plasma was immediately separated in a refrigerated centrifuge and frozen at 70°C. Plasma dopamine, epinephrine, and norepinephrine concentrations were measured by use of high-performance liquid chromatography with electrochemical detection. These concentrations were measured in 3 cats during administration of dopamine (samples not collected for the first cat) and 6 cats for each of the other treatments.

**Statistical analysis—Results**

Results within each drug treatment were analyzed by repeated-measures ANOVA followed by a Tukey-Kramer test to determine differences in responses with each dose. Differences among drugs were determined by a similar statistical approach comparing low, medium, high, and highest doses. Values of P < 0.05 were considered significant.

**Results**

Administration of dopamine caused a gradual increase in PCV, reaching a significant change from the baseline value during an infusion rate of 20 μg/kg/min (Table 1). There was a concomitant increase in the hemoglobin concentration, being significantly different.
from the baseline value during administration of dopamine at infusion rates of 10 and 20 μg/kg/min. This latter change contributed to the significant increase in CaO₂ and CVO₂ from baseline during infusion rates of 10 and 20 μg/kg/min because there was minimal change in Pao₂. Heart rate increased from an original value of 156 ± 16 beats/min to 242 ± 16 beats/min during the highest infusion rate of dopamine. The change in heart rate became significantly different from baseline during an infusion rate of 5 μg/kg/min. Cardiac index and oxygen consumption increased during administration of dopamine at infusion rates from 5 to 20 μg/kg/min; however, the oxygen utilization ratio and whole-blood lactate concentration did not change. Cardiac index and oxygen consumption increased during administration of dopamine at infusion rates from 5 to 20 μg/kg/min; however, the oxygen utilization ratio and whole-blood lactate concentration did not change. Cardiac index and oxygen consumption increased during administration of dopamine at infusion rates from 5 to 20 μg/kg/min; however, the oxygen utilization ratio and whole-blood lactate concentration did not change. The change in heart rate became significantly different from baseline during an infusion rate of 5 μg/kg/min. Cardiac index and oxygen consumption increased during administration of dopamine at infusion rates from 5 to 20 μg/kg/min; however, the oxygen utilization ratio and whole-blood lactate concentration did not change. Cardiac index and oxygen consumption increased during administration of dopamine at infusion rates from 5 to 20 μg/kg/min; however, the oxygen utilization ratio and whole-blood lactate concentration did not change.

The SVRI decreased and was significantly different from baseline during administration of dopamine at an infusion rate of 15 μg/kg/min. No significant changes in Pao₂, Paco₂, or pH were detected, although there was a gradual decrease in the bicarbonate concentration and an increase in base deficit that became significant 30 minutes after the end of the dopamine infusion. With the exception of these latter values, all other values had returned to baseline by 30 minutes after the end of the infusion. Measured concentrations of dopamine increased with increasing infusion rates; however, this was only significantly different from baseline during an infusion rate of 20 μg/kg/min (Table 2). In 4 of 5 cats, dopamine concentrations were decreased by 30 minutes after the infusion, whereas the concentration increased in 1 cat. Concentrations only decreased consistently in 1 cat from 30 to 90 minutes, whereas both increases and decreases were detected in the other cats. Concentrations of epinephrine and norepinephrine were less than the limit of detection in most of the samples.

Administration of dobutamine also increased PCV, being significantly different from the baseline value during an infusion rate of 5 μg/kg/min (Table 3). The hemoglobin concentration decreased during infusion rates of 10 and 15 μg/kg/min, and CaO₂ was increased at these infusion rates as well. Core body temperature

Table 1—Mean ± SD values for cardiopulmonary parameters in 5 isoflurane-anesthetized cats before and during IV infusion of incremental doses of dopamine and 30, 60, and 90 minutes after the end of the infusion.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Baseline</th>
<th>2.5</th>
<th>5</th>
<th>10</th>
<th>20</th>
<th>30</th>
<th>60</th>
<th>90</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCV (%)</td>
<td>29 ± 4</td>
<td>30 ± 5</td>
<td>32 ± 5</td>
<td>37 ± 5</td>
<td>37 ± 6</td>
<td>42 ± 10</td>
<td>32 ± 8</td>
<td>30 ± 7</td>
</tr>
<tr>
<td>Lactate (mM/L)</td>
<td>1.1 ± 0.4</td>
<td>1.0 ± 0.5</td>
<td>1.0 ± 0.4</td>
<td>1.0 ± 0.4</td>
<td>0.9 ± 0.2</td>
<td>1.3 ± 0.4</td>
<td>1.3 ± 0.3</td>
<td>1.3 ± 0.2</td>
</tr>
<tr>
<td>Temperature (°C)</td>
<td>37.8 ± 0.3</td>
<td>37.9 ± 0.2</td>
<td>38.1 ± 0.3</td>
<td>38.2 ± 0.2</td>
<td>38.3 ± 0.3</td>
<td>38.3 ± 0.3</td>
<td>38.3 ± 0.3</td>
<td>37.9 ± 0.4</td>
</tr>
<tr>
<td>pH</td>
<td>7.352 ± 0.038</td>
<td>7.381 ± 0.050</td>
<td>7.386 ± 0.052</td>
<td>7.347 ± 0.064</td>
<td>7.355 ± 0.089</td>
<td>7.377 ± 0.080</td>
<td>7.369 ± 0.056</td>
<td>7.352 ± 0.029</td>
</tr>
<tr>
<td>Pao₂ (mm Hg)</td>
<td>52.4 ± 5.8</td>
<td>53.3 ± 5.9</td>
<td>53.5 ± 5.8</td>
<td>53.7 ± 5.7</td>
<td>53.7 ± 5.6</td>
<td>54.0 ± 5.8</td>
<td>53.7 ± 5.6</td>
<td>53.1 ± 5.7</td>
</tr>
<tr>
<td>PaO₂ (mm Hg)</td>
<td>142.7 ± 19.2</td>
<td>143.3 ± 19.3</td>
<td>143.6 ± 19.3</td>
<td>144.8 ± 20.1</td>
<td>145.0 ± 20.3</td>
<td>145.2 ± 20.3</td>
<td>145.2 ± 20.3</td>
<td>145.2 ± 20.3</td>
</tr>
<tr>
<td>Pao₂ (mm Hg)</td>
<td>125.4 ± 25.5</td>
<td>126.3 ± 26.0</td>
<td>126.5 ± 26.0</td>
<td>126.6 ± 26.0</td>
<td>126.7 ± 26.0</td>
<td>126.8 ± 26.0</td>
<td>126.8 ± 26.0</td>
<td>126.8 ± 26.0</td>
</tr>
<tr>
<td>VO₂ (mL/min/m²)</td>
<td>3.1 ± 1.0</td>
<td>3.5 ± 0.9</td>
<td>3.6 ± 1.1</td>
<td>4.2 ± 1.2</td>
<td>3.8 ± 1.0</td>
<td>3.2 ± 1.5</td>
<td>2.8 ± 1.0</td>
<td>2.4 ± 0.9</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>156 ± 16</td>
<td>180 ± 19</td>
<td>193 ± 21</td>
<td>217 ± 17</td>
<td>234 ± 16</td>
<td>242 ± 16</td>
<td>169 ± 14</td>
<td>161 ± 15</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>68 ± 16</td>
<td>85 ± 19</td>
<td>94 ± 28</td>
<td>100 ± 22</td>
<td>105 ± 18</td>
<td>120 ± 31</td>
<td>89 ± 12</td>
<td>86 ± 12</td>
</tr>
<tr>
<td>CVP (mm Hg)</td>
<td>6 ± 2</td>
<td>6 ± 2</td>
<td>6 ± 2</td>
<td>6 ± 2</td>
<td>6 ± 2</td>
<td>6 ± 2</td>
<td>6 ± 2</td>
<td>6 ± 2</td>
</tr>
<tr>
<td>PAP (mm Hg)</td>
<td>16 ± 5</td>
<td>16 ± 5</td>
<td>16 ± 5</td>
<td>16 ± 5</td>
<td>16 ± 5</td>
<td>16 ± 5</td>
<td>16 ± 5</td>
<td>16 ± 5</td>
</tr>
<tr>
<td>CI (mL/min/m²)</td>
<td>1,202 ± 355</td>
<td>1,762 ± 379</td>
<td>2,056 ± 319</td>
<td>2,551 ± 562</td>
<td>2,703 ± 443</td>
<td>2,494 ± 150</td>
<td>1,262 ± 245</td>
<td>1,242 ± 212</td>
</tr>
<tr>
<td>SVI (mL/m²)</td>
<td>7.9 ± 2.9</td>
<td>9.9 ± 2.5</td>
<td>10.8 ± 2.4</td>
<td>11.8 ± 2.4</td>
<td>11.6 ± 2.6</td>
<td>10.4 ± 1.2</td>
<td>7.5 ± 2.7</td>
<td>7.8 ± 1.8</td>
</tr>
<tr>
<td>DO delivery</td>
<td>177 ± 350</td>
<td>254 ± 89</td>
<td>345 ± 93</td>
<td>478 ± 106</td>
<td>498 ± 99</td>
<td>472 ± 83</td>
<td>196 ± 41</td>
<td>178 ± 29</td>
</tr>
<tr>
<td>VO₂ (mL/min/kg/min)</td>
<td>18.8 ± 6.1</td>
<td>22.7 ± 4.8</td>
<td>24.7 ± 5.1</td>
<td>31.6 ± 4.9</td>
<td>34.0 ± 4.3</td>
<td>36.4 ± 12.7</td>
<td>26.1 ± 6.1</td>
<td>26.2 ± 2.9</td>
</tr>
<tr>
<td>Oxygen utilization ratio</td>
<td>0.126 ± 0.046</td>
<td>0.106 ± 0.049</td>
<td>0.082 ± 0.033</td>
<td>0.071 ± 0.025</td>
<td>0.071 ± 0.017</td>
<td>0.079 ± 0.031</td>
<td>0.136 ± 0.034</td>
<td>0.149 ± 0.020</td>
</tr>
<tr>
<td>SVRI</td>
<td>4,389 ± 1,884</td>
<td>3,689 ± 1,219</td>
<td>3,481 ± 1,183</td>
<td>3,167 ± 1,144</td>
<td>3,004 ± 623</td>
<td>3,700 ± 1,104</td>
<td>3,952 ± 833</td>
<td>4,033 ± 816</td>
</tr>
<tr>
<td>PVRI</td>
<td>431 ± 283</td>
<td>323 ± 228</td>
<td>395 ± 124</td>
<td>386 ± 110</td>
<td>414 ± 106</td>
<td>402 ± 123</td>
<td>449 ± 134</td>
<td>441 ± 119</td>
</tr>
</tbody>
</table>

aWithin a row, values with different superscript letters are significantly different (P < 0.05) from baseline values (a) and values obtained during administration of dopamine at infusion rates of 2.5 (b), 5 (c), 10 (d), 15 (e), and 20 (f) μg/kg/min.
was increased over the baseline value during an infusion rate of 10 μg/kg/min and remained greater than the baseline value throughout the infusion period and for 60 minutes after the infusion was discontinued. Heart rate increased significantly during the lowest infusion rate and reached a peak value of 235 ± 8 beats/min during the highest infusion rate. Cardiac index increased significantly during an infusion rate of 5 μg/kg/min; however, oxygen consumption did not change. The SVI was significantly different from the baseline value during administration of dobutamine at all infusion rates. The MAP increased in 3 cats by 20% during the lowest infusion rate of dobutamine; however, the MAP decreased in 2 cats. In one of those cats, MAP did not return to the baseline value even during the highest infusion rate of dobutamine. The overall increases in MAP were not significant. After the infusion was discontinued, the decrease in MAP was measured in 3 cats and the lowest MAP was 73 ± 20 mm Hg at 10.3 ± 1.2 minutes. The SVRI decreased from baseline and this was significant during the 3 highest infusion rates. Although changes in \( \text{PaCO}_2 \) were not significant, the gradual decrease in the values contributed to an increase in \( \text{pH} \), which became significant during infusion rates of 13 and 20 μg/kg/min. The concentration of bicarbonate decreased, whereas base deficit and lactate concentration increased. Concentrations of lactate and bicarbonate and pH and base deficit values were different from baseline values 30 minutes after the infusion was discontinued, where-

<table>
<thead>
<tr>
<th>Parameter Baseline 2.5 5 10 15 20 30 60 90*</th>
<th>Time after infusion (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dopamine concentrations (ng/mL)</td>
<td>0.61 ± 0.39 6.44 ± 5.38 14.27 ± 12.06 32.69 ± 20.80 42.91 ± 29.69 62.98 ± 59.68*</td>
</tr>
</tbody>
</table>

*Within a row, values with different superscript letters are significantly different from baseline values (a) and values obtained during administration of dopamine at an infusion rate of 2.5 μg/kg/min (b).

Table 3—Mean ± SD values for cardiothoracic parameters in 6 isoflurane-anesthetized cats before and during IV infusion of incrementnal doses of dobutamine and 30, 60, and 90 minutes after the end of the infusion.

<table>
<thead>
<tr>
<th>Parameter Baseline 2.5 5 10 15 20 30 60 90*</th>
<th>Time after infusion (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCV (%)</td>
<td>27 ± 3 33 ± 6* 38 ± 5* 39 ± 5* 41 ± 4* 39 ± 3*</td>
</tr>
<tr>
<td>Lactate (mmol/L)</td>
<td>1.0 ± 0.4 1.1 ± 0.4* 1.3 ± 0.4 1.5 ± 0.5 1.7 ± 0.5 2.0 ± 0.5*</td>
</tr>
<tr>
<td>Temperature (°C)</td>
<td>37.3 ± 0.3 37.8 ± 0.3 37.9 ± 0.3 38.0 ± 0.3* 38.0 ± 0.3 38.2 ± 0.3*</td>
</tr>
<tr>
<td>pH</td>
<td>7.375 ± 0.090 7.369 ± 0.082 7.387 ± 0.063 7.388 ± 0.074 7.391 ± 0.063* 7.392 ± 0.062*</td>
</tr>
<tr>
<td>( \text{Paco}_2 ) (mm Hg)</td>
<td>30.6 ± 7.5 35.8 ± 9.3 31.9 ± 6.9 30.5 ± 7.2 30.0 ± 6.4 28.3 ± 6.3</td>
</tr>
<tr>
<td>( \text{PaCO}_2 ) (mm Hg)</td>
<td>542.3 ± 36.5 558.9 ± 38.0 565.0 ± 25.0 568.9 ± 26.0 571.6 ± 24.2 566.5 ± 16.2</td>
</tr>
<tr>
<td>C(aO2) (mL/L)</td>
<td>134.4 ± 22.8 184.9 ± 31.4 177.5 ± 28.9 187.5 ± 27.6 193.3 ± 24.2 179.6 ± 32.0 149.6 ± 31.2 146.6 ± 18.4 148.6</td>
</tr>
<tr>
<td>( \text{PaCO}_2 ) (mm Hg)</td>
<td>63.8 ± 9.6 90.4 ± 22.9 109.8 ± 21.2 111.5 ± 16.6 111.3 ± 12.9 107.7 ± 15.6 61.7 ± 8.5* 60.2 5.2* 4.2</td>
</tr>
<tr>
<td>( \text{PaCO}_2 ) (mL/L)</td>
<td>118.7 ± 21.3 174.5 ± 32.1 170.6 ± 28.2 181.4 ± 27.1 187.1 ± 23.6 173.2 ± 30.1 129.3 ± 23.3 127.4 ± 14.9 128.7</td>
</tr>
<tr>
<td>Venous admixture (μL/dL)</td>
<td>2.9 ± 1.3 3.3 ± 1.4 5.5 ± 1.9 5.6 ± 1.4 5.5 ± 1.6 5.9 ± 2.3 2.4 ± 1.2* 2.2 ± 1.0 2.6</td>
</tr>
</tbody>
</table>

*Within a row, values with different superscript letters are significantly different from baseline values (a) and values obtained during administration of dopamine at an infusion rate of 2.5 μg/kg/min (b).

*Calculated by use of values from 4 cats. Samples in which the dopamine concentration was below the limit of detection were not used for these calculations.
as only base deficit and CVP values were different from baseline values 60 minutes after the infusion was discontinued. Unfortunately, data from only 1 cat was recorded 90 minutes after the infusion was discontinued; therefore, these data were not included in the analysis. Concentrations of dopamine, epinephrine, and norepinephrine were less than the limit of detection in most of the samples.

Administration of epinephrine increased PCV, hemoglobin concentration, and CaO₂ over baseline values at all infusion rates (Table 4). Core body temperature was increased over the baseline value at the highest infusion rate of epinephrine. Heart rate was increased at all infusion rates; however, the highest values were not as high as those detected during administration of dopamine and dobutamine. Cardiac index increased, with the change becoming significant at an infusion rate of 0.5 µg/kg/min. In 5 of 6 cats, the increase in cardiac output was > 30% at the lowest infusion rate, and the increase was > 30% in all cats at an infusion rate of 0.25 µg/kg/min. The SVI increased during all infusion rates of epinephrine, and infusion rates of 1 and 2 µg/kg/min caused a further increase in SVI over values obtained during the lowest infusion rate of 0.125 µg/kg/min. Oxygen consumption was increased significantly 30 minutes after the end of the epinephrine infusion but at no other time. The oxygen utilization ratio decreased during an infusion rate of 1 µg/kg/min and returned to baseline values by 30 minutes after the infusion. The MAP increased and was significantly different from the baseline value during administration of epinephrine at an infusion rate of 0.5 µg/kg/min. During the 2 lowest infusion rates of epinephrine, MAP increased by > 10% in 4 of 6 cats and increased by > 20% in all cats during an infusion rate of 0.5 µg/kg/min. After the infusion was discontinued, the decrease in MAP was measured in 4 cats and the lowest MAP was 44 ± 12 mm Hg at 3.8 ± 1 minutes. The SVRI did not change significantly. Mean pulmonary arterial pressure increased with the lowest infusion rate of epinephrine but had decreased to the baseline value by 30 minutes after the end of the infusion. The Paco₂ and PaO₂ did not change significantly, but pH was decreased by administration of epinephrine at infusion rates of 0.25 to 2 µg/kg/min. By 30 minutes after the end of the infusion, pH had increased so that it was no longer different from baseline values. The concentration of lactate increased and the concentration of bicarbonate decreased with the lowest infusion rate of epinephrine. Basic deficit increased during administration of epinephrine at an infusion rate of 0.125 µg/kg/min and remained significantly different from the baseline value 90 minutes after the end of the infusion. Sixty and 90 minutes after the end of the infusion, the base deficit had decreased significantly from the peak values detected during the infusion.

Table 4—Mean ± SD values for cardiopulmonary parameters in 6 isoflurane-anesthetized cats before and during IV infusion of incremental doses of epinephrine and 30, 60, and 90 minutes after the end of the infusion.

| Parameters | Baseline | 0.125 | 0.25 | 0.5 | 1.0 | 2.0 | 30 | 60 | 90*
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</thead>
<tbody>
<tr>
<td>PCV (%)</td>
<td>26 ± 3</td>
<td>26 ± 3</td>
<td>26 ± 3</td>
<td>26 ± 3</td>
<td>26 ± 3</td>
<td>26 ± 3</td>
<td>26 ± 3</td>
<td>26 ± 3</td>
<td>26 ± 3</td>
</tr>
<tr>
<td>Lactate (mmol/L)</td>
<td>1.0 ± 0.5</td>
<td>1.0 ± 0.5</td>
<td>1.0 ± 0.5</td>
<td>1.0 ± 0.5</td>
<td>1.0 ± 0.5</td>
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<tr>
<td>Hb (g/dL)</td>
<td>10.6 ± 1.4</td>
<td>10.6 ± 1.4</td>
<td>10.6 ± 1.4</td>
<td>10.6 ± 1.4</td>
<td>10.6 ± 1.4</td>
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<tr>
<td>Temperature (°C)</td>
<td>37.8 ± 0.3</td>
<td>37.8 ± 0.3</td>
<td>37.8 ± 0.3</td>
<td>37.8 ± 0.3</td>
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<tr>
<td>pH</td>
<td>7.395 ± 0.059</td>
<td>7.395 ± 0.059</td>
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<tr>
<td>Paco₂ (mm Hg)</td>
<td>36.0 ± 5.1</td>
<td>36.0 ± 5.1</td>
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<td>36.0 ± 5.1</td>
<td>36.0 ± 5.1</td>
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<tr>
<td>PaO₂ (mm Hg)</td>
<td>422.1 ± 154.6</td>
<td>422.1 ± 154.6</td>
<td>422.1 ± 154.6</td>
<td>422.1 ± 154.6</td>
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<tr>
<td>Cao₂ (mmol/L)</td>
<td>141.6 ± 19.7</td>
<td>141.6 ± 19.7</td>
<td>141.6 ± 19.7</td>
<td>141.6 ± 19.7</td>
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<tr>
<td>CVO₂ (mL/L)</td>
<td>124.9 ± 17.9</td>
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<td>124.9 ± 17.9</td>
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<tr>
<td>SVRI (dynes·cm⁻⁵)</td>
<td>5 83</td>
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<tr>
<td>VO₂ (mL/min/m²)</td>
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<td>19.6</td>
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<td>PAOP (mm Hg)</td>
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<td>9</td>
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<td>9</td>
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<tr>
<td>CI (mL/min/m²)</td>
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<td>1,221</td>
<td>1,221</td>
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<td>1,221</td>
<td>1,221</td>
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<tr>
<td>SV (mL/m²)</td>
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<td>7.4</td>
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<tr>
<td>O₂ delivery (mL/min/m²)</td>
<td>173</td>
<td>173</td>
<td>173</td>
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<td>173</td>
<td>173</td>
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<tr>
<td>O₂ utilization ratio</td>
<td>0.017 ± 0.003</td>
<td>0.017 ± 0.003</td>
<td>0.017 ± 0.003</td>
<td>0.017 ± 0.003</td>
<td>0.017 ± 0.003</td>
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<td>0.017 ± 0.003</td>
</tr>
<tr>
<td>SVRI (dynes·cm⁻⁵)</td>
<td>3,990</td>
<td>3,990</td>
<td>3,990</td>
<td>3,990</td>
<td>3,990</td>
<td>3,990</td>
<td>3,990</td>
<td>3,990</td>
<td>3,990</td>
</tr>
<tr>
<td>PVRI (dynes·cm⁻⁵)</td>
<td>452</td>
<td>452</td>
<td>452</td>
<td>452</td>
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*Within a row, values with different superscript letters are significantly (P < 0.05) different from baseline values (a) and values obtained during administration of epinephrine at infusion rates of 0.125 (b), 0.25 (c), 0.5 (d), 1.0 (e), and 2.0 (f) µg/kg/min.

*Calculated by use of values from 4 or 5 cats.

See Table 1 for key.
Concentrations of epinephrine increased with increasing infusion rates and decreased rapidly after the end of the infusion (Table 5). Concentrations of dopamine and norepinephrine were less than the limit of detection in most of the samples.

Administration of phenylephrine during the 2 highest infusion rates increased the PCV above baseline and increased the hemoglobin concentration during the highest infusion rate (Table 6). Heart rate was not altered significantly; however, the CI and SVI were significantly increased from baseline during the highest infusion rate of phenylephrine. There were significant changes in oxygen consumption, but no specific doses or times were significantly different. Oxygen utilization ratios did not change significantly. The MAP was increased from baseline during administration of phenylephrine at infusion rates of 1 and 2 μg/kg/min, and pulmonary arterial pressure was increased during infusion rates of 0.5 and 2 μg/kg/min. After the infusion was discontinued, the decrease in MAP was measured in 5 cats and the lowest MAP was 92 ± 19 mm Hg at 23.6 ± 4 minutes. The SVRI was also increased from baseline during administration of phenylephrine at infusion rates of 1 and 2 μg/kg/min, and there were significant changes in pulmonary vascular resistance; however, no specific values were significantly different. The pH, PaCO₂, and PaO₂ did not change significantly. The concentration of bicarbonate decreased and base deficit increased over time, with significant changes from baseline values detected 30 and 60 minutes after the end of the infusion. By 90 minutes, the change in the bicarbonate concentration was still significant;
however, the base deficit was not significantly different from the baseline value. All cardiovascular variables had returned to baseline values by 30 minutes after the end of the infusion. Concentrations of dopamine, epinephrine, and norepinephrine were less than the limit of detection in most of the samples.

Few arrhythmias were detected during the study. Two cats had a few premature ventricular contractions during administration of dobutamine, epinephrine, and phenylephrine but not dopamine. One cat had premature ventricular contractions during administration of dopamine at an infusion rate of 2.5 μg/kg/min but not at higher infusion rates, and another cat had arrhythmias during the baseline period and during administration of phenylephrine at an infusion rate of 0.5 μg/kg/min.

**Discussion**

Cats have been used as a model for studying the release of catecholamines and the mechanism of action of catecholamines; however, no published studies contain the full spectrum of cardiopulmonary effects of these catecholamines in cats anesthetized with inhalants. Doses of catecholamines used in our study were based on doses used clinically and are probably not in the range of a maximal effect. In a study in dogs anesthetized with pentobarbital, the maximum effect of dopamine was determined during an infusion rate of approximately 40 μg/kg/min, whereas dobutamine had not reached a plateau by an infusion rate of 160 μg/kg/min. In the study reported here, the peak effect on cardiac output occurred during an infusion rate of approximately 15 μg/kg/min; however, this could not be confirmed without further study of higher infusion rates.

Studies characterizing the effects of catecholamines in cats have been performed. In 1 study, dopamine caused a dose-dependent increase in MAP in cats anesthetized with α-chloralose; however, the purpose of the study was to evaluate the effect on renal function; therefore, no other measurements of cardiopulmonary function were made. In 5 cats anesthetized with isoflurane and breathing spontaneously, administration of dopamine significantly increased heart rate, blood pressure, and myocardial contractility during the highest dose (10 μg/kg/min) tested. In that study, myocardial contractility was measured by use of left ventricular pressure/time integration, whereas in the study reported here, an increase in myocardial contractility is inferred from an increase in stroke volume in the face of an increasing heart rate, with a significant increase occurring during administration of dopamine at an infusion rate of 5 μg/kg/min. In our study, dopamine did not cause an increase in systemic vascular resistance; in fact, during an infusion rate of 15 μg/kg/min, systemic vascular resistance was significantly lower than the baseline value. This suggested that, in cats, the vasoconstrictive properties of dopamine are not detected until the administration of doses higher than those used in our study. The lowest dose of dopamine used in our study did not cause significant cardiopulmonary changes; however, changes in oxygen delivery (increases in hemoglobin concentration, oxygen content, and cardiac output) and contractility became evident during an infusion rate of 5 μg/kg/min. Clinically, an MAP of 70 mm Hg is usually regarded as the lowest acceptable value during anesthesia, and a dose of 10 μg/kg/min was needed to exceed this value in all 6 cats. Cardiac index had increased by 65% for all cats at this dose. Part of the action of dopamine is thought to be that it causes an increase in the release of epinephrine from the adrenal gland; however, in the study reported here, no apparent increase in epinephrine concentrations was detected during dopamine infusion.

The concentration of dopamine increased with each successive increase in infusion rate, but although there was an initial rapid decrease in concentration, the values remained within the range detected with infusion rates of 10 and 15 μg/kg/min for the full 90-minute observation period after the end of the infusion. The pharmacokinetics of dopamine have not been studied in cats; however, in humans, the decrease in plasma concentrations of dopamine is initially rapid, but increased concentrations have been measured for as much as 24 hours after the end of an infusion. In humans, it is also recognized that plasma concentrations of dopamine are highly variable when using infusion rates based on body weight. Measured concentrations from the same infusion rate in healthy males varied by a factor of 75. In the study reported here, there was a 17-fold difference between concentrations measured in 2 cats during an infusion rate of 5 μg/kg/min. The highest dose (10 μg/kg/min) was needed to exceed this value in all 6 cats. Cardiac index had increased by 65% for all cats at this dose. Part of the action of dopamine is thought to be that it causes an increase in the release of epinephrine from the adrenal gland; however, in the study reported here, no apparent increase in epinephrine concentrations was detected during dopamine infusion.

Dobutamine has been used in a number of studies, and in 2 studies, dobutamine was used in cats anesthetized with inhalants. In cats anesthetized with pentobarbital, dobutamine increased contractility without increasing heart rate or blood pressure. This ability of dobutamine to separate the chronotropic and inotropic actions was ascribed to dobutamine's stimulation of α-adrenergic receptors (effect abolished by phentolamine). In another study of bilaterally vagotomized cats anesthetized with pentobarbital, dobutamine induced a dose-dependent increase in systolic blood pressure, which was reduced by propranolol. In a study in which halothane was used, dobutamine was used to reverse the cardiovascular effects of thiocarbamidation induced by epidural administration of lidocaine. In that study, dobutamine was administered at a fixed dose rate and MAP was used as the measure of cardiovascular function. In another study, dobutamine was tested in cats that had been deprived of taurine and it did not increase myocardial contractility in the taurine-deficient cats but it did in clinically normal cats. In that study, the effect of dobutamine on cardiac output, oxygen delivery, and oxygen consumption was not examined. Results of our study indicated that dobutamine increased heart rate and CI in a dose-dependent manner and the effect on cardiac output and contractility appeared to be greater than for dopamine or epinephrine at the doses used. Systemic vascular resistance decreased, as expected with this drug, as it has both β-2 adrenergic receptor agonist and α-1 adrenergic receptor agonist and antagonist effects. This decrease in systemic vascular resistance
contribute to the lack of a significant effect on MAP despite the increase in CI; however, all cats had an MAP > 70 mm Hg during administration of dobutamine at an infusion rate of 5 μg/kg/min.

Natural release of epinephrine in cats undergoing rapid blood loss during α-chloralose anesthesia attained plasma concentrations of 1.83 ± 0.27 ng/mL. This value is similar to the value measured during administration of the lowest dose of epinephrine used in our study. In 1 study, epinephrine was given at doses similar to those used in our study and there was a dose-dependent increase in arterial pressure and changes in portal pressure and hepatic blood volume. However, that study was performed in cats anesthetized with pentobarbital, and no measurements of cardiac output, oxygen extraction, and oxygen consumption were made. In a study in cats anesthetized with α-chloralose and pentobarbital, the effects of epinephrine on heart rate, MAP, and cardiac output were measured; however, cats had been vagotomized first, thus blunting the normal pressor reflexes present in an intact animal. With this anesthetic technique, baseline arterial pressures and cardiac output were higher than those recorded in our study; however, despite these differences in study conditions, the increase in heart rate during administration of epinephrine at an infusion rate of 1 μg/kg/min in that study was similar to that detected in our study. Epinephrine had a greater effect on MAP than dopamine or dobutamine because it did not significantly alter systemic vascular resistance. However, this effect came at a metabolic cost, with an increase in lactate concentration and a progressive metabolic acidosis that was more severe than that caused by administration of dopamine or dobutamine.

Phenylephrine is a synthetic noncatecholamine, although its chemical structure is similar to that of epinephrine. It has mostly α-1 adrenergic receptor agonist properties and a short plasma half-life. Phenylephrine is usually used to raise systemic blood pressure without increasing myocardial contractility. Norepinephrine has potent α-1 adrenergic receptor agonist properties but also has β-1 activity in the range of doses considered clinically normal; therefore, it is more likely to increase myocardial contractility than phenylephrine. In the study reported here, phenylephrine caused a significant increase in MAP during an infusion rate of 1 μg/kg/min with an associated increase in systemic vascular resistance and no change in cardiac output. During an infusion rate of 2 μg/kg/min, CI also increased and this was associated with an increased SVI, suggesting the onset of a positive inotropic action at this dose. This was probably mediated via cardiac α-1 adrenergic receptors, which have a positive inotropic effect in cats. In dogs, it is recognized that α-1 adrenergic receptor agonism does not normally have a positive inotropic effect at body temperatures considered normal and that high doses of phenylephrine are needed in dogs to detect this effect. In our study, pulmonary arterial pressure was significantly increased during administration of phenylephrine at an infusion rate of 0.5 μg/kg/min. This finding is similar to that in some people in which phenylephrine increased pulmonic vascular resistance more than norepinephrine at doses inducing a similar increase in systemic blood pressure. In our study, oxygen delivery was also increased during the highest dose of phenylephrine, which is attributable to the increases in hemoglobin concentration and CI.

After the infusions were terminated, cats became hypotensive after infusion of dopamine or epinephrine but not with dobutamine or phenylephrine. These differences were likely attributable to the rapid metabolism of dopamine and epinephrine. These results suggested that abrupt termination of dopamine or epinephrine administration may result in hypotension, and although not tested in our study, the authors have found that this may be avoided by reducing the infusion rate in a stepwise fashion.

Catecholamines have a thermogenic action. In a study in dogs, administration of dopamine and epinephrine induced an increase in body temperature but dobutamine did not. In that study, administration of all 3 catecholamines resulted in an increase in oxygen consumption. In the study reported here, administration of dobutamine and epinephrine was associated with significant increases in body temperature; however, maintenance of body temperature within a narrow range was part of the experimental protocol, thus mitigating the direct effect of the drugs. Oxygen consumption increased significantly during administration of dopamine; however, administration of epinephrine resulted in an ongoing increase, such that the significant change was measured 30 minutes after the end of the infusion. Administration of phenylephrine also induced significant changes in oxygen consumption. In studies in dogs, oxygen consumption increased during administration of dopamine, dobutamine, epinephrine, and norepinephrine at doses similar to those used in our study.

Hemoglobin concentrations increased during all infusions. Thejection of RBCs from the spleen is thought to be mediated by α-1 adrenergic receptors; therefore, increased hemoglobin concentrations would be expected during administration of phenylephrine and epinephrine. In dogs, administration of dopamine or dobutamine did not increase hemoglobin concentrations at doses ranging from 5 to 160 μg/kg/min. In our study, mean initial hemoglobin (14.1 ± 1.2 g/dL) and Hct (44.8 ± 3.2%) values, obtained from analysis of jugular venous blood from cats before the first experiment, were within reference ranges used at the veterinary medical teaching hospital (hemoglobin, 9 to 15.1 g/dL; Hct, 29% to 48%). During anesthesia, the measured hemoglobin values decreased under the influence of isoflurane, a phenomenon that is thought to result from an increase in blood volume. It is hypothesized that the decrease in hydrostatic pressure associated with anesthesia results in a net gain of fluid from the periphery with a concomitant reduction in Hct. It is unlikely that this was solely because of hemodilution with administered fluids because the lowest values were recorded at control points when cats had received 8.1 ± 2.9 mL/kg of lactated Ringer's solution. The increase in cardiac output and blood pressure associated with these catecholamine treatments is expected to restore the normal balance of
Starling forces, resulting in a return of blood volume towards normal.

Therefore, it would appear that dopamine is likely to be the most useful drug in clinical anesthesia in which the first aim is to increase blood pressure and perfusion. Results of the study reported here indicated that dopamine needed to be titrated to effect because there is variation in individual plasma concentrations. Dobutamine is at least as effective at increasing blood flow, but because it tends to decrease systemic vascular resistance, it is likely to have less effect on blood pressure. This is an important distinction in a clinical setting in which blood pressure is easy to measure and there is no simple method of measuring blood flow. Epinephrine is at least as effective as dopamine but has the negative effects of increasing lactic acid production and increasing base deficit. Phenylephrine is normally used in cases in which it is advantageous to increase blood pressure by vasoconstriction. This may be useful in animals with pronounced systemic vasodilation, such as in visceral vasoconstriction. This may be useful in animals with inflammation. It may also be used to increase blood pressure in cases in which increases in myocardial contractility may be disadvantageous, such as in hypotrophic cardiomyopathy. None of the infusions used in our study in healthy cats induced serious arrhythmias. Results of our study bore out our original hypothesis, except that phenylephrine, at the highest dose, increased cardiac output.

References