Hypotension during anesthesia has been reported in both humans and other animals. Factors such as dehydration, hemorrhage, and concurrent diseases are common causes of hypotension in anesthetized patients and can be further exacerbated by the negative cardiovascular effects of anesthetic agents. Isoflurane can cause dose-dependent decreases in systemic vascular resistance and blood pressure with minimal depression of cardiac function and heart rate. An MAP < 60 mm Hg and corresponding SAP < 80 to 90 mm Hg typically are interpreted as hypotension in anesthetized mammals. In contrast, reference ranges for SAP, DAP, and MAP in conscious animals are approximately 100 to 140, 60 to 100, and 80 to 90 mm Hg, respectively. It is accepted that vital organs, such as the brain and kidneys, are unable to adequately regulate blood flow when MAP is < 60 mm Hg. It has been speculated that organ blood flow decreases proportionally with decreases in blood pressure, with the extent of subsequent organ damage related to the duration of sustained hypotension. Rapid heart rates in combination with relatively large heart sizes and differences in cardiac anatomy contribute to higher cardiac outputs in birds than in mammals. In addition, birds have a lower total peripheral resistance and a higher arterial pressure than mammals to allow for an adequate cardiac output to meet required metabolic needs. Multiple studies have found that blood pressure is higher in Psittaci-
formes, Gruiformes, Accipitriformes, Falconiformes, and Strigiformes than in mammals and other avian species. In a study on conscious great horned owls, investigators reported a mean ± SD SAP of 232 ± 37 mm Hg, DAP of 178 ± 28 mm Hg, and MAP of 203 ± 28 mm Hg, which are substantially different than those reported for healthy conscious mammals. Hypotension is a commonly reported complication in small and large animals during general anesthesia. Management strategies are intended to reduce the depth of anesthesia and optimize cardiac output through treatment of arrhythmias, replacement of volume deficits, and administration of positive inotropic drugs such as dopamine hydrochloride and dobutamine hydrochloride.

Dopamine is an endogenous catecholamine that exerts its effects on the cardiovascular system through stimulation of dopaminergic, α-adrenergic, and β-adrenergic receptors. Dopamine has the ability to induce vasodilation via dopaminergic receptors as well as vasoconstriction via α-adrenergic receptors. It improves cardiac contractility via myocardial β-adrenergic receptors, which may lead to an increase in cardiac output to improve oxygen delivery to the body. The overall effect of dopamine on the cardiovascular system is determined by many factors, both intrinsic and extrinsic. Different doses induce differences in responses because the drug’s affinity for each receptor is affected by the plasma concentration. Because dopamine is rapidly metabolized (half-life, 2 minutes), it must be administered as a CRI to be effective. Dobutamine is a synthetic sympathomimetic catecholamine derived from isoproterenol. It acts directly as a β-adrenergic agonist, but it also exerts weak effects through β-receptors and potentially α-adrenergic receptors. Dobutamine exerts beneficial effects through its positive inotropic and chronotropic activity via its action on β-adrenergic receptors. At lower doses, dobutamine can improve contractility, stroke volume, and cardiac output without increasing heart rate. Higher infusion rates may result in tachycardia, vasoconstriction, and hypertension with the potential for arrhythmias. Similar to dopamine, the half-life of dobutamine is relatively short (2 to 3 minutes), and dobutamine must be administered as a CRI to be effective.

In 1969, investigators detected α-adrenergic and β-adrenergic receptors in Pekin ducks (Anas platyrhynchos domesticus), which suggested that other avian species may also have these receptors. The authors of that study also determined that norepinephrine and epinephrine are present in avian plasma and have similar cardiac effects, such as an increase in the rate of depolarization of pacemaker cells and myocardial contraction. In another study performed on anesthetized red-tail hawks (Buteo jamaicensis), investigators found that administration of norepinephrine at a rate of 0.4 to 5 μg/kg/min could cause systemic hypertension. To the authors’ knowledge, the effects of dopamine and dobutamine have not been evaluated in psittacine birds. Therefore, the objective of the study reported here was to determine the effects of dopamine and dobutamine for the treatment of isoflurane-induced hypotension in psittacine birds. Our goals were to establish an appropriate dose for these agents and to determine the dose that would be most effective in healthy parrots anesthetized with isoflurane. We hypothesized that birds would become hypotensive when administered isoflurane at a concentration of 2.5% and that dopamine and dobutamine would significantly increase arterial blood pressure and correct the hypotension.

Materials and Methods

Animals—Twelve adult Hispaniolan Amazon parrots (Amazona ventralis) with a mean body weight of 0.267 kg (range, 0.252 to 0.295 kg) were obtained from a colony maintained at Louisiana State University. Tap water and food were available ad libitum. Birds were considered healthy on the basis of medical history and results of physical examination and a CBC. Transeptolic cardiac ultrasonography was performed on the parrots of this colony as part of another research project and did not reveal any abnormalities. The protocol for the study was approved by the Louisiana State University Institutional Animal Care and Use Committee.

Anesthesia and instrumentation—Anesthesia was induced in all birds with 5% isoflurane in 100% oxygen delivered via a face mask at a flow rate of 1.5 L/min. Each bird was intubated with an uncuffed endotracheal tube (internal diameter, 3 mm), and anesthesia was maintained by administration of isoflurane (end-tidal expiratory concentration, 2%) via a Bain circuit with a flow rate of 200 mL/kg/min. A circulating hot water blanket and forced warm air blanket were used to provide supplemental heat for the duration of the anesthetic period. Heart rate, PETCO₂, esophageal temperature, respiratory rate, a lead II ECG, end-tidal expiratory concentration of isoflurane, and inspiratory fraction of oxygen were monitored by use of a multifunction monitor. An esophageal temperature probe was inserted until the tip was at the level of the heart; temperature was maintained between 37° and 41°C. Birds were ventilated with the aid of a mechanical ventilator set to deliver 8 breaths/min with a peak inspiratory pressure of 10 to 15 cm H₂O and a tidal volume of 10 mL/kg to ensure a stable PETCO₂ between 35 and 45 mm Hg throughout the procedures.

The medial aspect of the distal portion of a wing was aseptically prepared, and a 26-gauge, 19-mm catheter was placed percutaneously in the deep radial artery. The catheter was secured with tissue glue and a clear acrylic dressing. The arterial catheter was connected to a continuous multifunction monitor via a disposable pressure transducer system. The system was calibrated in accordance with the manufacturer’s recommendations. The system was flushed with saline (0.9% NaCl) solution, and the pressure transducer was calibrated with a zero value at the level of the right atrium. Blood pressure was assessed for stability and a consistent waveform. A second 24- to 26-gauge, 19-mm catheter was placed in the basilic vein of the contralateral wing for drug infusion. Throughout the anesthetic period, each bird was administered lactated Ringer’s solution at a rate of 10 mL/kg/h via an infusion pump to simulate standard surgical protocol while anesthetized.
Experimental design—The study was conducted in accordance with a randomized crossover design. The order of drug administration and the doses administered were determined for the birds by use of computer software. Each anesthetized bird received 3 doses of each drug during a treatment period of 20 min/dose. Treatments were CRIs of dobutamine* (5, 10, and 15 µg/kg/min) and dopamine* (5, 7, and 10 µg/kg/min). Doses for dobutamine and dopamine were extrapolated from doses recommended in small and large animal medicine. New bottles of dopamine and dobutamine were used on each day of the experiment. All drugs were diluted with sterile saline solution to the same concentration by the same investigator. Dopamine was diluted to a concentration of 0.04 mg/mL, and dobutamine was diluted to a concentration of 0.125 mg/mL. The volume of each drug administered was subtracted from the total amount of lactated Ringer’s solution infused each hour to ensure that birds received the same amount of fluids throughout the study. Birds were monitored for severe tachycardia, arrhythmias, and changes in blood pressure.

After the drug and order of doses was assigned for a specific anesthetized bird, isoflurane was increased to an end-tidal concentration of 2.5%. After the end-tidal concentration of isoflurane was stable at a concentration of 2.5%, after the end-tidal concentration of isoflurane was stable at a concentration of 2.5% for 10 minutes, a CRI of dobutamine or dopamine was initiated (time 0). Each CRI was continued for 20 minutes. At that time, the CRI was discontinued, and arterial pressure was allowed to equilibrate for 10 minutes; this 10-minute period also provided an appropriate amount of time for drug elimination to minimize carryover effects of the preceding infusion. This procedure was repeated until all 3 doses of the drug were administered. After a washout period of 20 minutes, the procedures were repeated for the other drug. The catheters were then removed from the artery and vein, and the birds were allowed to recover from anesthesia. All birds were closely monitored until they were able to perch without assistance. Total duration of anesthesia was 3 to 4 hours.

Data collection—Variables were recorded throughout drug administration. The $P_{ETCO_2}$, inspired and expired concentrations of isoflurane, heart rate, esophageal temperature, respiratory rate, inspiratory fraction of oxygen, and direct blood pressure measurements (SAP, DAP, and MAP) were recorded every minute by use of a multifunction monitor.

Statistical analysis—Distribution of data for outcome variables (SAP, DAP, MAP, heart rate, $P_{ETCO_2}$, and esophageal temperature) was tested for normality by use of a Shapiro-Wilk test; data distribution was also assessed for skewness, kurtosis, and normal quantile plots. Analysis revealed that 20% of the data were not normally distributed over the various time points. Therefore, data were logarithmically transformed, which resulted in a normal distribution. Raw data and logarithmically transformed data were analyzed via a repeated-measures ANOVA with a mixed linear model.*

### Table 1—Effects of dopamine and dobutamine on SAP, MAP, and DAP in 8 isoflurane-anesthetized Hispaniolan Amazon parrots (Amazona ventralis).

<table>
<thead>
<tr>
<th>Blood pressure</th>
<th>Treatment (µg/kg/min)*</th>
<th>Time of significant change (min)†</th>
<th>Change from 0 to 10 minutes (mm Hg)‡</th>
<th>Mean ± SD</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAP Dopamine</td>
<td>5</td>
<td>7</td>
<td>32.1 ± 10.7</td>
<td>11.1–53.1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>7</td>
<td>32.1 ± 10.7</td>
<td>11.1–53.1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>4</td>
<td>65.0 ± 10.7</td>
<td>51.0–93.0</td>
<td></td>
</tr>
<tr>
<td>Dobutamine</td>
<td>5</td>
<td>4</td>
<td>43.9 ± 10.7</td>
<td>22.9–91.3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>4</td>
<td>51.0 ± 10.7</td>
<td>30.0–72.0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>15</td>
<td>4</td>
<td>52.9 ± 10.7</td>
<td>31.6–73.6</td>
<td></td>
</tr>
<tr>
<td>MAP Dopamine</td>
<td>5</td>
<td>6</td>
<td>29.9 ± 8.8</td>
<td>12.5–47.2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>5</td>
<td>45.1 ± 8.8</td>
<td>27.8–62.5</td>
<td></td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>4</td>
<td>58.0 ± 8.8</td>
<td>40.6–75.4</td>
<td></td>
</tr>
<tr>
<td>Dobutamine</td>
<td>5</td>
<td>3</td>
<td>18.0 ± 8.8</td>
<td>9.4–51.4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>4</td>
<td>42.0 ± 8.8</td>
<td>24.8–59.4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>15</td>
<td>5</td>
<td>44.2 ± 8.8</td>
<td>26.9–61.6</td>
<td></td>
</tr>
<tr>
<td>DAP Dopamine</td>
<td>5</td>
<td>6</td>
<td>28.1 ± 8.8</td>
<td>10.9–45.4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>6</td>
<td>39.1 ± 8.8</td>
<td>21.9–56.4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>4</td>
<td>49.1 ± 8.8</td>
<td>31.9–66.4</td>
<td></td>
</tr>
<tr>
<td>Dobutamine</td>
<td>5</td>
<td>4</td>
<td>31.4 ± 8.8</td>
<td>14.1–48.6</td>
<td></td>
</tr>
<tr>
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<td>35.4 ± 8.8</td>
<td>18.1–52.6</td>
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</tr>
<tr>
<td></td>
<td>15</td>
<td>5</td>
<td>37.5 ± 8.8</td>
<td>20.2–54.7</td>
<td></td>
</tr>
</tbody>
</table>

*Each anesthetized parrot was treated with 3 doses of dopamine (CRI at 5, 7, and 10 µg/kg/min) and 3 doses of dobutamine (CRI at 5, 10, and 15 µg/kg/min). Each CRI was administered for 20 min; the CRI then was discontinued, and arterial blood pressure was allowed to equilibrate for 10 minutes before measurement. Time 0 was the initiation of a CRI. First time at which the change in blood pressure differed significantly (P ≤ 0.05) from the value at time 0. †Represents the value calculated for the difference between blood pressures at 0 and 10 minutes; the 10-minute time point was chosen because it appeared to be the time of maximum effects of the drugs for all treatments.
The power for the ANOVA was > 0.99. A compound symmetry covariance was used because it provided the best fit to the data according to the covariance matrix estimates. Thus, the variances were also considered homogenous among treatments. The specific hypothesis that dopamine and dobutamine had different effects was also tested by use of contrast statements. Post hoc multiple comparisons and difference estimates from time 0 (initiation of drug administration) were obtained with a least squares means method. A Tukey adjustment for multiple comparisons was used. We also calculated 95% confidence intervals for the difference estimates.

Results

Four of the 12 parrots were removed from the study because they developed marked second-degree atrioventricular block during the administration of dobutamine at a CRI of 15 µg/kg/min. The heart rates of these 4 birds were similar to those of the 8 other birds that received dobutamine at the CRI of 15 µg/kg/min. However, when the atrioventricular block occurred, the blood pressure dramatically decreased. The CRI of dobutamine was discontinued at that point, and within a few minutes, the birds returned to a normal cardiac rhythm. Data for these 4 parrots were excluded from analysis.

Because results for the repeated-measures ANOVA for raw (nontransformed) and logarithmically transformed data yielded identical results and ANOVAs are relatively robust for data with a mild departure from a normal distribution, only results of the analysis for the nontransformed data were reported. Similarly, plots for the nontransformed and logarithmically transformed data were identical.

Results

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Mean ± SD SAP, MAP, and DAP at time 0 were 132.9 ± 22.1 mm Hg, 116.9 ± 20.5 mm Hg, and 101.9 ± 22.0 mm Hg, respectively. Analysis via the repeated-measures ANOVA on the outcome variables did not reveal significant time-by-treatment interactions. There were significant (P < 0.001) effects of time and treatment on SAP, DAP, MAP, and PETCO₂. There was a significant (P < 0.001) effect of treatment but not of time on heart rate (P = 0.101) and esophageal temperature (P = 1.000). Overall, dopamine induced significantly (P < 0.001) higher values than did dobutamine for all variables, except for PETCO₂ (P = 0.056).
For direct arterial blood pressure measurements, changes from time 0 were significant 4 to 7 minutes after initiation of the CRI (Table 1). Multiple comparisons procedures revealed that dopamine at 7 and 10 µg/kg/min and dobutamine at 15 µg/kg/min had the greatest effects on blood pressure (Figure 1). Treatment effects were also plotted for heart rate (Figure 2). Treatment caused a significant change in blood pressure at 10 minutes, compared with the value at time 0. Effects of all treatments appeared to reach a plateau at 10 minutes after initiation of a CRI.

**Discussion**

Analysis of results of the study reported here indicated that the use of dopamine and dobutamine at all doses caused an increase in SAP, DAP, and MAP in Hispaniolan Amazon parrots during anesthesia maintained by administration of 2.5% isoflurane. Infusion of dopamine at 7 and 10 µg/kg/min and dobutamine at 5, 10, and 15 µg/kg/min increased blood pressure to MAP values previously reported in these birds. Blood pressures differed significantly between drugs and doses between 3 and 7 minutes after initiation of a CRI. At 10 minutes after initiation of a CRI, the effect of the treatments started to plateau. Dopamine at 10 µg/kg/min induced the greatest effect on SAP, DAP, and MAP, whereas dopamine at 5 µg/kg/min had the least effect on blood pressures.

Isoflurane is a vasodilator; however, the mechanism of isoflurane-induced vasodilation is not definitively known but is believed to be through isoflurane’s interaction with nitric oxide. The activity of the nitric oxide pathway is regulated by agonist-induced influx of calcium rather than calcium release from internal stores. A number of studies have been conducted on the effects of volatile anesthetics on cytoplasmic calcium concentration signaling in mammalian endothelial cells. The results of 1 study indicated that isoflurane caused a dose-dependent inhibition of calcium entry into cells. Isoflurane decreases arterial blood pressure and systemic vascular resistance during moderate and deep planes of anesthesia. In adult humans administered isoflurane at 1.5 times MAC, systemic vascular resistance and arterial blood pressure decreased to values 60% to 70% of those obtained in awake patients.

In Amazon parrots, the MAC of isoflurane is 1.49%, with surgical MAC defined as a concentration 1.3 to 1.5 times this value. To induce hypotension, we exceeded the surgical MAC and chose 1.7 times MAC (2.5% isoflurane) as a suitable concentration. Parrots were stabilized at the target inspired and expired isoflurane at 2.5% isoflurane (2.5% isoflurane) as a suitable concentration. Parrots were stabilized at the target inspired and expired isoflurane concentrations. Equilibration was assumed to be complete within a 10-minute period. Isoflurane causes a dose-dependent decrease in blood pressure associated with a decrease in systemic vascular resistance and cardiac index. These findings were supported by a significant reduction in SAP, DAP, and MAP in anesthetized parrots at 2.5% isoflurane, compared with values reported in a previous study that involved Hispaniolan Amazon parrots. In that study, mean ± SD SAP, DAP, and MAP of 163 ± 18 mm Hg, 148 ± 18 mm Hg, and 155 ± 18 mm Hg, respectively, were detected for birds anesthetized by administration of 2% isoflurane. In the study reported here, we regarded the values from that report as the reference ranges for normotension. To our knowledge, the actual blood pressures in conscious parrots are not known.

Alternatively, controlled hypotension is defined as a reduction of 30% from the baseline MAP in conscious humans. In the present study, blood pressure in parrots anesthetized by administration of 2.5% isoflurane was reduced from the baseline value by 30%. By use of this definition of controlled hypotension, data from the present study supported that we had adequately induced hypotension as indicated by a reduction in MAP of 30% from reference values reported elsewhere.

Multiple studies on arterial blood pressure measurements have found that arterial blood pressure in birds typically is significantly higher than that in mammals. However, the authors are not aware of any studies in psittacines that detected a correlation between values of MAP and inhibition of the vascular autoregulatory pathways. In 1 study performed in anesthetized Galliformes, investigators compared autoregulation of the glomerular filtration rate and blood pressure in chickens and found that chickens were able to maintain glomerular filtration rate when the MAP ranged between 60 and 110 mm Hg. However, when MAP decreased to < 50 mm Hg, chickens were unable to sustain their glomerular filtration rate, and urine output ceased altogether.

However, blood pressure and therefore autoregulation may not be similar in other avian species. For example, direct blood pressure measurements are higher in Psittaciformes, Gruiformes, Falconiformes, and Strigiformes than in pigeons, chickens, and ducks. Cardiovascular physiology may vary among avian species, variations in anesthetic protocols and experimental design among studies may have contributed to the differences in results. To the authors’ knowledge, the study reported here is the first in which use of dobutamine and dopamine as a CRI in psittacine birds has been described. The cardiovascular responses to the infusion of dopamine and dobutamine in Hispaniolan Amazon parrots are in agreement with the general description of dose-dependent effects on heart rate and blood pressure reported in other species. Doses for dobutamine and dopamine were extrapolated from doses recommended in small and large animal medicine. Although, to our knowledge, no data on clearance of these drugs have been reported in birds, the elimination half-life of dopamine and dobutamine in dogs is 2 to 3 minutes, which means that the plasma concentration of these drugs is expected to reach a steady state approximately 10 to 11 minutes after administration. Similar to the effects in dogs, blood pressure plateaued in parrots of the present study at 10 minutes with no further significant changes after that time for either drug or any infusion rate, which suggested that a steady state had been reached. Further pharmacokinetic studies should be performed to confirm the half-life and elimination of dopamine and dobutamine in avian species.
Comparisons between dopamine and dobutamine revealed that dopamine caused the greatest change in blood pressure. Results for the drug doses used in the present study suggested that dopamine may allow better pharmacological control of blood pressure in isoflurane-anesthetized parrots. Dopamine’s greater effects on arterial blood pressure, compared with the effects for dobutamine, may have been attributable to its higher affinity for α-adrenergic receptors. Given that hypotension was induced by isoflurane-induced vasoconstriction, the vasoconstrictive properties of dopamine would theoretically be more effective for these circumstances.

As expected, both drugs caused a significant increase in heart rate. The β₁-adrenergic receptor stimulation by both dopamine and dobutamine causes similar increases in dogs,17 cats,37 horses,39 and alpacas.38 The ability of both drugs to induce clinically important increases in heart rate at low and high infusion rates suggests that the lowest recommended dose should be used to avoid potential adverse effects such as arrhythmogenesis.17,22,40

Four parrots were removed from the study because they developed marked arrhythmia during the administration of dobutamine at a CRI of 15 μg/kg/min. The ECG and blood pressure waveforms were consistent with second-degree atrioventricular block. The CRI was discounted in each parrot, and the arrhythmia resolved within a few minutes. Atrioventricular blocks have been reported in small animal medicine during administration of high doses of dobutamine.17-40 Myocardial ischemia induced by tachycardia that results from dobutamine infusion is thought to be the most likely mechanism that causes second-degree atrioventricular block.22,40

The accurate determination of arterial blood pressure is critical in any species. Direct blood pressure measurement is the criterion-referenced standard. However, direct blood pressure measurements can be affected by numerous factors. Insertion of an arterial catheter in a peripheral artery rather than a central artery can affect blood pressure. Impedance of arteries increases as the distance from the aorta increases. High resistance to the distal site of the catheter used for arterial pressure determination may also affect measurements by reflection of the pulse wave and result in an apparent increase in SAP. There can be inappropriate damping of the arterial pulse signal as a result of differences in the length and diameter of arterial catheters, differences in compliance of the catheter wall, small air bubbles within the catheter or measurement system, and partial occlusion of the system.1 In the study reported here, all arterial catheters were placed in a deep radial artery, waveforms were assessed for their consistency before the start of each experiment, and each parrot served as its own control animal.

Throughout the administration of the agonists, respiration of each parrot was kept constant by the use of intermittent positive-pressure ventilation via a workstation ventilator. Other studies in sandhill cranes39 and Hispaniolan Amazon parrots12 revealed that ventilation had a direct positive effect on arterial blood pressure. To exclude this variable, ventilatory rate for each bird was set at 8 breaths/min with a positive-pressure ventilation of 10 cm H₂O. Throughout the administration of both drugs at all doses, PETCO₂ increased slightly, with some birds having a PETCO₂ > 50 mm Hg during treatment periods. However, these values were not significantly different. The PETCO₂ values decreased during the washout period between subsequent drug infusions. The degree of hypercapnia was considered unlikely to have an influence on the cardiovascular values. Thus, these increases in carbon dioxide possibly were a result of an increase in cardiac performance, such as increases in blood pressure that allowed better tissue perfusion and mobilization of carbon dioxide from tissues to the lungs.

One of the limitations for the present study was the inability to determine cardiac output in the parrots during the treatments. Several factors, including the size of the parrots and the presence of the keel bone, prevented us from assessing cardiac output via the lithium dilution technique or echocardiography, respectively. An increase in blood pressure may result from an increase in cardiac output secondary to increased stroke volume, peripheral resistance, or both. Unfortunately, without the measurement of cardiac output, we could not assess the effects of dobutamine and dopamine on systolic function. The increase in arterial blood pressures could have been attributable to the α-adrenergic effect of dopamine and possibly a minimal α-adrenergic action of dobutamine. However, at least for dobutamine, the increase was most likely attributable to direct stimulation of β₁-adrenergic receptors and the subsequent increase in cardiac output. Nonetheless, both drugs at all doses caused an increase in blood pressure.

Inotropic agents have been used in small and large animal veterinary medicine for treatment of hypotension.2 As advances are made in avian surgery and medicine, the array of routine avian surgical procedures expands, and the accuracy of blood pressure measurement in avian species improves, the duration of anesthesia will increase, thus increasing the potential incidence of hypotensive complications. Preexisting dehydration, intraoperative blood and fluid loss, sepsis, inflammatory vasoactive mediators, and therapeutic drugs may have deleterious effects on arterial blood pressure. The use of vasotensive drugs, appropriate crystalloid and colloid fluid solutions, and balanced anesthetic protocols is essential to maintain appropriate blood pressure and blood flow to vital organs throughout anesthesia.22 In the present study, the increase in blood pressure induced by dopamine and dobutamine administration indicated their positive pharmacodynamic properties during anesthesia in Hispaniolan Amazon parrots and could have been a reflection of their effects on cardiac output and arterial vasocostriction.

Although benefits for the administration of dopamine and dobutamine were detected in the present study, the clinical use of dopamine and dobutamine should be reserved for parrots unresponsive to the correction of underlying conditions, decreases in anesthetic agents, administration of anticholinergics, and administration of fluids. However, the present study set a precedent for the use of inotropic drugs in severely hypotensive birds and confirmed that both dobutamine and dopamine may improve vascular perfusion as indicated by an increase in MAP.

a. Kaytee Exact Rainbow pellets, Kaytee Inc, Chilton, Wis.


References


