SELECTED CLINICAL, BIOCHEMICAL, AND ELECTROLYTE ALTERATIONS IN ANESTHETIZED CAPTIVE TIGERS (PANTHERA TIGRIS) AND LIONS (PANTHERA LEO)


Published By: American Association of ZooVeterinarians

DOI: http://dx.doi.org/10.1638/2013-0202R.1


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SELECTED CLINICAL, BIOCHEMICAL, AND ELECTROLYTE ALTERATIONS IN ANESTHETIZED CAPTIVE TIGERS (PANTHERA TIGRIS) AND LIONS (PANTHERA LEO)


Abstract: A prospective study to assess changes in selected plasma biochemistry and electrolyte values, plasma insulin and aldosterone concentrations, and electrocardiography (ECG) was performed on eight female captive tigers (Panthera tigris) and three lions (Panthera leo) undergoing general anesthesia for elective laparoscopic ovariectomy. Each animal was sedated with medetomidine (18–25 µg/kg) and midazolam (0.06–0.1 mg/kg) intramuscularly, and anesthesia was induced with ketamine (1.9–3.5 mg/kg) intramuscularly and maintained with isoflurane. Venous blood samples were collected and analyzed for plasma biochemistry parameters and insulin and aldosterone concentrations. An ECG was recorded at the time of each blood sample collection. Mean plasma potassium, glucose, phosphorus, and aldosterone concentrations increased during anesthesia (P ≤ 0.05). One tiger developed hyperkalemia (6.5 mmol/L) 2.5 hr after anesthetic induction. Plasma insulin concentrations were initially below the low end of the domestic cat reference interval (72–583 pmol/L), but mean insulin concentration increased (P ≤ 0.05) over time compared with the baseline values. Three tigers and two lions had ECG changes that were representative of myocardial hypoxemia. Based on these results, continuous monitoring of clinical and biochemical alterations during general anesthesia in large nondomestic felids is warranted, and consideration should be given to reversal of medetomidine in these animals should significant changes in electrolytes or ECG occur.

Key words: Aldosterone, electrocardiogram, insulin, lion (Panthera leo), potassium, tiger (Panthera tigris).

INTRODUCTION

General anesthesia affects various organ systems and may exacerbate preexisting homeostatic imbalances. Depression of the central nervous and cardiopulmonary systems caused by general anesthesia can lead to decreased tissue perfusion and cellular metabolism, and changes in the endocrine response in cats and dogs. Similar physiologic alterations could be expected during general anesthesia in tigers and lions; however, the range of possible physiologic derangements in these animals is currently unknown. Inability to perform preanesthetic evaluation in these animals and the absence of species-specific reference intervals for biochemical analytes preclude modification of anesthetic protocols based on preexisting disease, and it may compromise detection and management of anesthesia-related complications.

Hyperkalemia was reported as an important complication in up to 20% of a population composed mainly of tigers (Panthera tigris), cheetahs (Acinonyx jubatus), and cougars (Felis concolor) undergoing general anesthesia. Although the authors of that report recommended frequent blood gas and electrolyte analysis and electrocardiogram (ECG) monitoring in anesthetized nondomestic large felids, the potential risk factors or pathophysiology for development of hyperkalemia were not elaborated.

There is no established definition for hyperkalemia in nondomestic felids; therefore, the defining potassium level for hyperkalemia has been extrapolated from domestic dogs, cats, and horses: ≥ 5.5 mmol/L. Prompting the current study was an observed consistent increase in plasma potassium concentration during anesthesia in six tigers, an African lion (Panthera leo), a snow leopard (Panthera uncia), and one liger (Panthera tigris–Panthera leo hybrid) under anesthesia for various surgical procedures at the authors’ hos-
pital. In six of these animals, ECG changes (bradycardia, spiked T-waves, and wide P waves and QRS complexes) were observed, and cardiac arrest occurred in three animals. Due to the increasing number of large, nondomestic felids undergoing general anesthesia at the same institution, and considering the significance of hyperkalemia in these animals, further investigation of these imbalances was deemed necessary.

This study prospectively characterized changes in plasma biochemistry, particularly potassium, venous blood gases, and plasma insulin and aldosterone concentrations, and changes in ECGs in captive tigers and lions undergoing general anesthesia for elective laparoscopic ovarioectomy.

**MATERIALS AND METHODS**

**Animals**

Eleven healthy, adult, intact female lions and tigers with a familial history of mammary gland carcinoma or a history of intermittent vaginal discharge underwent laparoscopic ovarioectomy, as has been described elsewhere. Exclusion criteria included evidence of vaginal discharge or mammary masses (any subcutaneous nodule appearing contiguous with the mammary chain) at the time of anesthesia, and presence of abnormalities on initial complete blood count or plasma biochemistry panel. The study was approved by the Institutional Animal Care and Use Committee (protocol no. 1990) at the University of Tennessee and was performed with the consent of the animals’ owner.

**Study design**

Food and water were withheld for 12 hr prior to anesthesia. Upon arrival at the hospital, each animal was sedated, via hand or remote injection, with medetomidine (target dose, 20 μg/kg i.m.; Wildlife Pharmaceuticals, Fort Collins, Colorado 80550, USA) and midazolam (target dose, 0.1 mg/kg i.m.; Hospira Inc. Lake Forest, Illinois 60045, USA), based on estimated weight. Once sedated, ketamine (target dose, 2 mg/kg i.m.; Bioniche Teoranta, Inverin, County Galway, Ireland) was administered for immobilization. This was followed by isoflurane (Isoflo, Abbott Laboratories, North Chicago, Illinois 60064, USA) in oxygen, delivered via mask, until the depth of anesthesia was adequate to place an oro-tracheal tube. Anesthesia was maintained with isoflurane in oxygen (2 L/min) using a rebreathing anesthesia circuit (Narkomed 2B, North American Drager, Telford, Pennsylvania 18969, USA). After intubation, a complete physical exam was performed, and animals were weighed. Animals were placed in dorsal recumbency on a padded table and were instrumented for continuous monitoring (Datex S5, Datex-Engstrom, Helsinki, Finland) of indirect blood pressure using an oscillometric technique, end-tidal CO₂, partial pressure, end-tidal isoflurane concentration, ECG, oxygen saturation, and esophageal temperature. An 18-ga, 5-cm i.v. catheter (Surflow i.v. catheter, Terumo Medical Corp., Somerset, New Jersey 08873, USA) was placed in a medial saphenous vein for fluid and drug administration, and a 16-ga, 4.5-cm i.v. catheter (BD Insyte, Becton Dickinson & Co. Infusion Therapy Systems, Sandy, Utah 84070, USA) was placed in the jugular vein for blood collection. Animals were ventilated to maintain an end-tidal CO₂ between 35 and 45 mmHg (4.7–6.0 kPa). Blood pressure was measured indirectly using an appropriately sized cuff (width approximately 40% of the limb circumference) placed over the distal forearm, and was considered normal if mean arterial pressure was ≥60 mmHg. Body temperature was maintained between 37.5°C and 38.5°C with a warm air blanket (Bair Hugger Augustine Medical, Eden Prairie, Minnesota 55344, USA).

Lactated Ringer’s solution (Hospira, Inc.) was administered throughout the anesthesia at 10 ml/kg/hr if the initial packed cell volume (PCV) was >40%, and at 3 ml/kg/hr if the initial PCV was ≤40%. Meloxicam (0.2 mg/kg s.c.; Boehringer Ingelheim Vetmedica, Inc., St. Joseph, Missouri 64506, USA) and ampicillin (15 mg/kg, i.v.; Baxter Healthcare, Brescia, Italy) were administered intravenously.

Jugular venous blood samples (6 ml) were collected immediately after catheter placement (time 0, approximately 30 min following anesthetic induction, corresponded to the start of isoflurane anesthesia); 1 hr after the start of isoflurane administration (time 1 hr), and every 30 min thereafter (times 1.5–3.5 hr) for venous blood gas analysis and sodium, potassium, glucose, and ionized calcium determination using a handheld blood analyzer (i-STAT System, Abbott Point of Care, Princeton, New Jersey 08540, USA). These samples were also analyzed directly for whole blood lactate concentration via a bedside analyzer (Accutrend Lactate Analyzer, Roche, Manheim, Germany), and for PCV by the microhematocrit centrifugation method (Microhematocrit, Fisher Scientific, Pittsburgh, Pennsylvania 15219, USA). In addition, an aliquot from each sample was collected into lithium heparin tubes (BD Vacu-
tainer, Becton Dickinson & Co., Franklin Lakes, New Jersey 07417, USA) for analysis of blood urea nitrogen, creatinine, albumin, globulins, calcium, phosphorus, chloride, bicarbonate, total protein, glucose, sodium, and potassium concentrations and creatinine kinase activity by an automated chemistry analyzer (instrument COBAS 501c, Roche Diagnostics Corp. Indianapolis, Indiana 46256, USA). Samples were also analyzed for insulin (Human insulin specific, Millipore, Billerica, Massachusetts 01821, USA) and aldosterone concentrations using a solid-phase radioimmunoassay (Siemens Healthcare Diagnostics, Los Angeles, California 90045, USA). The final blood sample (time 2.5 or 3.5 hr) was collected immediately before isoflurane was discontinued. Complete blood counts were performed (Advia 120, Siemens Healthcare Diagnostics) on samples collected at time 0 and at the end of anesthesia.

Six-lead ECGs were recorded with the cats in dorsal position, using the same lead placement and gain in each animal, at the same time points as the blood sample collection times. The ECGs were interpreted by a board-certified veterinary cardiologist (RG).

Upon completion of surgical procedures, isoflurane was discontinued, the animals were placed in a recovery cage, and atipamezole (0.09–0.13 mg/kg i.m.; Pfizer Animal Health, NY/Orion Corp., Espoo, Finland) was administered. The animals were extubated when they began to swallow and breathe normally, and had a palpebral reflex. Animals were closely observed until responsive to external stimulation and were then transported to their permanent holding facility for further observation.

Statistical analyses

A mixed-model analysis of variance (ANOVA) was used to examine the effect of time on blood values. The classification variables were cat and time, with time being the independent variable evaluated for its effect on the dependent variables (blood parameters). Individual cat was included as a random effect in all models. Normality was assessed by examining plotted results, performing the W statistic of the Shapiro-Wilk test, and observing stem leaf diagrams. Distributions of residuals from the models were used to ensure that model assumptions were met. Logarithmic transformation (natural log scale) of parameters with nonnormal distributions (phosphorus, potassium, partial pressure of venous oxygen [PvO₂]) was performed prior to inclusion in the mixed-model ANOVA. Pearson correlation coefficients were examined to assess correlation of the handheld blood analyzer with the automated chemistry analyzer for sodium, potassium, and glucose concentrations. Data are expressed as least squares mean ± SEM (SAS Institute Inc., Cary, North Carolina 27513, USA). A P value of ≤0.05 was considered significant.

RESULTS

Eleven felids (eight tigers and three lions) met the inclusion criteria for this study. The animals were all long-term captives, with a mean age of 8.8 yr (range = 4 to 11 yr) and mean weight of 135.5 kg

Table 1. Selected plasma biochemical analytes and insulin and aldosterone concentrations (expressed as least squares means ± SEM) from tigers and lions under general anesthesia over time. All animals (n = 11) were induced with medetomidine, midazolam, and ketamine and all samples obtained while the animals were maintained under isoflurane anesthesia. Values in each row with different uppercase alphabetical superscripts are significantly different (P ≤ 0.05). LSM = least square means, n = number of animals sampled at each time point.

<table>
<thead>
<tr>
<th>Analyte (reference interval)</th>
<th>0 hr</th>
<th>1 hr</th>
<th>1.5 hr</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potassium (2.8–4.8 mEq/L)</td>
<td>3.0 ± 0.1A</td>
<td>3.7 ± 0.2A,B</td>
<td>3.9 ± 0.2B,C</td>
</tr>
<tr>
<td>Calcium (9.5–11.2 mg/dL)</td>
<td>9.9 ± 0.1A</td>
<td>9.3 ± 0.1B</td>
<td>9.2 ± 0.1B</td>
</tr>
<tr>
<td>Phosphorus (2.2–5.5 mg/dL)</td>
<td>4.1 ± 0.2A</td>
<td>4.9 ± 0.5A,B</td>
<td>5.2 ± 0.4B,C</td>
</tr>
<tr>
<td>Aldosterone (194–388 pmol/L)</td>
<td>522.0 ± 256.0A</td>
<td>1067.0 ± 363.0A,B</td>
<td>1601.0 ± 331.0B,C</td>
</tr>
<tr>
<td>Insulin (72–583 pmol/L)</td>
<td>45.0 ± 5.0A</td>
<td>39.0 ± 7.0A</td>
<td>42.0 ± 7.0A</td>
</tr>
<tr>
<td>Glucose (88–183 mg/dL)</td>
<td>143.0 ± 15.5A</td>
<td>179.0 ± 25.6A,B</td>
<td>192.0 ± 19.4A,B</td>
</tr>
</tbody>
</table>

a University of Tennessee Clinical Pathology Laboratory’s automated chemistry analyzer reference intervals for domestic cats.
b Hours after initial blood sample.
c First blood sample collected after induction of anesthesia and jugular catheter placement.
d Michigan State University Diagnostic Center for Population and Animal Health’s reference intervals for domestic cats.
(range = 103 to 162 kg). All animals were healthy based on physical examination and clinical pathology performed upon induction of anesthesia. Induction of anesthesia and tracheal intubation were uneventful. All animals’ heart rate, temperature, end-tidal CO₂ partial pressure, and oxygen saturation remained within normal limits for domestic cats throughout anesthesia. Mean arterial blood pressure remained >60 mmHg in all animals except one lion. After actual weight determination, the administered drug dosages were 18–25 μg/kg for medetomidine, 0.06–0.1 mg/kg for midazolam, and 1.9–3.5 mg/kg for ketamine.

No statistically significant changes or clinically important findings were seen in complete blood count results or plasma blood urea nitrogen, creatinine, creatinine kinase, globulins, ionized calcium, sodium, chloride, or lactate concentrations for any animal at the beginning of anesthesia or over the course of anesthesia. Plasma calcium did decrease significantly over time (Table 1). Mean plasma potassium, phosphorus, glucose, aldosterone, and insulin concentrations did significantly increase over time (Table 1). One tiger developed hyperkalemia at sampling time 2.5 hr, with a potassium concentration of 6.5 mEq/L (initial potassium concentration in this animal was 2.9 mEq/L). No corrective measures were taken in that tiger because the sample was analyzed postextubation, and the animal’s recovery was uneventful.

Initial mean insulin concentrations (Table 1) were below the domestic cat reference interval (72–583 pmol/L); however, the concentrations increased overtime, and by time 3 hr the mean plasma insulin concentrations were within the reference interval (109.0 ± 8.0 pmol/L). Pearson correlation coefficients for the handheld blood analyzer and automated chemistry analyzer were strong for potassium ($R = 0.9$), sodium ($R = 0.7$), and glucose ($R = 0.97$). Despite some variations in venous blood gas values over time (Table 2), there were no significant changes in venous pH, PVO₂, partial pressure of venous carbon dioxide, total carbon dioxide, bicarbonate, base excess, or oxygen saturation of venous hemoglobin between the initial and final samples.

Five animals in this study had ECG abnormalities. Two animals (one tiger and one lion) had S–T segment changes (elevation and depression, respectively) detected between times 0 and 2 hr, but these changes resolved by time 2.5 hr. Two other tigers had an increase in T-wave height (0.6–0.7 mV) between times 0 to 3.5 hr, despite normal serum potassium concentration. In one lion, the ECG at time 0 had negative T-waves ($–0.2$ to $–0.1$ mV), but biphasic T-waves (alternating between negative and positive) were noted at time 1 hr, along with S–T segment depression ($–0.1$ mV). The ECG changes in the latter animal coincided with clinical hypotension (mean arterial pressure of 43 mmHg) and a potassium concentration of 3.6 mEq/L. Atipamezole (34 mcg/kg i.m.) was administered as a rescue procedure. The cat’s mean blood pressure normalized (85 mmHg) within 10 min, and the ECG recorded at time 2 hr (1 hr later) had positive T-waves (0.3–0.4 mV) and normal S–T segments.

**DISCUSSION**

Several statistically significant biochemical and hormonal alterations were observed during this study, but the most clinically important change was the simultaneous increase in plasma potassium and glucose concentrations with a decrease in initial plasma insulin concentration. The mean

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**Table 1.** Extended.

<table>
<thead>
<tr>
<th>Time point (hr)</th>
<th>2</th>
<th>2.5</th>
<th>3</th>
<th>3.5</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>LSM</strong></td>
<td>n</td>
<td><strong>LSM</strong></td>
<td>n</td>
<td><strong>LSM</strong></td>
</tr>
<tr>
<td>3.8 ± 0.2&lt;sup&gt;B,C&lt;/sup&gt;</td>
<td>5</td>
<td>4.3 ± 0.2&lt;sup&gt;C&lt;/sup&gt;</td>
<td>9</td>
<td>4.0 ± 0.2&lt;sup&gt;B,C&lt;/sup&gt;</td>
</tr>
<tr>
<td>8.8 ± 0.1&lt;sup&gt;C&lt;/sup&gt;</td>
<td>5</td>
<td>9.1 ± 0.1&lt;sup&gt;B&lt;/sup&gt;</td>
<td>9</td>
<td>9.2 ± 0.1&lt;sup&gt;B&lt;/sup&gt;</td>
</tr>
<tr>
<td>6.0 ± 0.5&lt;sup&gt;B,C&lt;/sup&gt;</td>
<td>5</td>
<td>6.3 ± 0.4&lt;sup&gt;C&lt;/sup&gt;</td>
<td>9</td>
<td>6.1 ± 0.6&lt;sup&gt;B,C&lt;/sup&gt;</td>
</tr>
<tr>
<td>2188.0 ± 362.0&lt;sup&gt;C&lt;/sup&gt;</td>
<td>5</td>
<td>2080.0 ± 287.0&lt;sup&gt;C&lt;/sup&gt;</td>
<td>8</td>
<td>2118.0 ± 405.0&lt;sup&gt;B,C&lt;/sup&gt;</td>
</tr>
<tr>
<td>51.0 ± 7.0&lt;sup&gt;A,B&lt;/sup&gt;</td>
<td>5</td>
<td>62.0 ± 6.0&lt;sup&gt;B&lt;/sup&gt;</td>
<td>8</td>
<td>109.0 ± 8.0&lt;sup&gt;C&lt;/sup&gt;</td>
</tr>
<tr>
<td>281.0 ± 22.9&lt;sup&gt;C&lt;/sup&gt;</td>
<td>5</td>
<td>238.0 ± 17.1&lt;sup&gt;B,C&lt;/sup&gt;</td>
<td>9</td>
<td>205.0 ± 25.6&lt;sup&gt;B&lt;/sup&gt;</td>
</tr>
</tbody>
</table>
plasma potassium concentrations increased progressively over time in all animals, and although the final mean potassium values did not exceed the reference interval for domestic cats (2.8–4.8 mEq/L), one tiger did become hyperkalemic (6.5 mEq/L) by time 2.5 hr.

Plasma potassium concentration can increase due to a shift in potassium from intracellular to extracellular fluid, or as a result of inorganic acidemia, decreased renal excretion, or insulin deficiency.5 A decrease in insulin secretion is the most likely differential for the potassium changes seen in these nondomestic felids. Insulin, via activation of Na+/K+ ATPase pump, causes the influx of potassium into the intracellular space and has an important role in acute regulation of changes in plasma potassium concentrations.10 Mean plasma insulin concentration was initially less than the domestic cat reference interval (72–583 pmol/L); however, insulin concentrations increased over the course of anesthesia. In the hyperkalemic tiger, hyperkalemia coincided with an insulin concentration (46 pmol/L) much lower than the group’s mean value at that time point, and a glucose concentration (407 mg/dL) greater than that of all the other animals at that time point.

The observed increase in plasma glucose concentrations concurrent with the initially decreased plasma insulin concentrations is consistent with the reported effect of medetomidine administration. α-2 agonists inhibit the production of insulin by binding to the α-2 receptors on the β-cells of the pancreas,14 resulting in decreased cellular glucose uptake and increased plasma glucose concentration. Dose-dependent, medetomidine-induced hypoinsulinemia and hyperglycemia have been reported in dogs26 and cats, with more profound hyperglycemia occurring in cats.10 In the latter study, the peak increase in plasma glucose concentration and maximum decrease in plasma insulin concentration occurred at about 2 hr and 1 hr, respectively, following administration of medetomidine. These are similar to the findings of the present study. Dose-dependent changes in glucose concentrations are also reported in Bengal tigers immobilized with xylazine.22 The simultaneous alterations in insulin and glucose occurring with increased potassium concentrations support our hypothesis that increases in plasma potassium concentrations are a result of the decrease in plasma insulin concentrations caused by medetomidine.

There is no reference interval for plasma aldosterone in nondomestic felids. However, mean plasma aldosterone concentrations were all greater than the reference interval for domestic cats (194–388 pmol/L) reported by the analyzing laboratory (Diagnostic Center for Population and Animal Health, Michigan State University, Lansing, Michigan 48910-8104, USA), and aldosterone concentrations increased progressively during the course of anesthesia. Factors such as increase in plasma potassium concentration and stressful conditions, such as surgery, can increase aldosterone release from the adrenal gland. A linear increase in plasma aldosterone concentration has been observed with a dietary-induced increase in serum potassium concentration in people.3 In that study, a small increase (0.2 to 0.3 mEq/L) in potassium concentration caused an approximately 25% increase in aldosterone plasma aldosterone concentration. Similar increases

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**Table 2.** Venous blood gas analyses (expresses as least squares mean ± SEM) from tigers and lions under general anesthesia over time. All animals (n = 11) were induced with medetomidine, midazolam, and ketamine and all samples obtained while the animals were maintained under isoflurane anesthesia. Samples were collected from a free-flowing jugular catheter and tested using a handheld blood analyzer. Values in each row with different uppercase alphabetical superscripts are significantly different (P ≤ 0.05).

<table>
<thead>
<tr>
<th>Analyte (reference interval)</th>
<th>0* (n = 10)</th>
<th>1 (n = 11)</th>
<th>1.5 (n = 10)</th>
<th>2 (n = 11)</th>
<th>2.5 (n = 7)</th>
<th>3 (n = 3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH (7.27–7.41)</td>
<td>7.26 ± 0.01A</td>
<td>7.27 ± 0.01A</td>
<td>7.27 ± 0.01A</td>
<td>7.24 ± 0.01A</td>
<td>7.26 ± 0.02A</td>
<td>7.25 ± 0.02A</td>
</tr>
<tr>
<td>PCO₂ (33–45 mmHg)</td>
<td>41.0 ± 1.3A,B</td>
<td>38.0 ± 1.3A</td>
<td>39.0 ± 1.3A,B</td>
<td>41.0 ± 1.3A</td>
<td>42.0 ± 1.6A,B</td>
<td>42.0 ± 2.4A,B</td>
</tr>
<tr>
<td>PO₂ (45–65 mmHg)</td>
<td>67.0 ± 7.0A</td>
<td>75.0 ± 7.5A,B</td>
<td>89.0 ± 9.3A,B</td>
<td>94.0 ± 9.3A</td>
<td>96.0 ± 11.9A</td>
<td>86.0 ± 16.4A,B</td>
</tr>
<tr>
<td>TCO₂ (16–24 mmol/L)</td>
<td>20.0 ± 0.5A</td>
<td>19.0 ± 0.5A</td>
<td>19.0 ± 0.5A</td>
<td>19.0 ± 0.5A</td>
<td>18.0 ± 0.7A</td>
<td>20.0 ± 1.0A</td>
</tr>
<tr>
<td>BE (–10–10)</td>
<td>–8.6 ± 0.7A</td>
<td>–9.0 ± 0.5A</td>
<td>–8.9 ± 0.6A</td>
<td>–9.4 ± 0.6A</td>
<td>–8.6 ± 0.7A</td>
<td>–8.7 ± 1.1A</td>
</tr>
<tr>
<td>Bicarb (18–23 mEq/L)</td>
<td>18.0 ± 0.5A</td>
<td>18.0 ± 0.5A</td>
<td>18.0 ± 0.5A</td>
<td>18.0 ± 0.5A</td>
<td>19.0 ± 0.6A</td>
<td>19.0 ± 0.9A</td>
</tr>
<tr>
<td>SvO₂ (60–80%)</td>
<td>88.0 ± 1.5A</td>
<td>92.0 ± 1.4A,B</td>
<td>94.0 ± 1.5A</td>
<td>94.0 ± 1.5A</td>
<td>95.0 ± 1.8A</td>
<td>93.0 ± 2.8A,B</td>
</tr>
</tbody>
</table>

* Hours after initial blood sample.

* First blood sample collected after induction of anesthesia and jugular catheter placement.

A decrease in insulin secretion is the most likely differential for the potassium changes seen in these nondomestic felids. Insulin, via activation of Na+/K+ ATPase pump, causes the influx of potassium into the intracellular space and has an important role in acute regulation of changes in plasma potassium concentrations. Mean plasma insulin concentration was initially less than the domestic cat reference interval (72–583 pmol/L); however, insulin concentrations increased over the course of anesthesia. In the hyperkalemic tiger, hyperkalemia coincided with an insulin concentration (46 pmol/L) much lower than the group’s mean value at that time point, and a glucose concentration (407 mg/dL) greater than that of all the other animals at that time point.

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in plasma aldosterone concentration secondary to increased plasma potassium have been reported in dogs. Surgery and anesthesia have also been shown to increase aldosterone production in people. In the current study, the stress associated with surgery and anesthesia, and the concurrent increase in plasma potassium concentrations were the likely factors responsible for the increase in plasma aldosterone concentration.

In the present study, plasma phosphorus concentration increased over the course of anesthesia. Possible etiologies for development of hyperphosphatemia include translocation of phosphorus due to tissue trauma, metabolic acidosis, or decreased phosphorus excretion. An increase in phosphorus concentration is reported in dogs with metabolic acidosis. A mild decrease in venous blood pH (7.24–7.26) and decrease in base excess values were observed in this study. The latter is indicative of a metabolic acidemia; however, other causes of hyperphosphatemia, such as tissue destruction due to the surgical procedure and recumbency, cannot be completely ruled out. Considering the lack of significant increase in creatinine concentrations and normal blood urea nitrogen concentrations in these animals, a renal etiology for the observed hyperphosphatemia is unlikely.

Five animals in this study had ECG abnormalities (elevation and/or depression of the S–T segment, increased height of the T-wave, and changes in the polarity of T-wave). The changes in T-wave polarity have previously been reported in tigers and lions anesthetized with ketamine 10 mg/kg i.m. and xylazine 1–2 mg/kg i.m. The changes in S–T segments and the T-waves in the present study are suggestive of myocardial hypoxia, such as is seen in domestic cats. All of the animals in this study were intubated, received 100% oxygen throughout the procedure, and maintained PvO₂ values greater than 65 mmHg. α-2 agonists can decrease coronary blood flow, increase coronary vascular resistance, and contribute to myocardial ischemia; it is likely medetomidine caused the observed ECG changes in this study. In a report of dogs under enflurane anesthesia, dexmedetomidine (1–4 μg/kg i.v.) caused a dose-dependent decrease in coronary blood flow and an increase in coronary vascular resistance. In that study, coronary blood flow changes were associated with an increase in myocardial oxygen extraction and a decrease in coronary sinus oxygen saturation. The cardiovascular effects of medetomidine in dogs, rats, and cats have been reversed by the use of atipamezole.

CONCLUSION

Increases in plasma potassium, glucose, phosphorous, and aldosterone concentrations, along with a decrease in insulin concentrations and ECG changes, occurred in healthy, large, nondomestic felids during isoflurane general anesthesia after induction with medetomidine, midazolam, and ketamine. Therefore, serial monitoring of the ECG, blood pressure, electrolytes, glucose, and acid-base status of anesthetized nondomestic felids is warranted. Use of the lowest possible dose of medetomidine and minimizing the duration of anesthesia may aid in prevention of potentially severe plasma electrolyte and hormonal changes. Additionally, partial or full reversal of α-2 agonists, once the animal is at an adequate depth of anesthesia, may be considered. Evaluation of the effects of other anesthetic drugs, doses, and drug combinations on biochemical values and clinical parameters of large, nondomestic felids under general anesthesia is warranted.

Acknowledgments: The authors thank Marylynn Haven and Debbie Chaffins of Tiger Haven for their cooperation in the study, and Jessica Konzer Birdwell and Virginia Martin Nystrom for their technical assistance. Funding by University of Tennessee Companion Animal Fund.

LITERATURE CITED


Received for publication 25 August 2013