LETTER TO THE EDITOR

**Acute bradycardia associated with positional change in a hyperkalaemic cheetah (*Acinonyx jubatus*)**

An otherwise healthy, captive 5-year-old male Cheetah (*Acinonyx jubatus*) weighing approximately 40 kg, presented for diagnostic imaging and orthopaedic assessment of the left pelvic limb.

The cheetah was starved for 24 hours and sedated for travel by zoo staff with an intramuscular (IM) injection of medetomidine (0.05 mg kg-1, Medator; Virbac Limited, UK) and ketamine (4 mg kg-1, Anaestamine; Animalcare, UK), with an additional bolus of medetomidine (0.013 mg kg-1) and ketamine (0.75 mg kg-1) administered during transit. Upon presentation for anaesthesia at our facility, an 18-gauge intravenous (IV) cannula was placed in the right cephalic vein, and anaesthesia was induced with propofol (1.5 mg kg-1, PropoFlo Plus; Abbott, UK). Topical lidocaine (Intubeaze 20mg/mL Oromucosal spray, Dechra Veterinary Products, UK) was applied to the larynx, the trachea was intubated using a 12mm endotracheal tube which was connected to a circle breathing system. Anaesthesia was maintained with isoflurane (Isoflo, Zoetis, UK) in oxygen, initially at a vaporiser setting of 2%. This was adjusted as required during the procedure based on our assessment of anaesthetic depth. Intravenous fluid therapy with Hartmann’s solution was started at a rate of 180 mL hour-1. Monitoring consisted of capnography, pulse oximetry, electrocardiogram (ECG), oscillometric blood pressure, and oesophageal temperature on a multi-parameter monitor.

The cheetah remained stable under anaesthesia for 90 minutes (HR 80 beats minute-1 , *f*R 10 breathes minute-1, systolic arterial pressure (SAP) 130mmHg, mean arterial pressure (MAP) 100mmHg, diastolic arterial pressure (DAP) 85mmHg, oesophageal temperature 37.6°C) spending 60 minutes in right lateral recumbency to take radiographs and to facilitate examination of the left pelvic limb.

The cheetah was re-positioned into left lateral recumbency to radiograph the contralateral limb. Immediately the heart rate decreased rapidly to 24 beats minute-1, with an increase in *f*R to 24 breathes minute-1. The ECG changed from a normal sinus rhythm to ventricular escape rhythm with absent p-waves. Pulses were confirmed and were hyperdynamic in nature but oscillometric blood pressure failed to measure, and no Doppler device was available. The vaporiser was turned off and, believing the bradycardia to be associated with a cardio-vagal reflex, 0.015 mg kg-1 atropine (Atropine sulfate, Hameln Pharmaceuticals Ltd, UK) was administered IV. This was repeated twice with no response. Subsequently 0.005 mg kg-1 of glycopyrrolate glycopyrrolate (Glycopyrronium bromide, Martindale Pharmaceuticals Ltd, UK) was administered IV. This caused a transient response with the heart rate increasing to 80 beats minute-1 with intermittent 2nd degree AV blocks. An intra-arterial catheter was placed in the dorsal pedal artery confirming normotension (SAP 110 mmHg, MAP 75 mmHg, DAP 58 mmHg).

Arterial blood gas analysis revealed a moderate acidaemia secondary to a respiratory acidosis, and hypoxaemia (table 1). It also revealed a serum potassium of 6 mmol L-1 and blood glucose of 18.4 mmol L-1. Mechanical ventilation (MV) was started to correct the respiratory acidosis and acidaemia. Care was taken not to produce airways pressures over 15 cmH2O to avoid further increases vagal tone.

Thirty minutes after the initial arterial blood gas the analysis was repeated. Although the heart rate was now 38 beats minute-1 the ECG complexes remained the same. The respiratory acidosis had been corrected (table 1), however the hyperkalaemia and hyperglycaemia had worsened to 7.6 mmol L-1 and 31.5 mmol L-1 respectively. An infusion of calcium gluconate (Calcium gluconate 10%, Hameln Pharmaceuticals Ltd, UK) was started at 100 mg kg-1 over 20 minutes, and sodium bicarbonate was drawn up to be administered, however the cheetah started to swallow at this point, prompting swift disconnection from all infusions and extubation. Flow-by oxygen was delivered via mask during recovery. The heart rate increased to 60 beats minute-1 and the cheetah was moved to a cage. The cheetah went on to make an unremarkable recovery.

Several interesting points can be taken from this case which may aid future anaesthetic management of large felidae. Although the profound bradyarrhythmia was undoubtedly associated with hyperkalaemic myocardial toxicity, there may have been other contributing factors.

The initial management of bradycardia was for a cardio-vagal reflex following a change in recumbency. The duration of the effect, combined with normotension and a lack of response to anticholinergics, suggested a cardio-vagal reflex was not entirely to blame.

At this point mild hyperkalaemia was diagnosed. Hyperkalaemia can be associated with acidaemia; therefore we initially hypothesised the elevation in serum potassium would reduce if the respiratory acidosis was reversed. Acidosis may also directly be a cause of bradycardia due to decreased conduction speeds through the AV node. Arterial blood gas analysis was rechecked 30 minutes later after starting MV, which revealed the acidaemia and respiratory acidosis has resolved. Despite this the bradycardia was still present and the hyperkalaemia had worsened.

There are reports of alpha-2 agonist based anaesthesia leading to hyperkalaemia in about 20% of exotic felids, but the mechanism for this is unknown (Steeil et al. 2013; Reilly et al. 2014; Ramsey 2014). One potential mechanism is alpha-2 agonist inhibition of insulin release (Sinclair 2003), although stimulation of the alpha-adrenergic receptors also increases serum potassium through activation of Ca2+-dependent-K+-channels in the liver (Moratinos & Reverte 1993).

In such cases atipamezole administration may well reverse the hyperkalaemia and bradycardia (Reilly et al. 2014). Unfortunately, the mode of transportation available to move the cheetah back to the zoo was not suitable to allow a safe unsedated recovery so atipamezole could not be administered.

Why the bradycardia occurred immediately following turning is unclear. It is possible there were alterations in vagal tone or stimulation during turning which caused an abrupt decrease in heart rate which could not then be resolved due to the hyperkalaemia. While hyperkalaemia has been reported in large cats previously, the addition of a vagal component may be an important differential in the future.

**References**

Moratinos J, Reverte M (1993) Effects of catecholamines on plasma potassium: the role of alpha-and beta-adrenoceptors. Fundam Clin Pharmacol 7, 143–153.

Reilly S, Seddighi MR, Steeil JC, et al. (2014) Selected Clinical, Biochemical, And Electrolyte Alterations In Anesthetized Captive Tigers (Panthera Tigris) And Lions (Panthera Leo). J Zoo Wildl Med 45, 328–334.

Sinclair MD (2003) A review of the physiological effects of alpha2-agonists related to the clinical use of medetomidine in small animal practice. Can Vet J 44, 885–897.

Steeil J, Ramsay EC, Aczm D, Schumacher J (2013) Hyperkalemia in Exotic Felids Anesthetized with an Alpha-2 Adrenoceptor Agonist, Ketamine, and Isoflurane. In: AAZV Annu Conf 2013 (Abstract)