Etorphine-halothane anaesthesia in two five-year-old African elephants (Loxodonta africana)

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ABSTRACT
Anaesthesia of 2 five-year-old female African elephants (Loxodonta africana) was required for dental surgery. The animals were each premedicated with 120 mg of azaperone 60 min before transportation to the hospital. Before offloading, 1 mg etorphine was administered intramuscularly (i.m.) to each elephant to facilitate walking them to the equine induction/recovery room. For induction, 2 mg etorphine was administered i.m. to each animal. Induction was complete within 6 min. Surgical anaesthesia was induced with halothane-in-oxygen after intubation of the trunk. During surgery the mean heart rate was 61 and 45 beats/min respectively. Systolic blood pressures increased to 27.5 and 25.6 kPa respectively, and were treated with intravenous azaperone. Blood pressure decreased thereafter to a mean systolic pressure of 18.1 and 19.8 kPa, respectively. Rectal temperature was 35.6 and 33.9 °C at the onset of surgery, and decreased to 35.3 and 33.5 °C, respectively, at the end of anaesthesia. Etorphine anaesthesia was reversed with 5 mg diprenorphine at the completion of 90 min of surgery.

Key words: anaesthesia, azaperone, elephant, etorphine, halothane, Loxodonta africana.


INTRODUCTION
Anaesthesia of 2 female, 5-year-old African elephants (Loxodonta africana) was required for treatment of a fractured tusk in each of the animals at the Faculty of Veterinary Science, University of Pretoria. The elephants had been in captivity for 8 months, and their estimated body mass was 1200 (Case 1) and 1000 kg (Case 2), respectively. The elephants were treated 7 days apart. For sedation, 120 mg azaperone (Stresnil, Janssen Animal Health) was administered intramuscularly (i.m.) 60 min before crating and transport. On arrival at the Veterinary Academic Hospital about 60 min later, the elephants were each injected i.m. with 1 mg of etorphine (M99, Logos Agvet) while in the crate. This was done to facilitate walking the elephants into the equine induction/recovery room, a distance of 40 m. Ataxia from the etorphine was first noted when the induction room was entered about 6 minutes later.

MONITORING
The anaesthetic depth was monitored by observing the percentage anaesthetic delivered by the vaporiser, the palpebral reflex, eyeball rotation and the muscle tone of the trunk. Venous access was obtained with a 18 G teflon catheter (Jelco, Ethicon) after venipuncture of an ear vein on the medial surface and sutured in place with 2/0 nylon. A balanced electrolyte solution (PlasmaVet, SABAX) was infused at a rate of 10 ml/kg/hr during anaesthesia. In total, 15 l of the electrolyte solution were administered during anaesthesia in both animals. Multi-parameter physiological monitors were used to monitor vital signs during anaesthesia.

Heart rate, electrocardiogram (ECG), blood pressure and temperature were monitored with a CardioCap II monitor (Datex Instrumentarium Corporation). Disposable ECG electrodes were attached to the skin with the negative lead to the scapular area on the right side. A 20 G teflon catheter (Jelco, Ethicon) was introduced into an artery on the medial surface of the upper (right) ear and sutured in place with 2/0 nylon (Ethilon, Ethicon) for direct measurement of blood pressure. Systolic and diastolic pressures were measured with a calibrated strain gauge transducer connected to the physiological monitor. The temperature probe was placed in the rectum. The Capnomac Ultima (Datex Instrumentarium Corporation, Helsinki) was used to monitor ventilation rate and end-tidal carbon dioxide concentration (ETCO2), measured from the endotracheal tube placed in the trunk. The cardiopulmonary variables are shown in Table 1. Arterial blood was collected anaerobically in heparinised 5 ml syringes at 30 min intervals during anaesthesia, and stored in iced water until analysis. Arterial blood-gas analysis was performed with an ABL3 (Acid-Base Laboratory, Radiometer). Blood-gas values were corrected to body temperature and are given in Table 2. The monitored variables were recorded on a standard anaesthetic monitoring sheet during anaesthesia.

ANAESTHESIA
The equine induction/recovery room consisted of a darkened room with padded walls and floor. For induction an additional 2 mg of etorphine was administered i.m. to each animal, which was left alone in the room. Progress during induction was observed through a small window in the door. Anaesthesia was maintained after intubation by a large animal circle anaesthetic machine with carbon dioxide absorption (North American Dräger, Large Animal Control Center), out-of-circuit vaporiser (Halothane Vapor 19.3), and a 30 l reservoir bag. Ventilation was spontaneous. Initially only oxygen was delivered to the breathing circuit at 10 l/min. Halothane (Fluothane, Zeneca) was introduced into the circuit when surgery commenced. The concentration varied between 1 and 2 % during maintenance of anaesthesia, and was adjusted to keep the muscles of the trunk relaxed. To be able to maintain anaesthesia, additional increments of 0.5 mg etorphine were administered intravenously (i.v.) when required, i.e. when the anaesthetic plane decreased.

Case 1
The elephant went down into lateral recumbency within 6 min after injecting the etorphine. Breathing was laboured...
with audible inspiratory sounds, and steps were taken to rectify ventilatory obstruction that resulted from partial occlusion of the trunk as it was folded below the head of the animal. Oxygen was insufflated at 15 l/min with a 15 mm latex tubing, introduced for a distance of 30 cm down the trunk passage (nostril). This resulted in abolition of inspiratory sounds. The upper (right) front and hind limbs were suspended in a horizontal position during anaesthesia. Attempts at orotracheal intubation were not successful, as the oropharynx was too narrow to allow manual palpation of the larynx. A lubricated 16-mm silicon endotracheal tube was introduced down the other trunk passage and connected to the anaesthetic machine. Twenty mg of azaperone was injected i.v., and the blood pressure thereafter decreased to 14.4/7.1 kPa within 15 min after administration. At this time heart rate decreased from 60 to 51 beats/min. During stable anaesthesia, the mean and standard deviation (±SD) heart rate was 61 (8) beats/min, and the mean systolic (SD) and diastolic (SD) blood pressures 18.1(3.7)/12.1(3.5) kPa, with the maximum pressure at 19.7/15.7 and the minimum 13.3/10 kPa. A gradual increase in the arterial blood pressure was observed during this period until arousal, when it suddenly increased to 21.7/13.1 kPa. The heart rate increased to 80 beats/min at this time, and an additional dose of etorphine was administered. The blood pressure thereafter decreased to 18.9/12.8 kPa. The ETCO2 concentration varied between 6.5 and 8.5 %, and the arterial partial pressure of CO2 (PaCO2) increased to a maximum of 7.3 kPa, indicating hypoventilation. The partial pressure of oxygen in arterial blood (PaO2) increased to 21.5 kPa after intubation, and oxyhaemoglobin saturation increased to 99 % as result of oxygen supplementation. Rectal temperature decreased during anaesthesia from 35.5 after induction to 35.3 °C at the end of surgery. The duration of anaesthesia was 1 hr and 45 min.

**Table 1**: Intra-operative cardiopulmonary variables in 2 African elephants anaesthetised with an etorphine-halothane combination.

<table>
<thead>
<tr>
<th>Time</th>
<th>Case 1</th>
<th>Case 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HR</td>
<td>SYSDIAS</td>
</tr>
<tr>
<td>09:30</td>
<td>56</td>
<td>29.2</td>
</tr>
<tr>
<td>09:35</td>
<td>47</td>
<td>19.3</td>
</tr>
<tr>
<td>10:40</td>
<td>52</td>
<td>24.7</td>
</tr>
</tbody>
</table>

*HR = heart rate (beats/min); SYS = systolic blood pressure (kPa); DIAS = diastolic blood pressure (kPa); VR = ventilation rate (breaths/min); ET = percentage end-tidal carbon dioxide concentration.

**Table 2**: Intra-operative arterial blood-gas variables in 2 African elephants anaesthetised with etorphine-halothane.

<table>
<thead>
<tr>
<th>Time</th>
<th>Case 1</th>
<th>Case 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>pH</td>
<td>PaO2</td>
</tr>
<tr>
<td>10:10</td>
<td>7.31</td>
<td>21.5</td>
</tr>
<tr>
<td>10:30</td>
<td>7.33</td>
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<td>11:10</td>
<td>7.37</td>
<td>19.1</td>
</tr>
<tr>
<td>09:30</td>
<td>7.34</td>
<td>18.5</td>
</tr>
<tr>
<td>10:00</td>
<td>7.26</td>
<td>35.2</td>
</tr>
<tr>
<td>10:30</td>
<td>7.24</td>
<td>36.9</td>
</tr>
</tbody>
</table>

**PaO2** = partial pressure of oxygen in arterial blood (kPa); **PaCO2** = partial pressure of carbon dioxide in arterial blood (kPa); **HCO3** = plasma bicarbonate concentration (meq/l); **SBE** = standard base excess; **SAT** = percentage oxyhaemoglobin saturation.
6 min, and appeared to be more complete, as it resisted crating more than the 1st elephant. After crating, an additional 40 mg of azaperone was administered i.m. in both animals for sedation during transport.

**DISCUSSION**

The use of halothane to maintain anaesthesia after etorphine induction has been described previously. Recommended drugs for immobilisation of African elephant are etorphine, carfentanil or a combination of xylazine and ketamine. The last combination is recommended for juveniles. The first 2 agents are highly potent opioid agonists that will induce analgesia, immobilisation and anaesthesia in the elephant. The recommended dose for the African elephant varies between 1 mg and 1.5 μg/kg body mass, or 5 mg for a small calf and 7 mg for a medium-sized calf. The additional dose of 2 mg was sufficient in this instance as the animals went down with minimal excitement or stumbling. The total dose of 3 mg etorphine appears low, but may have been influenced by the earlier administration of azaperone, hypothermia, and the fact that these animals were relatively tame and accustomed to being handled by people. Although etorphine is a potent analgesic, it will not induce surgical anaesthesia on its own. Surgical anaesthesia was therefore obtained with halothane-in-oxygen. Tamas and Geiser required up to 2.5 % halothane to maintain anaesthesia on a semi-closed breathing circuit (partial rebreathing) with the trunk bilaterally intubated.

Etorphine is a potent ventilatory depressant, and when combined with mechanical impairment of ventilation in the sternally-recumbent elephant, results in hypoxia and hypercapnoea. Vigilant monitoring and action during the induction period is required to ensure lateral recumbency with an extended trunk to prevent ventilatory obstruction from external compression or flexing of the trunk. Changes observed in sedated, laterally-recumbent elephants included a decrease in the partial pressure of oxygen to 7.3 kPa and an increase in the partial pressure of CO₂ to 7.4 kPa. Nasal oxygen insufflation proved valuable to restore arterial oxygen partial pressures. Etorphine-halothane anaesthesia in Case 2 resulted in hypoventilation with a PaCO₂ of 7.9 kPa and an end-tidal CO₂ concentration of 8.9 %. A PaCO₂ of 5.8 kPa in conscious laterally-recumbent elephants did not indicate hypoventilation compared to a PaCO₂ of 5.9 in normal standing elephants. Carfentanil-halothane anaesthesia results in a gradual increase in PaCO₂ from 6.9 to 9.9 kPa. Etorphine-isoflurane anaesthesia resulted in increases in the PaCO₂ in excess of 8.7 kPa. The use of a demand valve to improve minute ventilation increased the partial pressure of oxygen significantly. In this study the maximum PaO₂ of 36.9 kPa was less than the maximum value of 53.3 kPa for carfentanil and halothane-in-oxygen anaesthetised elephants. The PaO₂ decreased during lateral recumbency in unsedated elephants from 12.8 to 11.2 kPa, and from 33.3 to 33.2 kPa during anaesthesia. In this study a similar decline from 21.5 to 19.1 kPa in the PaO₂ was observed during halothane anaesthesia and was probably the result of hypoventilation, ventilation-perfusion mismatch, pulmonary shunts, and altitude (Pretoria 1400 m). The PaO₂ of 21.5 kPa that was obtained in Case 1 was much lower than the 36.9 in Case 2. Air may have entered the lungs during inhalation around the tubes at the trunk opening to dilute the inhaled halothane-oxygen mixture, resulting in the lower partial pressures obtained.

Arterial hypertension and potentially fatal pulmonary oedema may occur in elephants during capture and immobilisation. Possible factors identified in the aetiology of this condition are herding and forced exercise, the administration potent opioids, recency of a combination of these factors. Systolic and diastolic blood pressures in standing unsedated elephants are 23.8 and 15.8 kPa, respectively. In laterally-recumbent, unsedated elephants the systolic and diastolic blood pressures increased respectively to 30.1 and 20.1 kPa. A systolic blood pressure of >26.7 kPa was recorded after carfentanyl immobilisation. A similar situation was observed in this case, and the vasodilatory effect of azaperone appeared to have worn off by induction, as hypertension was observed after instrumentation. To prevent a further increase in blood pressure, additional azaperone was administered i.v. The decline in the systolic and diastolic pressure was clinically significant, from 27.5/20.5 to 14.4/7.1 kPa7, after 40 mg azaperone in the first case. The mechanism whereby azaperone decreases blood pressure is through α-adrenergic antagonism resulting in peripheral vasodilatation. The rectal temperature after induction was previously reported to be 37.1 °C and decreased to 36.4 °C at the end of surgery.

Hypothermia was observed in both cases after induction, with rectal temperatures of 35.5 and 33.9 °C. This may have been the result of the azaperone administration before transport. Azaperone inactivates the hypothalamic thermal control centre and dilates peripheral blood vessels, which may result in rapid heat loss when ambient temperature is low during transport in a crate on an open truck.

**REFERENCES**