HYPERTENSION FOLLOWING ETORPHINE ANESTHESIA IN A RHINOCEROS (DICEROS SIMUS)

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CASE REPORT

A 2,000 kg, 30-year-old male white rhinoceros (Diceros simus) at the Milwaukee County Zoo had a 4-square-inch chronic draining tract on the bottom of the right rear foot. General anesthesia was required for exploration and treatment of the lesion.

Etorphine HCl (2.8 mg) was injected by pole syringe in the biceps muscles of the left rear leg. Within 20 min, the rhinoceros became ataxic and went down to a position between right lateral and sternal recumbency. Head pressing preceded recumbency. An air dunnage bag was positioned beneath the rhinoceros as it became recumbent to avoid nerve and/or muscle damage during anesthesia. One sterile 20-gauge 2-inch catheter was cannulated into a vein in the right ear and another was placed into an artery on the medial surface of the left ear. The venous catheter was kept patent by infusion of lactated Ringer’s solution. The arterial catheter was connected to a transducer and the resulting pressure waves, as well as electrocardiographic tracings, were displayed on a dual channel video monitor. Flushing of the arterial line with heparinized saline was performed as needed.

The foot lesion was scrubbed, explored, and debrided. Despite the inflation of a pneumatic tourniquet on the right rear leg, extensive hemorrhage occurred. Arterial bleeding was difficult to control. Approximately 8 liters of blood loss occurred during the procedure. The animal remained immobilized and appeared analgesic during the surgical period. No obvious nidus for infec- 

fection was located in the 4-inch tract and necrotic tissue was excised around the lesion. Finally, the foot was bandaged with a tight wrap and the tourniquet removed. Total tourniquet time was 40 min.

Arterial pressure readings were taken within 15 min of recumbency. Initial systolic/diastolic pressures (300/250 mm Hg) preceded tourniquet application. These pressures gradually decreased and stabilized at 240/200 mm Hg for the remainder of the procedure. Mean arterial pressure (MAP) ranged from 280 to 210 mm Hg. The heart rate remained between 80 and 100 beats/min. Respiratory rate ranged from 15 to 20 breaths/min and rectal temperature was constant at 36°C. Occasional unifocal premature ventricular depolarizations (1–2/min) were observed.

The narcotic was reversed (120 min post-administration) using 5.6 mg diprenorphine administered intravenously. Both catheters were removed at this time. Within 3 min, the rhinoceros rolled into sternal recumbency. The animal remained in this position for an additional minute before rising on the first attempt with no signs of ataxia.

DISCUSSION

This report documents hypertension in a nondomestic ungulate following immobilization with etorphine. Etorphine-induced hypertension has been reported in the following domestic ungulates: ponies, swine, and horses. Arterial blood pressure in these reports was measured via a catheter placed directly into an artery. Clinical assessment of increased blood pressure in domestic cattle and zebras has been suggested following etorphine administration. Based upon the cardiovascular responses of
these species, etorphine was presumed to be hypertensive in the nondomestic ungulate.9 

There is little information available about the normal blood pressure in the nondomestic ungulate. Blood pressure measurements in these animals are reported during anesthesia. Intravenous carfentanil alone produced elevated blood pressure in an Indian elephant.7 Systolic blood pressure measured in an auricular artery was greater than 200 mm Hg during carfentanil anesthesia but stabilized at 160–190 mm Hg during halothane anesthesia. Lower direct arterial pressures (MAP = 105–140 mm Hg) and heart rates (25–35 beats/min) were recorded from the auricular artery of an Asian elephant following etorphine anesthesia.12 However, the concurrent administration of xylazine with etorphine in this elephant may have influenced arterial pressures. Blood pressure measurement in the rhinoceros has not been reported.

The presence of a tourniquet can influence arterial blood pressure. Application of a pneumatic tourniquet to the distal limb of horses for 120 min resulted in a 20% increase in arterial blood pressure.11 Elevated arterial pressures in the present case were recorded before, during, and after tourniquet application. Therefore the tourniquet was not considered significant in the development of hypertension in this animal. This tourniquet was only partially effective in reducing hemorrhage at the surgical site. Presumably the elevated blood pressure and thick skin of the rhinoceros contributed to the tourniquet’s ineffectiveness.

There is evidence that the cardiovascular effect of etorphine in ungulates is caused by increased activity of the sympathetic nervous system. Etorphine-induced tachycardia and hypertension were accompanied by a sixfold plasma norepinephrine increase in horses.3 Furthermore, the cardiovascular responses to etorphine were eliminated in ponies pretreated with the beta-adrenergic blocking agent propranolol.6 The dose of etorphine used in this animal was appropriate for anesthesia based upon immobility and appearance of analgesia. However, sympathetic stimulation is still possible during surgical operation despite an apparent adequate plane of anesthesia.5

In summary, this case documents hypertension in a nondomestic ungulate following anesthesia with etorphine alone. Since hypertension exasperates hemorrhage, this complication should be considered when the potential for massive hemorrhage exists.

PRODUCTS MENTIONED IN TEXT


b. Gould Disposable Transducer, Oxnard, California.

c. Spacelabs 514T, Hillsboro, Oregon.


REFERENCES


9. Lumb, W. V. and Jones, E. W.: Veterinary an-

BOOK REVIEW


This book, one of a series in the “Library of Veterinary Practice” published by the Blackwell organization, will be a useful addition to the library of the veterinary practitioner and student, although it, like others in the field, has its shortcomings. One of these is a lack of international perspective. On the other hand, the price puts this book more realistically than some within reach of the veterinary student or recent graduate.

The first part of the book is a series of chapters on clinical examination, aids to diagnosis, postmortem examination, medication, anesthesia, surgery, nursing and after care, breeding problems, and the release of casualty wild birds. These topics are covered in 207 pages, and the last three sections do a good job of covering material that is not only important but does not seem to have been dealt with as thoroughly in other texts.

The second half of the book consists of a series of ready reference tables. Some of these are more useful than others. The first two list weights of birds likely to be seen in general practice and the incubation and fledging periods of selected birds. The next seven tables cover infectious, parasitic, and toxic diseases of birds and are very comprehensive. Unfortunately they seem to lack any sort of logical structure, other than being divided according to the traditional breakdown of bacterial, viral, mycotic, and so forth. The first column gives the name of the disease, but these are not in any clearly recognizable order. It is not until the fifth column that clinical signs are described. It would have been much more useful to this reviewer if these tables had been designed as a keying-out system enabling the clinician to observe symptoms, collect samples, and eliminate differentials as one progressed through the chart.

The listing of poisons likely to affect birds in table 9 is a useful addition to the increasing complexity of avian disease diagnosis, but tables 10 and 12 have little or no relevance to anyone outside the United Kingdom.

Like the curate's egg, this book is good in parts and can be used as a quick reference for a variety of purposes. The price makes it a realistic addition to the library of the student or established avian practitioner.—Reviewed by J. C. Haigh