

Tourniquet-induced hypertension in an ostrich

Janyce L. Cornick-Seahorn, DVM, MS; George S. Martin, DVM, MBA; Tom N. Tully, DVM, MS; James M. Morris, DVM

- Monitoring of ratites during general anesthesia should be performed with the same standard of care that is applied to mammalian species.
- Because of the paucity of information regarding arterial blood pressures in awake and anesthetized ostriches, interpretation of these values in the individual ostrich can be difficult.
- Tourniquet-induced hypertension during general anesthesia, as defined by a 30% increase in systolic or diastolic arterial pressures compared with the first values recorded after incision, may develop in ostriches.

A 3-year-old 110-kg male ostrich (*Struthio camelus*) was examined because of lameness of the left limb and swelling and drainage in the area of the left tarsometatarsal bone. The ostrich had lacerated the skin over the cranial aspect of this bone 1 month earlier and, although the laceration had healed, swelling and discharge persisted, and lameness had developed. Radiography revealed periosteal proliferation and a sequestrum (20 cm long) of the middle third of the dorsal aspect of the tarsometatarsal bone. Surgical removal of the sequestrum was planned.

Food was withheld from the ostrich for 6 hours prior to surgery. Anesthesia was induced in a dark, padded stall by use of diazepam^a (0.1 mg/kg of body weight, IM) and zolazepam/tiletamine^b (4.4 mg/kg, IM). The ostrich was transported to the radiology suite for additional radiographic views. A 12-mm ID endotracheal tube^c was inserted 15 minutes after injection of the induction drugs. A small-animal anesthetic machine with a semiclosed-circle system was used to deliver isoflurane (2.5 to 5.0%) in oxygen (2.0 L/min). The ostrich was moved to the surgery suite and positioned in right lateral recumbency on a padded surgery table. Its head was positioned to be slightly elevated. A 16-gauge catheter was placed in the left brachial vein for administration of lactated Ringer's solution (10 ml/kg/h, IV) during surgery. A 20-gauge catheter was placed in the left brachial artery to measure arterial pressure and collect arterial blood for pH and gas analyses. The catheter was connected to a pressure transducer,^d which was calibrated at the level of the sternum. An ECG was continuously recorded in lead II.^d Initial recordings after instrumentation included heart rate (38 beats/min), respiratory rate (24 breaths/min), mean arterial pressure (MAP, 165 mm of Hg), systolic arterial pressure (SAP, 210 mm of Hg), and diastolic arterial pressure (DAP, 131 mm of Hg). A pneumatic tourniquet^e was placed 8 cm proximal to the left tibiotarsal-tarsometatarsal joint and inflated to 600 mm of Hg after expelling venous blood from the distal regions of

the limb with a sterile elastic wrap. A blood gas analysis performed at this time revealed hypercapnia and metabolic alkalosis (Fig 1). Surgery and intermittent positive pressure ventilation (IPPV), using a volume-cycled ventilator,^f were initiated simultaneously 15 minutes after tourniquet placement. Settings for IPPV included a tidal volume of 1,400 ml, a respiratory rate of 12 breaths/min, and a peak inspiratory pressure of 12 cm of H₂O. Movement of the ostrich's head was observed at the beginning of surgery. Butorphanol (0.02 mg/kg) and ketamine (0.2 mg/kg) were administered IV to improve analgesia and maintain immobilization. The vaporizer setting was increased from 3 to 5% isoflurane. Blood pH and gases were reassessed after 15 minutes of IPPV. Isoflurane concentration was decreased to 4% for 20 minutes and then to 3.5%, where it was maintained until 10 minutes before the end of surgery. Voluntary movement did not recur. Arterial blood pressure began a gradual increase approximately 45 minutes after tourniquet inflation and was still gradually increasing 105 minutes after tourniquet inflation. The tourniquet was released 15 minutes before the end of anesthesia (105 minutes after tourniquet inflation). Arterial blood pressure decreased immediately and remained stable for the last 10 minutes of anesthesia. Isoflurane and IPPV were discontinued, and 100% oxygen was delivered. Ventilation was manually assisted at a rate of 2 breaths/min until spontaneous respiration returned. Diazepam was administered (0.05 mg/kg, IM) to smooth the recovery, and the ostrich was moved to a dark, padded recovery stall and extubated when swallowing was observed. The ostrich was sternal and standing at 40 and 70 minutes after the end of anesthesia, respectively.

Tourniquet-induced hypertension (TIH) has been described in human beings¹⁻³ and horses.^{4,5} Intraoperative hypertension in human beings has been defined as a 30% increase in SAP or DAP, compared with the first pressure recording after incision.¹ The frequency of intraoperative hypertension in human beings was 11% when a tourniquet was used, compared with 1% when a tourniquet was not applied.¹ Tourniquet pain in awake human beings reaches maximal severity after 45 to 60 minutes.² Findings in 1 study³ indicated a higher prevalence of TIH in people undergoing general anesthesia, compared with that for spinal or epidural anesthesia.

The increase in SAP (220 to 298 mm of Hg, 35%) after tourniquet inflation and the immediate decrease (298 to 204 mm of Hg, 31.5%) after tourniquet release suggested TIH in this ostrich. Reference values for arterial blood pressures in awake and anesthetized ostriches have not been established; therefore, use of the term hypertension in this case refers to the relative increase observed. On the basis of another report⁶ (range of maximal values recorded from 6 isoflurane-anesthetized ostriches: MAP, 105 to 232 mm of Hg; SAP, 148 to 275 mm of Hg; DAP, 91 to 197 mm of Hg) and personal

From the Department of Veterinary Clinical Sciences, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA 70803.

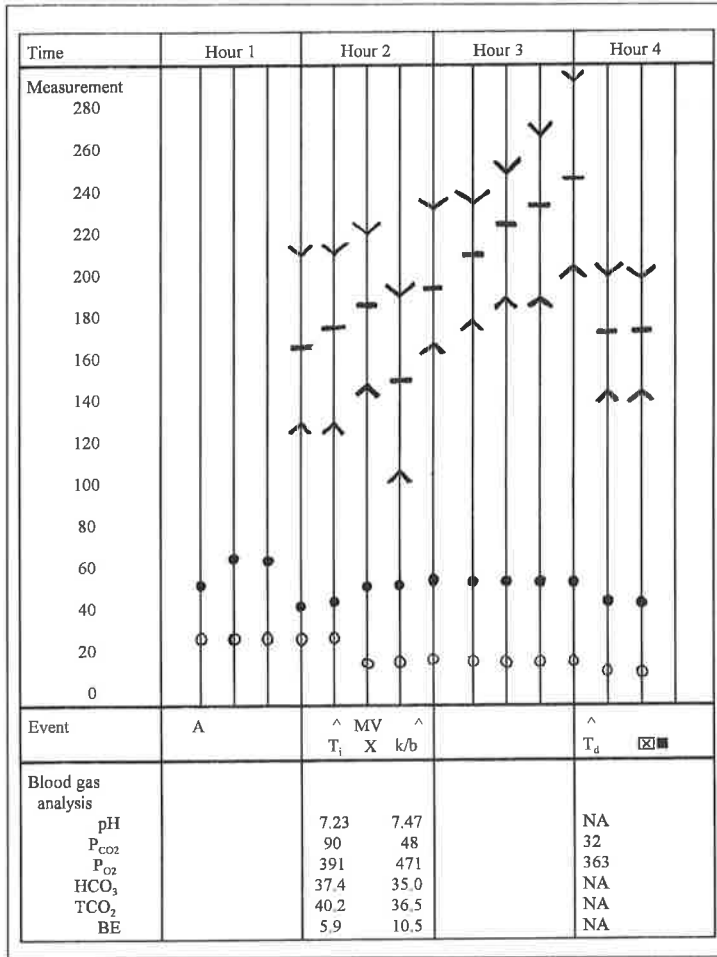


Figure 1—Graphic representation of data recorded, including heart rate (●, beats/min); respiratory rate (○, breaths/min); mean (—), systolic (∇), and diastolic (△) arterial blood pressures (mm of Hg); and events that occurred during general anesthesia of an ostrich for sequestrum removal. A = begin isoflurane delivery; T₁ = tourniquet inflation; ^ = blood collected for pH and gas analysis; MV = begin mechanical ventilation; X = begin surgery; k/b = IV administration of ketamine and butorphanol; T_d = tourniquet deflation; ☒ = end surgery; ■ = end isoflurane delivery; HCO₃ = bicarbonate concentration mEq/L; TCO₂ = total CO₂ concentration mmol/L; BE = base excess mEq/L. NA = not available.

observations⁸ (range of maximal values recorded from 5 isoflurane-anesthetized ostriches: MAP, 112 to 218 mm of Hg; SAP, 138 to 262 mm to Hg; DAP, 91 to 197 mm of Hg), arterial blood pressure values in isoflurane-anesthetized ostriches are higher than values established for some commonly studied domestic species.⁷⁻⁹ The ostrich of this report had arterial pressures within the range of maximal values that have been reported⁶; however, the arterial pressures recorded just before tourniquet release exceeded those values.

No change was detected in heart rate of the ostrich throughout the anesthetic period. An increase in heart rate concurrent with the increase in arterial blood pressure has been reported in 1 horse with TIH.⁴ In a study of human beings¹ and in a controlled study of horses,⁵ an increase in heart rate did not appear to be associated with TIH.

The increase in arterial blood pressure in the ostrich of this report was preceded by a decrease that occurred 30 minutes after tourniquet inflation. This transient decrease was probably attributable to a combination of factors, including initiation of IPPV, an IPPV-induced decrease in PaCO₂, an increased inspired concentration of isoflurane and administration of ketamine (both having a direct negative inotropic effect),¹⁰⁻¹³ and administration of butorphanol which, along with ketamine and increased isoflurane delivery, may have enhanced analgesia

and obtunded a physiologic response to surgical stimulation.

Causes of an increase in arterial blood pressure during general anesthesia include inadequate anesthesia, hypercapnia, hypoxemia, and hyperthermia. Other than voluntary movement at the start of surgery, there was no further indication of inadequate anesthetic depth during the surgical procedure. Hypercarbia developed during the first hour of anesthesia but was corrected to near normal limits before the steady increase in arterial blood pressure became apparent. Hypoxemia was ruled out from the results of serial blood gas analyses. Hyperthermia was not ruled out because body temperature was not recorded; however, there was no evidence, such as accelerated soda lime exhaustion or development of metabolic acidosis, to suggest the development of hyperthermia.

The rapid decrease in blood pressure with tourniquet deflation suggested that tourniquet application was responsible for the increase in arterial blood pressure observed during surgery. Detrimental effects were not apparent as a result of the high arterial pressures recorded.

^aDiazepam Injectable, Rugby Laboratories Inc, Rockville Centre, NY.

^bTelazol, Fort Dodge Laboratories Inc, Fort Dodge, Iowa.

^cBivona Inc, Gary, Ind.

^dModel 90602A, Spacelabs Inc, Redmond, Wash.

^eModel 400, Kidde Inc, Mebane, NC.

^fMetomatic Veterinary Ventilator, Ohio Medical Products, Madison, Wis.

^gMedical records, Louisiana State University Teaching Hospital and Clinic, Baton Rouge, La.

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Book Review:

Restraint and Handling of Wild and Domestic Animals. Second Edition. By Murray E. Fowler. 383 pages; illustrated. Iowa State University Press, 2121 S State Ave, Ames, IA 50014-8300. 1995. Price \$64.95.

Dr. Fowler's first edition of this book, published in 1978, had 9 printings before publication of the second edition. The prefaces to both editions of the book state its goal: "It is only through an enlightened understanding of restraint principles that humane handling with the least amount of stress will be possible for any animal." There is no question that both editions provide the information in an organized and concise form to aid those individuals who need to restrain or handle various wild and domestic animals.

The depth of the book goes beyond the "Art of Restraint," with appropriate limited discussion on chemical restraint and anesthesia in addition to other practical management techniques for various species. The text also is laced with the author's experiences, positive and negative, of restraint episodes. An additional strength is the safety aspect for the animals and human beings associated with a restraint procedure. This safety aspect may seem overly stressed, but it places into proper perspective the fact that some domestic and many wild animals can cause serious or fatal injury to inexperienced or careless handlers.

The second edition again is divided into 3 parts (general concepts, domestic animals, wild animals). There are 26 chapters that are well illustrated. Most of the pictures were published in the first edition, but the photos are much clearer in the second edition. Many of the chapters are the same, which is to be expected when the basic concepts and techniques of restraint are discussed; however, there are timely updates on anesthetic drugs and techniques that currently are available, and discussions on emerging drugs that will be available (ie, etorphine, medetomidine, atipamezole). There are expanded sections dealing with restraint techniques in camelids and ruminants.

The book remains a classic textbook on all aspects of restraint. The second edition is a must for veterinary technicians, animal owners, veterinary students, and veterinarians who work with domestic or wild species and who do not own the first edition, and is a useful addition to those who do.

Mitchell Bush, DVM