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DEATHS FROM EXERTIONAL MYOPATHY AT THE NATIONAL ZOOLOGICAL PARK FROM 1975 TO 1985

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ABSTRACT: A retrospective study was conducted to determine the incidence of deaths from exertional myopathy (EM) in the hoofstock collection at the National Zoological Park (NZP) from 1975 to 1985. The diagnosis of EM was based on history, clinical signs, clinicopathological findings, gross and microscopic lesions. Only bovids, cervids, and equids were included in the study. Data were derived from the medical and pathological records of the NZP. There were 10 cases of EM during this period, but only seven deaths were directly attributable to EM. Five deaths occurred after immobilization. Two deaths occurred after improper transport, but the anesthetic history was not known in these cases. Pathologic changes consistent with EM were incidental findings in an animal killed by dogs, and in two animals euthanized because of suspected infection with *Parelaphostrongylus tenuis*. Only cervids and bovids were affected. There was no age or sex predilection for developing EM and deaths occurred throughout the year. Deaths associated with immobilization occurred most often after anesthesia with xylazine and etorphine. These are the drugs used most frequently in bovid and cervid anesthesia at the NZP. The overall incidence of deaths from EM after immobilization was 0.25% for the 10-yr period.

Key words: Exertional myopathy, exotic hoofstock, review, anesthetic complication, restraint, muscle enzymes.

INTRODUCTION

Exertional myopathy (EM), also known as capture myopathy, or exertional rhabdomyolysis has been reported in man (Geller, 1974; Gitin and Demos, 1974), birds (Young, 1967; Brannian et al., 1981; Windingstad et al., 1983) and a variety of domestic (Jones and Hunt, 1983) and exotic mammals (Munday, 1972; Basson and Hofmeyr, 1973; Lewis et al., 1977; Kocan et al., 1980; Chalmers and Barrett, 1982). Pathologically, EM resembles malignant hyperthermia of swine, posttransport myopathy of cattle, and equine rhabdomyolysis (Jones and Hunt, 1983). It generally occurs after vigorous muscle exertion. Although the specific pathogenesis is poorly understood, it is thought that anaerobic metabolism caused by intense muscular activity leads to an accumulation of lactic acid, severe metabolic acidosis and secondary muscle necrosis (Basson and Hofmeyr, 1973; Harthoorn and Young, 1974; Jones and Hunt, 1983). Clinical signs include pain, stiffness, inability to rise, oliguria and depression. When muscle injury is severe, the urine may be discolored a dark brown with myoglobin. Acute renal failure may result from a combination of ischemia, acidosis and myoglobinuria (Harthoorn and Young, 1974; Harthoorn, 1975; Brannian et al., 1981; Chalmers and Barrett, 1982). Clinicopathological changes include an initial acidemia and hyperkalemia, followed later by azotemia and increased levels of aspartate aminotransferase (AST), creatinine kinase (CK) and lactic dehydrogenase (LDH) (Harthoorn, 1975; Chalmers and Barrett, 1982). While not entirely muscle specific, elevations of these enzymes indicate muscle destruction. Urinalysis may show hyposthenuria, proteinuria, glucosuria and casts (Bartsch et al., 1977).

Treatment for EM consists of intravenous fluids, sodium bicarbonate, corticosteroids, calcium channel blockers, and supplemental vitamin E and selenium (Harthoorn and Young, 1974; Harthoorn, 1975; Chalmers and Barrett, 1982; Blood et al., 1983). Physiological changes are often irreversible and despite drastic supportive measures, affected animals often die. Death may occur acutely, within a few hours, with rapidly progressing depression as the only sign. Usually death occurs 2–

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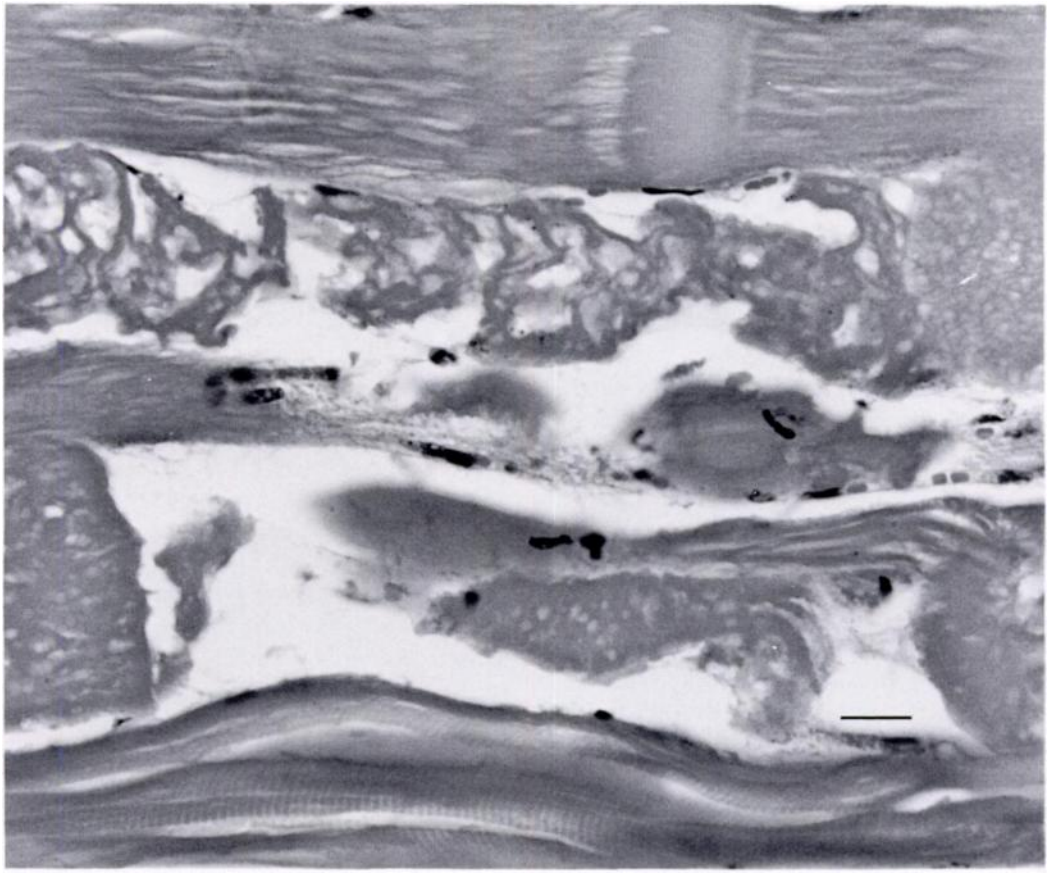


FIGURE 1. Histological section of skeletal muscle of a yellow-backed duiker (Case 1) showing myonecrosis with hyalinization and disruption of fibers, loss of cross striations, and pyknotic sarcolemmal nuclei. H&E. Bar = 100 μm.

4 days after the onset of signs and results from a combination of renal failure, prolonged acidemia and hyperkalemia, and pulmonary edema. Chronically affected animals may die 2–4 wk later from complications such as heart failure, and inanition caused by persistent pain and paresis (Basson and Hofmeyr, 1973; Harthoorn and Young, 1974; Chalmers and Barrett, 1982).

In acute cases, skeletal muscle is grossly edematous and hemorrhagic, with red-black streaks clearly demarcated from normal tissue. If death occurs after 3–4 days the lesions become pale. The large muscle masses of the rear legs are most often affected. Pale streaking may also be seen in the myocardium. The kidneys are often swollen and dark brown (Bartsch et al., 1977; Lewis et al., 1977; Kocan et al., 1980; Brannian et al., 1981; Chalmers and Barrett, 1982). Microscopic findings depend on the duration of illness, but include degeneration, necrosis, mineralization and reparative fibrosis of both skeletal and cardiac muscle, as well as dilated renal tubules, renal tubular necrosis and intratubular pigmented casts (Geller, 1974; Bartsch et al., 1977; Lewis et al., 1977; Kocan et al., 1980; Brannian et al., 1981; Chalmers and Barrett, 1982).

The present survey was undertaken to determine the number of deaths from EM which occurred in the hoofstock collection

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at the NZP. Histories, clinical and pathological findings were evaluated.

METHODS

Data for this study were obtained from the medical and pathological records of the NZP from 1975 to 1985. Medical records reported clinical and anesthetic history. The total number of immobilizations performed on Bovidae, Cervidae, and Equidae were taken from an anesthetic log. All necropsies and histopathological preparations were performed by the Department of Pathology at the NZP.

CASE HISTORIES

Case 1

A 6-yr-old male yellow-backed duiker (*Cephalophus sylvicultor*) was shipped to the NZP in a small crate. Anesthetic history for loading and transport was not available. The animal arrived recumbent, unable to stand. Self-inflicted abrasions were apparent, indicating that struggling in the crate had occurred. Blood samples taken upon arrival revealed a blood urea nigrogen (BUN) of 53 mg/dl and a creatinine of 1.4 mg/dl. Lactate dehydrogenase (LDH) and aspartate aminotransferase (AST) levels exceeded 36,000 I.U. and 35,000 I.U., respectively. Twenty-four hours later the BUN, creatinine and AST had increased slightly and the creatine kinase (CK) exceeded 1×10^6 I.U. Hematologic values remained normal. Death occurred 36 hr after arrival despite treatment with intravenous fluids and corticosteroids. At necropsy, pale streaking was present in the myocardium and in many skeletal muscles, with the muscles of the rear legs most severely affected. The kidneys and urine in the bladder appeared normal. Histologic examination revealed myonecrosis of skeletal muscle (Fig. 1) and acute renal tubular necrosis with intratubular casts (Fig. 2).

Case 2

A 16-mo-old female sable antelope (*Hippotragus niger*) arrived at the NZP after transport by truck for 18 hr. Anesthetic history prior to arrival was not available. It was unable to stand the next morning, and despite treatment with oral and intravenous fluids, corticosteroids, muscle relaxants and supplemental vitamin E and selenium, euthanasia was performed 48 hr after arrival. Hematologic and electrolyte values remained normal until death. BUN and creatinine values were slightly elevated and did not change greatly over 48 hr. Immediately prior to euthanasia their values were 48 mg/dl and 3 mg/dl, respectively.

At necropsy, pale streaks with mild hemorrhage were seen in the muscles of the rear legs (Fig. 3) and in several other muscle groups. The kidneys appeared normal. Histologically, various stages of myopathy were seen. Some muscle fibers were acutely necrotic while others showed more chronic changes with focal mineralization and macrophage proliferation. Sarcolemmal proliferation indicated regenerative attempts. Nephrosis with tubular dilation and casts and moderate pulmonary congestion were also evident.

Case 3

An 8-mo-old female scimitar-horned oryx (*Oryx dammah*) was immobilized with xylazine (Rompum®, 100 mg/ml, Haver-Lockhart, Bayvet Division, Miles Laboratories Inc., Shawnee, Kansas 66201, USA) and etorphine HCl (M99, 1 mg/ml, Lemmonson Company, Sellersville, Pennsylvania 18960, USA) for transport between zoo facilities. Full relaxation was never attained and hyperthermia (43.3 C) was noted 30 min after anesthetic reversal with diprenorphine (M50-50, 2 mg/ml, Lemmonson Company). The animal was treated with intravenous fluids, corticosteroids, sodium bicarbonate and antibiotics and was cooled to a temperature of 40 C before transport. On arrival it was unable to stand and died 12 hr later despite further treatment. LDH and AST were 3,605 I.U. and 1,090 I.U., respectively, imme-

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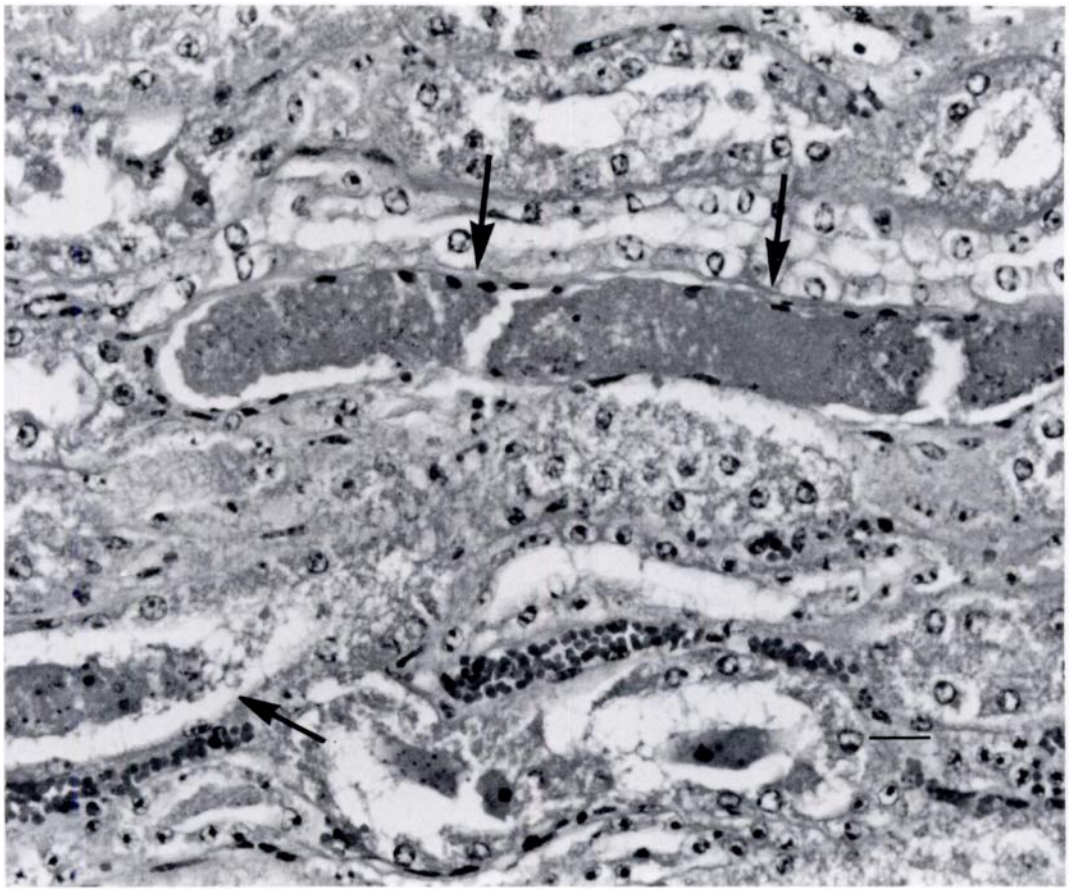


FIGURE 2. Histological section of the kidney of a yellow-backed duiker (Case 1) showing tubular necrosis with intratubular casts (arrows). H&E. Bar = 100 μm.

diately prior to death. Hematologic values remained normal.

Necropsy revealed bilateral pale mottling and hemorrhage of the quadriceps muscles and pulmonary congestion. Histologically, acute changes in skeletal muscle were evident. Edema and hemorrhage were present, and individual muscle fibers were degenerate with vacuolization and loss of cross striations.

Case 4

A female sable antelope greater than 12 yr old was immobilized with carfentanyl (Carfentanyl Citrate, 5 mg/ml, experimental drug, Janssen Pharmaceutical, B-2340 Beerse, Belgium) and azaperone (Azaperone, 40 mg/ml, Pitman-Moore, Washington Crossing, New Jersey 08560, USA) for a hoof trim. The level of anesthesia was not sufficient. Therefore, manual restraint with ropes was required. After anesthetic reversal with diprenorphine, the animal had trouble standing. Radial paralysis of the right foreleg was apparent, and bilateral abduction of the rear limbs occurred. In 24 hr, the animal was unable to stand. Treatment with intravenous fluids, corticosteroids, supplemental vitamin E and selenium, and antibiotics was begun, and the animal was slung in a standing position. Death occurred 30 hr after immobilization. Hematologic, serum enzyme and electrolyte values remained normal.

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FIGURE 3. Cross-section of the thigh of a sable antelope (Case 4) showing sharp demarcations between normal muscle (left) and pale affected muscle.

Bilateral hemorrhage of the adductor muscles of the rear legs and flexor muscles of the hips were seen at necropsy. Edema and congestion of the lungs were present. Histologically, acute myonecrosis with hemorrhage, and pulmonary edema and congestion were seen. There were no gross or microscopic renal changes.

Case 5

An 11-mo-old male Reeves' muntjac (*Muntiacus reevesi*) was immobilized with etorphine HCl and halothane (Halothane®, Halocarbon Laboratories, Inc., Hackensack, New Jersey 07601, USA) for electroejaculation and surgical vasectomy. During the procedure, the animal became hyperthermic (43 C) and was immersed in cold water. Diprenorphine was used for anesthetic reversal. Postoperative treatment consisted of intravenous fluids and banamine, a non-steroidal anti-inflammatory drug. The following day, the animal was lethargic and obtunded. Death occurred approximately 48 hr after surgery. Hematologic and serum chemistry values were not obtained.

Both cardiac and skeletal muscle appeared normal at necropsy. Pulmonary edema was present. The urinary bladder was distended with green-black urine. There was also hemorrhage in the rectal

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lumen. Histologically, skeletal myonecrosis and acute renal tubular necrosis with tubular casts were seen.

Case 6

A 17-yr-old male wisent (*Bison bonasus*) was immobilized with xylazine and etorphine HCl for reproductive and hormonal studies. The animal did not stand after anesthetic reversal with diprenorphine, yohimbine (Yohimbine, 3.75 mg/ml, Sigma Chemical Company, St. Louis, Missouri 63178, USA) and 4-aminopyridine (4-aminopyridine, 3.75 mg/ml, Sigma Chemical Company) and unilateral radial paralysis was suspected. While attempting to rise, entanglement in a fence occurred which exacerbated the animal's struggles. Treatment consisted of steroids and antibiotics. Euthanasia was performed 72 hr after anesthesia. A blood sample was not obtained.

Pale streaking of the muscles of all legs was evident grossly. The myocardium appeared diffusely pale. There were omasal and abomasal ulcers with extensive upper gastrointestinal hemorrhage. Histologic examination revealed skeletal myodegeneration and necrosis with hemorrhage, and renal tubular degeneration with occasional tubular casts.

Case 7

A 7-yr-old male dama gazelle (*Gazella dama*) was immobilized with xylazine and etorphine HCl for a hoof trim. The animal remained quiet during the procedure, and stood well after anesthetic reversal with yohimbine and naloxone (Naloxone, 20 mg/ml, Wildlife Laboratories, Fort Collins, Colorado 80525, USA). Difficulty standing and stiffness in the rear legs were noted over the next 48 hr. Treatment consisted of intravenous fluids, corticosteroids, sodium bicarbonate, supplemental vitamin E and selenium, and antibiotics. Death occurred 6 days postimmobilization following complications from a ruptured urinary bladder. The CK and AST exceeded 20,000 I.U. and 3,400 I.U., respectively, prior to death. The BUN and creatinine levels were also greatly elevated, but it is unclear to what extent this was a result of EM or the ruptured bladder.

Unilateral edema, hemorrhage and a large area of necrosis were apparent in the large muscle mass of the rear leg. Hydroperitoneum and rectal prolapse associated with urinary bladder rupture were also present. Severe myonecrosis with hemorrhage and cellular infiltration, and pulmonary congestion were seen histologically.

EM was an incidental finding in a 19-mo-old male scimitar-horned oryx and a 4-yr-old female sable antelope, both affected with progressive hind limb ataxia. Neither animal responded to supportive treatment and both were euthanized 96 hr after the onset of signs. In the oryx, hematologic, electrolyte, BUN and creatinine levels remained normal but the LDH increased from 820 to 73,000 I.U., and the AST from 92 to 1,600 I.U., over 96 hr. The sable had a slight neutrophilia without leukocytosis. The AST increased from 73 to 1,600 I.U. over 96 hr. CK levels were not measured for either animal.

Pale streaks were evident bilaterally in the rear leg muscles of the sable, but skeletal muscles in the oryx were normal grossly. Histologic examination revealed myodegeneration and necrosis in both cases, with mineralization occurring the oryx. Myocardial degeneration was present in the sable. Infection with *Parelaphostrongylus tenuis* was positively diagnosed as the cause of ataxia in the sable. Cross-sections of adult nematodes were found in the brain and degenerative tracts associated with parasite migration were evident throughout the spinal cord. *Parelaphostrongylus tenuis* was also the suspected cause of ataxia in the oryx. Although nematodes were not found, changes con-

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TABLE 1. Number of immobilizations, postimmobilization deaths, and postimmobilization deaths from exertional myopathy that occurred in hoofstock at the National Zoological Park from 1975 to 1985.

Animal group	Number of imobilizations	Number of deaths	Number of deaths from EM	% Mortality from EM
Bovidae	859	8	4	0.47
Cervidae	902	8	1	0.11
Equidae	203	0	0	0
Total	1,964	16	5	0.25

sistent with parasite migration tracts were present in the spinal cord.

In both cases, EM probably resulted from ataxia and the struggle to rise and maintain balance. Although both were immobilized for transport to the hospital, EM associated with anesthesia was not considered the cause of death. However, anesthesia may have contributed to the lesions in the musculature.

Skeletal muscle hyalinization and necrosis consistent with EM were incidental microscopic findings in a 7-yr-old male blesbok killed by dogs.

RESULTS AND DISCUSSION

Only five confirmed deaths from EM after immobilization occurred at the NZP during the 10-yr period from 1975 to 1985. This accounts for 31% of all anesthetic related deaths that occurred during nearly 2,000 immobilizations of hoofstock performed in the same period (Table 1). The remaining cases of EM were associated with a variety of circumstances. There was no apparent age or sex predilection and deaths occurred throughout the year. Equids seem less likely to develop the disease than bovids and cervids, but fewer equid immobilizations were performed. The actual incidence of EM may be higher, but unless an animal died, and the diagnosis was confirmed grossly and microscopically, it was not included in this survey.

Clinicopathological parameters did not consistently indicate EM. Blood samples were not obtained for assay in three cases, and CK was not routinely measured. LDH and AST levels were usually elevated and were a good indication of muscle necrosis. Because preimmobilization data are not available, however, it is possible that these animals had higher levels prior to handling, indicating increased susceptibility to EM. Only three cases showed elevated BUN and creatinine. There were no hematologic, electrolyte, or serum enzyme abnormalities in one case.

Traditionally, the occurrence of EM in wildlife and exotic species has been associated with overexertion related to pursuit, capture, restraint with or without immobilizing drugs, and transport (Young, 1967; Harthoorn, 1975; Chalmers and Barrett, 1982). The sable in Case 4 required manual restraint because of an insufficient level of anesthesia. This, compounded by radial paralysis, probably led to EM. EM can occur regardless of the capture and restraint history, however. Neither the wisent in Case 6 nor the gazelle in Case 7 struggled before or during anesthesia. It is unclear why EM occurred in these cases. In the wisent, the massive weight placed on specific muscle groups during anesthesia may have caused muscle damage as well as radial paralysis. His struggles to extricate himself from the fence would have exacerbated the muscle lesions and led to EM. It has been suggested that fear and anxiety trigger EM, and that overexertion and transport simply worsen the condition (Harthoorn and Young, 1974; Chalmers and Barrett, 1982). Hyperthermia has also been implicated. Anesthetic drugs that cause hyperthermia have been associated with clinical signs and lesions consistent with EM, and increased mortality occurs when immobilizations are performed when the ambient temperature is warm (Harthoorn, 1975; Lewis et al., 1977; Chalmers and Barrett, 1982). Hyperthermia occurred in both Cases 3 and 5 and may have

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triggered EM. In addition, the oryx in Case 3 never attained full relaxation under anesthesia.

EM has been attributed to poor transport conditions. If the crate is too small, if an animal fights the crate in which it is contained, or if it struggles to maintain its balance during transport EM may develop as occurred in Case 1.

The pathologic lesions seen in nutritional myopathies related to vitamin E and selenium deficiencies are indistinguishable from those seen in EM (Harthoorn and Young, 1974; Chalmers and Barrett, 1982; Jones and Hunt, 1983). Although there is controversy whether nutritional deficiency is directly related to EM, deficiencies in vitamin E and/or selenium may predispose animals to it (Brady et al., 1978; Chalmers and Barrett, 1982; Ullrey et al., 1984; Robbins et al., 1985). The sable in Case 2 had pathologic changes compatible with both exertional and nutritional myopathies. The more chronic changes antedated the animal's arrival at the zoo, suggesting an underlying vitamin E and selenium deficiency. Unfortunately, vitamin E and selenium levels in blood and tissues were not determined.

The above cases illustrate that EM results from a variety of circumstances and that vigorous pursuit and chemical restraint are not prerequisite. All immobilizations of affected animals were performed in small, confined areas where exertion from pursuit was minimal. However, whether from inadequate anesthesia, transport, or secondary to an underlying disease process, physical exertion did occur in most cases.

Every effort should be made to ensure that animals are caught and restrained as quickly and quietly as possible, to minimize fear, anxiety and physical exhaustion. Immobilizations should be performed in cool ambient temperatures to reduce heat stress. Obtaining an adequate plane of anesthesia is preferable to using manual restraint to control a struggling animal. If possible, supplemental vitamin E and selenium should be given to animals suspected of having or bordering on deficiency, well before immobilization and transport. Recently, oral supplementation with sodium bicarbonate was used successfully to prevent recurring episodes of exertional rhabdomyolysis in a horse (Robb and Kronfeld, 1986). Consideration of anatomic variations is also important; EM localized to the neck muscles was the cause of death in a giraffe recovering from anesthesia. This animal was not part of the NZP collection, but the necropsy and histopathology were performed at the NZP. Assessment of crate size and travelling conditions is mandatory to ensure that an animal has adequate room to stand during transport. Additionally, it should be kept in mind that EM may occur anytime an animal undergoes stress and physical exertion, regardless of the anesthesia and transport history. Unfortunately, the restraint required to treat exotic hoofstock may simply exacerbate EM by prolonging the insult.

Absolute prevention of EM is unrealistic, but with care, its occurrence can be dramatically reduced. Once an animal exhibits signs of exertional myopathy, the prognosis is poor.

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